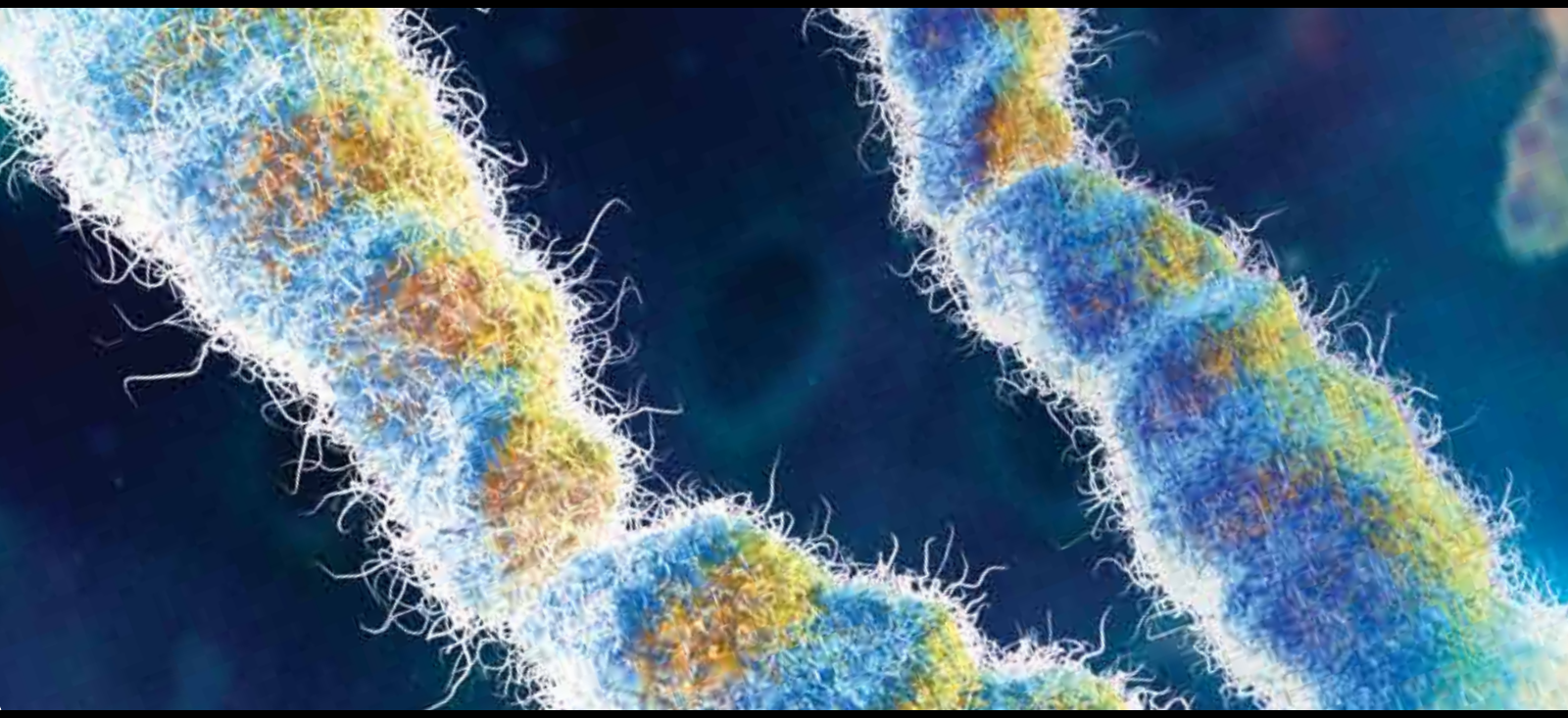
Journal of Aging Research

Behavioral Factors of Longevity

Guest Editors: Peter Martin, Leonard W. Poon, and Bo Hagberg



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Editorial Board

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Editorial
Behavioral Factors of Longevity

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The majority of studies on longevity have focused disproportionally on biomedical aspects of longevity [1]. While biomedical aspects undoubtedly play an important role in determining the length and quality of life, there are also a number of important social, psychological, and behavioral factors associated with longevity. Recent research has demonstrated that distal experiences such as education [2] and childhood personality [3] as well as proximal behaviors such as nutritional behaviors [4], coping with stress [5], and social support [6] are all important components in determining mortality, longevity, and quality of life among very old people.

This special issue on behavioral factors of longevity attempts to highlight important behavioral factors associated with longevity. We pose two important questions. The first question concerns predictors of longevity. Why do some people live to a very long life whereas others do not? The second question concerns the quality of life for individuals who have lived a very long life. What is life like when one reaches nonagenarian or centenarian status? We were, therefore, guided by two separate but interrelated aspects of longevity research: studies discussing factors contributing to longevity and studies discussing behavioral aspects among long-lived individuals.

The articles in the special issue cover a number of important topics. The first highlights the importance of biopsychosocial models in longevity research (Picard). With regard to the mortality and survivorship aspect, papers include gender differences (M. Poulain et al.; F. Balard et al.), minority representation and socioeconomic status (L. S. Ka'opua et al.; Yao and Robert), season of birth (Gavrilov and Gavrilova), obesity (Cohen-Mansfield and Perach; J. Nocera et al.), alcohol consumption (E. K. Howie et al.), dental health (A. Paganini-Hill et al.), personality (B. P. Chapman et al.), stress trajectories (C. M. Aldwin et al.), depression and cognition (D. Paulson et al.), and cognitive beliefs (Fry and Debats). The second topic of life quality includes the importance of dietary patterns (D. B. Hausman et al.), physical and psychological well-being (J. Cho et al.), social support (Ailshire and Crimmins; G. K. Randall et al.), and life satisfaction (A. J. Bishop et al.).

It is clear from these contributions that there are many behavioral factors contributing to longevity. Broadly speaking, they include health and health behaviors, individual characteristics, such as gender, ethnicity, and socioeconomic status, stress, cognitive beliefs, and cognition, as well as social and environmental support, mental health, and life satisfaction. In a previous publication, we included all these factors in the Georgia adaptation model [7]. An extension of this model would include important distal variables.

The studies included in this volume highlight and introduce specific behaviors that are associated with longevity. Surely, there might be other specific behaviors of importance for longevity that are not covered here but the components proposed to be of importance in this issue allow us to take a much broader perspective on aging that goes beyond biomedical explanations. We hope this special issue will stimulate more research on behavioral factors of longevity.

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References

[1] L. W. Poon, P. Martin, A. Bishop et al., "Understanding centenarians' psychosocial dynamics and their contributions to

health and quality of life," *Current Gerontology and Geriatrics Research*, vol. 2010, Article ID 680657, 13 pages, 2010.

[2] J. K. Montez, M. D. Hayward, D. C. Brown, and R. A. Hummer, "Why is the educational gradient of mortality steeper for men?" *Journals of Gerontology B*, vol. 64, no. 5, pp. 625–634, 2009.

[3] H. S. Friedman, J. S. Tucker, C. Tomlinson-Keasey, J. E. Schwartz, D. L. Wingard, and M. H. Criqui, "Does childhood personality predict longevity?" *Journal of Personality and Social Psychology*, vol. 65, no. 1, pp. 176–185, 1993.

[4] B. J. Willcox, K. Yano, R. Chen et al., "How much should we eat? The association between energy intake and mortality in a 36-year follow-up study of Japanese-American men," *Journals of Gerontology A*, vol. 59, no. 8, pp. 789–795, 2004.

[5] L. Tafaro, M. T. Tombolillo, N. Brükner et al., "Stress in centenarians," *Archives of Gerontology and Geriatrics*, vol. 48, no. 3, pp. 353–355, 2009.

[6] D. G. Blazer, "How do you feel about…? Health outcomes in late life and self-perceptions of health and well-being," *The Gerontologist*, vol. 48, no. 4, pp. 415–422, 2008.

[7] L. W. Poon, G. M. Clayton, P. Martin et al., "The Georgia Centenarian Study," *International Journal of Aging and Human Development*, vol. 34, no. 1, pp. 1–17, 1992.

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Research Article

Season of Birth and Exceptional Longevity: Comparative Study of American Centenarians, Their Siblings, and Spouses

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This study explores the effects of month of birth (a proxy for early-life environmental influences) on the chances of survival to age 100. Months of birth for 1,574 validated centenarians born in the United States in 1880–1895 were compared to the same information obtained for centenarians' 10,885 shorter-lived siblings and 1,083 spouses. Comparison was conducted using a within-family analysis by the method of conditional logistic regression, which allows researchers to control for unobserved shared childhood or adulthood environment and common genetic background. It was found that months of birth have significant long-lasting effect on survival to age 100: siblings born in September–November have higher odds to become centenarians compared to siblings born in March. A similar month-of-birth pattern was found for centenarian spouses. These results support the idea of early-life programming of human aging and longevity.

1. Introduction

Studies of centenarians (persons living to age 100 and over) are useful in identifying factors leading to long life and avoidance of fatal diseases. These studies may be a sensitive way to find genetic, familial, environmental, and life-course factors associated with lower mortality and better survival [1, 2]. Several theoretical concepts suggest that early-life events and conditions may have a significant long-lasting effect on survival to advanced ages. These concepts include (but are not limited to) the idea of fetal origin of adult diseases also known as the Barker hypothesis [3, 4] and the related idea of early-life programming of aging and longevity; the theory of technophysio evolution [5], the reliability theory of aging, and the high initial damage load (HIDL) hypothesis in particular [6, 7]. These ideas are supported by the studies suggesting significant effects of early-life conditions on late-life mortality [3, 8–10]. Finch and Crimmins [11] suggested that historical decline in chronic inflammation (due to decreasing exposure to early-life infections) has led to a decrease in morbidity and mortality from chronic conditions at old age. They showed that both childhood mortality and cardiovascular diseases of old age may share common infectious and inflammatory causes rooted in the external environment [12].

Month of birth often is used by epidemiologists as a proxy characteristic for environmental effects acting during in-utero and early infancy development. These early effects include temperature and sun exposure during in-utero and early postnatal period, nutritional status during early development, exposure to infectious agents, and other factors [3, 13, 14]. Previous studies demonstrated that life expectancy may be influenced by person's month of birth [15–18]. However, studies of month-of-birth effects on longevity face significant difficulties in finding appropriate data on differential mortality by season of birth. Longitudinal data with information about season of birth are the optimal data for study of month-of-birth effects on longevity [19]. Such longitudinal data were available for population of Denmark and showed that the remaining life expectancy at age 50 was higher for persons born in October-November compared to persons born in April–June [15]. In other studies, the effects of

month of birth on late-life mortality were estimated indirectly using information on mean age at death from cross-sectional collection of death certificates [19–22].

Little information is available on the month of birth association with exceptional longevity. To our knowledge, there is only one study that examines the effects of month of birth on longevity [23]. In this study, month-of-birth distribution of 925 age-validated German semi-supercentenarians (persons aged 105+ years) was compared to seasonal distribution of births in the German Empire at the time of semi-supercentenarians' birth (1880–1900). It was found that more semi-supercentenarians than expected were born in December while the proportion of semi-supercentenarians born in June was low. This study suggests that the December-born have a significantly higher risk of surviving up to age 105+ compared to the June-born [23] although it cannot be indicated unequivocally if month-of-birth pattern among semi-supercentenarians is due to seasonality of infant mortality or later-life month-of-birth effects. Additional problems in the studies of month-of-birth effects on longevity arise from possible confounding due to between-family variation in childhood socioeconomic conditions [24–26] and parental genetic background [27]. One possible solution to these challenges is to compare associations within sibships taking into account that socioeconomic and genetic background is similar for siblings from the same family [14, 28].

In this study, we analyze the effects of month of birth on survival to age 100 years using a large set of centenarians born in the United Sates in 1880–1895 and their shorter-lived siblings and spouses. Siblings share early childhood conditions including parental socioeconomic status, genetic background, and geographical location while spouses share common adulthood environment. It was shown that longevity has a significant familial component [29–32] suggesting the need to control for this important factor. Comparing month-of-birth characteristics of adult siblings or spouses with that of centenarians provides an opportunity for obtaining net effects of month-of-birth on survival and control for unobserved confounding factors.

2. Methods

2.1. Data Collection. This study compares centenarians to their shorter-lived siblings (who share common childhood conditions and genetic background) and spouses (who share common adulthood environment) using a large set of computerized family histories. Family histories (genealogies) proved to be a useful source of information for studies in historical demography [33] and biodemography [34, 35]. In this study, data were collected through a search of over 400,000 online family histories available at Rootsweb (http://wc.rootsweb.ancestry.com), which is one of the largest publicly available repositories of online genealogies. Search for centenarians in the Rootsweb database was conducted with assistance of the web-automation technique [36], which allows researchers to run automated queries (using program scripts in PHP language) and search online databases for individuals with desired properties (persons who lived 100+ years in our case). Applying this technique helps researchers to save time and effort on routine data collection from online resources. Application of the web-automation technique to the Rootsweb publicly available online resource identified over 40,000 records of centenarians born in 1880–1895 with known names of their parents. However, in many cases, one and the same centenarian appeared in two or more genealogies. After removing these duplicates, we obtained 23,127 records for centenarians born in 1880–1895 with detailed information on their birth and death dates as well as birth and death dates of their parents. According to the past experience with computerized genealogies [34], availability of detailed information on vital events ensures a good quality of collected genealogies. However, a significant proportion of records for siblings in the obtained genealogies did not contain information about death dates that we needed for the within-family analysis of human longevity. So the next step was to indentify the most informative families with complete information on birth and death dates for siblings. As a result of this identification procedure, we found 2,834 families where information on birth and death dates was known for more than 80 percent of siblings in a family. This procedure resulted in a set of families having higher-than-average sibship size and hence providing more control records (siblings) for the matched case-control study. During this data refining procedure, the proportion of male centenarians in genealogies dropped from 28.2% to 23.2% (see Table 1) and became close to the proportions reported in the USA censuses (19.3–24.0%) [37], which indicates an improvement in quality for the selected genealogies.

2.2. Data Verification. Previous studies demonstrated that age misreporting and age exaggeration in particular are more common among long-lived individuals [38, 39]. Therefore, the primary focus of data cleaning in this study was on the age verification for long-lived individuals. We followed the approach of age verification and data linkage [38, 40], which we applied previously on another dataset of centenarians [41]. This approach involves data consistency checks, death date verification through the linkage to the Social Security Administration Death Master File (DMF) and birth date verification through the linkage to early USA censuses. DMF is a publicly available data resource (available at the Rootsweb.com website), which covers deaths that occurred in the period 1937–2010 and captures about 95% of deaths recorded by the National Death Index [42]. More details about the procedure of centenarian age validation were published elsewhere [41]. Validation of centenarian death and birth dates produced 1,574 centenarians. Information on siblings and spouses of validated centenarians was collected using the web-automation technique described earlier. Table 1 shows the steps of data collection and cleaning for this study. Note that the proportion of males among validated centenarians found in genealogies (23%) is close to the official reports (19–24%) for centenarians in the United States based on the census data [37].

We used only those records of centenarians whose age was successfully confirmed through the DMF (with matched birth and death years). We added only few cases where death year was different from that found in the DMF (however,

Table 1: Number of centenarians and their siblings at different stages of data collection and cleaning.

Type of records	Centenarians			Number of shorter-lived siblings
	Males	Females	Total	
All initial nonduplicate records for centenarians born in 1880–1895 with names of parents available	7,174	18,277	25,451	
Centenarians having detailed information on birth and death dates of their parents	6,370	16,757	23,127	172,091
Centenarians having detailed information on birth and death dates of their parents and siblings	707	2,127	2,834	21,893
Centenarians after data cleaning with confirmed death dates through the linkage to DMF	365	1,209	1,574	10,885

in these cases, the individual still had a centenarian status). Our previous work with centenarian data cleaning showed that incorrect death dates was the main source of errors in genealogical records of centenarians [41]. At the same time, birth dates were correctly reported in practically all records that had correct death dates and good consistency of birth and death dates for parents and siblings. Therefore, in this study we conducted a birth date verification procedure for a portion of approximately 15% of records. In all cases, birth years of centenarians agreed well with information reported in 1880, 1900, or 1910 censuses (as well as information about birth years of siblings). In addition to that, partial verification of centenarian birth dates was already accomplished through the linkage to DMF.

As a result of data quality checks, we found 1,574 records of centenarians born in 1880–1895 with verified birth and death dates. Given the fact that longevity is often clustered in families, we found other centenarians in the studied families (born outside the 1880–1995 time window) so that the total number of centenarians increased to 1,945 persons. Distribution of centenarians according to their lifespan is presented in Table 2. Note that the majority of centenarians lived less than 103 years and there are no claims of extraordinary high longevity (above 112 years) in the sample.

2.3. Life Span Data Reconstruction for Siblings and Spouses. Birth dates were reconstructed for all centenarian siblings using information available in computerized genealogies and early censuses. The procedure of death date verification using DMF is not feasible for validating death dates of shorter-lived siblings or spouses (used as controls) because data completeness of DMF is not very high for deaths occurred before the 1970s [43]. State death indexes, cemetery records, and obituaries cover longer periods of time. Taking into account that exact ages of death for controls (siblings) are not particularly important for comparison (it is sufficient to assume that they lived less than 100 years), we relied on death date information recorded in family histories for siblings and spouses not found in external sources. This approach was used previously in the Utah Population Database study for individuals died before 1932 [30]. Death dates were reconstructed for 99.99% of siblings using the social security death master file, state death indexes, and online genealogies (only 124 out of 13,654 cases were left unresolved).

Table 2: Distribution of centenarians born in 1880–1895, by age at death.

Age at death	Centenarians having siblings		
	Men	Women	Both sexes
100	132	398	530
101	92	266	358
102	52	214	266
103	43	137	180
104	16	71	87
105	18	58	76
106	9	38	47
107	2	15	17
108	0	5	5
109	1	3	4
110	0	3	3
111	0	0	0
112	0	1	1
Total:	365	1,209	1,574

2.4. Study Population. Data for 10,885 siblings of 1,574 centenarians were used in this study. As a result, each case (centenarian) had about 7 control siblings on average. The sibship size (eight siblings on average) in the studied centenarian families is higher than the average number of children in American families reported by the 1900 USA Census: 5.12 ± 0.01; data obtained from the 5% sample of the US 1900 Census from the integrated public use microdata series (IPUMS) [44]. Larger sibship size in the centenarian families compared to the general population can be explained by the fact that genealogies are more likely to be compiled for larger families and that longer-lived individuals in the United States were born more often in rural areas with higher fertility [41, 45]. This difference in sibship size with the general population is not critical for the within-family design of this study when appropriate control group (shorter-lived siblings raised in the same family or spouses) is selected. Table 3 presents characteristics of the final sample used in this study. 171 siblings and 4 centenarians had unknown month of birth, so their records were excluded from the statistical analyses. As expected, spouses have higher age at death than siblings whose age at death was not conditioned on survival

Table 3: Characteristics of centenarians born in 1880–1895 and their siblings and spouses. Values are numbers (percentages) or means (standard deviations).

Characteristic	Men	Women	Both Sexes
Number of records (percent)			
Centenarians, total	365 (23.2)	1,209 (76.8)	1,574 (100.0)
Centenarians with spouses	231 (23.9)	737 (76.1)	968 (100.0)
Siblings of centenarians	5,731 (52.7)	5,154 (47.3)	10,885 (100.0)
Spouses of centenarians	814 (75.2)	269 (24.8)	1,083 (100.0)
Mean age at death, years (standard deviation)			
Centenarians, total	101.5 (1.7)	101.8 (1.9)	101.7 (1.9)
Centenarians with spouses	101.5 (1.8)	101.8 (2.0)	101.8 (1.9)
Siblings of centenarians	62.9 (29.3)	66.1 (30.7)	64.3 (29.9)
Spouses of centenarians	72.69 (14.7)	77.8 (17.1)	73.9 (15.5)
Mean year of birth (standard deviation)			
Centenarians, total	1887.0 (5.5)	1888.6 (5.5)	1888.2 (5.5)
Centenarians with spouses	1887.4 (5.0)	1888.8 (4.7)	1888.4 (4.8)
Siblings of centenarians	1888.6 (10.4)	1889.0 (10.3)	1888.8 (10.4)
Spouses of centenarians	1885.4 (7.5)	1892.2 (7.2)	1887.1 (8.0)

to ages eligible for marriage. Centenarians and siblings were born in about the same year on average. Spouses of male centenarians were approximately 5 years younger and spouses of female centenarians were about 3 years older on average than their long-lived mates (see Table 3). 5% sample of the US 1900 Census (with information on month of birth) was used for comparisons with the general population [44].

2.5. Research Design. This study explored the effects of month of birth on the likelihood of survival to age 100. Centenarians (cases) were compared to their "normal" shorter-lived siblings (controls) or spouses using a within-family analysis. This approach allows investigators to study the within-family differences, not being confounded by the between-family variation. Long-lived persons born in 1880–1895 were used as cases. Siblings were born in a wider time window than centenarians but on average in the same year. Taking into account relatively high child mortality in the 19th century, we conducted analyses with different lifespan cut-offs in order to study late-life survival to advanced ages and evaluate the stability of results. The main approach used in this study is based on the comparison of children within rather than across families. A similar approach was applied for comparison of centenarians to their spouses.

2.6. Statistical Analyses. Differences in the month-of-birth distributions between centenarians or their siblings and the general population according to the 1900 US Census were assessed with the chi-square test. Standardized residuals were calculated in order to determine which months of birth may be major contributors to rejection of the null hypothesis (in the case it is rejected). When the absolute value of the residual is greater than 2.00, it indicates that it was a major influence on a significant chi-square test statistic. The chi-square test was also used to examine whether gender or longevity is related to the month of birth.

Statistical analyses of the within-family effects for 1 : n matched study were performed using a conditional multiple logistic regression model (fixed-effect model) to investigate the relationship between an outcome of being a case (long-lived person) and a set of prognostic factors [46, 47]. The likelihood of survival to advanced ages (to be in the centenarian group) is used as a dependent variable and month of birth and gender are used as explanatory variables. All analyses were conducted using Stata statistical software, release 11 [48]. Adjustment for multiple comparisons was conducted by the Bonferroni method. However, the Bonferroni adjustment is often criticized by statisticians as being too conservative [49, 50]. A technique proposed by Benjamini and Hochberg offers a more powerful alternative to the traditional Bonferroni method [51]. This technique is based on controlling the false discovery rate (FDR)—the proportion of significant results that are actually false positives. According to the Benjamini and Hochberg procedure, the null hypothesis is rejected when ordered individual P values (from smallest to largest) are lower than $(i/m)Q$, where i is a rank of P value, m is the total number of tests, and Q is the chosen FDR. The level of FDR in the Benjamini and Hochberg procedure was set to 0.10.

3. Results

Comparison of month-of-birth distributions for centenarians and their shorter-lived siblings with month-of-birth distribution for persons born in 1880–1890 and enumerated by the 1900 US Census showed statistically significant differences ($P < 0.001$ for both centenarians and their siblings). Table 4 shows month-of-birth distribution for centenarians, their siblings survived to adulthood and the general population. In the case of centenarians, absolute values of standardized residuals exceeded the critical value of 2 in six cases: there is an excess of centenarians born in

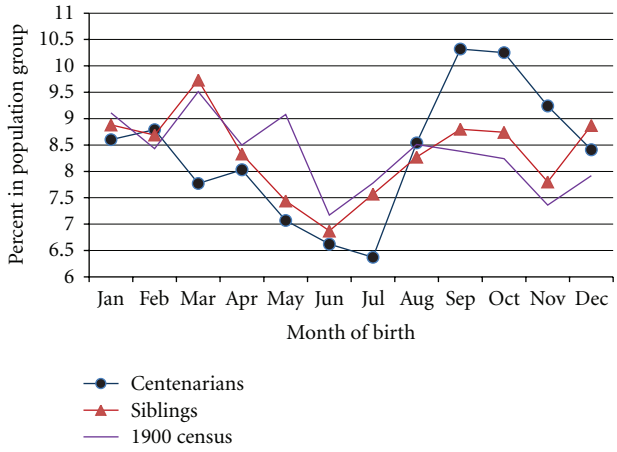

Figure 1: Distribution of individuals by month of birth in percent: centenarians, their shorter-lived siblings survived to age 20 and the USA population born in 1880–1895 according to the 1900 US Census.

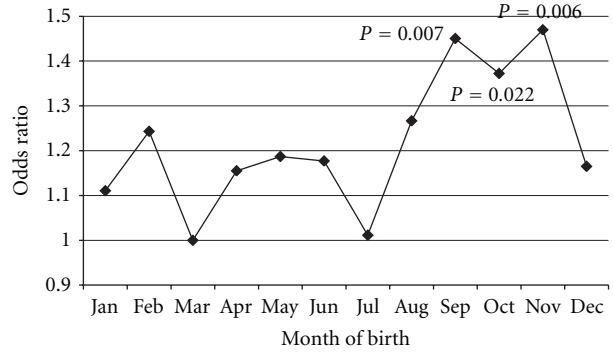

Figure 2: Month of birth and odds ratios for becoming a centenarian. A within-family-study of centenarians and their siblings survived to age 50 (9,724 studied persons). Being born in March is used as a reference level. Unadjusted P values are shown.

September–November and a lack of centenarians born in March, May, and July. Figure 1 shows that the excess of centenarians born in the fall months is particularly high compared to the general population. For siblings, absolute values of standardized residuals exceed the critical value only for May-born and December-born individuals. Overall, the seasonal pattern of births for siblings is closer to that for the general population compared to the seasonal pattern of births for centenarians (Figure 1). In the general population, more persons were born in the first half of the year (51.8%) while more centenarians were born in the second half of the year (53.12%). Centenarian siblings occupy an intermediate position with 49.94% being born in the first half of the year. These differences in birth seasonality (being born in the first or the second half of a year) between centenarians and their shorter-lived siblings (survived to age 20) are statistically significant (chi-square test statistic = 5.03, $df = 1$; $P = 0.025$). As shown in Table 4 and Figure 1, month-of-birth distribution for centenarians also departs from the distribution of their shorter-lived siblings and this difference is statistically significant (all siblings: chi-square test statistic = 19.99, $df = 11$, $P = 0.045$; siblings survived to age 20: chi-square test statistic 19.50, $df = 11$, $P = 0.053$). At the same time, we found no statistically significant association between month of birth and gender.

To analyze the effects of month-of-birth on exceptional longevity, which are not confounded by birth and infant death seasonality, childhood conditions, or genetic background, a within-family study was conducted. To discriminate between the effects due to differential survival early in life from the late-life effects, we analyzed survival to age 100 among siblings conditional on their survival to different adult ages. Table 5 presents the odds ratios to become a centenarian for siblings born in different months and survived to 30, 50, and 70 years of age. These results demonstrate that persons born in September–November have significantly higher chances of exceptional longevity than persons born in March. This survival advantage of persons born in the fall months is consistent across different lifespan cut-offs suggesting long-lasting influence of season of birth on longevity.

Being born in the spring months was associated with decreased chances of survival to age 100 while birth in the fall months significantly increases chances to become a centenarian. Figure 2 depicts the general pattern of month-of-birth effects on longevity after age 50. This pattern suggests that persons born during the fall months have higher chances of survival to age 100 compared to March-born individuals who have the lowest chances of achieving longevity. July-born individuals also show low odds of survival to age 100 compared to individuals born in fall.

It was suggested that month-of-birth effects on mortality may become weaker for later-born cohorts [19]. To test whether the season-of-birth effects are weaker in later-born cohorts, we split the sample of centenarians and siblings into two approximately equal groups: those who were born before 1899 and those who were born after this year. The effects of month-of-birth on survival after age 50 for these two cohorts are presented in Table 6. For group born before 1899, the odds of survival to 100 are significantly higher for persons born in November compared to persons born in March. For later-born cohorts, the month-of-birth effect is much weaker and not statistically significant after adjustment for multiple comparisons.

In order to control for living conditions during the adult life, we compared centenarians with their spouses. The results of these comparisons are shown in Table 7 and confirm the month-of-birth pattern in longevity found in previous analyses. Again, individuals born in October-November have a significantly higher likelihood of survival to age 100 compared to individuals born in April. These effects are long-lasting and can be visible after age 50.

4. Discussion

4.1. Comparison with Previous Studies. We found that persons born in the fall months are more represented among centenarians compared to the general population while

Table 4: Month-of-birth distributions (in percent) for the US 1900 Census population, centenarians, and their siblings[a].

Month of birth	1900 Census, 5% sample Persons born in 1880–95	Centenarians		Siblings survived to age 20	
	N = 1,320,328	N = 1,570	Standardized residuals	N = 9,175	Standardized residuals
January	9.11	8.60	−0.671	8.88	−0.721
February	8.43	8.79	0.491	8.69	0.847
March	9.51	7.77	−2.235	9.73	0.693
April	8.50	8.03	−0.645	8.33	−0.568
May	9.08	7.07	−2.643	7.44	−5.200
June	7.17	6.62	−0.808	6.87	−1.086
July	7.78	6.37	−2.004	7.57	−0.704
August	8.51	8.54	0.034	8.27	−0.780
September	8.38	10.32	2.653	8.80	1.375
October	8.24	10.25	2.781	8.74	1.672
November	7.36	9.24	2.739	7.80	1.567
December	7.92	8.41	0.687	8.87	3.240

[a]Month-of-birth distributions for both centenarians and their siblings differ from the month-of-birth distribution for the general population (individuals enumerated in the 1900 census and born in 1880–1895); difference significant at $P < 0.001$.

Table 5: Odds ratios (P values) to become a centenarian as predicted by conditional logistic regression (fixed effects) for different age cut-off subgroups. Effects of month of birth[a].

Variable	All siblings	Siblings survived to age 30	Siblings survived to age 50	Siblings survived to age 70
Month of birth:				
January	1.13 (0.387)	1.11 (0.472)	1.11 (0.463)	1.09 (0.537)
February	1.25 (0.101)	1.25 (0.109)	1.24 (0.124)	1.16 (0.303)
March	Reference	Reference	Reference	Reference
April	1.15 (0.320)	1.15 (0.337)	1.16 (0.320)	1.09 (0.567)
May	1.20 (0.218)	1.17 (0.288)	1.19 (0.251)	1.15 (0.373)
June	1.20 (0.229)	1.00 (0.254)	1.18 (0.284)	1.11 (0.486)
July	1.03 (0.855)	1.19 (0.991)	1.01 (0.941)	1.00 (0.990)
August	1.25 (0.110)	1.24 (0.125)	1.27 (0.100)	1.21 (0.198)
September	**1.44 (0.006)**[c]	**1.43 (0.009)**[c]	**1.45 (0.007)**[c]	**1.39 (0.022)**
October	**1.43 (0.008)**[c]	**1.37 (0.021)**[c]	**1.37 (0.022)**[c]	1.27 (0.099)
November	**1.51 (0.003)**[b,c]	**1.48 (0.005)**[c]	**1.47 (0.006)**[c]	**1.41 (0.017)**
December	1.17 (0.266)	1.13 (0.380)	1.17 (0.283)	1.11 (0.486)
Female sex	3.77 (<0.001)	3.82 (<0.001)	3.80 (<0.001)	3.41 (<0.001)
Pseudo R^2	0.0811	0.0861	0.0871	0.0766
Number of observations	12,132	10,393	9,724	8,123

[a]Statistically significant effects ($P < 0.05$) are highlighted in bold.
[b]Statistically significant after Bonferroni adjustment.
[c]Statistically significant after Benjamini-Hochberg procedure.

persons born in the first half of the year are less represented among the group of long-lived individuals. Centenarians, their siblings, and the general population show decreased proportion of persons born during the summer months, which is probably related to seasonal distributions of births and infant deaths in the past [52]. The month-of-birth pattern among centenarians in this study is compared to the month-of-birth distribution of persons aged 5–20 years in the US 1900 Census and hence the results of this comparison are not affected by seasonal distribution of infant deaths.

Table 6: Odds ratios (P values) to become a centenarian as predicted by conditional logistic regression (fixed effects), by different birth cohort subgroups for siblings survived to age 50. Effects of month of birth[a].

Variable	Born before 1889	Born in 1889 or later
Month of birth:		
January	1.42 (0.109)	0.90 (0.628)
February	1.41 (0.118)	1.02 (0.911)
March	Reference	Reference
April	1.09 (0.717)	1.18 (0.467)
May	1.27 (0.312)	0.86 (0.500)
June	1.30 (0.289)	1.35 (0.192)
July	1.14 (0.579)	1.00 (0.984)
August	1.19 (0.446)	1.23 (0.360)
September	1.31 (0.209)	**1.55 (0.042)**
October	**1.61 (0.027)**	1.13 (0.578)
November	**1.78 (0.008)**[b]	1.23 (0.357)
December	1.33 (0.185)	0.94 (0.772)
Female sex	3.32 (<0.001)	4.74 (<0.001)
Pseudo R^2	0.0842	0.1249
Number of observations	3,279	3,441

[a]Statistically significant effects ($P < 0.05$) are highlighted in bold.
[b]Statistically significant after Benjamini-Hochberg procedure.

Table 7: Odds ratios (P values) to become a centenarian as predicted by conditional logistic regression (fixed effects), by different age cut-off and gender subgroups for spouses of centenarians. Effects of month of birth[a].

Variable	All spouses	Spouses survived to age 50
Month of birth:		
January	1.27 (0.358)	1.29 (0.346)
February	1.65 (0.066)	**1.83 (0.032)**
March	1.58 (0.084)	1.65 (0.067)
April	Reference	Reference
May	1.37 (0.263)	1.46 (0.190)
June	1.46 (0.176)	1.63 (0.099)
July	1.61 (0.096)	1.66 (0.086)
August	1.52 (0.117)	1.56 (0.112)
September	1.58 (0.079)	1.66 (0.063)
October	**2.17 (0.004)**[b,c]	**2.24 (0.004)**[b,c]
November	**2.22 (0.003)**[b,c]	**2.22 (0.004)**[b,c]
December	1.21 (0.487)	1.32 (0.332)
Female sex	3.40 (<0.001)	3.42 (<0.001)
Pseudo R^2	0.2192	0.2226
Number of observations	1,921	1,800

[a]Statistically significant seasonal effects ($P < 0.05$) are highlighted in bold.
[b]Statistically significant after Bonferroni adjustment.
[c]Statistically significant after Benjamini-Hochberg procedure.

In the previous study of German semi-supercentenarians, seasonal distribution of birth dates of long-lived individuals was compared to the seasonal distribution of births 105 years earlier, so this comparison may be influenced by seasonality of infant deaths in the past. At the same time, our results demonstrate some similarity with the results of semi-supercentenarian study: persons born in the second half of the year are over-represented among German semi-supercentenarians [23] as it was shown in this study. The within-family multivariate analysis demonstrated a survival advantage of individuals born in September–November compared to individuals born in March. A similar pattern of season-of-birth and longevity was also found for spouses of centenarians, which reinforces the findings obtained for centenarian siblings. These results are in agreement with previous publications on the effects of month-of-birth on lifespan in the countries of the Northern hemisphere [15, 16, 21, 22, 53] and in the United States in particular [19, 54]. These earlier studies show better survival for persons born in September–December compared to persons born in the middle of the year.

At the same time, the results of this study show that individuals born in March or April have similar low odds of achieving longevity as individuals born during the summer months and persons born during the winter months do not live longer than the March-born individuals. This is different from the results of other studies, which showed decline in mean age at death for persons born during the summer months and relatively high mean age at death for persons born during the winter months [19, 21, 22]. These differences in month of birth pattern between our study and other publications can be partially explained by changes in seasonality of births and infant deaths over time. Births usually peak in March and hence March-born individuals are overrepresented among both living and dead persons (this is the reason why March-born individuals are highly represented in the general population, see Figure 1). Studies based on the analysis of cross-sectional death certificates do not have information about population at risk [19] and hence may be affected by secular changes in seasonality of births and infant deaths. Although these secular effects probably do not significantly modify the overall month-of-birth pattern in life expectancy, they can affect amplitudes of seasonal effects for specific months. It would be reasonable to suggest that decline of summer infant deaths over time resulted in increased representation of summer-born individuals in the later-born cohorts, which led to an apparent drop in the mean age at death for persons born in these months.

Study of the earlier-born and the later-born groups found that the season-of-birth effects fade in the later born cohorts (Table 6), which is consistent with previous reports [19] and can be explained by improving nutrition and sanitation over time. We found no gender differences in month-of-birth distributions for both centenarians and their siblings, which is consistent with previous publications [19].

It should be noted that another study of season-of-birth effects on life span in the single-year USA birth cohorts (based on the USA Social Security Administration data) found that life expectancy at age 80 depends on month of birth [54]. In this study, 80-year olds born in May-June showed significantly lower life expectancy compared to

individuals born in the end of the year and this seasonal pattern repeats itself in every studied birth cohort. This month-of-birth pattern of life expectancy is similar to the pattern reported earlier for mean age at death obtained on the basis of the USA death certificates [19]. However, in the study of centenarians and their siblings, we do not find a specific survival advantage for persons born in the winter months. It is possible that certain unobserved socioeconomic or other characteristics of parents (such as possible preferential winter births for wealthier social groups), which are controlled for in the case-sibling design of our study, may result in apparently better survival of winter-born individuals in the general population. Further research is needed for better explanation of this phenomenon.

4.2. Strengths and Limitations. Our within-family study follows centenarians and their siblings from birth until the end of their life while previous studies analyzed a cross-sectional sample of the USA death certificates for persons belonging to multiple birth cohorts. For this reason, our results do not depend on the secular changes in seasonality of births and infant deaths. Another advantage of this study is its within-family design, which controls for unobserved characteristics of childhood conditions and parental genetic background. This study confirms the existence of month-of-birth effects on longevity and shows that these effects can be observed even after controlling for unobserved between-family variation.

Some limitations of this study should be mentioned. Due to the data collection from computerized genealogies, we cannot be certain that centenarians (and controls) represent a random population sample. This limitation is not crucial for the analytical approach applied in this study, which tests specific hypothesis of seasonal birth effects on longevity, but may pose a question about generalizability of results. It is believed that the RootsWeb source of online family histories has more individuals with larger families and better offspring survival. Indeed our sample of centenarians has larger families compared to the general population. This deviation from the general population may potentially affect the results of univariate analyses when month-of-birth distributions for centenarians and siblings are compared to the general population. However, in the within-family analyses, we compare siblings with each other rather than with the general population, so the difference in family size does not affect the results of hypothesis testing about the month-of-birth effects on longevity [55]. Also, comparison with the general population shows better similarity of month-of-birth pattern for siblings rather than centenarians suggesting that shorter-lived siblings are closer to the general population in terms of month-of-birth distribution.

Another problem is that some month-of-birth effects become not statistically significant after adjustment for multiple comparisons. For example, month-of-birth effects become nonsignificant when survival of siblings after age 70 is studied. However, the overall pattern of month-of-birth effects on longevity shows consistency across different age cut-offs suggesting a stability of the observed seasonal pattern. In addition to that, independent analyses on centenarian spouses demonstrated a similar pattern of month-of-birth effects on longevity. Finally, the conditional logistic regression analyses suggest that despite significant effects of months of birth on relative survival the effect sizes of month-of-birth effects on survival to 100 are small and explain about 2% of the variance of becoming a centenarian. This small percentage of explained variance is related to very high variability of individual lifespan, which has a substantial stochastic component [6].

4.3. Existing Explanations of Month-of-Birth Effects on Longevity

4.3.1. Maternal and Child Nutrition in the Past. There are several possible explanations of why month of birth may affect mortality and health later in life. One explanation suggests that nutritional status of mother during pregnancy may affect fetal development in utero [3, 56]. Nutritional deficiencies during early development may have long-lasting effects on mortality later in life [3]. This explanation is supported by the Ames theory [57] that micronutrient deficiencies play a major role in DNA damaging, human aging, and premature deaths from cancer and heart disease. Recent review suggests that both improper diet stimulating chronic inflammation and dietary deficiencies and nutrient imbalances may be strong sources of mutagenesis [58]. So it is reasonable to hypothesize that seasonal vitamin deficiency during the critical periods of fetus and infant development may affect later health and longevity of the deficiency-exposed birth cohorts.

Birth weight often serves as an indicator of nutritional status during early development and was shown to be dependent on month of birth. For example, in Greece, infants born during the autumn and winter seasons of the year had significantly increased birth weight and gestation age [20]. Recent review of birth weight seasonality in developed countries shows a tendency of infants born during the fall and winter seasons in European countries to have higher birth weights [59]. There are also reports that premature births show a slight excess of incidence during the months of June–August [60].

4.3.2. Seasonal Infections. Early seasonal impacts on subsequent adult lifespan may include not only seasonal vitamin deficiency, but also other seasonal impacts, such as infectious diseases. Seasonal peaks of disease occurrence are typical for many infections [61]. The most drastic effects of infectious agents in pregnancy, which probably represent the tip of the iceberg of the damage to progeny, include the following: cardiac malformation, deafness, cataracts, glaucoma, and tooth defects for the rubella virus (German measles); growth retardation, blindness, mental retardation, and deafness for cytomegalovirus; skin scarring, muscle atrophy, and mental retardation for varicella (chickenpox) [62, 63]. It was shown that poliovirus epidemics peak in July-August and exposure to this virus in the second trimester of gestation seem to produce subsequent adult schizophrenia in February birth cohorts [64].

4.3.3. Environmental Temperature at Birth and Conception. Effect of environmental temperature during the time of birth or conception may be another possible explanation for low proportion of centenarians among individuals born during the summer and spring months. For example, British women experiencing higher summer temperatures during their first year of life and hence suffering severe diarrhea and dehydration in infancy had higher blood pressure at older ages [65]. High ambient temperature was also associated with higher risk of preterm delivery in the recent study of a large sample of California births [66]. There are reports that high temperatures may be implicated in lower sperm quality [67, 68], particularly among smokers [69]. This may result in a less viable progeny born during the spring months. On the other hand, cold outdoor temperature at birth during the winter months is associated with coronary heart disease, insulin resistance, and poor lung function at older age [70].

4.3.4. The Deadline Hypothesis. It was suggested that schools or other professional training organizations, which have a deadline for admission, may favor children who are somewhat older compared to their peers (usually children born in the fall months). This so-called deadline hypothesis [19] predicts that the relative advantage in school achievement has cumulative effects over the life course. In the case of historical data, the deadline hypothesis should be more relevant to survival of men whose social status is dependent on their own achievements. For women, their social status in the past was predominantly determined by the social status of their husbands. In the case of centenarians, who are predominantly women, the deadline hypothesis looks like a less likely explanation of the observed month-of-birth effects on longevity.

4.4. Explanation of Survival Advantage for Persons Born in the Fall Months. Analysis of the existing literature suggests that persons born in the fall months in the United States could avoid extremes of very high and very low ambient temperatures during their first month of life as well as high summer temperatures during conception. Persons born during the fall months also did not experience an early exposure to infectious diseases, which were common during summer, early winter, or spring months in the past. Seasonal pattern of the USA mortality for children below age one month in the past supports this suggestion. According to the USA statistics, mortality below age one month in 1940 was the lowest in September–November [52] suggesting lower infectious load during this period of the year, because most infant deaths in the past were caused by infections. Better maternal nutrition during the last trimester of pregnancy also contributed to the survival advantage of individuals born during the fall season. All these three factors (mild ambient temperatures, better maternal nutrition, and low infectious load) helped persons born in the fall months to avoid accumulation of excessive number of defects by body systems very early in life. These results are consistent with the high initial damage load (HIDL) hypothesis [6, 7], which emphasizes the importance of the initial level of damage in determining future human longevity. More specific explanation of the observed month-of-birth effects on longevity can be provided by the inflammation hypothesis suggested by Finch and Crimmins [11]. According to this hypothesis, a strong acute-phase inflammatory response to childhood infections initiates chronic inflammation, which promotes chronic diseases of aging. Reduced lifetime exposure to infection and subsequent inflammation may explain both declining mortality at older ages and decreasing amplitude of month-of-birth effects on lifespan over time. The results obtained in this study suggest that optimizing the process of early development can potentially result in avoiding many diseases in later life and significant extension of healthy life span. More research is needed to determine more specific factors of seasonal birth effects on longevity.

5. Conclusions

This is the first study of association between month of birth and exceptional longevity, which controls for early-life shared conditions and common genetic background. We developed a large sample of validated centenarians, their siblings, and spouses to study early-life seasonal effects on human longevity. We found significant associations between month of birth and longevity: individuals born in September–November have higher likelihood of becoming centenarians compared to March-born individuals. These results are consistent with the reports of higher life expectancy for persons born in the end of the year [16, 19, 21, 22] and the study of mortality after age 80 in several single-year USA birth cohorts [54]. The results of this study demonstrate that month-of-birth effects on exceptional longevity persist after controlling for shared childhood environment and unobserved shared characteristics of parents. Association of month-of-birth with exceptional longevity appears to be stronger for earlier birth cohorts born before 1899. Similar month-of-birth effects on longevity were found for centenarian spouses: individuals born in October-November were more likely to live to 100 compared to individuals born in April. The results of this study suggest that early-life environmental conditions may have long-lasting effects on human aging and longevity.

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References

[1] N. Barzilai and A. R. Shuldiner, "Searching for human longevity genes: the future history of gerontology in the post-genomic era," *Journals of Gerontology A*, vol. 56, no. 2, pp. M83–M87, 2001.

[2] D. C. Willcox, B. J. Willcox, H. Todoriki, J. D. Curb, and M. Suzuki, "Caloric restriction and human longevity: what can we

learn from the Okinawans?" *Biogerontology*, vol. 7, no. 3, pp. 173–177, 2006.

[3] D. J. P. Barker, *Mothers, Babies, and Disease in Later Life*, Churchill Levingstone, London, UK, 1998.

[4] D. Kuh and B. Ben-Shlomo, *A Life Course Approach to Chronic Disease Epidemiology*, Oxford University Press, Oxford, UK, 1997.

[5] R. W. Fogel, "Technophysio evolution and the measurement of economic growth," *Journal of Evolutionary Economics*, vol. 14, no. 2, pp. 217–221, 2004.

[6] L. A. Gavrilov and N. S. Gavrilova, *The Biology of Life Span: A Quantitative Approach*, Harwood Academic Publisher, New York, NY, USA, 1991.

[7] L. A. Gavrilov and N. S. Gavrilova, "Early-life programming of aging and longevity: the idea of high initial damage load (the HIDL hypothesis)," *Annals of the New York Academy of Sciences*, vol. 1019, pp. 496–501, 2004.

[8] L. A. Gavrilov and N. S. Gavrilova, "Early-life factors modulating lifespan," in *Modulating Aging and Longevity*, S. I. S. Rattan, Ed., pp. 27–50, Kluwer Academic, Dodrecht, The Netherlands, 2003.

[9] M. D. Hayward and B. K. Gorman, "The long arm of childhood: the influence of early-life social conditions of men's mortality," *Demography*, vol. 41, no. 1, pp. 87–107, 2004.

[10] K. R. Smith, G. P. Mineau, G. Garibotti, and R. Kerber, "Effects of childhood and middle-adulthood family conditions on later-life mortality: evidence from the Utah Population Database, 1850–2002," *Social Science and Medicine*, vol. 68, no. 9, pp. 1649–1658, 2009.

[11] C. E. Finch and E. M. Crimmins, "Inflammatory exposure and historical changes in human life-spans," *Science*, vol. 305, no. 5691, pp. 1736–1739, 2004.

[12] E. M. Crimmins and C. E. Finch, "Infection, inflammation, height, and longevity," *Proceedings of the National Academy of Sciences of the United States of America*, vol. 103, no. 2, pp. 498–503, 2006.

[13] D. A. Lawlor, H. Clark, G. Ronalds, and D. A. Leon, "Season of birth and childhood intelligence: findings from the Aberdeen Children of the 1950s cohort study," *British Journal of Educational Psychology*, vol. 76, no. 3, pp. 481–499, 2006.

[14] G. D. Smith, S. Leary, A. Ness, and D. A. Lawlor, "Challenges and novel approaches in the epidemiological study of early life influences on later disease," in *Early Nutrition Programming and Health Outcomes in Later Life: Obesity and Beyond*, B. Kolatzko, T. Decsi, D. Molnar, and A. DeLaHunty, Eds., pp. 1–14, Springer, Dordrecht, The Netherlands, 2009.

[15] G. Doblhammer and J. W. Vaupel, "Lifespan depends on month of birth," *Proceedings of the National Academy of Sciences of the United States of America*, vol. 98, no. 5, pp. 2934–2939, 2001.

[16] L. A. Gavrilov and N. S. Gavrilova, "Season of birth and human longevity," *Journal of Anti-Aging Medicine*, vol. 2, no. 4, pp. 365–366, 1999.

[17] A. M. Vaiserman and V. P. Voitenko, "Early programming of adult longevity: demographic and experimental studies," *Journal of Anti-Aging Medicine*, vol. 6, no. 1, pp. 11–20, 2003.

[18] E. Hungtington, *Season of Birth*, John Wiley & Sons, New York, NY, USA, 1938.

[19] G. Doblhammer, *The Late Life Legacy of Very Early Life. Demographic Research Monographs*, Springer, Heidelberg, Germany, 2004.

[20] A. D. Flouris, Y. Spiropoulos, G. J. Sakellariou, and Y. Koutedakis, "Effect of seasonal programming on fetal development and longevity: links with environmental temperature," *American Journal of Human Biology*, vol. 21, no. 2, pp. 214–216, 2009.

[21] A. Lerchl, "Month of birth and life expectancy: role of gender and age in a comparative approach," *Naturwissenschaften*, vol. 91, no. 9, pp. 422–425, 2004.

[22] A. M. Vaiserman, A. C. Collinson, N. M. Koshel, I. I. Belaja, and V. P. Voitenko, "Seasonal programming of adult longevity in Ukraine," *International Journal of Biometeorology*, vol. 47, no. 1, pp. 49–52, 2002.

[23] G. Doblhammer, R. Scholz, and H. Maier, "Month of birth and survival to age 105+: evidence from the age validation study of German semi-supercentenarians," *Experimental Gerontology*, vol. 40, no. 10, pp. 829–835, 2005.

[24] M. Kihlbom and S. E. Johansson, "Month of birth, socioeconomic background and development in Swedish men," *Journal of Biosocial Science*, vol. 36, no. 5, pp. 561–571, 2004.

[25] M. Bobak and A. Gjonca, "The seasonality of live birth is strongly influenced by socio-demographic factors," *Human Reproduction*, vol. 16, no. 7, pp. 1512–1517, 2001.

[26] F. L. Goodenough, "Month of birth as related to socio economic status of parents," *Pedagogical Seminary and Journal of Genetic Psychology*, vol. 59, pp. 65–76, 1941.

[27] D. T. A. Eisenberg, B. Campbell, J. MacKillop, J. K. Lum, and D. S. Wilson, "Season of birth and dopamine receptor gene associations with impulsivity, sensation seeking and reproductive behaviors," *PLoS ONE*, vol. 2, no. 11, Article ID e1216, 2007.

[28] L. A. Gavrilov and N. S. Gavrilova, "Epidemiology of human longevity: the search for appropriate methodology," *Journal of Anti-Aging Medicine*, vol. 4, no. 1, pp. 13–30, 2001.

[29] N. S. Gavrilova, L. A. Gavrilov, G. N. Evdokushkina et al., "Evolution, mutations, and human longevity: European royal and noble families," *Human Biology*, vol. 70, no. 4, pp. 799–804, 1998.

[30] R. A. Kerber, E. O'Brien, K. R. Smith, and R. M. Cawthon, "Familial excess longevity in Utah genealogies," *Journals of Gerontology A*, vol. 56, no. 3, pp. B130–B139, 2001.

[31] R. Pearl and R. D. W. Pearl, *The Ancestry of the Long-Lived*, John Hopkins University Press, Baltimore, Md, USA, 1934.

[32] B. J. Willcox, D. C. Willcox, Q. He, J. D. Curb, and M. Suzuki, "Siblings of Okinawan centenarians share lifelong mortality advantages," *Journals of Gerontology A*, vol. 61, no. 4, pp. 345–354, 2006.

[33] J. W. Adams and A. B. Kasakoff, "Estimates of census underenumeration based on genealogies," *Social Science History*, vol. 15, pp. 527–543, 1991.

[34] L. A. Gavrilov, N. S. Gavrilova, S. J. Olshansky, and B. A. Carnes, "Genealogical data and the biodemography of human longevity," *Social Biology*, vol. 49, no. 3-4, pp. 160–173, 2002.

[35] K. R. Smith, G. P. Mineau, and L. L. Bean, "Fertility and postreproductive longevity," *Social Biology*, vol. 49, no. 3-4, pp. 185–205, 2002.

[36] D. Sklar and A. Trachtenberg, *PHP Cookbook*, O'Reilly, 2002.

[37] C. A. Krach and V. A. Velkoff, *Centenarians in the United States*, Government Printing Office, Washington, DC, USA, 1999.

[38] I. T. Elo, S. H. Preston, I. Rosenwaike, M. Hill, and T. P. Cheney, "Consistency of age reporting on death certificates and social security records among elderly African Americans," *Social Science Research*, vol. 25, no. 3, pp. 292–307, 1996.

[39] B. Jeune and J. Vaupel, *Validation of Exceptional Longevity*, Odense University Publisher, Odense Denmark, 1999.

[40] S. H. Preston, I. T. Elo, I. Rosenwaike, and M. Hill, "African-American mortality at older ages: results of a matching study," *Demography*, vol. 33, no. 2, pp. 193–209, 1996.

[41] N. S. Gavrilova and L. A. Gavrilov, "Search for predictors of exceptional human longevity: using computerized genealogies and internet resources for human longevity studies," *North American Actuarial Journal*, vol. 11, no. 1, pp. 49–67, 2007.

[42] H. D. Sesso, R. S. Paffenbarger, and I. M. Lee, "Comparison of National Death Index and World Wide Web death searches," *American Journal of Epidemiology*, vol. 152, no. 2, pp. 107–111, 2000.

[43] K. Faig, *Reported Deaths of Centenarians and Near-Centenarians in the U.S. Social Security Administration's Death Master File*, Orlando, Fla, USA, 2001, Paper presented at the Society of Actuaries "Living to 100" International Symposium.

[44] S. Ruggles, J. T. Alexander, K. Genadek et al., *Integrated Public Use Microdata Series (IPUMS): Version 5.0 [Machine-readable database]*, University of Minnesota, Minneapolis, Minn, USA, 2010.

[45] S. H. Preston, M. E. Hill, and G. L. Drevenstedt, "Childhood conditions that predict survival to advanced ages among African-Americans," *Social Science and Medicine*, vol. 47, no. 9, pp. 1231–1246, 1998.

[46] D. W. Hosmer and S. Lemeshow, *Applied Logistic Regression*, John Wiley & Sons, New York, NY, USA, 2001.

[47] N. E. Breslow and N. E. Day, *Statistical Methods in Cancer Research*, vol. 1 of *The Analysis of Case-Control Studies*, Oxford University Press, Oxford, UK, 1993.

[48] StataCorp, *Stata Statistical Software: Release 11*, StataCorp LP, College Station, Tex, USA, 2009.

[49] T. V. Perneger, "What's wrong with Bonferroni adjustments," *British Medical Journal*, vol. 316, no. 7139, pp. 1236–1238, 1998.

[50] K. J. Rothman, "No adjustments are needed for multiple comparisons," *Epidemiology*, vol. 1, no. 1, pp. 43–46, 1990.

[51] Y. Benjamini and Y. Hochberg, "Controlling the false discovery rate—a practical and powerful approach to multiple testing," *Journal of the Royal Statistical Society B*, vol. 57, pp. 289–300, 1995.

[52] F. E. Linder and R. D. Grove, *Vital Statistics Rates in the United States 1900–1940*, Goverment Printing Office, Washington, USA, 1943.

[53] E. L. Abel and M. L. Kruger, "Birth month affects longevity," *Death Studies*, vol. 34, no. 8, pp. 757–763, 2010.

[54] L. A. Gavrilov and N. S. Gavrilova, "Mortality measurement at advanced ages: a study of the Social Security Administration Death Master File," in *Living to 100 and Beyond: Survival at Advanced Ages [online monograph]*, The Society of Actuaries, Shaumburg, Ill, USA, 2008.

[55] M. Woodward, *Epidemiology. Study Design and Data Analysis*, Chapman & Hall/CRC, Boca Raton, Fla, USA, 2005.

[56] P. E. Watson and B. W. McDonald, "Seasonal variation of nutrient intake in pregnancy: effects on infant measures and possible influence on diseases related to season of birth," *European Journal of Clinical Nutrition*, vol. 61, no. 11, pp. 1271–1280, 2007.

[57] B. N. Ames, "Micronutrients prevent cancer and delay aging," *Toxicology Letters*, vol. 102-103, pp. 5–18, 1998.

[58] L. R. Ferguson, "Dietary influences on mutagenesis-Where is this field going?" *Environmental and Molecular Mutagenesis*, vol. 51, no. 8-9, pp. 909–918, 2010.

[59] G. Chodick, S. Flash, Y. Deoitch, and V. Shalev, "Seasonality in birth weight: review of global patterns and potential causes," *Human Biology*, vol. 81, no. 4, pp. 463–477, 2009.

[60] S. M. Kevan, "Season of life—season of death," *Social Science and Medicine*, vol. 13, no. 4, pp. 227–232, 1979.

[61] J. P. Fox, C. E. Hall, and L. Elveback, *Epidemiology. Man and Disease*, The Macmillan Company, London, UK, 1970.

[62] K. L. Moore, *The Developing Human. Clinically Oriented Embriology*, WB Saunders, Philadelphia, Pa, USA, 4th edition, 1988.

[63] A. Ornoy and A. Tenenbaum, "Pregnancy outcome following infections by coxsackie, echo, measles, mumps, hepatitis, polio and encephalitis viruses," *Reproductive Toxicology*, vol. 21, no. 4, pp. 446–457, 2006.

[64] J. Suvisaari, J. Haukka, A. Tanskanen, T. Hovi, and J. Lönnqvist, "Association between prenatal exposure to poliovirus infection and adult schizophrenia," *American Journal of Psychiatry*, vol. 156, no. 7, pp. 1100–1102, 1999.

[65] D. A. Lawlor, G. D. Smith, R. Mitchell, and S. Ebrahim, "Adult blood pressure and climate conditions in infancy: a test of the hypothesis that dehydration in infancy is associated with higher adult blood pressure," *American Journal of Epidemiology*, vol. 163, no. 7, pp. 608–614, 2006.

[66] R. Basu, B. Malig, and B. Ostro, "High ambient temperature and the risk of preterm delivery," *American Journal of Epidemiology*, vol. 172, no. 10, pp. 1108–1117, 2010.

[67] G. M. Centola and S. Eberly, "Seasonal variations and age-related changes in human sperm count, motility, motion parameters, morphology, and white blood cell concentration," *Fertility and Sterility*, vol. 72, no. 5, pp. 803–808, 1999.

[68] Z. Chen, T. Toth, L. Godfrey-Bailey, N. Mercedat, I. Schiff, and R. Hauser, "Seasonal variation and age-related changes in human semen parameters," *Journal of andrology*, vol. 24, no. 2, pp. 226–231, 2003.

[69] R. Künzle, M. D. Mueller, A. W. Huber, H. Drescher, and N. A. Bersinger, "Seasonality in human semen quality of smokers and non-smokers: effect of temperature," *Asian Journal of Andrology*, vol. 6, no. 3, pp. 243–247, 2004.

[70] D. A. Lawlor, G. Davey Smith, R. Mitchell, and S. Ebrahim, "Temperature at birth, coronary heart disease, and insulin resistance: cross sectional analyses of the British women's heart and health study," *Heart*, vol. 90, no. 4, pp. 381–388, 2004.

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Research Article

Are Men Aging as Oaks and Women as Reeds? A Behavioral Hypothesis to Explain the Gender Paradox of French Centenarians

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Since the 1990s, several studies involving French centenarians have shown a gender paradox in old age. Even if women are more numerous in old age and live longer than men, men are in better physical and cognitive health, are higher functioning, and have superior vision. If better health should lead to a longer life, why are men not living longer than women? This paper proposes a hypothesis based on the differences in the *generational habitus* between men and women who were born at the beginning of the 20th century. The concept of *generational habitus* combines the generation theory of Mannheim with the *habitus* concept of Bourdieu based on the observation that there exists a way of being, thinking, and doing for each generation. We hypothesized that this *habitus* still influences many gender-linked behaviours in old age. Men, as "oaks," seem able to delay the afflictions of old age until a breaking point, while women, as "reeds," seem able to survive despite an accumulation of health deficits.

"*The winds that trees and mountains tear alarm not me—unbroken still I bend .*"
(Jean de la Fontaine, Fables 1, 22)

1. Introduction

The title of this paper builds on the famous fable of Jean de la Fontaine (1621–1695) *The Oak and the Reed* in which he compares the two plants facing the natural elements (Table 2). The moral of the fable is that the oak remains immovable while the reed bends into the wind, but when the wind becomes stronger, the oak is uprooted while the flexible reed survives. This metaphor illustrates the hypothesis we develop to explain part of the gender paradox noted by Allard and Robine [1] as a result of a large national survey on centenarians: "*…women are more prevalent in old age but in poorer overall health. It leads to an impossible challenge: if you want to become a centenarian, be a woman, you're more likely to achieve the breakthrough but once you get there, be a man, as you will be in better shape.*" Comparing the responses of the reeds to the wind with the adaptation of elderly to the inherent frailty process that progresses with age, we defend the hypothesis that men, as oaks, have the capacity to cope with the challenges of old age until a breaking point, whereupon they die, while women, as reeds, are more flexible and can adapt and survive.

2. Materials and Methods

2.1. Data from Previous Epidemiological Studies on French Oldest Old. In order to illustrate our hypothesis, we put into perspective various observations related to the French

oldest old population coming from three different studies: *In the Search of the Secret of Centenarians* (1990–2000) (A la recherche du secret des centenaires (SIECLE and Ipsen Foundation)), *European Challenge for Healthy Ageing* (ECHA, 2003-2004), and *GEnetics of Healthy Aging* (GEHA, 2004–2010). The observation of the gender paradox was made over a ten-year period by Allard and Robine based upon a study of 3500 centenarians alive in France in 1990. Indeed, the Ipsen survey (1990–2000) followed 800 centenarians, 95 men and 705 women, including Mme Jeanne Calment (a women!), the oldest person who ever existed and who lived until 122 years old. Women were more numerous than men in the centenarian population in 1990, and it is still the case today.

The figures (Table 1) show that women in old age are more numerous than men, with a sex-ratio (i.e., number of women divided by the number of men) strongly increasing with age. Moreover, women live longer according to French National Institute of Statistics (INSEE): life expectancy at birth, in 2010, was 84.8 years for women and 78.1 for men (Bilan démographique 2010, Insee Première 1332—January 2011). The longevity gap was 6.7 years between men and women. Beyond the French context, when we look at the living supercentenarians (people having reached the age of 110 years) around the world in the Gerontology Research Group database (as of January 17, 2011 http://www.grg.org/Adams/E.HTM), we observe that there are 79 women for 4 men, a sex-ratio of 19.8. Therefore, women live longer and are more numerous than men, and this situation becomes more pronounced with increasing age. If health, that is, the absence of disease or good functionality, contributes to longevity, then logically old women should be in better health than old men, that is, have less disease and disability. On the contrary, Allard and Robine showed that "*in the three standard dimensions of functional abilities, physical (strength, mobility, agility), sensory (vision and hearing) and cognitive (memory, attention, mental performance, and affect), men aged over 100 years have better performance than women*." It was one of the first observations of the gender disability gap.

First, let us summarize the results of the Ipsen survey, the first French national survey on centenarians. A thorough standardized clinical examination was performed by the general practitioner (GP) of the centenarians. According to this medical examination, 65.2% of men were evaluated as having a "good" or "very good" health status, while only 56.5% of women were this healthy while 10.7% of women were evaluated as having a "poor" or "very poor" health while only 3.4% of men were at this bottom end of the health spectrum. Regarding vision, 35.1% of men had "good" or "very good" vision, while only 27.19% of women were in a similar condition. Centenarians totally blind or with bad vision were 22.0% for women and 19.1% for men. Unlike the other data, the centenarians who had "good" or "very good" hearing were more numerous in the female population, 28.2% against 21.1% in the male population. In the Ipsen survey, the cognitive health of the centenarians was tested using the 10 questions of the Pfeiffer test. This test is a screening test, similar to the MMSE (though shorter) for possible cognitive impairments and dementia. Men who made no errors numbered 39.1% while women numbered only 12.9%.

Table 1: Breakdown of the French population by sex and age, by January 1st 2011.

Birth cohort	Age	Total	Males	Females	Sex_ratio
1920	90	159965	45659	114306	2.5
1915	95	28413	5461	22952	4.2
1910	100	6926	694	6232	9.0

Source: INSEE, 2011.

Since this study, no comparable survey on centenarians has been run in France, but we can examine the French data of two European studies: *European Challenge for Healthy Aging* (ECHA) [2] and *GEnetics of Healthy Aging* (GEHA), regarding activity limitations and cognitive impairments. ECHA involved centenarians or near centenarians and included 55 French men and 77 women. The use of the Mini-Mental State Examination (MMSE) resulted in an average score of 17.6 for men and 10.7 for women. Regarding the Activities of Daily Living (ADL) [3], 30.1% of men displayed no activity limitation while women numbered only 11.8% in this case. Similarly, while only 20.4% of men had difficulties with 4 or more ADL activities (the ADL evaluated were feeding, dressing, transferring, toileting, and bathing), 48% of women showed difficulties. The GEHA participants are younger because this study involved nonagenarian siblings. The average age of the 201 French male participants was 92.7 years old, and the average age of the 461 French female participants was 93.4 years. The MMSE average scores were 23.7 for men and 21.8 for women. Regarding the ADL, men without difficulties reached 66.2%, while women numbered only 46.4%. Men having difficulties with 4 or more ADL activities numbered 9.5%, while 16.2% of women showed difficulties. Even if we do not have specific data for the centenarians, 754,000 people were affected by dementia in France in 2010, 72% of them were women and 28% men, according to the last estimations [4].

The data presented here on health differences between men and women could be discussed in terms of data collection methodology or representativeness of the samples; however, they are sufficient to illustrate our main point, that is, "women live longer than men and they are more numerous in old age but men are in better health." There are numerous studies in many countries confirming the above observations, and there are many hypotheses to explain the two components of these observations as we will explain below.

The literature dealing with the longevity gender gap is considerable. Each scientific discipline has developed several hypotheses to explain the causes of this phenomenon. Facts come from descriptive sciences, demography, epidemiology, and other fields, and from both the social sciences and the biological sciences. They can be linked with the whole life course of the subjects: we know that men are more affected by violent death (war, murder, work-related, traffic accidents, suicide, etc.), and their behaviour patterns expose them to risk factors such as excessive alcohol intake, tobacco and other drug consumption, or other risk raking behaviour.

Table 2

De la Fontaine, J (1668). *Les Fables choisies*. Julaud, J-J. (2010). *Les Fables de la Fontaine* (new Ed), Paris: First Editions.	Thomson, R (1806). *La Fontaine's fables*. Now translated from the French; Paris: Doyen.
Le Chêne et le Roseau (I, 22)	The Oak and Reed (I, 22)
Le Chêne un jour dit au Roseau:	The oak one day addressed the reed,
"Vous avez bien sujet d'accuser la Nature;	"Nature you may accuse indeed;
Un Roitelet pour vous est un pesant fardeau.	A wren for you's a heavy load,
Le moindre vent, qui d'aventure	The softest breeze that stirs abroad,
Fait rider la face de l'eau,	That ruffles but the water's bed,
Vous oblige à baisser la tête:	Compels you to hang down your head;
Cependant que mon front, au Caucase pareil,	While I, like some proud mountain's brow,
Non content d'arrêter les rayons du soleil,	Not only stop the solar ray,
Brave l'effort de la tempête.	But brave the blasts that round me play:
Tout vous est Aquilon, tout me semble Zéphyr.	Loud rowing storms to you, to me like zephyrs blow.
Encor si vous naissiez à l'abri du feuillage	Now, did you spring within the shade I throw,
Dont je couvre le voisinage,	Were you beneath my sheltering foliage found,
Vous n'uriez pas tant à souffrir:	You would not suffer from the north unkind;
Je vous défendrais de l'orage;	I could defend you from the tempests round;
Mais vous naissez le plus souvent	But ye are seldom, save in marshy ground,
Sur les humides bords des Royaumes du vent.	Upon the borders of the realms of wind.
La nature envers vous me semble bien injuste.	Nature to you I really think unjust."
-Votre compassion, lui répondit l'Arbuste,	"Your pity," answered him the reed, "I trust
Part d'un bon naturel; mais quittez ce souci.	From goodness springs, but pray that pity spare;
Les vents me sont moins qu'à vous redoutables.	The winds that trees and mountains tear
Je plie, et ne romps pas. Vous avez jusqu'ici	Alarm not me—unbroken still I bend.
Contre leurs coups épouvantables	You hitherto, 'tic true, unshaken bear
Résisté sans courber le dos;	Their mighty blasts—but wait the end."
Mais attendons la fin. "Comme il disait ces mots,	Just as he spoke,
Du bout de l'horizon accourt avec furie	A tempest from the far horizon broke;
Le plus terrible des enfants	Ne'er from the bowels of the north,
Que le Nord eût portés jusque-là dans ses flancs.	Till then, came such a son of fury forth;
L'Arbre tient bon; le Roseau plie.	The oak stood fast; the reed bowed down again.
Le vent redouble ses efforts,	The winds then bursting with redoubled roar,
Et fait si bien qu'il déracine	Up by the roots the boasting giant tore,
Celui de qui la tête au Ciel était voisine	Whose cloud-capped head so proud did reign,
Et dont les pieds touchaient à l'Empire des Morts	Whose feet sank down to death's domain.

Accordingly, the survival selection is stronger for men than for women. Many hypotheses come from biological sciences: of course, physiologies of men and women are different. Genetic, hormonal, and phenotypical differences, such as body size, are well known. Other hypotheses come from psychological and social sciences: behaviours, education, and social roles are obviously gender-related; social inequalities between men and women during childhood and work life could lead to differing social and medical support, and so forth [1]. Box 1 lists some of the proposed hypotheses suggesting the complexity of the explanations of the differentials in health and longevity between genders.

Eventually the survival advantage for women results in a larger percentage of women reaching older ages. For all age groups, more women survive, but despite living longer, they display more comorbid conditions. The mechanisms reported in Box 1 could explain the higher rates of disability among women. However, it is also possible that, for any morbidity level, men remain more active, outwardly displaying less disability, ignoring pain, discomfort, and risk, and this behaviour pattern contributes to a shortening of their life. It is this last aspect of the gender gap that we aim to explore in this paper from an anthropological perspective. Although some reviews are available on the gender gap (as briefly referred to in Box 1), there are far fewer that attempt to disentangle the relative contribution of the various biological and social factors and even fewer that examine this issue for the oldest old. Our goal is not to dissect the utility of the above hypotheses, but to suggest another one, based upon gender-linked behaviour patterns of the oldest old, which can contribute to partially explain the gender paradox in healthy survival among the oldest old.

2.2. In-Depth and Semistructured Interviews of Oldest Old Informants. For this purpose, we used qualitative data from comprehensive [5] and semistructured interviews of centenarians held between 2003 and 2007. Balard [6] met more than 100 centenarians or near centenarians (people at least 95 years old) while undertaking doctoral dissertation research under the framework of the ECHA and GEHA studies. He

Increase in life expectancy at birth was accompanied by the emergence of a gap in favour of women which progressively widened to about 6-7 years: the longevity gender gap. On the other hand, excess of disability is currently observed to the detriment of women, cancelling their longevity advantage: the disability gap. Many researchers simply conclude that women live longer but in poorer health than men [42, 43]. The female survival advantage can be tracked back to conception with a very large ratio of male per female conception leading to the actual ratio of 105 males for 100 females at birth [44].

A small longevity gap, between 1 and 3 years, in favour of women has been observed in pre-transitional societies in line with studies suggesting the existence of a small female survival advantage in many species possibly due to similar genetic and hormonal processes impacting on cholesterol levels and immune functions and possibly also related to parenting and child rearing. In fact, the widening of the human gender gap is a very recent phenomenon, probably due to 20th century social and economic transformation, benefiting apparently women more than men. The widening of the gap corresponds to a transitional period between conditions when the greatest danger to life was starvation and infectious diseases to modern conditions when the greatest danger is opulence and high caloric intake leading to metabolic imbalance. The question then is why women can derive a greater or a faster benefit from these changes in living conditions. Another question is what will be the residual gap when men will possibly have caught up with the transition [45–47].

A number of biological and social factors have been put forward to explain gender-specific behaviours, habits and beliefs which can lead to the observed longevity gaps. Most researchers focus only on the longevity gap and favour a simple biological or social theory such as the chromosomal, hormonal, oxidative and replicative theories on the biological side, and stress-related job and gender roles, smoking and risk behaviour, social constructionist and feminist theories on the social science side [45, 48–50]. Only a few look for a coherent explanation working both for the longevity and the disability gaps.

Indeed, everything that distinguishes men and women can contribute to explain the longevity and disability gap [42]. A number of these conditions have changed over time and can contribute to explain the widening of the gaps. On the other hand, women have a lower level of functioning. Studies consistently show that a greater percentage of women are disabled compared to men. Although it is not clear whether there are gender differences in the reporting of disability, the prevalence of total disability in older women can be estimated to be approximately 50 percent higher than in men. At every level of co-morbidity women have greater disability [47]. Women have been presented as the sicker gender. The use of behavioural indices (bed rest, sick leave, number of contacts, health care utilisation, self reported morbidity) will confound our understanding of morbidity because they actually represent how men and women cope with illness rather that representing their true health status. Depression provides a good example. Despite the fact suicide rates are much higher for men than for women, depression is thought of as a female problem because women are seeking more help for depression. Instead, men tend to engage in private activities, including drinking and drug abuse, designed to alleviate their depression [47, 50].

Several hypotheses have been raised on the biological side to explain the gender gaps [46, 51–56]. But none of the basic biological factors has significantly changed through the 20th century and cannot contribute to explain the widening of the gaps [45]. According to Stindl, the longevity gap could be explained by the difference in body size between men and women, needing a different number of replications and then leading to a different length of telomeres. Change in body size through the 20th century would explain the widening of the gaps, men being closer to their replicative limits [57]. Women may also be better able to cope with overnutrition than men. Indeed, female advantage for survival may arise in prosperous countries by innate ability of the female body to mobilise and transport nutrients for the benefit of the foetus in pregnancy, giving them a better excretory system. In this case caloric restriction may be more efficient for males [58].

On the social science side, smoking has been the main explanation for many years. According to the tobacco theory, the longevity gap should continue reducing as women approach the same total smoking years as men [45]. While men and women engage in different social practices to demonstrate their masculinities and femininities respectively, male beliefs and behaviours undermine their health whereas female beliefs and practices reinforce their longevity. In traditional Western societies, men should be independent, strong and tough, taking risks (from street violence to skydiving, according to social class), refusing to acknowledge physical discomfort and need of help, and refusing positive health behaviour such as using sunscreen. Moreover, health care utilization and positive health beliefs or behaviors can be viewed as a form of idealized femininity [50].

Box 1: A sampling of hypotheses regarding the gender gap in health and longevity.

led comprehensive talks, in order to collect alternative points of view on the centenarians' daily lives, to be compared with the pictures painted by the epidemiologic questionnaires. The method was inductive, close to "grounded theory" [7]. The comprehensive talks were used to "*explore and examine research participants' concerns*" [8]. In addition to these comprehensive talks, Balard performed in-depth interviews [9] with a group of 14 key informants who were followed for 4 years and were subjected to 5 to 10 in-depth interviews each.

In this context, the word "informant" means a person able to "bear witness to his or her group, society, and culture" [10]. All of our informants were aged 95 or older when we met them. Complementary interviews were also performed with their nearest relatives or proxies. Some of the informants died during the fieldwork and were replaced by new informants. The key informants were chosen according to their ability to communicate and their comfort in interacting. Even if people with dementia were not *a priori* excluded,

people in advanced stages of dementia were not interviewed for practical and ethical reasons. Most of the interviews were face-to-face talks to avoid interference between the elderly and his/her family circle. The interviews were as close as possible to informal talks, without using a real interview guide but trying to "*go with the flow*" [9] and to follow the recurring concerns evoked by the centenarians during the comprehensive talks. As Bourdieu [11] recommended, it is "*better to listen to than to question*." Listening is vital to understand the meaning of a discourse [12]. In this view, the qualitative interviews should provide to the researcher an access to the mental representations of the interviewees, to understand "*the meaning of respondents' experiences and life worlds*" [13]. As Charmaz [8] put it: "*Qualitative interviewing provides an open-ended, in-depth exploration of an aspect of life about which the interviewee has substantial experience, often combined with considerable insight*." In our research, the particularity of our key informants was to be one of the oldest old, that is, a centenarian (or near centenarian).

In this context, the main themes of the interviewees were their life stories, current daily lives, thoughts, representations of old age, sense of purpose in life (*raison d'être*), assessment of the quality of their lives, and strategies to cope with frailty. Our posture was consistent with the advice of Goffman [14] to "*Subjectize yourself*" (to enter the subjectivity of the speaker).

The goal was to translate the thoughts or, in Lapantine's words, the "representations" of the speaker, obtained from interviews centred on the interviewee's conceptions, reasoning, and subjective logic [15]. Centenarians were considered as "*meaning makers*" [16]. To facilitate the words of the informants, the interviewer opted to introduce himself as a student who meets the elderly to learn from their experience of life. This introduction turned out to be highly facilitating because it produced a dissymmetrical relationship in which the informant was in a position of superiority. The elderly was positioned as a teacher, imparting knowledge to a novice, thus preventing him/her from doubting statements given or perceiving the interview as a test, which may cause anxiety. In accordance with qualitative research, all interviews with key informants were tape-recorded and transcribed verbatim. Fieldwork and interviews were stopped when the data saturation level was obtained. The coding and analysis of the interviews were based on the meaning node obtained by the categories given by the informants' discourses. As a result of which, the usual theoretical frameworks on quality of life (psychological indices, life satisfaction, subjective well-being, etc.) and successful aging based on health and autonomy were not used.

3. Results and Discussion

> "*Someone once asked me what he had to do to get old. Do not do anything, it comes. Old age comes without being noticed*" (Mme Emilia, 98 years old). (for ethical reasons, the names of the informants have been changed.) "*Physicians, families and all the others know a lot of things thanks to the intelligence of their brain and sometimes thanks to the intelligence of their heart. But we, we live inside old age. We feel old age and feeling is much more than knowledge.*" (Words of Mme B, quoted by C. Memin, 2001).

The analysis of the interviews revealed that the definitions of aging (vieillissement) and old age (vieillesse), given by the oldest old, did not really differ between men and women. While avoiding a deep discussion of ethnolinguistic concepts, we nonetheless should clarify one point. In French, there are two words, "*vieux*" (old) and "*âgé*" (aged), which could be translated as "old person." The oldest old, and probably most people accept to be designated as "*âgé*" but refuse the word "*vieux*" which seems to be a taboo for them. In their representation, "*âgé*" refers to the aging process while "*vieux*" refers to an identity. When they are talking about their experience, our key informants explained that they feel they are "*aging but they are not old*." Concerning the aging process, they describe it by saying "*It started there shortly*." They feel the aging process through different symptoms.

The first and probably the most important one is the *ability to walk*. For the oldest old, walking is a kind of index of the aging process. They assess their degree of aging by their performances and ability to walk. So the consciousness of the beginning of the aging process appears when they feel the first difficulties walking. Thereafter, when they have to reduce the distance they used to walk, or when they have to sit down several times during their usual walk, they consider that they are further in their aging process. In their representations, the one who cannot walk is "*the old one*" (le vieux) that's why M. Pierre (98 years old) could say when talking about the other residents of his nursing home "*Here, things have changed, more and more old ones (vieux) entered, you know people in wheelchairs*." The second symptom that the oldest old consider as a marker of their aging process is the *feeling of fatigue*. They explain fatigue is at first occasional and then appears when they do usual activities: "*I am tired when I walk*" (M. Léon, 99 years old). Then fatigue comes also in the absence of activity. The mere presence of many people around them, and worse if they are engaged in discussion with them, causes fatigue. Just before becoming old (vieux), the oldest old explain "*even by doing nothing, we feel tired*" (M. Georges, 96 years old). The third symptom the oldest old consider as a marker of the aging process is the *feeling of vulnerability* that they express by saying "*Now I have to be careful with everything*." Depending on the informants, "*having to be careful*" could focus on weather: "*I have to be careful not to get cold. I never go out when it rains or it is windy*" (M. Léon). It could also refer to the fear of falling, "*I am careful when I walk… if I fell, it would be terrible*" (M. Louis, 100 years). Some of the informants express their vulnerability related to others: "*Now, I have to be careful, if someone, if somebody attacks me, I could not defend myself*" (Mme Marie, 96 years old). The fourth and last symptom that oldest old recognize as markers of their aging process could be summarized as the *feeling of weakening.* They talk about the decrease of their senses: "*Today, I have difficulties to see*"; "*I do not hear very well*." They admit being always

impaired by small health problems while they were not used to be so in the past.

Through these different symptoms, the oldest old admit they are aging but they defend themselves against being labelled old (vieux). The old one (le vieux) is not aging, he is at the end of the aging process. In their representations, the old one is a human being living under a death sentence. According to the representation of the oldest old, the old one (le vieux) is first and foremost the one who is "*useless*." In accordance with the social values of his/her generation, the oldest old consider that the social utility and the sense of life of human beings are in work and family. M. Aimé, 95 years old said "*Now I am old (vieux), I am unfit, useless... I cannot even start my rotary tiller. It does not matter if I die*." Close to social utility, another marker of old age for the oldest old is the feeling of "*not being listened to*" by their entourage. Mme Emilia (99 years old) said "*The old ones (vieux) are the ones who are no more listened to*." For the oldest old, "*being listened to*" is a real mark of social utility because as Mme Anna (96 years old) says "*I am not 96 years old without reason*." A lot of them consider that today their social role is to pass on their experience to young people. In this view, those who cannot pass on their experience are useless and old (vieux). We have already explained that the old one (le vieux) is the one who cannot walk, he/she is also "*the one who lost one's head*." This idea is close to the concept of "social validity". Those who are demented are like the old ones (le vieux), they are marginalized and socially disqualified. The last qualifier used by the informants to describe the old one is close to the concept of dependence but it is expressed differently by the oldest old. As Mme Anna said "*You are old when you are at the mercy of everyone*." Everybody depends on other persons but being "*at the mercy of somebody*" means to be at risk to lose control of one's life, to be forced or manipulated.

In the representation of the aging process expressed by the oldest old and in their ideas of old age, we find many elements of a cross-cultural definition of age already put forward by anthropologists [17–21]. Health, safety, functioning (mobility, capacity to act), and individual productivity (cf. "productive if it creates societal value, whether or not it is reimbursed") [22] are some elements that many societies include in their definitions of aging. Aging implies a frailty process, according to Linda Fried, "*a physiologic state of increased vulnerability to stressors that results from decrease of physiologic reserves, and even dysregulation, of multiple physiologic systems*" [23]. According to our observations, frailty is not only physiological. In old age, frailty is also social, cultural, and psychological, involving the sense of identity. Indeed, the oldest old explain they suffer from the lag between "*their time*" and the society of today. They also suffer from a break in their identity, a sense of "diminished self" or what Levi-Strauss called when he turned 90 years old "*the feeling of being a broken hologram* (Reconstitution of memories of Roger-Pol Droit published in *Le Monde* 29/01/1999. Discourses of Claude Levi-Strauss pronounced on 25/01/1999 in Collège de France.)" The old anthropologist described his life as a dialogue between his "*real self and his virtual self*." To apply this concept to our informants, the *real self* is the frail old man or woman they see in the mirror and feel when confronting the challenges that come with "aged bodies" while the *virtual self* is the representation they have of themselves, which still preserves a living idea of the whole and a continuity with the past (almost a timeless identity). It is in this context that we hypothesize that men and women are not equal in their ability to face the various symptoms and related challenges of old age and dimensions of frailty. We hypothesise that, while men try to fight against the stigma of old age until death, women try to find a way to accept it and continue to live on. Through these gender-specific adaptive strategies, the gap between the "real self" and "virtual self" (in Levi-Strauss terminology) is more easily bridged for most of the oldest old women of this generation when compared to the men.

According to the concept of "*generational habitus*" suggested by Mauger [24] and built on from the work of Mannheim [25] and Bourdieu [11], we hypothesize that the differences in behaviour between men and women, even at the oldest ages, are coming from a "*generational mentality*," that is, "the typical ways in which people think and their attitudes toward life." This theoretical position extends the notion of "*social generation*" [26] (i.e., "people whose unity comes from a particular mentality"). The concept of "*generational habitus*" is close to the idea of the existence of contemporaneous connections between individuals. "The generation creates a rather narrow circle of individuals who despite the diversity of the other factors at play, are linked into a whole by the fact that they lived the same major events and changes during their receptivity period" [27]. Mauger used the concepts of "*mental tools*" [28] and "*habit-shaping force*" [29] to illustrate how people are influenced by their generational membership. Over the life course, the main receptivity period is during childhood, when people acquire the gender stereotypes of their culture. Of course, the way of thinking, being, and doing evolves over the whole life course, but these cultural landmarks regularly resurface, especially when identity, sense of self, and self-esteem are threatened, as in old age, when gradual or sudden decline in physical capacity, loss of control over one's environment, or other age-related challenges emerge.

In order to illustrate the influence of the "*generational habitus*" for the behaviour patterns of the oldest old, we can evoke the "community of thought" which exists among our informants. Indeed, it appears that our informants share many values despite their difference in social status (occupation, level of education, and income). For them, family and work are of utmost importance. They severely reject behaviours jeopardizing the family such as divorce and remarriage. They have a very different conception of care than their children do. For them, care is part of the traditional system of mutual help and supportive relationships that they have known in their village or neighbourhood. In this view, care should not be a professional task and/or something that is paid for, except for specific medical care services. They consider that it is the role of their nearby family or close relatives to help them with daily tasks and provide support in old age. They cannot accept that they should pay for cleaning and cooking whereas they have (or they think they have) previously helped others in the past.

This "*generational habitus*" still influences them in their daily choices, values, and thinking.

The analysis of the interviews showed a clear gap between the representation of the social role of men and women for our informants. The large majority of people born during the first two decades of the 20th century in France were "country folk" even if some of them spent their adulthood in towns. As showed by Segalen [30], this rural environment was characterised by an important distinction between men and women concerning social identity, social role, and production functions. These findings are close to the work of Guttman [31] who describes men as "breadwinners," active, and dominant, and women as "homemakers" who provide emotional support and accept dependency as part of their social role, that is confined mainly to the domestic sphere. In this context, Segalen [30] showed that despite the appearance of male domination, there was mutual support, solidarity, complicity, and reciprocity between men and women. Lebra [32] referred to the exchange of such mutual obligations as *reciprocal dependency* where there exists a reciprocity or exchange of dependency. Others have recognized that despite the appearance of subjugation and economic dependency in relation to their husbands, women wield considerable power and status within the domestic sphere that accompanies their role monopoly as full time homemaker. Feminist anthropological approaches have analyzed these social exchanges within the context of a public and private dichotomy where men wield political power as the "public face" of society, while the private space is the female space. Ortner [33] elaborated on this theme when she published a landmark article likening *female to male as nature is to culture*. This theory dealt with the perception that men are the upholders of culture whereas women are more associated with nature. Women are seen as closer to nature in reference to three dimensions: (1) women's bodies are seen as more *natural* since they are more involved with childbirth and childrearing; (2) the social roles of women that intertwine with child rearing are viewed as closer to nature, specifically confining women to the domestic realm; (3) social perceptions of the female psyche portray woman as closer to nature.

Of course, the power, status, and roles of men and women vary between cultures and time periods. Moreover, men and women have multiple overlapping and contextualized identities based upon gender, age, ethnicity, class, regional, and other differences that evolve over the life course. Some authors like Guttman [31] describe an androgyny process. Following his theory, the traditional gender role is stronger during what he called "*the parental imperative*" (i.e., to ensure the survival of children and the handing down of the social values. Once the children have reached adulthood, the parents could express the "*other-gender*" side of their personality). This phenomenon seems to be due to the softening of the traditional gendered roles that occurs with the aging process. Men become less authoritarian and more sensitive, whereas women accept less to be dominated and become more powerful. Guttman [31] used the image of the warrior who became a peaceful chief, or that of the "virile older woman." Many anthropologists have shown that during adulthood, and especially after menopause, women's social roles, status, and identity change. For example in Gabon, according to Bikoma [34], "The non-menstruating woman takes part in strictly masculine demonstrations. She is integrated into the guard and is part of the Wise." Many works have described how women's status changes, often rising as they grow older [35, 36]. Thus, it appears that the gender differences in behaviour change over the life course, most often appearing as a softening of the traditional gender characteristics. The child of one of our informants confirms "*Today it is different but when I was a child, my father was terrifying*."

Does very old age cause an increase in the androgyny process observed in middle-life or is there a specific moment in the life course in which the traditional gender role reappears? We consider that the answer is significantly different between traditional societies and postindustrial ones, such as the France of today.

The finding of Singleton [20] that "In some societies, when you get older, you see better (the author refers to the mystical and magical abilities often attributed to the oldest old in traditional societies) and you are better seen" does not seem to hold in our postindustrial societies. Indeed, the image of very old age is quite negative in France. It conveys the ideas of illness, handicap, and senility. As we explained earlier, the oldest old worry about losing their identity as well as becoming "*an old person*" (*un vieux*). They suffer from the feeling of no longer being part of society. It is possible that, facing this unknown and stressful event that represents very old age, one important adaptational strategy consists in taking refuge in what they consider the most important things (i.e., what they value most) in their life and identity. Men try to show that they are still strong enough, helpful (useful), cognitively aware and intact, and able to make decisions for themselves, while women concentrate on issues such as being emotionally close to their family members, seeing them happy in life, and maintaining other important human relationships. Thus, it appears that the components of the social identity of women are less challenged by the frailty process, offering them more possibilities to adapt to the losses associated with the aging process when compared to men.

3.1. Men as Oaks: Preserving Their Virtual Identity at All Costs.

> "*Frailty implies to be no longer able to be the same while feeling the necessity to be oneself*" [37] (Journée d'études internationales: L'âge et le pouvoir en question. Intégration et exclusion des personnes âgées dans les décisions publiques et privées, Paris, 10 et 11 Septembre 2007).

The interviews with the male informants reveal that preserving their "virtual self" is one of the leitmotives of their dialogues and a constancy of their behaviour. They give much weight to the past when making current assessments of self. Their self-identity is based on their past roles and statuses as worker, breadwinner, and head of family. They accumulate anecdotes on their competence as workers. Some of them presented themselves as a kind of "stakhanovist" (exceptionally diligent and productive). M. Aimé "*With my horse, I worked every day from sunrise to sunset, I worked*

for me and for the others too." M. Léon: "*When I was 86, I used to prune vines 5 or 6 hours a day.*" Physical strength and physical endurance are two important dimensions of the self-identity they make efforts to lay claim to. Others have insisted on their skill at work like M. Henri (96 years old) who explained that he made a piece out of wood to replace a part of a tank engine during World War II. Generally, they are keen to report anecdotes which highlight their productive skills. They did their best to work as long as possible even if they were officially retired. For them, it is vitally important to prove that they are still valid, competent, autonomous, and able. With an interlocutor, their self-presentation leans towards "self-staging" [14] or presenting themselves in the light of their ideal self-image. When they became unable to go on with their work, they tried to find activities in which they could continue to prove they were competent and useful like gardening, or repairing and improving things around the home. Within their family, the men of this generation are used to being in authority and to be listened to, even if these characteristics weaken over time and with age. During the interviews, they insisted on showing that their opinion is still listened to and respected by their children to whom they continue to give advice. To survive in old age, the men in our study seemed to choose to stay immovable. They did not like to deal with their new and vulnerable self-identity and refused to be compared to the old ones (les vieux). However, there comes a time when the gap between their "real self" and the image they have of themselves is too large to continue to self-stage. Thus, when walking becomes too difficult, when it is impossible to preserve a useful activity in conformity to their identity, when they have the feeling that they are losing their authority because they are not able to manage daily affairs, men at these oldest old ages express a kind of surrender. We have already quoted the sentence of M. Aimé: "*Now I am old (vieux), I am unfit, useless… I cannot even start my rotary tiller. It does not matter if I die*." M. Léon, who was a very positive centenarian during the first interviews, confessed 3 months before his death: "*Now I get to the end… I am too old*." The events that precipitate the surrender and probably the "*premature*" death of the oldest men are various and personal. Some of them could not endure the death of their wife. In addition to the emotional suffering, they appear helpless without the person devoted to take care of their daily personal needs. Others could not deal with the idea of being physically disabled. Usually oriented to outdoor activities, they found it extremely difficult to accept that they must now stay home. When they felt they were losing control over their environment, their self-sufficiency, and perhaps most importantly their continuity in self, the oldest men seem to prefer to give up and die rather than accept a state of disability and/or the idea of further gradual decline. To use the terminology of Evert et al. [38], men seem to have the ability to survive as "delayers." Therefore, as long as they can delay significant physical limitations and severe disability, they have the will to continue to keep going and literally stay alive. The behaviour of oldest old men is highlighted by their efforts to remain physically and mentally strong and active. This may contribute to explain their better physical and cognitive functional health status, compared to women, but also their tendency to take more risks and their excess mortality. Because they are giving such significance to their functionality and physical performance, the oldest men make a great deal of effort to walk even if it is dangerous for them and this risk-taking behaviour may lead to falls and fatal events. M. Aimé's daughter explained that the neighbour found her father lying in his garden after a fall on three separate occasions.

Ultimately some of them may prefer to die. Two of our male informants explained that they thought about committing suicide; the first because he could not endure the thought of himself as degraded and useless, and the second because he was obliged to leave his home to enter a nursing home. Statistics confirm the tendency for men to commit suicide more often than women. For instance, at age 85 and over, the suicide mortality rate in France is 6.5 times higher for men than for women [39].

We saw that men can train themselves more than women to perform various physical tasks. We also wonder whether they are not tempted to exaggerate when reporting on their health status and functional abilities. Do they fool themselves when self-staging? Do they try to bias reality when reporting their self-perceived health or ADL abilities? Cognitive tests or physical tests, such as the hand grip test, are *a priori* objective tests. Such behaviour implies they might be on the razor's edge. They delay or seem to delay the old one (le vieux) stigma until a breaking point. This behaviour may partly explain why they are less numerous among older ages and why they live shorter lives. Possibly, the stronger selection effect that occurs for men reinforces their behaviour and the feeling that they are in better health. Compared to other men of their generation, they are the survivors, the chosen ones. This "relative appreciation" [40] may result in a self-appraisal that they were the most robust and/or able of their birth cohort, it may be that they literally could not see themselves otherwise.

3.2. Women as Reeds: "Let Live" to Survive. The oldest old women report very different behaviour compared to men. Their "*generational habitus*" also influences their social roles. Although playing their major social role within the domestic sphere, contrary to their husbands, they were never the ultimate authority, even in their own home. When they were children, they lived under their father's authority and then, as a spouse, under their husband's authority. Thus, to be in authority is not something cultural, it is not a habit. In this generation, many women have worked as housewives. Their family role was firstly "*to take care*" of others, housework, childrearing, and help their husband in his activity. In their life, "*taking care of others*" is what better illustrates their self identity. When they were children, they used to help their mother at home. Then, when they were mother and wife, they were responsible for the health and the blossoming of their children and husband. Most of them were also helping their husband in his work, taking care of several tasks. Later, these women were in charge of their old parents or step-parents. The representations attached to their identity were neither physical strength, nor being in authority, but the ability to listen to and understand the needs of others,

to be a person full of maternal affection for others. Their "*working*" role was mainly oriented towards the household and associated tasks [30]. The activities linked to their social identity were very often cooking, doing the washing or other household tasks, and childrearing. Their ability in sewing was a gender-specific ability that was highly valued. M. Aimé's daughter explains "*the death of my mother raised the issue of my father being alone. I mean when she was alive, she was really a servant for him. She did cooking; she prepared his clothes for grooming… she did everything for him*." The daughter of a couple of nonagenarians testified in a letter "*My mother is 90. She weighs only 37 kg. Her entire existence was conducted according to her husband. She has to prepare his medicine, read for him (very painful), especially to serve the meal for him*." The daughter explains that her mother sacrificed herself for her husband. Referring to this point we notice that this could partly explain why the protective effect of marriage in France is only valid for men in this generation [41]. Mme Emilia (99 years old) confirms that in her generation the role of women was dedicated to support other family members: "*When I was a child, I helped my mother… I had to take care of my young brothers and sisters […] I don't want to live with my children because I know what taking care of an old person implies, I have done that for my father and my mother-in-law*."

According to Cool [59], we note that there is "continuity in women's core social role of housewife" for this generation and we agree that women seem to show an ability to adapt to discontinuity and/or conflicting roles throughout the life course. Therefore, women of this generation appear more flexible and adaptable than men. Indeed, if the collected material shows a general continuity in the social role of the women of our sample, it also shows that they had to face many discontinuities, upheavals and challenges over the life course. We hypothesize that their greater flexibility and ability to adapt is partially explained by their "generational *habitus*" shaping them as "good girl" and "exemplary spouse." As Segalen [30] expressed it: "Authority to men and power to women." Women have learned not to fight against their "environment" but to adapt to it and to find the best ways to live in it. When they grew old, women did not feel the need to fight against old age stigma as men did. Physical strength, being in authority, and an identity based on their ability to be the head *of the family are not female prerogatives for this generation.* We observed that women more easily accepted having difficulties with some activities, such as gardening, walking outside the house, and could more easily give up such activities. We also observed that women were not emotionally injured when their children made decisions for them. They explained that as an old person, the most important thing for them was to see their children and grandchildren. They appreciated it when they were still able to cook for them. As Mme Emilia, and many oldest-old women explained "*now, I let myself live*." What she means is that she chose "*not to fight against old age*" but to "*live with it*." Confronted by a number of losses, such as memory or mobility, Mme Germaine says "*I do not care about that today; it is not the most important… what really matters is to see my children and their families in good health*." Women seem to have a high ability to accept not being able to fully control their life, and their body, their fate. When they fail in the MMSE test, they appear less uncomfortable with this than men do. They do not consider that their identity and pride are at stake. In conclusion, women seem to have a better ability to accept and live with the various symptoms of old age and frailty. Therefore, even if they accumulate health problems, chronic degenerative diseases, functional limitations, and activity restrictions, even if they are no longer performing as they once did, they can survive. Referring to Evert et al.'s [38] terminology, women seem to be closer to the "survivors." Not focusing on their losses, they are able to continue to live without experiencing a large gap between their "virtual identity" and their "real identity." This behaviour was reported by all six oldest old female informants in our study. We can also notice that, while the majority of the men in the French sample of the GEHA study did their best to be successful with the physical and cognitive tests that were proposed, women seemed not to consider them so important. It is possible that for the women in GEHA, the interaction and the presence of someone to communicate with was more important than the tests *per se* and could partially contribute to explaining why they were less successful in the tests when compared to men.

The hypothesis developed here is close to *identity theory,* as proposed by Burke [60]. We insisted on the "*generational habitus*" that influences the gender behaviour in old age and that we observed in the current cohorts of the oldest old people. Being much less self-centred than men, women of these cohorts found a way to relativize their frailty and live with it, instead of ignoring or concealing it as men seemed to do. The main part of these gender-based behavioural or adaptational differences in regard to frailty and old age possibly came from the "*generational habitus*" which constructed the physiological reserve, psychological attitudes, ways of thinking, values, and social behaviour of the oldest old men and women in a very different manner. Men and women of these birth cohorts have grown up, lived, and grown old as completely different social beings. Their gender-specific behaviour in old age continues to influence their longevity and quality of life in different ways.

However, looking at the current homogenization of male and female social roles and behaviours (especially health-related behaviors), we can postulate that the gender gaps observed today among centenarians may decrease over time. More recent cohorts of women have partially adopted risky behaviours, such as smoking or drinking alcohol, which were the prerogative of men in the past, and increasing participation in what was once considered exclusively male occupations (transport, military, law enforcement, etc.) may expose women to more violent deaths due to accidents, homicide, and/or suicide. Indeed, in line with decreasing polarization of gender roles, the size of women's life expectancy advantage over men has been steadily shrinking in most developed nations (some exceptions such as Japan exist) for the past few decades. Moreover, men in developed nations seem to be paying more attention to their health in recent years when compared to men of previous generations, with recent gains in male life expectancy (in comparison to female) appearing to be related more to an acceleration of progress for males, rather than a worsening of health situation for women [61].

Decreasing male deaths from cancer (especially lung) and cardiovascular disease are the major mortality-related contributors [61, 62]. In this context, the gender gap in longevity will likely continue to decrease in the future as more women take up smoking and other health risk behaviors. But will this new "*generational habitus*" change the gender differences for the oldest old? The 200,000 centenarians who will live in France in 2060, according to the latest population forecasts, will have a "*generational habitus*" constructed in the postwar period, shaped by social, cultural, technological, economic, and behavioural forces far different than that of today's centenarians. They will have a much higher level of medical literacy. But will the behaviour of men and women be more alike as they enter the ranks of the oldest old? Of course only time will tell. The gender gap in longevity and disability will likely remain in a state of flux for some time due to the continual shifts in the social, economic, and behavioural dynamics that determine health and longevity for both women and men.

4. Conclusions

French people, who were born at the beginning of the 20th century (i.e., before 1915), display a "*generational habitus*" which clearly juxtaposes men and women in terms of social identity, family role, and sense of purpose in life. This *habitus* still influences many gender-linked behaviours and adaptational strategies, even at the oldest ages. We suggest that these differences in behaviour favour functional health for men and longevity for women. The "*generational habitus*" influences the representation of the "*raison d'être*" for men and women. The male "*raison d'être*," for these cohorts, is more self-centric and oak-like explaining why, when the gap between their real and virtual identity and self-image as the provider and pillar of their family and community becomes too wide and difficult to sustain, they find it nearly impossible to go on. On the contrary, the female "*raison d'être*," for the same cohort, is based more on their status in relation to others, in particular as givers of care, even if it is reduced to their mere presence next to those they love. They are more flexible, adaptive, and reed-like; therefore, even if more severely disabled than men, women can continue to survive.

Quote from Claude-Levi Strauss on the Occasion of His 90th Birthday. (Note that he continued on until he reached 100 years old, a centenarian, as did most of the subjects in this study.)

"Montaigne dit que la vieillesse nous diminue chaque jour et nous entame de telle sorte que, quand la mort survient, elle n'emporte plus qu'un quart d'homme ou un demi-homme. Montaigne est mort à 59 ans, et ne pouvait avoir idée de l'extrême vieillesse où je me trouve aujourd'hui. Dans ce grand âge que je ne pensais pas atteindre et qui constitue une des plus curieuses surprises de mon existence, j'ai le sentiment d'être comme un hologramme brisé. Cet hologramme ne possède plus son unité entière et cependant, comme tout hologramme, chaque partie restante conserve une image et une représentation complète du tout. [. . .]Ainsi y-a-t-il aujourd'hui pour moi un moi réel, qui n'est plus qu'un quart ou la moitié d'un homme, et un moi virtuel, qui conserve encore vive une idée du tout. Le moi virtuel dresse un projet de livre, commence d'en organiser les chapitres et dit au moi réel: "C'est à toi de continuer". Et le moi réel, qui ne peut plus, dit au moi virtuel: "C'est ton affaire. C'est toi seul qui vois la totalité." Ma vie se déroule à présent dans ce dialogue très étrange. [. . .] Je vous suis très reconnaissant d'avoir, pour quelques instants, grâce à votre présence aujourd'hui et votre amitié, fait cesser ce dialogue en permettant un moment à ces deux moi de coïncider de nouveau. Je sais bien que le moi réel continue de fondre jusqu'à la dissolution ultime, mais je vous suis reconnaissant de m'avoir tendu la main, me donnant ainsi le sentiment, pour un instant, qu'il en est autrement."

"Montaigne says that old age diminishes us so that, when death arrives, it claims only a quarter or a half a man. Montaigne died at fifty-nine and surely had no idea of the extreme old age, in which I find myself today. Having lived to a ripe old age which I never expected to attain, and which is one of the strangest surprises I have experienced, I feel like a shattered hologram. This hologram no longer possesses its entire unity, yet, as with any hologram, each surviving shard preserves an image and full representation of the whole.

So today for me there is a real self, which is but a quarter or half a man, and a virtual self, which preserves a living idea of the whole. The virtual self is planning a new book and beginning to organize its chapters, and to the real self it says, "Your job is to carry on." But the real self, which cannot carry on, says to the virtual self, "That's your problem. Only you see the thing whole." My life nowadays is defined by this very strange dialogue.

I am very grateful to you whose presence here today and whose friendship have for a short time silenced this dialogue and allowed these two selves to coincide again briefly. I know full well that the real self will continue to melt away until the moment of final dissolution, but I thank you for reaching out and for an instant giving me the sense that it might not be so."

Claude Levi-Strauss speaking extemporaneously to friends assembled to celebrate his 90th birthday (January 1999). (Translated from the French by Arthur Goldhammer.) Source: http://buffleheadcabin.com/post/6614364005/.

References

[1] M. Allard and J.-M. Robine, *Les Centenaires Français*, Serdi, Paris, France, 2000.

[2] B. Jeune, A. Skytthe, A. Cournil et al., "Handgrip strength among nonagenarians and centenarians in three European regions," *Journals of Gerontology, Series A*, vol. 61, no. 7, pp. 707–712, 2006.

[3] S. Katz, T. Downs, H. Cash, and R. Grotz, "Index of activities of daily living," *The Gerontologist*, vol. 20, no. 1, p. 301, 1970.

[4] T. Mura, J.-F. Dartigues, and C. Berr, "How many dementia cases in France and Europe? Alternative projections and scenarios 2010–2050," *European Journal of Neurology*, vol. 17, no. 2, pp. 252–259, 2010.

[5] J.-C. Kaufmann, *L'enquête et ses Méthodes, L'entretien Compréhensif*, Armand Colin, Paris, France, 2007.

[6] F. Balard, *Les Plus Âgés des Âgés, Une Culture Vivante aux Portes de la Mort*, Editions Universitaires Européennes, Sarrebruck, Germany, 2010.

[7] B. G. Glaser and A. Strauss, *Discovery of Grounded Theory. Strategies for Qualitative Research*, Transaction, Aldine, Tex, USA, 1967.
[8] K. Charmaz, "Qualitative interviewing and grounded theory analysis," in *Handbook of Interview Research, Context and Method*, J. F. Gubrium and J. A. Holstein, Eds., pp. 675–695, Sage, London, UK, 2001.
[9] J. M. Johnson, "In-depth interviewing," in *Handbook of Interview Research, Context and Method*, J. Gubrium and J. A. Holstein, Eds., pp. 103–121, Sage, London, UK, 2001.
[10] J. Poirier, *Récits de Vie, Théorie et Pratique*, Presses Universitaires de France, Paris, France, 1983.
[11] P. Bourdieu, *Le Sens Pratique*, Editions de minuit, Paris, France, 1980.
[12] H. J. Rubin and I. S. Rubin, *Qualitative Interviewing. The Art of Hearing Data*, Sage, Thousand Oaks, Calif, USA, 1995.
[13] C. Warren, "Qualitative interviewing," in *Handbook of Interview Research, Context and Method*, J. Gubrium and J. Holstein, Eds., pp. 83–103, Sage, London, UK, 2001.
[14] E. Goffman, "On fieldwork," *Journal of Contemporary Ethnography*, vol. 18, pp. 123–132, 1989.
[15] F. Laplantine, *Anthropologie de la Maladie*, Payot, Paris, France, 1993.
[16] J. A. Holstein and J. F. Gubrium, "Interpretive practice," in *The Sage Handbook of Qualitative Research*, Y. S. Lincoln, Ed., pp. 483–506, Sage, Thousand Oaks, Calif, USA, 2005.
[17] J. Keith, C. L. Fry, A. P. Glascock, C. Ikels, J. Dickerson-Putman, and H. C. Hadening, *The Aging Experience: Diversity and Commonality across Cultures*, Sage, Thousand Oaks, Calif, USA, 1994.
[18] C. L. Fry, J. Dickerson-Putman, P. Draper, C. Ikels, J. Keith, and A. P. Glascock, "Culture and the meaning of a good old age," in *The Cultural Context of Aging: Worldwide Perspectives*, J. Sokolovsky, Ed., pp. 99–123, Bergin and Garvey, London, UK, 1997.
[19] J. Sokolovsky, "Culture, aging and context," in *The Cultural Context of Aging: Worldwide Perspectives*, J. Sokolovsky, Ed., pp. 1–15, Bergin and Garvey, London, UK, 1997.
[20] M. Singleton, "Devenir vieux—ailleurs et autrement," in *Proceedings of the Vivre et Soigner La Vieillesse Dans Le Monde Conference*, Genova, Italy, 2003.
[21] N. S. Consedine, C. Magai, and F. Conway, "Predicting ethnic variation in adaptation to later life: styles of socioemotional functioning and constrained heterotypy," *Journal of Cross-Cultural Gerontology*, vol. 19, no. 2, pp. 97–131, 2004.
[22] D. C. Willcox, B. J. Willcox, J. Sokolovsky, and S. Sakihara, "The cultural context of "successful aging" among older women weavers in a Northern Okinawan village: The role of productive activity," *Journal of Cross-Cultural Gerontology*, vol. 22, no. 2, pp. 137–165, 2007.
[23] L. P. Fried, L. Ferrucci, J. Darer, J. D. Williamson, and G. Anderson, "Untangling the concepts of disability, frailty, and comorbidity: implications for improved targeting and care," *Journals of Gerontology, Series A*, vol. 59, no. 3, pp. 255–263, 2004.
[24] G. Mauger, "La théorie des générations de K. Mannheim et la théorie de l'habitus," *Annales de Vaucresson*, vol. 30-31, pp. 59–78, 1989.
[25] K. Mannheim, *Le Problème Des Générations*, Nathan, Paris, France, 1928.
[26] F. Mentré, *Les Générations Sociales*, Thèse, Bossard, Paris, France, 1920.
[27] W. Diltey, *Le Monde de L'esprit, Histoire des Sciences Humaines*, vol. 1, Aubier-Montaigne, Paris, France, 1942.
[28] L. Fèbvre, *La Terre et L'Évolution Humaine*, Albin Michel, Paris, France, 1922.
[29] E. Panofsky, *Architecture Gothique et Pensée Scolastique*, Editions de Minuit, Paris, France, 1951.
[30] M. Segalen, *Mari et Femme dans la Société Paysanne*, Flammarion, Paris, France, 1980.
[31] D. Guttman, *Reclaimed Power, Men and Women in Later Life*, Northwestern University Press, Evanston, Ill, USA, 1994.
[32] T. S. Lebra, "The dilemma and strategies of aging among contemporary Japanese women," *Ethnology*, vol. 18, no. 4, pp. 337–353, 1979.
[33] S. B. Ortner, "Is female to male as nature is to culture?" in *Women, Culture, and Society*, M. Z. Rosaldo and L. Lamphere, Eds., pp. 73–83, Stanford University Press, 1974.
[34] F. Bikoma, *Socialisation de la Femme Accomplie chez les Ndzebi du Gabon*, Ph.D. Thesis, Université Montpellier III, Montpellier, France, 2004.
[35] J. Dickerson-Putman and K. J. Brown, *Women among Women: Anthropological Perspectives on Female Age Hierarchies*, University of Illinois Press, Urbana, Ill, USA, 1998.
[36] V. Kerns and K. L. Brown, *In Her Prime: New Views of Middle-Aged Women*, University of Illinois Press, Urbana, Ill, USA, 1991.
[37] J.-F. Bickel, "Etre actif dans le grand âge: un plus pour le bien être?" *Retraite et Société*, vol. 52, pp. 84–108, 2007.
[38] J. Evert, E. Lawler, H. Bogan, and T. Perls, "Morbidity profiles of centenarians: survivors, delayers, and escapers," *Journals of Gerontology, Series A*, vol. 58, no. 3, pp. 232–237, 2003.
[39] J. Andrian, "Le suicide des personnes âgées," *Gérontologie et Sociétés*, vol. 90, pp. 49–69, 1999.
[40] L. K. George and E. C. Klipp, "Subjective components of aging well: researchers need to consider," *Generations*, vol. 15, no. 1, pp. 57–60, 1991.
[41] A. Desesquelles and N. Brouard, "Le réseau familial des personnes âgées de 60 ans ou plus vivant à domicile ou en institution," *Revue Population*, vol. 58, pp. 201–209, 2003.
[42] B. M. Barer, "Men and women aging differently," *International Journal of Aging and Human Development*, vol. 38, no. 1, pp. 29–40, 1994.
[43] C. Franceschi, L. Motta, S. Valensin et al., "Do men and women follow different trajectories to reach extreme longevity? Italian Multicenter Study on Centenarians (IMUSCE)," *Aging*, vol. 12, no. 2, pp. 77–84, 2000.
[44] K. L. Moore and T. V. N. Persaud, "The beginning of human development," in *The Developing Human: Clinically Oriented Embryology*, K. L. Moore and T. V. N. Persaud, Eds., pp. 14–39, WB Saunders, Philadelphia, Pa, USA, 1998.
[45] G. H. Miller, "Is the longevity gender gap decreasing?" *New York State Journal of Medicine*, vol. 86, no. 2, pp. 59–60, 1986.
[46] D. W. E. Smith, "Is greater female longevity a general finding among animals?" *Biological Reviews*, vol. 64, no. 1, pp. 1–12, 1989.
[47] A. B. Newman and J. S. Brach, "Gender gap in longevity and disability in older persons," *Epidemiologic Reviews*, vol. 23, no. 2, pp. 343–350, 2001.
[48] K. R. Boucot, "Wanted: more men to live longer," *Archives of Environmental Health*, vol. 11, no. 5, pp. 611–612, 1965.
[49] J. E. Wallace, "Gender differences in beliefs of why women live longer than men," *Psychological Reports*, vol. 79, no. 2, pp. 587–591, 1996.
[50] W. H. Courtenay, "Constructions of masculinity and their influence on men's well-being: a theory of gender and health," *Social Science and Medicine*, vol. 50, no. 10, pp. 1385–1401, 2000.

[51] A. Oksuzyan, K. Juel, J. W. Vaupel, and K. Christensen, "Men: good health and high mortality. Sex differences in health and aging," *Aging Clinical and Experimental Research*, vol. 20, no. 2, pp. 91–102, 2008.
[52] J. Viña, J. Sastre, F. Pallardó, and C. Borrás, "Mitochondrial theory of aging: importance to explain why females live longer than males," *Antioxidants and Redox Signaling*, vol. 5, no. 5, pp. 549–556, 2003.
[53] A. Benetos, K. Okuda, M. Lajemi et al., "Telomere length as an indicator of biological aging: the gender effect and relation with pulse pressure and pulse wave velocity," *Hypertension*, vol. 37, no. 2, pp. 381–385, 2001.
[54] S. Brouilette, R. K. Singh, J. R. Thompson, A. H. Goodall, and N. J. Samani, "White cell telomere length and risk of premature myocardial infarction," *Arteriosclerosis, Thrombosis, and Vascular Biology*, vol. 23, no. 5, pp. 842–846, 2003.
[55] J. M. Y. Wong and K. Collins, "Telomere maintenance and disease," *Lancet*, vol. 362, no. 9388, pp. 983–988, 2003.
[56] C. Franceschi and N. Fabris, "Human longevity: the gender difference," *Aging*, vol. 5, no. 5, pp. 333–336, 1993.
[57] R. Stindl, "Tying it all together: Telomeres, sexual size dimorphism and the gender gap in life expectancy," *Medical Hypotheses*, vol. 62, no. 1, pp. 151–154, 2004.
[58] S. Seely, "The gender gap: why do women live longer than men?" *International Journal of Cardiology*, vol. 29, no. 2, pp. 113–119, 1990.
[59] L. Cool, "Role continuity or crisis in later life? A Corsican case," *The International Journal of Aging and Human Development*, vol. 13, no. 3, pp. 169–181, 1981.
[60] P. J. Burke, "Identity processes and social stress," *American Sociological Review*, vol. 56, pp. 836–849, 1991.
[61] F. Meslé, "Gender gap in life expectancy: the reasons for a reduction of female advantage," *Revue d'Epidemiologie et de Sante Publique*, vol. 52, no. 4, pp. 333–352, 2004.
[62] F. C. Pampel, "Cigarette use and the narrrowing sex differential in mortality," *Population and Development Review*, vol. 28, no. 1, pp. 77–104, 2002.

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Research Article

A Population Where Men Live As Long As Women: Villagrande Strisaili, Sardinia

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Usually women live longer than men and female centenarians largely outnumber male centenarians. The findings of previous studies identifying a population with a femininity ratio close to 1.0 among centenarians in the mountainous region of Sardinia was the starting point of an in-depth investigation in order to compare mortality trajectories between men and women in that population. The exceptional survival of men compared to women emerges from the comparison with similar Italian data. Age exaggeration for men has been strictly excluded as a result of the age validation procedure. The discussion suggests that besides biological/genetic factors, the behavioral factors including life style, demographic behavior, family support, and community characteristics may play an important role. No single explanation is likely to account for such an exceptional situation and a fully integrated multidisciplinary approach is urgently needed.

1. Introduction

In developed countries, it has been widely documented that females live longer than males [1–3]. This female advantage in survival, the so-called Longevity Gender Gap (LGG), results from lower female death rates throughout the lifespan [4–6]. In traditional societies, the LGG was less pronounced and even inversed [7] and a secular trend of increasing LGG is generally observed. Nevertheless the trend has changed in the recent decades resulting in a slightly decreasing LGG [8].

In general, it has been observed that during the life course, the occurrence of certain diseases such as cardiovascular diseases, lung cancer and accidents are more frequent among males [9] and this trend is confirmed when extending the comparison to the top 12 causes of deaths [10]. The cumulative effect of these differences throughout the life course, results in a greater proportion of females surviving at more advanced ages. In populations experiencing a low mortality, the femininity ratio (F/M) among centenarians is usually above 4, that is, there are more than 4 female centenarians per male centenarian (data source: *Human Mortality Database*). However, recent research has shown that in certain populations the F/M ratio among the oldest olds may be remarkably lower, and when this occurs it is often the result of a reduced excess of male mortality rather than a higher mortality of women [11–13]. Among these populations, the one living in the Mediterranean island of Sardinia certainly represents an interesting case study. This region has been suspected to be characterized by a particularly high male longevity [14]. Considering the importance of age validation in longevity studies, the demographic data were carefully checked by using all available data sources in order to avoid cases of age exaggeration [15]. Based on these validation results, an area with significantly higher levels of longevity and lower F/M ratio among centenarians has been identified in the central-eastern part of the island and was called the *Blue Zone* [16]. As a result the topic of research has been switched from individual longevity to population longevity aiming to facilitate the identification of longevity determinants shared by people living in such area.

This longevity *Blue Zone* located in the provinces of Ogliastra and Nuoro in the mountainous region of Sardinia

shows a value of the Extreme Longevity Index (ELI[1]) computed for the newborns between 1880 and 1900 that is more than twice as high as that of whole Sardinia. Strikingly the F/M ratio among centenarians in this population is close to one, as 47 male centenarians were found and only 44 female centenarians. Thus the LGG usually observed among the oldest olds in other populations is virtually nonexistent in the longevity *Blue Zone*.

From these findings, the following question arises: *How is it possible that in this population the proportion of men reaching the extreme end of age spectrum is the same as for women?* In order to answer this question, the population of the municipality of Villagrande Strisaili was selected, as it is the municipality with the highest ELI value in Sardinia (10.8 centenarians per 1,000 newborns) and an extraordinary low F/M ratio; among the 30 centenarians found in Villagrande 16 are male. The life trajectory of each individual born in the village during the period 1876–1912[2] was followed from birth to death.

The analysis of the collected data allows the comparison of the mortality trajectory for men and women. Moreover the life table estimates of Villagrande have been compared with the corresponding ones for Italy extracted from the *Human Mortality Database* (HMD) [17]. The analysis of mortality trajectories allows the identification in more detail of the critical age interval for determining between-gender level differentials in longevity among the populations considered.

In the discussion, possible explanations for the lack of LGG observed in the *Blue Zone* will be reviewed in light of existing literature. As we will suggest, besides biological/genetic factors, numerous behavioral characteristics may count for explaining the male advantage. Accordingly only multidisciplinary investigations could explain the exceptional survival of males in the population under study. Having in mind this objective the few empirical evidences presented here should be considered as a starting point for further investigations.

2. Setting, Data, and Methods

2.1. Setting. The village of Villagrande is located at 700 meters above sea level in the province of Ogliastra, but the altitude of its territory ranges from sea level to *Punta La Marmora* at 1,834 meters. On 1 January 2010, 3,441 inhabitants lived in Villagrande (ISTAT) where agropastoral-pastoral activities and traditional life style are still prevalent. Despite the fact that until the 1960s this region was among the poorest within the island, recent economic developments brought the population of this area close to the average welfare standard of the Italian population.

2.2. Data. The database developed for the present study includes all individuals born in Villagrande from 1876 to 1912. For each individual we traced the exact date at death or the proof that he/she was still alive at the date of investigation. The data was gathered from civil registers (which record all births, marriages, and deaths), parish registers and the population register (*anagrafe*). All information has been collected in the municipality population registration office and was cross-checked with information reported in the military register and orally reported information from any relatives of these persons. With regard to those who died outside the village, the information was recovered by using annotations on date and place of death reported in the margin of the birth certificate or transcription of the date of death in the *anagrafe*. For those who emigrated and for whom no death has been reported, the survival status has been verified with the municipality of current residence.

A total number of 1,957 persons born in Villagrande during the years 1876 to 1912[3] have been considered in this study of which:

(i) 1,624 who died in Villagrande itself;

(ii) 206 who died outside Villagrande;

(iii) 20 who were still alive at the date of investigation;[4]

(iv) 107 for whom the date of death or the survival is unknown.

2.3. Coverage. The data collection method enables to reduce significantly the number of missing dates of death due to lack of information (107 cases). The latter group of newborns has been divided between outmigrants with the documented date of emigration and those where no indication of emigration was available. For those without information on emigration, their date of marriage or the latest trace in administrative documents, for example, military reports was considered as partial information. As a result, there were only 26 newborns for whom no information at all on survival was found, whereas for other 81 we found at least information attesting their survival at the time of marriage, military examination, or emigration. A final coverage rate of above 98 per cent was reached. This should be considered exceptional in the context of historical demography and family reconstruction. Moreover such level of coverage contributed to the complete validation of the database and the strength of the subsequent analysis.

2.4. Methods. The presence of still alive newborns and newborns with partial information implied the setting up of censoring strategies in order not to lose precious information. The general rule was to take the last information available that attested survival as censoring time.

The method utilized in this study is the classical cohort life table construction privileging as output the mortality rates and the cumulative survival curve. The computation of the survival curve takes into account the contribution of all censored newborns up to their exit of observation. The exclusion of the 26 persons without information and the censoring of the 101 others (20 alive and 81 with partial information) can hardly affect the validity of our findings. The distribution of the 1957 persons by gender, five years age groups at death or survival is displayed in Table 1.

For comparative purposes, we utilized data for Italy collected from birth cohort life tables available in the HMD for the same birth cohorts 1876–1912 considered for

Table 1: Observed number of deaths (showing also censored cases) by gender and age groups and cohorts of newborns including those who were alive at the time of investigation (2006).

	Males					Females				
Age completed	1876–1889	1890–1900	1901–1912	Alive	Total	1876–1889	1890–1900	1901–1912	Alive	Total
0	27 (1)	37 (1)	58 (1)	—	125	38 (1)	31 (1)	34 (1)	—	106
1–4	36 (2)	41	29 (1)	—	109	48	42	35	—	125
5–9	9	11	17	—	37	21	7	9	—	37
10–14	7	7	12	—	26	4	2	11	—	17
15–19	6	14 (6)	13 (3)	—	42	4	5	4 (2)	—	15
20–24	6 (1)	32 (5)	14 (2)	—	60	6	17	6	—	29
25–29	7 (5)	9 (1)	8 (1)	—	31	9 (2)	9	8	—	28
30–34	12 (7)	5 (4)	9 (3)	—	40	8 (1)	9	7 (2)	—	27
35–39	7 (2)	1 (1)	7 (3)	—	21	10 (1)	2	4	—	17
40–44	4 (1)	4	11 (2)	—	22	10	5	5	—	20
45–49	3	9	8 (1)	—	21	7	4	5	—	16
50–54	7	2	3	—	12	3 (1)	6	4	—	14
55–59	13	5	5	—	23	5	5	11	—	21
60–64	10	6	6 (1)	—	23	13	5	11	—	29
65–69	14	2	8	—	24	9	13	11	—	33
70–74	9	12	15 (2)	—	38	11	9	9	—	29
75–79	24	25	30 (1)	—	80	16	18	18	—	52
80–84	9	13	29 (2)	—	53	29	25	28 (4)	—	86
85–89	30 (1)	22	35	—	88	26	38	52 (2)	—	118
90–94	25	15	29 (2)	2	73	20	17	37	4	78
95–99	12	10	11	6	39	7	6	9	5	27
100+	3	3	2	1	9	7	1	1	2	11
Total	**280 (20)**	**285 (18)**	**359 (25)**	**9**	996	**311 (6)**	**276 (1)**	**319 (11)**	**11**	935
Unknown	**3**	**3**	**5**	—	11	**6**	**3**	**6**	—	15

Villagrande and involving separately males and females by single year of birth and age.

3. Results

The survival curves from birth till age 100 are shown in Figure 1 separately for men and women by considering all birth cohorts 1876–1912 together. The corresponding data are reported in Table 2.

At age 5, one newborn out of four has already died and an additional tenth did not reach the age of 18. At age 50, 46.4% of males and 53.3% of females are surviving. From age 50 to 75, males and females seem to be exposed to the same mortality risk. Between age 75 and 90, men record a higher level of survival compared to females so that finally at age 90 about 12% of the newborns—both for males and females—are surviving.

The comparison with strictly comparable data for Italy extracted from the Human Mortality Database is presented in Figures 2(a) and 2(b) separately for men and women. Compared to the Italian population the situation is really exceptional in Villagrande. For infant and child mortality two major demographic features emerge; in Villagrande a lower mortality up to 2 years old than in Italy was recorded, while from age 2 to 5 years old mortality estimates increase. This particular trend was observed by Coletti for Sardinia

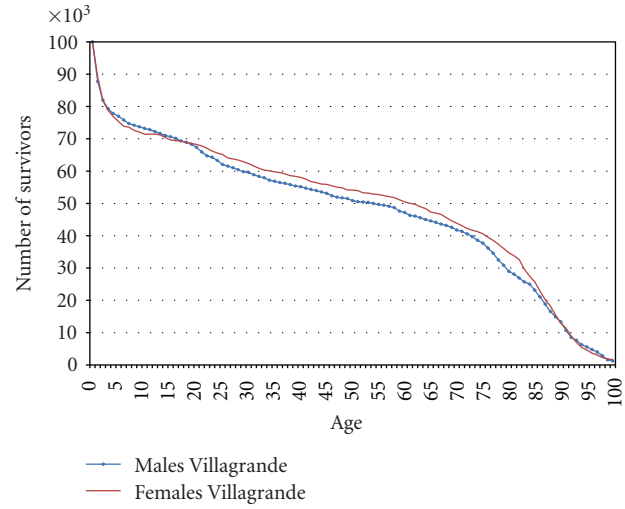

Figure 1: Comparing survival curves for males and females in Villagrande for a hypothetical cohort of 100,000 newborns of the years 1876–1912.

already in 1908 [18]. This low trend in early mortality could not be explained in terms of major economic development, given the poor conditions of Sardinian population at the end of 19th century, but the author hypothesizes that rather it was determined by the positive effect of the common practice

Table 2: Comparison of survival curves for males and females in Villagrande with the corresponding data extracted from the *Human Mortality Database* for the whole Italy (birth cohorts from 1876 till 1912 included).

Age	Villagrande		Italy	
	Males	Females	Males	Females
0	100000	100000	100000	100000
5	76506	75294	68333	69639
10	72791	71337	65950	67028
15	70181	69519	64851	65706
20	65964	67914	62907	63940
25	59940	64813	59438	61947
30	56827	61818	56845	60087
35	52811	58930	54591	58368
40	50703	57112	52648	56795
45	48494	54973	50797	55320
50	46386	53262	48773	53801
55	45181	51765	46176	51927
60	42871	49519	42676	49483
65	40562	46417	37929	46079
70	38153	42888	31743	41275
75	34337	39786	24114	34525
80	26305	34225	15779	25754
85	20984	25027	8258	16098
90	12149	12406	3162	7694
95	4819	4064	777	2480
100	904	1176	105	456
	Probability to survive from 50 to 75			
	74,0%	74,7%	49,4%	64,2%

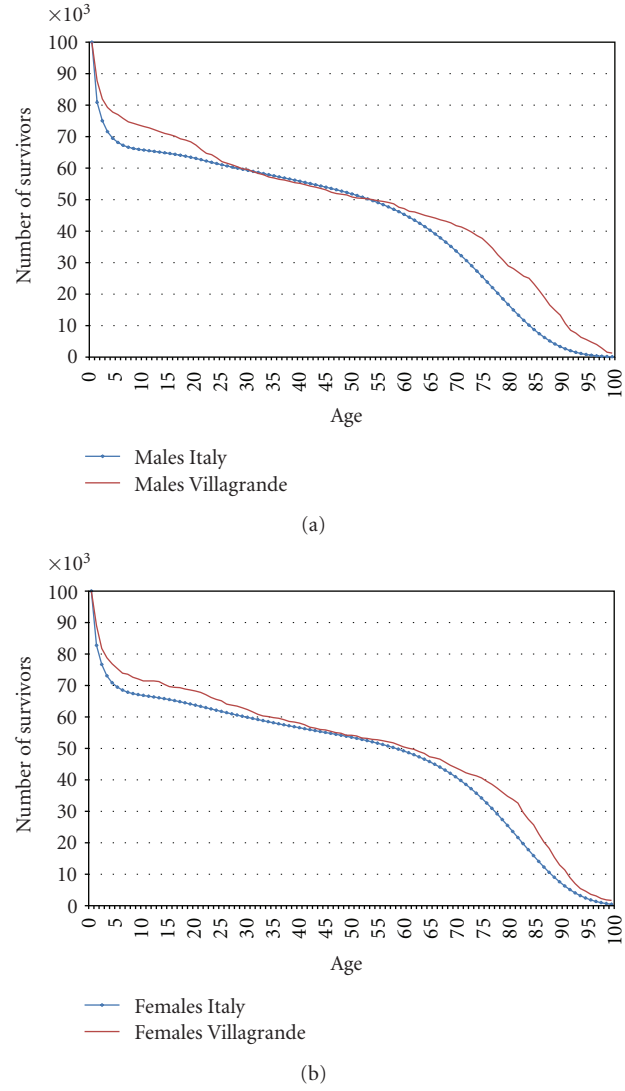

Figure 2: (a) Comparison of survival curves for males in Villagrande with the corresponding data extracted from the Human Mortality Database for the whole of Italy (birth cohorts from 1876 till 1912 included). (b) Comparing survival for females in Villagrande with strictly comparable data extracted from the Human Mortality Database for the whole of Italy (birth cohorts from 1876 till 1912 included).

of prolonged breastfeeding and the limited participation of Sardinian women to external works. Subsequent studies on regional data [19, 20] and on selected villages confirmed this trend of low early mortality [21, 22].

Considering the mortality rates from age 5 until age 18, it emerges that once a newborn managed to reach 5 years old, he/she faced more favourable conditions for survival. Nevertheless we observe in Villagrande a higher risk of death from age 6 to 10 compared to the mainland, while estimates until 18 are similar in the two populations. Therefore the survival advantage at early ages of individuals born in Villagrande Strisaili is not maintained, but generates overmortality at subsequent ages. This changing trend might be interpreted in terms of different timing in the selection process of frail individuals. In the Italian population frails are eliminated in the first years of life, while in Villagrande Strisaili this process appears to be postponed.

The analysis of adult mortality appears to be more complex since the latter is mainly due to external causes such as losses in WW I for men and maternal mortality for women. Military data show that no less than 40 males born in Villagrande died during WW I and that most of them were born in the period 1890–1900. For women, maternal mortality was also high during the reproduction period. Orrù and Putzolu [23], in their study on the diffusion of professional assistants for delivery, pointed out that in 1899 there was no professional assistant in Villagrande and in the surrounding area. However, in the village, a maternal mortality rate varying between 10.6 and 11.1 per 1,000 births was estimated [24], which is in line with the levels recorded in other Sardinian villages and in other European countries for the same period [25]. Due to the higher mortality risk for both sexes between 18 and 50, the proportions of 46.4% of men and 53.3% of women surviving at age 50 are similar to the ones observed for the whole of Italy (Figures 2(a) and 2(b)).

About three out of four males and females surviving at age 50 finally reached age 75. When comparing these figures with the ones of the Italian population a lower mortality risk is evident mostly for men. (For men, 74% of those aged

50 survive up to 75 for Villagrande compared to 49% in Italy. For women these proportions are, respectively, 75% and 64%.) The lower level of the mortality risk for men is confirmed when comparing the mortality rates (Figures 3(a) and 3(b)). For men the mortality rates are significantly lower starting at age 60 (considering a confidence interval of 0.01) while for women mortality rates are also lower but significant only from 70 to 85 and no longer significant after that age. Finally, it is worth noting that the proportion of those surviving at 90 years in the life table computed for Villagrande is balanced almost evenly between men and women (10,7% for men and 11,4% for women), and this result must be considered exceptional compared with the situation in Italy.

4. Discussion

Why do men usually live shorter than women? This question has been addressed in several studies where potential explanatory factors are examined [10, 26–33]. Why men live as long as women in Villagrande and, more generally, in the Sardinian longevity *Blue Zone,* is a related question emerging from the results of this study. To discuss this question more in detail, we will review several factors proposed in the literature to explain the LGG advantaging females and try to understand why they seem not to work in the population under concern. In addition some other factors that may directly favor male longevity will be considered.

4.1. Biological/Genetic Factors. The first group of factors invoked to explain the female advantage in longevity relates to biological traits. An increased level of homozygosity in the population, which may reduce the variability of the genetic pool, has been reported to favor male longevity [34]. As almost everywhere in the whole *Blue Zone*, the population of Villagrande was characterized by a high level of endogamy until WW II [35, 36]. In addition, the role of gender-specific genes in longevity has been largely investigated. Women have two X chromosomes, one from their father and the other from their mother, while males have only one X chromosome and one Y chromosome. Because of this, women have two cell lines and if any recessive allele detrimental for survival is inherited, the allele on the second X chromosome could compensate the expression of the first one, a protective strategy that is lacking in males [37]. On the other hand, an increased mutation load in the X chromosome may result from the higher paternal age at reproduction, a condition relatively common in central Sardinia, and that may contribute to reduce life span only in daughters [38], as sons do not inherit the paternal X chromosome. One possibility is that in genetically homogeneous populations such as the one living in Central Sardinia [39], the probability that females carry the same alleles in both X chromosomes (homozygosity) is so high as to reduce the protective effect of having two X chromosomes. Alternatively, specific X-linked recessive alleles may exist in the *Blue Zone* population, as a result of local selective pressures, that may extend the life expectancy of men via different mechanisms. This may be

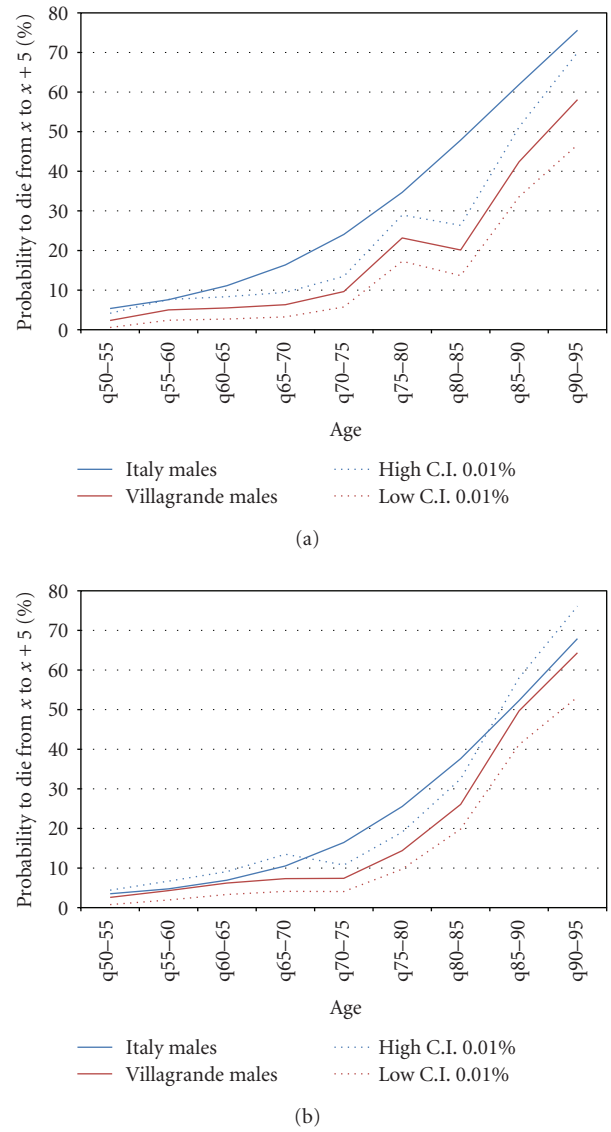

Figure 3: (a) Compared five years' probability of dying for males in Villagrande with strictly comparable data extracted from the HMD for the whole Italy (birth cohorts from 1876 till 1912 included). (b) Compared five years' probability of dying for females in Villagrande with strictly comparable data extracted from the HMD for the whole Italy (birth cohorts from 1876 till 1912 included).

the possible effect of G6PD deficiency, an X-linked disorder quite common in the island [40] that was hypothesized to explain Sardinian longevity for males [41]. Unfortunately the few available epidemiological data [42] do not support the existence of a higher prevalence of G6PD deficiency in villages with increased longevity. Recent findings suggest that an X-linked component affects the telomere length and that brings an additional potential explanation for the LGG as the telomere length is highly inherited, longer in women and linked to survival [43, 44].

It has also been hypothesized that Y-chromosome allelic variants may affect male survival, probably influencing the circulating levels of testosterone and hence its biological effects on vasculature and other tissues. Men with higher

testosterone levels show reduced markers of inflammation and metabolic syndrome and decreased risk of mortality for all causes independently from the overall health status [45]. Although the study of the endocrine profile of Sardinian oldest olds showed markedly higher testosterone serum levels and lower estradiol than in younger controls [46] the real significance of these results is still to be clarified. Moreover, a study carried out in a limited number of Sardinian male centenarians and younger control subjects failed to find any significant association between longevity and Y-related markers [47], although an additional set of markers and a larger sample may be required to further clarify the role of Y chromosome on increased survival.

The role of mitochondrial DNA on gender differential longevity has also been investigated. In particular, the frequency of a polymorphic variant called J haplogroup was reported to be increased in male centenarians from continental Italy [48]. In the Ogliastra population, the frequency of such haplogroup in the whole population is more than twice the average frequency in Sardinia (18.3% versus 6.7%) [49], making this polymorphism a valuable candidate to explain the reduced excess male mortality.

Other genetic traits have been claimed to favor male longevity, such as the β-thalassemia trait that confers some "protection" against the development of premature cardiovascular diseases [50]. Caselli and Lipsi [51] showed that the population in the *Blue Zone* presents the lowest mortality in Sardinia but also the lowest cardiovascular mortality. They concluded that these findings indicate the existence of a specific genetic or environmental factor that protects Sardinian men, further research being needed to clarify the exact nature of these factors.

Potential explanatory factors in this first group also include anthropometric differences often observed between males and females. The difference in average height between male and female may be lower in the *Blue Zone* population, while the average body mass index of females may be relatively higher compared to males. If these two hypotheses are confirmed this would lead to a longevity advantage for males as some studies have shown that there is an inverse relationship between both height and body mass index and longevity [52, 53]. Evidence derived from military conscripts shows a shorter height on average for men in the *Blue Zone* compared to those in the whole of Sardinia [54], whereas no such data are available for women, thus precluding the possibility to test this hypothesis.

4.2. Behavioral and Sociocultural Factors. The second group of factors is related to individual behavior and Sociocultural traits of the population. First of all it is important to mention that migration does not introduce any bias in our results as all and only individuals born in Villagrande are included in the study even though some of them later emigrated outside the village and did not die in the village. By evidence these persons who emigrated from the village could be different with regard to behavior and/or social characteristics from those who stay living in the village all their life. Nevertheless it has a limited impact as emigrants represent less than 10% of the newborns.

Other individual behaviors include possible differences in nutrition between males and females, but also in physical activity and energy balance. Such differences in behavior are mostly linked to the different occupations of males and females as well as other aspects of their life style. Although no specific study up to now ascertained any gender-related differences in Sardinian nutrition, it may be hypothesized that the distinct role of women within the families and the prevalent occupation of men may have induced appreciable differences in dietary patterns between genders. Also hypothetical difference in feeding boys and girls in their childhood should be investigated. Drinking red wine from Ogliastra was claimed to have a positive effect on longevity due to its higher content of antioxidants [55]. Combined with the traditional Sardinian higher consumption of wine by males compared to females, this may hypothetically contribute to male longevity. Besides, as in the longevity *Blue Zone* men were mostly shepherds while women were mostly responsible for all domestic tasks including animal breeding and vegetable and cereal cultivation [56], men likely had different physical activities compared to women during their active life that lasted until advanced ages. The additional circumstance of living in a mountainous area increases the daily energy expenditure of most men compared to women, also favoring longevity [57, 58]. Women were the main responsible for the care of goods and property but also the trustees of transmission of the ethical values, including self-defense, implying a certain degree of aggressivity [59]. Some specific character traits of males in the *Blue Zone* compared to females like a better tolerance to stress, a high level of extraversion and sociability, and possibly a better ability to take advantage of the positive aspects of the health transition, may also play a positive role in their longevity especially by lowering cardiovascular mortality [60]. However, data on personality characteristics of men in Villagrande is currently not available and only future research will allow testing this hypothesis.

Accordingly, the absence of a gender gap in mortality risk might also be attributed to differences in men's and women's social behavior within a rather archaic society that acknowledges distinct roles to males and females. In particular, the population of central Sardinia during the centuries experienced a kind of matriarchal organization [61] that lasted till the onset of industrialization in the late 1950s. Nevertheless the concept of a "matriarchal organization" in Sardinia, as it was put by Pitzalis-Acciaro in 1978 [61], is a controversial one and has been challenged by anthropologists thereafter [62, 63]. Anyway, during the last few centuries Sardinian society was clearly dominated by men, as elsewhere in the Mediterranean countries, especially among rural communities of farmers. The situation may have been slightly different among pastoral communities, where the chronic absence of men increased the workload and responsibility of women. This does not mean, however, that women had a real power; instead they had to shoulder the toughest jobs and accordingly it could be better to refer to a "women-centered" society, rather than "matriarchal" [64]. This specific role of women within the family, and hence within the community, probably implied an increased

"chronic stress burden" compared to men, which ultimately may have contributed to disfavor female longevity [63].

Some specific Sociocultural traits of the population living in the *Blue Zone* may also favor male longevity. Some gender stereotypes and related social norms may favor men compared to women and this seems to be the case in Ogliastra [59] where oldest men are the target of intense attention within the family but also in the local community, which may bring them support for living longer. Also it is well established that a stronger family support and long-standing living arrangement in a married couple may help to live longer [65]. If men marry younger women, they will largely escape widowhood that has a negative impact for living longer at least for men. Moreover, a high level of remarriage among widowed men increases the age difference with their last spouse and keeps a high proportion of men still married in old ages. Among the 324 ever-married men of Villagrande who died above age 80, 41 (13%) have been married more than once and the age difference with their (last) spouse, for 136 of them (39%), was larger than 10 years (median 9.1) while only 25 (7%) had an older spouse. Such larger age differences between spouses are favorable for male longevity [66]. In addition, after widowhood, older women tend to live more often alone while older men will live preferably with an unmarried child taking into account that entering a nursing home is an exception for the population concerned. Overall this situation also favors male longevity.

Furthermore the possible role of fertility and more specifically the impact of the timing of the reproductive period on women's survival have to be considered. Historically, Sardinian women were characterized by high fertility and an older age at birth of their children [67]. Astolfi et al. [68] shows that the spatial pattern of late reproduction behavior in Sardinia display areas that are very similar to the longevity *Blue Zone* assuming that there exists some common explanation linking late reproduction behavior and longevity. Such association has been proved in other populations [69, 70] but only as far as female longevity is concerned. Accordingly such argument cannot be considered as improving longevity of males compared to females. Nevertheless a more recent investigation on large historical databases [71] shows that late reproductive behavior is not only associated with female postmenopausal longevity but also with survival past age 50 of brothers of late-fertile women. According to these authors, these results support the hypothesis that both the late female fertility and the slow somatic aging for both men and women may be promoted by the same genetic variants. These arguments could explain the relative higher longevity observed in the *Blue Zone* but not the male advantage identified in the population under study. Nevertheless the similarities observed between the spatial patterns of late fertility and high longevity could be related to higher endogamy.

5. Conclusion

An intensive data collection and complete family reconstruction allowed finding the date of death or confirming the survival at the date of survey of almost all 1957 children born in Villagrande during the years 1876–1912. The cohort life table has been estimated and the mortality trajectory identified separately for men and women. The survival curves and mortality rates have been compared with similar ones for Italy extracted from the HMD. The exceptional situation of men in Villagrande emerges from that comparison, as men are living as long as women do, which is unusual elsewhere. Three conclusions can be drawn from our preliminary investigations. First age exaggeration for men has been clearly excluded as a result of the age validation exercise among the possible reasons explaining why men apparently live as long as women in Villagrande, as well as in the Sardinian *Blue Zone*. Secondly, we consider that the above-mentioned factors may work jointly in a population characterized by a sort of "matriarchal" organization but the overall impact of these factors should be strong enough to explain why men are living as long as women do in that population, a significantly different situation compared to all populations in developed countries.

Thirdly, it is unlikely that a single explanation can be found for justifying such an exceptional situation. Quoting Franceschi et al. [11] "*besides the classical biomedical disciplines such as gerontology, geriatrics, genetics and immunology among others, a new integrated approach, including demography, historical demography, anthropology, and other social sciences, appears to be necessary to disentangle the complex interaction between the environmental/cultural and biological/genetic components responsible for sex differences in centenarians.*"

The results of this investigation indicate that the usually observed higher mortality for men is not valid for the population of Villagrande and, by extension, to the one of the *Blue Zone*. Among the whole list of potential factors only few evidences have been found so far. The explanatory research should be continued keeping in mind that only a multidisciplinary approach will help finding the underlying set of explanatory factors.

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Endnotes

1. ELI (extreme longevity index) is the proportion of newborns in a given place who became centenarian wherever and thus estimates the probability for

a newborn in a given place to reach the age 100. In the *Blue Zone*, 91 centenarians were found among the 17,865 newborns of the years 1880–1900 while 1132 centenarians were counted among the 516,276 newborns in the whole Sardinia. The ELI values are, respectively, 5.1 centenarians per 1,000 newborns in the *Blue Zone* compared to 2.1 for Sardinia.

2. Although the data are available since 1866, the year when the civil registers were first held in Sardinian municipalities, only the cohorts born after 1876 are considered, as the data related to the earlier birth cohorts are less complete. The last year of observation, 1912 has been chosen in order to include the cohorts of those who were at least 95 years old at the time of investigation and to exclude those whose infant mortality could be increased during WW I.
3. From the totality of those born in Villagrande were excluded only those babies (120 out of 2077) that clearly were accidentally born in the village, where their parents were living at that time for work-related reasons. To identify these newborns we utilized the origins of both parents and their occupation (miners, workers involved in the construction of roads and train networks, police officers) as usually reported in the birth certificate.
4. As of 5 September 2006.

References

[1] Q. R. Hazzard, "The sex differences in longevity," in *Geriatric Medicine and Gerontology*, W. R. Hazzard, Ed., McGraw-Hill, New York, NY, USA, 1994.

[2] R. Crose, *Why Women Live Longer than Men?* Jossey-Bass, San Francisco, Calif, USA, 1997.

[3] J. Clarke, *The Human Dichotomy: The Changing Numbers of Males and Females*, Pergamon, Amsterdam, The Netherlands, 2000.

[4] R. H. Daw, "The comparison of male and female mortality rates," *Journal of the Royal Statistical Society, Series A (General)*, vol. 124, no. 1, pp. 20–43, 1961.

[5] S. P. Phillips, "Risky business: explaining the gender gap in longevity," *Journal of Men's Health and Gender*, vol. 3, no. 1, pp. 43–46, 2006.

[6] R. Jacobsen, A. Oksuzyan, H. Engberg, B. Jeune, J. W. Vaupel, and K. Christensen, "Sex differential in mortality trends of old-aged Danes: a nation wide study of age, period and cohort effects," *European Journal of Epidemiology*, vol. 23, no. 11, pp. 723–730, 2008.

[7] D. J. Kruger and R. N. Nesse, "An evolutionary life-history framework for understanding sex differences in human mortality rates," *Human Nature*, vol. 17, no. 1, pp. 74–97, 2006.

[8] F. Mesle, "Progrès récents de l'espérance de vie en France : ies hommes comblent une partie de leur retard," *Population*, vol. 61, pp. 437–462, 2006.

[9] J. M. Guralnik, J. L. Balfour, and S. Volpato, "The ratio of older women to men: historical perspectives and cross-national comparisons," *Aging-Clinical and Experimental Research*, vol. 12, no. 2, pp. 65–76, 2000.

[10] S. Austad, "Why women live longer than men: sex differences in longevity," *Gender Medicine*, vol. 3, no. 2, pp. 79–92, 2006.

[11] C. Franceschi, L. Motta, S. Valensin et al., "Do men and women follow different trajectories to reach extreme longevity? Italian multicenter study on centenarians (IMUSCE)," *Aging-Clinical and Experimental Research*, vol. 12, no. 2, pp. 77–84, 2000.

[12] G. Passarino, C. Calignano, A. Vallone et al., "Male/female ratio in centenarians: a possible role played by population genetic structure," *Experimental Gerontology*, vol. 37, no. 10-11, pp. 1283–1289, 2002.

[13] J.-M. Robine, G. Caselli, D. Rasulo, and A. Cournil, "Differentials in the femininity ratio among centenarians: variations between northern and southern Italy from 1870," *Population Studies*, vol. 60, no. 1, pp. 99–113, 2006.

[14] L. Deiana, L. Ferrucci, G. M. Pes et al., "AKEntAnnos. The sardinia study of extreme longevity," *Aging Clinical and Experimental Research*, vol. 11, no. 3, pp. 142–149, 1999.

[15] M. Poulain, G. M. Pes, C. Carru et al., "The validation of exceptional male longevity in Sardinia," in *Human Longevity, Individual Life Duration and the Growth of the Oldest-Old Population*, J. M. Robine, E. M. Crimmins, S. Horiuchi et al., Eds., chapter 7, pp. 146–166, Springler & Kluwer, New York, NY, USA, 2006.

[16] M. Poulain, G. M. Pes, C. Grasland et al., "Identification of a geographic area characterized by extreme longevity in the Sardinia island: the AKEA study," *Experimental Gerontology*, vol. 39, no. 9, pp. 1423–1429, 2004.

[17] Human Mortality Database (Wilmoth J., University of California, Berkeley and Shkolnikov V., Max Planck Institute for Demographic Research), 2011, http://www.mortality.org.

[18] F. Coletti, *La Mortalità nei Primi Anni d'età e la Vita Sociale in Sardegna*, Fratelli Bocca, Torino, Italy, 1908.

[19] L. Pozzi, *La lotta per la vita. Evoluzione e Geografia della Sopravvivenza in Italia fra '800 e '900*, Forum, Udine, Italy, 2000.

[20] A. M. Gatti, "Livelli e caratteristiche della mortalità nella diocesi di Ales in Sardegna (1801–1825)," in *Omaggio a Danilo Giori*, G. Bottazi, Ed., Giuffré, Milano, Italy, 1990.

[21] A. M. Gatti, "La mortalità infantile tra ottocento e novecento. La Sardegna nel panorama italiano," in *Quaderni del Dipartimento di Ricerche Economiche e Sociali*, vol. 13, University of Cagliari, Cagliari, Italy, 2002.

[22] M. Breschi, S. Mazzoni, P. M. Melis, and L. Pozzi, "Nuove indagini per l'analisi della mortalità nei primi anni di vita in Sardegna," in *Salute, Malattia e Sopravvivenza in Italia fra '800 e '900*, M. Breschi and L. Pozzi, Eds., pp. 191–216, Forum, Udine, Italy, 2007.

[23] L. Orrù and F. Putzolu, *Il Parto e la Nascita in Sardegna. Tradizione Medicalizzazione Ospedalizzazione*, CUEC, Cagliari, Italy, 1993.

[24] L. Salaris, *Searching for Longevity Determinants: Following Survival of Newborns in In-Land Village in Sardinia (1866–2006)*, Ph.D. thesis, Presses Universitaires de Louvain, 2009.

[25] A. M. Gatti, "Nascita dell'ostetricia e mortalità materna in Sardegna (XVII-XIX secolo)," in *Bollettino di Demografia Storica*, vol. 30-31, pp. 79–94, Si.De.S. Società Italiana di Demografia Storica, Roma, Italy, 1999.

[26] D. L. Wingard, "The sex differential in morbidity, mortality, and lifestyle," *Annual Review of Public Health*, vol. 5, pp. 433–458, 1984.

[27] I. Waldron, "What do we know about causes of sex differences in mortality? A review of the literature," *Population Bulletin of the United Nations*, no. 18, pp. 59–76, 1985.

[28] L. M. Verbrugge and D. L. Wingard, "Sex differentials in health and mortality," *Women and Health*, vol. 12, no. 2, pp. 103–145, 1987.

[29] F. M. Antonini, "Perché le donne sono più longeve degli uomini?" *Giornale di Gerontologia*, vol. 39, no. 4, pp. 177–178, 1991.

[30] B. B. Kalben, "Why men die younger: causes of mortality differences by sex," *North American Actuarial Journal*, vol. 4, pp. 83–111, 2000.

[31] A. Case and C. Paxson, "Sex differences in morbidity and mortality," *Demography*, vol. 42, no. 2, pp. 189–214, 2005.

[32] A. Oksuzyan, K. Juel, J. W. Vaupel, and K. Christensen, "Men: good health and high mortality. Sex differences in health and aging," *Aging Clinical and Experimental Research*, vol. 20, no. 2, pp. 91–102, 2008.

[33] R. G. Rogers, B. G. Everett, J. M. Onge, and P. M. Krueger, "Social, behavioral, and biological factors, and sex differences in mortality," *Demography*, vol. 47, no. 3, pp. 555–578, 2010.

[34] M. Bonafè, M. Cardelli, F. Marchegiani et al., "Increase of homozygosity in centenarians revealed by a new inter-Alu PCR technique," *Experimental Gerontology*, vol. 36, no. 7, pp. 1063–1073, 2001.

[35] A. Cannas, Biddamanna. Vida longa. Villagrande Strisaili, paese di longevi, Cagliari, 2007.

[36] A. Moroni, A. Anelli, W. Anghinetti et al., "La consanguineità umana nell'isola di Sardegna dal secolo XVIII al secolo XX," *L'Ateneo Parmense*, vol. 1, supplement 8, pp. 69–92, 1972.

[37] K. Christensen, K. H. Ørstavik, and J. W. Vaupel, "The X chromosome and the female survival advantage: an example of the intersection between genetic, epidemiology and demography," *Annals of the New York Academy of Sciences*, vol. 954, pp. 175–183, 2001.

[38] L. A. Gavrilov, N. S. Gavrilova, G. N. Evdokushkina et al., "Determinants of human longevity: parental age at reproduction and offspring longevity," *Longevity Report*, vol. 10, no. 54, pp. 7–15, 1996.

[39] L. L. Cavalli Sforza, P. Menozzi, A. Piazza et al., *The History and Geography of Human Genes*, Princeton University Press, Princeton, NJ, USA, 1994.

[40] E. Sanna, G. G. Cosseddu, G. Floris, R. Bruno, A. Salis, and M. Silvetti, "Present-day G-6-PD deficit in Sardinia with respect to malarial morbidity and mortality in the past," *Zeitschrift fur Morphologie und Anthropologie*, vol. 78, no. 2, pp. 257–267, 1990.

[41] A. G. Schwartz and L. L. Pashko, "Dehydroepiandrosterone, glucose-6-phosphate dehydrogenase, and longevity," *Ageing Research Reviews*, vol. 3, no. 2, pp. 171–187, 2004.

[42] E. Sanna, G. G. Cosseddu, G. Floris et al., "Micromapping the distribution of G6PD deficiency in Sardinia with data collected from the 1950s to the 1980s," in *Adaptation to Malaria. The Interaction of Biology and Culture*, L. S. Greene and M. E. Danubio, Eds., pp. 293–322, New York, NY, USA, 1997.

[43] A. Aviv, J. Shay, K. Christensen, and W. Wright, "The longevity gender gap: are telomeres the explanation?" *Science of Aging Knowledge Environment*, vol. 2005, no. 23, article pe16, 2005.

[44] T. S. Nawrot, J. A. Staessen, and J. P. Gardner, "Telomere length and possible link to X chromosome," *The Lancet*, vol. 363, no. 9408, pp. 507–510, 2004.

[45] G. A. Laughlin, E. Barrett-Connor, and J. Bergstrom, "Low serum testosterone and mortality in older men," *Journal of Clinical Endocrinology and Metabolism*, vol. 93, no. 1, pp. 68–75, 2008.

[46] G. Delitala, F. Sanciu, G. Fanciulli et al., "Gonadal hormones and adrenal steroidogenesis in centenarians," *Biochimica Clinica*, vol. 30, p. S37, 2006.

[47] G. Passarino, P. A. Underhill, L. L. Cavalli-Sforza et al., "Y chromosome binary markers to study the high prevalence of males in Sardinian centenarians and the genetic structure of the Sardinian population," *Human Heredity*, vol. 52, no. 3, pp. 136–139, 2001.

[48] G. De Benedictis, G. Rose, G. Carrieri et al., "Mitochondrial DNA inherited variants are associated with successful aging and longevity in humans," *The FASEB Journal*, vol. 13, no. 12, pp. 1532–1536, 1999.

[49] C. Fraumene, E. Petretto, A. Angius, and M. Pirastu, "Striking differentiation of sub-populations within a genetically homogeneous isolate (Ogliastra) in Sardinia as revealed by mtDNA analysis," *Human Genetics*, vol. 114, no. 1, pp. 1–10, 2003.

[50] M. Gallerani, C. Scapoli, I. Cicognani et al., "Thalassaemia trait and myocardial infarction: low infarction incidence in male subjects confirmed," *Journal of Internal Medicine*, vol. 230, no. 2, pp. 109–111, 1991.

[51] G. Caselli and R. M. Lipsi, "Survival differences among the oldest old in Sardinia: who, what, where, and why," *Demographic Research*, vol. 14, pp. 267–294, 2006.

[52] T. T. Samaras, H. Elrick, and L. H. Storms, "Is height related to longevity?" *Life Sciences*, vol. 72, no. 16, pp. 1781–1802, 2003.

[53] L. Salaris, M. Poulain, I. S. Piras et al., "Height and longevity among males born in Villagrande Strisaili, (1866–1915)," in *Proceedings of the 43rd Riunione Scientifica SIS*, Section C24—Mortality, Health and Poverty, pp. 649–652, CLUEP Padova, Torino, Italy, June 2006.

[54] L. Salaris, M. Poulain, and T. T. Samaras, "Height and survival at older ages among males born in an in-land village in Sardinia (Italy), 1866–2006," submitted.

[55] R. Corder, W. Mullen, N. Q. Khan et al., "Oenology: red wine procyanidins and vascular health," *Nature*, vol. 444, no. 7119, p. 566, 2006.

[56] L. Edelsward, *Highlands Visions. Recreating Rural Sardinia*, Ph.D.Deissertation, Department of Anthropology. McGill University, 1995.

[57] D. E. Warburton, C. W. Nicol, and S. S. Bredin, "Health benefits of physical activity: the evidence," *Canadian Medical Association Journal*, vol. 174, no. 6, pp. 801–809, 2006.

[58] G. M. Pes, F. Tolu, and M. Poulain, "Lifestyle and nutrition related to male longevity in Sardinia: an ecological study," *Nutrition, Metabolism & Cardiovascular Diseases*. In press.

[59] L. Assmuth, *Women's Work, Women's Worth: Changing Life courses in Highland Sardinia*, vol. 39 of *Transactions of the Finnish Anthropological Society*, Finnish Anthropological Society, 1997.

[60] E. J. Giltay, J. M. Geleijnse, F. G. Zitman, T. Hoekstra, and E. G. Schouten, "Dispositional optimism and all-cause and cardiovascular mortality in a prospective cohort of elderly Dutch men and women," *Archives of General Psychiatry*, vol. 61, no. 11, pp. 1126–1135, 2004.

[61] M. Pitzalis-Acciaro, *In Nome della Madre. Ipotesi sul Matriarcato Barbaricino*, Feltrinelli Economica, Milano, Italy, 1978.

[62] M. G. Da Re, *La casa e I Campi. La Divisione Sessuale del Lavoro nella Sardegna Tradizionale*, CUEC, Cagliari, Italy, 1990.

[63] A. Oppo, "Where there's no woman there's no home: profile of the agro-pastoral family in nineteenth-century Sardinia," *Journal of Family History*, vol. 15, no. 1, pp. 483–502, 1990.

[64] C. Eller, *The Myth of a Matriarchal Prehistory*, The University Press Group, 2011.

[65] J. Gaymu, P. Festy, M. Poulain, and G. Beets, *Future Elderly Living Conditions in Europe*, INED, Paris, Farnce, 2008.

[66] D. Foster, L. Klinger Vartabedian, and L. Wispe, "Male longevity and age differences between spouses," *Journals of Gerontology*, vol. 39, no. 1, pp. 117–120, 1984.

[67] L. Bernardi and A. Oppo, *Fertility and Family Configurations in Sardinia*, MPIDR Working Paper WP 2007-033, Max Planck Institute for Demographic Research, 2007.

[68] P. Astolfi, G. Caselli, O. Fioranic et al., "Late reproduction behaviour in Sardinia: spatial analysis suggests local aptitude towards reproductive longevity," *Evolution and Human Behavior*, vol. 30, no. 2, pp. 93–102, 2009.

[69] H. G. Müller, J. M. Chiou, J. R. Carey, and J. L. Wang, "Fertility and life span: late children enhance female longevity," *Journals of Gerontology, Series A Biological Sciences and Medical Sciences*, vol. 57, no. 5, pp. 202–206, 2002.

[70] Y. Zeng and J. W. Vaupel, "Association of late childbearing with healthy longevity among the oldest-old in China," *Population Studies*, vol. 58, no. 1, pp. 37–53, 2004.

[71] K. R. Smith, A. Gagnon, R. M. Cawthon, G. P. Mineau, R. Mazan, and B. Desjardins, "Familial aggregation of survival and late female reproduction," *Journals of Gerontology, Series A Biological Sciences and Medical Sciences*, vol. 64, no. 7, pp. 740–744, 2009.

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Research Article

Psychosocial Factors Associated with Longevity in the United States: Age Differences between the Old and Oldest-Old in the Health and Retirement Study

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Recent growth in the number of adults surviving to advanced ages raises questions about the quality of life associated with increased longevity. Psychosocial factors have received relatively little attention in research on quality of life among the oldest-old. This study uses nationally representative data on older US adults to examine how social relationships, feelings of loneliness, and satisfaction with life and the aging experience differ between the oldest-old, those who have survived to age 90 or older, and older adults in their 70s. We find that the oldest-old are able to maintain social relationships with family and friends and receive more social support than younger elderly adults. Yet, the oldest-old are more likely to feel lonely due to their greater rates of widowhood. Satisfaction with life was higher among the oldest-old, but the oldest-old had more negative perceptions of the aging experience. Psychosocial dimensions of longevity should be considered in research on quality of life among the oldest-old.

1. Introduction

As a result of recent demographic changes, such as declining mortality rates among older adults, reaching advanced old age has become an increasingly common experience in the US and around the world [1–3]. The rapid growth in the number of very old people raises questions about the quality of additional years of life lived by those achieving exceptional longevity. The World Health Organization has declared that "increased longevity without quality of life is an empty prize" [4]. Although much of the research thus far on longevity and quality of life has emphasized health and functioning, we argue that a more comprehensive understanding of quality of life at advanced old ages could be achieved by additionally considering psychosocial well-being.

Traditional approaches to studying quality of life among those who reach advanced old age are rooted in a biomedical paradigm that emphasizes the avoidance of disease and cognitive and physical declines [5]. There is concern, for instance, that those who survive to very old age spend their remaining years in a state of poor health and functioning and, therefore, have a poor quality of life [6, 7]. Prior research on long-lived individuals indeed confirms that among individuals who survive to exceptional old age there is a high prevalence of disease and disability as well as impaired cognitive performance [8–12].

However, aging is a multidimensional concept, and psychosocial factors that assess psychological and social well-being should be included in conceptual frameworks used to understand the aging process [13]. It is possible, for instance, that those experiencing health and functioning declines that accompany the aging process are still able to maintain a high quality of life with respect to social and psychological well-being. Thus, while the predominant conceptual framework for understanding the aging process places a strong emphasis on health and functioning, a more inclusive conceptualization of quality of life in advanced ages should also focus on psychosocial well-being.

Gerontologists have been interested in the psychosocial dimensions of quality of life in older adults for some time. In

his conceptualization of the "good life", Lawton argued that psychosocial indicators of well-being are inextricably linked with health and functioning in determining quality of life among older adults [14]. Psychosocial dimensions of well-being, such as social relationships, feelings of loneliness, and satisfaction with life, are important factors to consider in the measurement of quality of life among older adults [14, 15]. Prior research has established that social relationships, feelings of loneliness, and a sense of life satisfaction are not only predictive of longevity [16–18], but are also important for determining health-related quality of life among older adults [19–21]. Social connectedness and feeling positive about one's life may be particularly important for quality of life in very old age when health-related quality of life has declined. Yet, there has been very little empirical research on the psychosocial factors associated with longevity.

Because there are few sources of data on the oldest-old, and even less research on the psychosocial characteristics of the longest lived, our understanding of longevity and quality of life is limited. A few studies have compared the psychosocial well-being of US centenarians to younger elderly adults, finding that centenarians have fewer social relationships and less frequent social contact than their younger elderly counterparts [22] and that loneliness is more prevalent in extreme old age [23]. These studies of centenarians and older adults living in the state of Georgia indicate that long-lived individuals living in this southeastern state in the US have comparatively worse quality of life than younger old adults. In contrast, a study comparing the psychosocial well-being of Italian centenarians to younger elderly Italians found that centenarians reported having more social support and greater life satisfaction [24].

Studies of social and psychological factors among older US adults indicate that quality of life is worse among the oldest-old compared to the younger elderly. Age-related differences in social characteristics and health status may explain why the oldest-old have worse quality of life. For instance, as the oldest-old outlive family and friends they experience a contraction in their network of social relationships and, consequently, may have fewer social contacts and feel more socially isolated than younger adults. Furthermore, higher rates of disease and disability among the oldest-old [8–12] may impede their ability to live independent, socially engaged lives, which could result in fewer social interactions, greater feelings of loneliness, and less satisfaction with their lives.

The present study uses a nationally representative sample of older US adults to examine age differences in psychosocial characteristics between the old, those in their 70s, and oldest-old, those aged 90 or older. This study has two aims. The first is to determine whether social relationships, feelings of loneliness, satisfaction with life, and perceptions of the aging experience differ for those who have achieved exceptional longevity, compared with younger elderly adults. Based on findings from prior research on psychosocial factors associated with longevity, we expect that the oldest-old adults will be less socially connected but will report more social support than their younger counterparts. In addition, the oldest-old will express greater life satisfaction compared to younger elderly adults, though we expect the oldest-old will be less satisfied with the aging process. The second aim is to identify social and health characteristics that contribute to age differences in social and psychological well-being. We expect that age differences in feelings of loneliness, satisfaction with life, and perceptions of the aging experience are largely the result of age-related changes in social factors and health status.

2. Methods

2.1. Data. This study uses data from the Health and Retirement Study (HRS), a nationally representative ongoing survey of US adults over the age of 50. The HRS is designed to monitor changes in physical, functional, and cognitive health associated with aging. In 2006, the HRS also began collecting information on psychosocial characteristics of older adults. A random one-half of HRS households were selected to complete a self-administered psychosocial questionnaire in 2006, with the other half of the sample selected for participation in 2008.

The psychosocial questionnaire was administered to 8,568 respondents in 2006 and 7,500 respondents in 2008 who were living in the community. Because the primary aim of this study is to characterize the psychosocial factors of longevity, we focus on the oldest-old respondents who were between 90 and 104 years of age at the time of the interview. Survival to age 90 and beyond is relatively uncommon in older cohorts. Among those born in 1900, for instance, only about 5% of men and 14% of women survived to age 90 according to cohort life tables [1]. As a comparison, we also examine psychosocial characteristics of respondents who have achieved average survival and were age 70–79 at the time of the interview. The analytic sample comprised 4,187 older adults, aged 70–79, and 281 oldest-old adults, aged 90–104, who completed the psychosocial questionnaire.

2.2. Measures. Sociodemographic measures used to characterize the sample include gender, race/ethnicity, educational attainment, marital status, and living arrangements.

Health status is assessed with the number of comorbidities and the number of limitations in activities of daily living (ADLs). Number of comorbidities counts the number of doctor diagnosed diseases and chronic conditions reported by respondents or their proxies, including (1) high blood pressure or hypertension, (2) diabetes or high blood sugar, (3) cancer or a malignant tumor of any kind except skin cancer, (4) chronic lung disease such as chronic bronchitis or emphysema (excluding asthma), (5) heart attack, coronary heart disease, angina, congestive heart failure, or other heart problem, and (6) stroke. ADL limitations were assessed with a count of the number of six major life activities the respondent had difficulty performing, including walking across a room, dressing, bathing, eating, getting in and out of bed, and using the toilet.

We assess social relationships and quality of life among older and oldest-old adults with measures that reflect the extent and quality of respondents' relationships with others,

their feelings of loneliness, and their satisfaction with life in general and the aging experience in particular.

2.2.1. Social Relationships. We examine measures that capture the extent of social relationships, including frequency of contact with family and friends, the number of close relationships, and levels of social support and strain.

Frequency of social contact includes contact with nonhousehold children, nonhousehold family members, and friends. Questions about these forms of social contact were only asked in the 2008 survey. We created separate scales of frequency of contact with children, family, and friends. The scales were created by averaging responses to the following two questions: "On average, how often do you meet up with any of your children/family members/friends, not counting any who live with you?" and "On average, how often do you speak on the phone with any of your children/family members/friends, not counting any who live with you?" The response options were less than once a year or never = 1, once or twice a year = 2, every few months = 3, once or twice a month = 4, once or twice a week = 5, and three or more times a week = 6. Scale scores ranged from 1 to 6, with higher scores indicating more contact. The alpha coefficient of reliability for each of the scales was .66 for contact with children, .75 for contact with family, and .65 for contact with friends.

We also included separate measures for the number of close relationships with children, family members, and friends. Respondents were asked about their children, family members, and friends, "How many. . .would you say you have a close relationship with?" We coded responses so the number of close relationships ranged from 0 to 10 or more.

We assessed the quality of social relationships with measures of social support and relationship strain from spouses/partners, children, other family members, and friends. Social support was measured with the following three items: (1) How much do they really understand the way you feel about things? (2) How much can you rely on them if you have a serious problem? (3) How much can you open up to them if you need to talk about your worries? Relationship strain was measured with the following four items: (1) How often do they make too many demands on you? (2) How much do they criticize you? (3) How much do they let you down when you are counting on them? (4) How much do they get on your nerves? Response categories were not at all = 1, a little = 2, some = 3, and a lot = 4. Responses to items from each scale were averaged to create a total score, with higher scores indicating more social support or strain. The alpha coefficients for reliability of the social support scales were .81 for spouse/partner, .83 for children, .86 for family, and .84 for friends. The reliability coefficients of the relationship strain scales were .79 for spouse/partner, .79 for children, .79 for family, and .76 for friends.

2.2.2. Loneliness. We used a three-item scale that was developed to assess loneliness in large-scale surveys and that has been shown to have discriminant and convergent validity and to be related to objective measures of social isolation [25]. Respondents were asked about the frequency with which they felt they lacked companionship, felt left out, and felt isolated. Response options were often = 1, some of the time = 2, and hardly ever or never = 3. Items were reverse scored to make all items measure more frequent feelings of loneliness. The items were then averaged to create a total score ranging from 1 to 3, with higher scores indicating more feelings of loneliness. The alpha coefficient of reliability for the scale is .81.

2.2.3. Life Satisfaction. We used Diener's 5-item measure of life satisfaction, an established measure of subjective well-being [26, 27]. Respondents were asked how much they agreed or disagreed with the following five statements: (1) in most ways my life is close to ideal; (2) the conditions of my life are excellent; (3) I am satisfied with my life; (4) I have gotten the important things I want in life; and (5) if I could live my life again, I would change almost nothing. Response options were strongly disagree = 1, somewhat disagree = 2, slightly disagree = 3, slightly agree = 4, somewhat agree = 5, and strongly agree = 6. The items were averaged to create a total score ranging from 1 to 6, with higher scores indicating greater life satisfaction. The alpha coefficient for the reliability of the scale is .87.

2.2.4. Perception of Aging Experience. We measured attitudes toward the aging experience with eight items, five of which are based on the "Attitude Toward Own Aging" subscale of the Philadelphia Geriatric Center Morale Scale [28]. Questions about perceptions of the aging experience were only asked in the 2008 survey. Respondents were asked how much they agreed or disagreed with the following eight statements: (1) things keep getting worse as I get older; (2) I have as much pep as I did last year; (3) the older I get, the more useless I feel; (4) I am as happy now as I was when I was younger; (5) as I get older, things are better than I thought they would be; (6) so far, I am satisfied with the way I am aging; (7) the older I get, the more I have had to stop doing things that I like; and (8) getting older has brought with it many things that I do not like. Response options were strongly disagree = 1, somewhat disagree = 2, slightly disagree = 3, strongly agree = 4, somewhat agree = 5, and slightly agree = 6. The first, third, seventh, and eighth items were reverse scored to make all items measure positive perceptions of aging. The items were then averaged to create a total score ranging from 1 to 6, with higher scores indicating more positive perceptions of aging. The alpha coefficient for the reliability of the scale is .82.

2.3. Statistical Analyses. We first examined differences in sociodemographic characteristics, living arrangements, and health status by age group. We then examined differences in social relationships between those aged 70–79 and those aged 90–104. We also examined age group differences in loneliness, life satisfaction, and perceptions of aging and show differences in both the scale items and scale means. We conducted tests of differences using the Wald chi square statistic for categorical variables and t tests from bivariate ordinary least squares (OLS) regression for interval variables

(e.g., scale scores). Finally, we used OLS regression to determine if social relationships, health status, and psychosocial factors accounted for age group differences in loneliness, life satisfaction, and perceptions of the aging experience.

Analyses were performed using Stata software version 11 [29]. Due to the complex survey design of the HRS, we used Stata's survey prefix commands (SVY), which fit statistical models that account for the complex survey design of the HRS. All analyses, therefore, are adjusted to account for household sampling and are weighted using baseline sample weights that correct for differential probability of household selection and nonresponse and that make adjustment to the 1990 sex and age distribution of the U.S.

3. Results

3.1. Sample Characteristics. The demographic characteristics and health status of the sample by age group are presented in Table 1. Women made up the majority of the sample with more women represented among the oldest-old. The sample was mostly white and the overall racial/ethnic distribution was similar for both age groups. Among adults aged 70–79, only 22% had less than a high school education, compared to 33% of adults aged 90–104. About 61% of adults aged 70–79 were married at the time of the survey, while nearly 80% of those aged 90–104 were widowed. The high rate of widowhood among the oldest-old partly accounts for their higher rates of living alone. Both age groups had similar numbers of comorbidities, but the oldest-old had more ADL limitations.

Table 1: Sample characteristics by age group.

	Age 70–79 *N* (%)	Age 90–104 *N* (%)	*P* value
Female	2,374 (56.3)	200 (69.6)	<.000
Male	1,813 (43.7)	81 (30.4)	
Race/ethnicity			
White	3,332 (85.3)	230 (86.5)	n.s.
Black	510 (7.9)	29 (6.8)	
Hispanic	272 (4.9)	19 (5.9)	
Other	73 (1.9)	3 (0.8)	
Completed education			
Less than high school	929 (21.9)	92 (32.6)	<.000
High school	1,662 (39.4)	96 (33.6)	
College or more	1,593 (38.7)	93 (33.8)	
Marital Status			
Married/partnered	2,698 (61.1)	49 (16.5)	<.000
Divorced/separated	387 (10.2)	7 (2.6)	
Widowed	994 (25.7)	222 (79.9)	
Never married	108 (2.9)	3 (0.9)	
Lives alone	1,053 (27.0)	170 (59.1)	<.000
Comorbidities			
0	680 (16.7)	36 (11.5)	n.s.
1	1,400 (33.2)	94 (32.4)	
2	1,211 (28.6)	95 (33.1)	
3	623 (14.7)	41 (17.4)	
4+	273 (6.9)	15 (5.7)	
ADL limitations			
0	3,478 (81.9)	149 (49.7)	<.000
1	364 (8.9)	58 (21.1)	
2	160 (4.0)	32 (11.3)	
3	88 (2.3)	20 (7.9)	
4+	97 (2.9)	22 (10.0)	
Total *N*	$N = 4,187$	$N = 281$	

Note: Figures shown are weighted sample sizes with percentages in parentheses. *P* values denoting statistical significance of age differences were obtained using Wald chi square tests.
ADLs: activities of daily limitations.

3.2. Social Relationships. Table 2 shows mean values for measures of social relationships by age group. Mean scores on the frequency of social contact measures indicate that respondents had contact with children who did not live with them on a weekly basis and had contact with other family members with whom they did not live about once or twice a month, with the oldest-old reporting more contact with children and family. Both age groups reported being in contact with friends about once or twice a month. The oldest-old reported having fewer close relationships with children but more close relationships with family members. Both age groups reported having about four close friends.

In general, respondents reported receiving social support some of the time or a lot of the time from spouses/partners, children, family members, and friends. Those aged 90+ reported getting less social support from spouses and partners compared to the younger age group. However, compared to those aged 70–79, the oldest-old characterized their relationships with their children and other family members as having more social support and less strain. Those aged 70–79 reported having more supportive relationships with friends compared to the oldest-old.

3.3. Loneliness. In Table 3, we examined age differences in subjective assessments of one's sense of loneliness. About 17% of adults aged 90+ reported often feeling they lacked companionship, compared to only 10% of adults aged 70–79. The oldest-old also more often felt they were isolated compared to their younger counterparts. Age-related differences in these markers of loneliness resulted in higher scores on the loneliness scale among those aged 90+.

3.4. Life Satisfaction. Table 4 shows age differences in life satisfaction. We combined the somewhat and slightly agree categories and the somewhat and slightly disagree categories to create a more parsimonious comparison of item responses. There were no age differences in the amount of agreement with any of the statements about life satisfaction, except the statement that life is close to ideal. About 21% of those aged 70–79 strongly agreed with this statement compared to 17% of those aged 90+. Both age groups reported similar levels of overall life satisfaction.

Table 2: Age differences in social relationships between the old (age 70–79) and the oldest-old (age 90–104), HRS 2006/2008.

	Age 70–79			Age 90–104			P
	N	Mean	(s.d.)	N	Mean	(s.d.)	
Social contact							
Children	1933	4.54	(1.28)	108	4.93	(1.20)	<.001
Family	1931	3.80	(1.45)	114	4.10	(1.60)	.048
Friends	1939	4.36	(1.25)	113	4.38	(1.19)	n.s.
Close relationships							
Children	3854	2.85	(2.11)	226	2.45	(2.22)	.009
Family	3789	3.43	(3.17)	226	3.98	(3.50)	.020
Friends	3761	3.79	(3.29)	233	4.07	(3.71)	n.s.
Relationship quality							
Spouse/partner							
Social support	2710	3.50	(0.70)	65	3.31	(0.75)	.048
Strain	2735	1.95	(0.77)	69	1.82	(0.82)	n.s.
Children							
Social support	3813	3.35	(0.77)	225	3.54	(0.66)	<.001
Strain	3840	1.61	(0.66)	230	1.50	(0.68)	.025
Family							
Social support	3834	2.91	(0.98)	225	3.10	(0.88)	<.001
Strain	3847	1.48	(0.62)	228	1.36	(0.54)	<.001
Friends							
Social support	3894	3.03	(0.85)	238	2.95	(0.89)	n.s.
Strain	3819	1.37	(0.50)	243	1.31	(0.43)	.031

Note: Figures shown are weighted sample sizes and means with standard deviation in parentheses. P values denoting statistical significance of age differences were obtained using ANOVA F tests.
Social contact with children, family, and friends was measured in 2008 only.

Table 3: Differences in loneliness between the old (age 70–79) and the oldest-old (age 90–104), HRS 2006/2008.

	N	Often	Sometimes	Hardly ever/Never	P
Lack companionship					
Age 70–79	4,109	9.9	34.0	56.1	<.000
Age 90–104	269	17.1	39.7	43.2	
Feel left out					
Age 70–79	4,095	6.1	33.3	60.6	n.s.
Age 90–104	265	7.4	37.4	55.2	
Feel isolated					
Age 70–79	4,082	6.3	26.1	67.7	.085
Age 90–104	265	10.3	26.8	62.9	
Loneliness scale		Mean (s.d.)			
Age 70–79	4,123	1.46 (0.61)			<.001
Age 90–104	274	1.58 (0.61)			

Note: Figures shown are weighted sample sizes and percentages and weighted scale means with standard deviation in parentheses. P values denoting statistical significance of age differences were obtained using Wald chi square tests for the items and t tests from bivariate OLS regression for the scale mean.

3.5. Aging Experience. We also examined age differences in overall perceptions of and satisfaction with the aging experience. Table 5 shows the age-specific distributions of agreement/disagreement to statements about the aging experience. We again combined the somewhat and slightly agree categories and the somewhat and slightly disagree categories to create a more parsimonious comparison of item responses. Compared to those aged 70–79, fewer oldest-old adults agreed with the positive statements about the aging experience, but more of the oldest-old agreed with negative statements about the aging experience. For instance, 19% of those aged 90+ strongly agreed with the statement "The older I get the more useless I feel" compared to only 6% of adults aged 70–79. In addition, 40% of the oldest-old strongly agreed that they have had to stop doing the things they like to do as they got older, while only 17% of those aged 70–79 felt this way. However, mean differences in the aging experience scale indicate that in general, the oldest-old adults had a less positive overall perception of their aging experience.

3.6. Multivariate Analysis. Table 6 presents coefficients from the OLS regression models for loneliness (Panel A), life satisfaction (Panel B), and perceptions of the aging experience (Panel C). Panel A shows the results for loneliness. The first model includes dichotomous indicators for age group, gender and race/ethnicity, and a continuous measure of years of education. Loneliness was higher among the oldest-old and women and declined with increasing education.

The second model adds marital and living status as indicators of social contact. Although we also examined other indicators of social contact, marital status had consistently

Table 4: Differences in life satisfaction between the old (age 70–79) and the oldest-old (age 90–104), HRS 2006/2008.

	N	Strongly agree	Somewhat/ slightly agree	Somewhat/ slightly disagree	Strongly disagree	*P*
Life is close to ideal						
Age 70–79	3,780	20.9	55.4	17.0	6.7	.063
Age 90–104	240	16.8	56.0	20.6	6.6	
Conditions of life are excellent						
Age 70–79	3,866	20.9	52.5	19.3	7.3	n.s.
Age 90–104	243	20.4	52.6	19.6	7.4	
Satisfied with life						
Age 70–79	3,974	36.3	47.4	11.7	4.6	n.s.
Age 90–104	253	34.6	47.3	13.0	5.2	
Gotten important things in life						
Age 70–79	3,967	33.1	52.5	10.9	3.6	n.s.
Age 90–104	260	35.5	51.0	9.4	4.2	
Would not change life						
Age 70–79	3,926	19.5	46.3	22.5	11.6	n.s.
Age 90–104	251	25.9	42.4	21.4	10.3	
Life satisfaction scale			Mean (s.d.)			
Age 70–79	4,095		4.40 (1.45)			n.s.
Age 90–104	269		4.39 (1.27)			

Note: Figures shown are weighted sample sizes and percentages and weighted scale means with standard deviation in parentheses. *P* values denoting statistical significance of age differences were obtained using Wald chi square tests for the items and *t* tests from bivariate OLS regression for the scale mean.

stronger and more significant associations with the outcome measures (results not shown). Not being married was associated with more frequent feelings of loneliness, even with an adjustment for living alone. After accounting for differences in marital status, the age difference in loneliness was reduced and no longer statistically significant. Nearly 80% of the oldest old were widowed, which seems to explain why the oldest-old report more frequent feelings of loneliness compared to those aged 70–79. This explanation seems particularly plausible considering that one of the items in the loneliness scale asks about lack of companionship which tends to be provided by a spouse or partner.

After adjusting for comorbidities and ADL limitations in the next model, the coefficient for the age difference became negative and was marginally significant ($-.07$, $P < .10$). The number of comorbidities and ADL limitations was associated with greater feelings of loneliness. The number of ADL limitations increases with age, suggesting that feelings of loneliness may be less frequent among the oldest-old who have similar ADL profiles as those aged 70–79.

Panel B presents the results for the OLS regression model for life satisfaction. The first column shows that life satisfaction was lower among Blacks and that satisfaction increased with increasing education. However, there were no age differences in life satisfaction.

The second model adds social contact indicators and shows that not being married was associated with less life satisfaction. Moreover, after adjusting for social contact, the coefficient for the age differences increased in magnitude and became statistically significant (.19, $P < .05$). This suggests that the oldest-old may have greater life satisfaction than younger elderly adults when differences in marriage and widowhood are accounted for. The age difference increased further in the next model, which adjusts for differences in comorbidities and ADL limitations, and shows that greater levels of disease and functional limitation were associated with less life satisfaction. Taken together, these two models suggest that differences in life satisfaction between the old and oldest-old are underestimated when age-related differences in marital and health status are not considered.

In the final model, we also adjusted for loneliness. Greater feelings of loneliness were associated with less life satisfaction. The coefficients for comorbidities and ADL limitations were reduced after including loneliness, which indicates that the negative associations between health status and life satisfaction may operate partially through increased feelings of loneliness among those who experience poor health and activity limitations.

Panel C shows the results for perceptions of the aging experience. The first column shows that oldest-old adults had more negative perceptions of their aging experience and that having more years of education was associated with more positive perceptions of the aging experience. The second column shows that those who were not married had more negative perceptions of aging, but that accounting for differences in marital status did not explain the age difference.

The third model also included comorbidities and ADL limitations and shows that individuals who had worse physical health and functioning reported more negative perceptions of their aging experience. After accounting for differences in health status, the coefficient for the age difference was reduced by about 50%. The final model shows that

Table 5: Differences in perceptions of the aging experience between the young old (age 70–79) and the very old (age 90–104), HRS 2008.

	N	Strongly agree	Somewhat/ slightly agree	Somewhat/ slightly disagree	Strongly disagree	*P*
Things get worse as I get older						
Age 70–79	2,054	9.3	49.3	24.7	16.6	.019
Age 90–104	135	12.1	56.6	21.4	9.9	
I have as much pep as last year						
Age 70–79	2,061	16.2	39.1	34.9	9.7	<.000
Age 90–104	137	10.7	29.4	40.8	19.2	
The older I get the more useless I feel						
Age 70–79	2,050	5.9	25.8	29.9	38.4	<.000
Age 90–104	134	18.5	34.5	28.1	18.9	
I am as happy now as I was when I was younger						
Age 70–79	2,062	24.8	36.9	26.0	12.3	<.000
Age 90–104	135	10.2	34.2	38.4	17.2	
As I get older things are better than I thought they would be						
Age 70–79	2,057	22.2	48.3	22.4	7.2	n.s.
Age 90–104	135	19.4	44.2	23.1	13.4	
I am satisfied with the way I am aging						
Age 70–79	2,068	31.2	49.4	13.7	5.8	n.s.
Age 90–104	137	38.2	45.0	11.2	5.6	
The older I get, the more I have had to stop doing things that I liked						
Age 70–79	2,069	17.2	49.0	21.7	12.2	<.000
Age 90–104	135	40.4	47.4	9.1	3.1	
Getting older has brought with it many things that I do not like						
Age 70–79	2,069	17.3	52.4	19.9	10.5	<.000
Age 90–104	136	31.2	54.7	7.9	6.2	
Aging experience scale			Mean (s.d.)			
Age 70–79	2,073		3.81 (1.25)			<.000
Age 90–104	137		3.30 (0.94)			

Note: Figures shown are weighted sample sizes and percentages and weighted scale means with standard deviation in parentheses. *P* values denoting statistical significance of age differences were obtained using Wald chi square tests for the items and *t* tests from bivariate OLS regression for the scale mean. Perceptions of the aging experience were measured in 2008 only.

feelings of loneliness were associated with more negative perceptions of aging, but that loneliness did not account for age differences in perceptions.

4. Discussion

The purpose of this study was to gain insight into the social and psychological well-being of the oldest-old US adults, using a younger group of older adults as a comparison. We add to previous research that has used traditional biomedical-based models for understanding aging and longevity by examining psychosocial factors associated with longevity.

We found that the oldest-old had frequent social contact with family and friends and relatively high levels of social support. The high amount of social contact reported by the oldest-old in our study is consistent with other samples of oldest-old from the US and England [30, 31]. However, our finding that the oldest-old report having more contact with children and family than younger elderly adults is contrary to what has been reported in a prior study of US oldest-old adults [32]. Differences between our results and results from previous research may be due to differences in the measure but may also be due to differences in the study sample; ours was a national study of US adults aged 90 and older and the previous study focused on centenarians living in Georgia.

Compared to younger elderly adults, the oldest-old in our study reported receiving more positive support from their children and family. This finding is consistent with previous research [24]. The oldest-old also reported less strained relationships with their children. Overall, our results

Table 6: OLS regression models for loneliness, life satisfaction, and perceptions of the aging experience.

	Base model Coeff. (SE)	+ Social contact Coeff. (SE)	+ ADLs Coeff. (SE)	+ Loneliness Coeff. (SE)
Panel A. Model for Loneliness				
Age 90–104	.09 (.04)*	−.02 (.04)	−.07 (.04)$^{+}$	
Female	.09 (.02)***	.01 (.02)	.01 (.02)	
Black[a]	.06 (.03)$^{+}$	.01 (.03)	−.01 (.03)	
Hispanic[a]	.05 (.05)	.04 (.05)	.04 (.05)	
Other[b]	−.07 (.06)	−.07 (.06)	−.06 (.06)	
Education, yrs	−.02 (.00)***	−.02 (.00)***	−.01 (.00)***	
Not married		.24 (.03)***	.22 (.03)***	
Lives alone		.02 (.03)	.04 (.03)	
Comorbidities			.03 (.01)***	
1 ADL limitation[b]			.15 (.03)***	
2+ ADL limitations[b]			.20 (.03)***	
Constant	1.68 (.05)***	1.59 (.04)***	1.46 (.05)***	
R squared	0.030	0.075	0.100	
Panel B. Model for Life Satisfaction				
Age 90–104	.03 (.08)	.19 (.08)*	.33 (.09)***	.26 (.08)***
Female	−.05 (.04)	.08 (.04)*	.06 (.04)	.06 (.04)$^{+}$
Black[a]	−.23 (.07)***	−.17 (.07)*	−.11 (.07)	−.11 (.07)
Hispanic[a]	.16 (.10)	.17 (.10)$^{+}$	.16 (.10)	.13 (.09)
Other[b]	.34 (.12)**	.34 (.12)**	.33 (.11)**	.26 (.11)*
Education, yrs	.04 (.01)***	.04 (.01)***	.03 (.01)***	.02 (.01)*
Not married		−.38 (.07)***	−.30 (.07)***	−.12 (.07)$^{+}$
Lives alone		−.04 (.08)	−.10 (.07)	−.07 (.07)
Comorbidities			−.12 (.02)***	−.09 (.02)***
1 ADL limitation[b]			−.39 (.07)***	−.28 (.07)***
2+ ADL limitations[b]			−.52 (.08)***	−.38 (.08)***
Loneliness				−.79 (.04)***
Constant	3.88 (.11)***	4.03 (.11)***	4.45 (.11)***	5.61 (.12)***
R squared	0.017	0.040	0.081	0.193
Panel C. Model for Aging Experience				
Age 90–104	−.47 (.09)***	−.42 (.09)***	−.21 (.09)*	−.24 (.08)**
Female	.02 (.05)	.06 (.05)	.04 (.05)	.02 (.04)
Black[a]	.04 (.08)	.08 (.09)	.16 (.07)*	.15 (.07)*
Hispanic[a]	.13 (.11)	.14 (.11)	.09 (.11)	.16 (.10)
Other[b]	−.11 (.18)	−.11 (.18)	−.11 (.18)	−.13 (.17)
Education, yrs	.06 (.01)***	.06 (.01)***	.04 (.01)***	.03 (.01)***
Not married		−.18 (.08)*	−.01 (.08)	.12 (.07)
Lives alone		.08 (.08)	−.06 (.08)	−.04 (.07)
Comorbidities			−.17 (.02)***	−.49 (.08)***
1 ADL limitation[b]			−.62 (.08)***	−.66 (.08)***
2+ ADL limitations[b]			−.84 (.08)***	−.16 (.02)***
Loneliness				−.68 (.04)***
Constant	3.05 (.13)***	3.10 (.13)***	3.68 (.13)***	4.73 (.14)***
R squared	0.042	0.045	0.175	0.282

Notes: Numbers are coefficients with standard errors in parentheses.
[a]Reference group is white; [b]Reference group is no ADLs.
***$P < .001$, **$P < .01$, *$P < .05$, $^{+}P < .10$ (two-tailed test).

indicate that social relationships remain intact in the oldest-old. Research shows that social relationships are generally beneficial for health and well-being and thus, maintaining relationships with family and friends may be a key component of quality of life in advanced old age [16].

We also found evidence that perceptions of quality of life are lower among the oldest-old. Consistent with prior work [23], we found that the oldest-old felt lonely more often than younger elderly adults. By looking at multiple indicators of loneliness, we additionally found that age differences in loneliness are particularly pronounced with respect to lacking companionship and feeling isolated. However, the results indicate that the oldest-old have greater feelings of loneliness, because they do not have a spouse or partner to provide companionship, and because they have health limitations that may limit their social contact.

In accordance with prior research, we found very old adults were more satisfied with life than younger elderly adults [24] but only after accounting for marital status, comorbidities and ADL limitations, and loneliness. The age difference in life satisfaction was underestimated when social and health factors were not considered. This suggests that the social and health conditions that typically accompany old age, for instance losing a spouse and having difficulty with daily activities, decrease life satisfaction among the oldest-old, but that in the absence of these factors, long-lived individuals are more satisfied with their lives than younger elderly adults.

We also found that the oldest-old had more negative perceptions of their aging experience. For instance, 69% of the oldest-old agreed that things had gotten worse as they aged, 53% agreed that they felt more useless as they got older, and 88% agreed that they had to stop doing things they like to do. The higher burden of activity limitations among the oldest-old partially accounted for their more negative perceptions of the aging experience. Declines in physical functioning can prevent older adults from doing the things they want to do and may be a primary reason they feel useless and that their lives have gotten worse.

Even though the oldest-old were less satisfied with the aging experience, they reported greater life satisfaction. This may reflect the tendency of the oldest-old to reconstitute how they view themselves and their experiences to be more consistent with the realities of their lives [33]. So, even though the oldest-old express dissatisfaction with particular aspects of the aging experience, they may still believe that their overall life situation is as good as that of younger elderly adults.

Taken together, the results show that the oldest-old have similar, if not more, social contact than younger old adults but that the oldest-old still feel more lonely and socially disconnected, which is most likely to due to decreased social contact and interaction as a result of their higher rates of widowhood, disease, and disability. The results also show that the oldest-old have greater overall life satisfaction but more negative perceptions of the aging experience.

This study is the first to examine social and psychological well-being associated with aging and longevity in a national sample of US adults. We use several measures of psychosocial well-being to provide a comprehensive understanding of quality of life at advanced old age. The findings from this study make important contributions to the growing body of research on longevity and quality of life. In particular, this study highlights the importance of considering psychosocial factors associated with longevity in addition to other social and health factors that characterize the aging experience.

This study has some limitations. First, although we used a representative sample of US adults, information on psychosocial characteristics was not obtained from older adults living in nursing homes. Nursing home residents are more likely to have functional and cognitive impairments, and this sample may, therefore, have better health and functioning than the general population of adults aged 70–79 and especially adults aged 90 and older. Thus, the study results cannot be generalized beyond the community-dwelling older adult population as it is possible that quality of life in long-lived nursing home residents differs from that of community-dwelling oldest-old [22].

Another limitation is that the age differences reported in this study may be confounded with cohort differences. Although our results may indicate that psychosocial factors change with age, it is also possible that the age differences found in our study additionally or, instead, reflect cohort differences in how psychosocial factors relate to age.

5. Conclusions

Our findings suggest that the oldest-old are able to maintain quality of life with respect to social relationships and that while their aging experience to that point has been difficult, they are as satisfied with their lives as younger elderly adults. Our study contributes to the growing research on quality of life among the very old.

A consideration of psychosocial factors associated with longevity is essential not only for predicting longevity, but also for understanding quality of life among the longest lived individuals. Complex associations exist between health and functioning and psychosocial well-being [34], and the joint influence of these factors on longevity and quality of life should be considered in future longevity research [35].

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References

[1] F. C. Bell and M. L. Miller, *Life Tables for the United States Social Security Area, 1900–2100*, Social Security Administration, Office of the Chief Actuary, 2002.

[2] J. Oeppen and J. W. Vaupel, "Broken limits to life expectancy," *Science*, vol. 296, no. 5570, pp. 1029–1031, 2002.

[3] J. W. Vaupel, J. R. Carey, K. Christensen et al., "Biodemographic trajectories of longevity," *Science*, vol. 280, no. 5365, pp. 855–860, 1998.

[4] World Health Organization, *The World Health Report: Report of the Director-General*, World Health Organization, Geneva, Switzerland, 1997, http://www.who.int/whr/1997/en/whr97_djmessage_en.pdf/.
[5] J. W. Rowe and R. L. Kahn, "Human aging: usual and successful," *Science*, vol. 237, no. 4811, pp. 143–149, 1987.
[6] E. M. Crimmins and H. Beltran-Sanchez, "Mortality and morbidity trends: is there compression of morbidity?" *The Journals of Gerontology Series B*, vol. 66B, no. 1, pp. 75–86, 2010.
[7] E. M. Crimmins, M. D. Hayward, A. Hagedorn, Y. Saito, and N. Brouard, "Change in disability-free life expectancy for americans 70 years old and older," *Demography*, vol. 46, no. 3, pp. 627–646, 2009.
[8] K. Andersen-Ranberg, M. Schroll, and B. Jeune, "Healthy centenarians do not exist, but autonomous centenarians do: a population-based study of morbidity among Danish centenarians," *Journal of the American Geriatrics Society*, vol. 49, no. 7, pp. 900–908, 2001.
[9] J. Evert, E. Lawler, H. Bogan, and T. Perls, "Morbidity profiles of centenarians: survivors, delayers, and escapers," *The Journals of Gerontology*, vol. 58, no. 3, pp. 232–237, 2003.
[10] B. Hagberg, B. Bauer Alfredson, L. W. Poon, and A. Homma, "Cognitive functioning in centenarians: a coordinated analysis of results from three countries," *The Journals of Gerontology Series B*, vol. 56, no. 3, pp. 141–151, 2001.
[11] J. S. Kim, M. H. Bramlett, L. K. Wright, and L. W. Poon, "Racial differences in health status and health behaviors of older adults," *Nursing Research*, vol. 47, no. 4, pp. 243–250, 1998.
[12] D. F. Terry, P. Sebastiani, S. L. Andersen, and T. T. Perls, "Disentangling the roles of disability and morbidity in survival to exceptional old age," *Archives of Internal Medicine*, vol. 168, no. 3, pp. 277–283, 2008.
[13] C. M. Aldwin and D. F. Gilmer, *Health, Illness, and Optimal Aging: Biological and Psychosocial Perspectives*, Sage, Thousand Oaks, Calif, USA, 2004.
[14] M. P. Lawton, "Environment and other determinants of well-being in older people," *The Gerontologist*, vol. 23, no. 4, pp. 349–357, 1983.
[15] B. L. Neugarten, R. J. Havighurst, and S. S. Tobin, "The measurement of life satisfaction," *Journal of Gerontology*, vol. 16, no. 2, pp. 134–143, 1961.
[16] J. S. House, K. R. Landis, and D. Umberson, "Social relationships and health," *Science*, vol. 241, no. 4865, pp. 540–545, 1988.
[17] H. Koivumaa-Honkanen, R. Honkanen, H. Viinamäki, K. Heikkilä, J. Kaprio, and M. Koskenvuo, "Self-reported life satisfaction and 20-year mortality in healthy finnish adults," *American Journal of Epidemiology*, vol. 152, no. 10, pp. 983–991, 2000.
[18] A. C. Patterson and G. Veenstra, "Loneliness and risk of mortality: a longitudinal investigation in Alameda County, California," *Social Science and Medicine*, vol. 71, no. 1, pp. 181–186, 2010.
[19] C. F. Mendes de Leon, D. T. Gold, T. A. Glass, L. Kaplan, and L. K. George, "Disability as a function of social networks and support in elderly African Americans and Whites: the Duke EPESE 1986–1992," *The Journals of Gerontology B*, vol. 56, no. 3, pp. S179–S190, 2001.
[20] G. McAvay and J. Rodin, "Determinants of change in perceived health in a longitudinal study of older adults," *The Journals of Gerontology*, vol. 47, no. 6, pp. P373–P384, 1992.
[21] J. T. Cacioppo, L. C. Hawkley, L. E. Crawford et al., "Loneliness and health: potential mechanisms," *Psychosomatic Medicine*, vol. 64, no. 3, pp. 407–417, 2002.
[22] G. K. Randall, P. Martin, M. McDonald, and L. W. Poon, "Social resources and longevity: findings from the Georgia centenarian study," *Gerontology*, vol. 56, no. 1, pp. 106–111, 2010.
[23] B. S. Fees, P. Martin, and L. W. Poon, "A model of loneliness in older adults," *The Journals of Gerontology B*, vol. 54, no. 4, pp. P231–P239, 1999.
[24] M. D. Buono, O. Urciuoli, and D. De Leo, "Quality of life and longevity: a study of centenarians," *Age and Ageing*, vol. 27, no. 2, pp. 207–216, 1998.
[25] M. E. Hughes, L. J. Waite, L. C. Hawkley, and J. T. Cacioppo, "A short scale for measuring loneliness in large surveys," *Research on Aging*, vol. 26, no. 6, pp. 655–672, 2004.
[26] E. Diener, R. A. Emmons, R. J. Larsen, and S. Griffin, "The satisfaction with life scale," *Journal of Personality Assessment*, vol. 49, no. 1, pp. 71–75, 1985.
[27] W. Pavot, E. Diener, C. R. Colvin, and E. Sandvik, "Further validation of the satisfaction with life scale: evidence for the cross-method convergence of well-being measures," *Journal of Personality Assessment*, vol. 57, no. 1, pp. 149–161, 1991.
[28] M. P. Lawton, "The Philadelphia geriatric center morale scale: a revision," *The Journals of Gerontology*, vol. 30, no. 1, pp. 85–89, 1975.
[29] StataCorp, *Stata Statistical Software: Release 11.0.*, Stata Press, College Station, Tex, USA, 2007.
[30] M. Bury and A. Holme, "Quality of life and social support in the very old," *Journal of Aging Studies*, vol. 4, no. 4, pp. 345–357, 1990.
[31] M. G. Kovar and R. I. Stone, "The social environment of the very old," in *The Oldest Old*, R. M. Suzman, D. P. Willis, and K. G. Manton, Eds., 1992.
[32] P. Martin, B. Hagberg, and L. W. Poon, "Predictors of loneliness in centenarians: a parallel study," *Journal of Cross-Cultural Gerontology*, vol. 12, no. 3, pp. 203–224, 1997.
[33] C. L. Johnson and B. M. Barer, *Life Beyond 85 Years*, Prometheus Books, Amherst, NY, USA, 2003.
[34] A. J. Bishop, P. Martin, and L. Poon, "Happiness and congruence in older adulthood: a structural model of life satisfaction," *Aging and Mental Health*, vol. 10, no. 5, pp. 445–453, 2006.
[35] L. W. Poon, P. Martin, A. Bishop et al., "Understanding centenarians' psychosocial dynamics and their contributions to health and quality of life," *Current Gerontology and Geriatrics Research*, vol. 2010, Article ID 680657, 13 pages, 2010.

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Review Article

The Impact of Behavioral Intervention on Obesity Mediated Declines in Mobility Function: Implications for Longevity

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A primary focus of longevity research is to identify prognostic risk factors that can be mediated by early treatment efforts. To date, much of this work has focused on understanding the biological processes that may contribute to aging process and age-related disease conditions. Although such processes are undoubtedly important, no current biological intervention aimed at increasing health and lifespan exists. Interestingly, a close relationship between mobility performance and the aging process has been documented in older adults. For example, recent studies have identified functional status, as assessed by walking speed, as a strong predictor of major health outcomes, including mortality, in older adults. This paper aims to describe the relationship between the comorbidities related to decreased health and lifespan and mobility function in obese, older adults. Concurrently, lifestyle interventions, including diet and exercise, are described as a means to improve mobility function and thereby limit the functional limitations associated with increased mortality.

1. Introduction

The term longevity can be used to refer to a "long life" for an individual or more broadly to life expectancy within a population. In recent years, scientists have devoted much attention to finding ways to increase longevity. To date, much of this work has focused on understanding the biological processes that may contribute to aging and age-related disease conditions. A number of potential biological targets have been identified during the past few decades, and a wide range of intervention approaches are currently being developed. The types of interventions considered to have the potential to affect the aging process include biochemical and genetic techniques, hormonal treatments, and behavioral approaches to reduce age-related comorbidities and thereby increase mean lifespan. Although biological approaches may have significant potential in the future, the effects of behavioral interventions on age-related conditions can be more immediately evaluated and put into practice at the present time.

To determine the effectiveness of behavioral interventions and other treatment approaches to increase longevity, a benchmark is needed that enables scientists to determine whether or not the intervention was successful. Because it is unrealistic to conduct lifespan studies in humans, a surrogate endpoint is needed to estimate the effect interventions could have on longevity. Recently, gait (i.e., walking) speed—a simple, valid, and reliable clinical test—was considered to be such an endpoint [1]. A growing body of literature has identified walking speed as a strong predictor of major health outcomes and mortality in older adults [2–4]. In a pooled analysis of nine cohort studies with follow-up periods ranging from 6 to 21 years, declines in gait speed were found to be directly associated with decreased survival [1]. Specifically, hazard ratios for survival revealed that there was a 12% risk reduction in mortality for every 0.10 meters per

second increase in gait speed. As recently noted by Cesari [5], the findings of Studenski et al.'s pooled analysis provide the statistical foundations to estimate expected survival in older adults based solely on gait speed [1]. Based on these consistent and robust findings, we argue that gait speed may represent a useful marker of overall health status and may also be a useful prognostic indicator of mean lifespan in older adults. Further, because walking places demands on multiple organ systems and demands on input from the central nervous system, it may serve as the ideal target to evaluate the efficacy of interventions aimed at improving health and increasing lifespan.

Against the backdrop of these findings on the importance of mobility function, a growing body of literature indicates that obese, older adults are at particularly high risk of functional decline, marked by reductions in mobility (i.e., walking speed). This is of significant concern because the number and proportion of obese, older adults has increased dramatically during the past two decades [6]. Recent estimates indicate that an alarming 35% of older adults are obese and another 33% are overweight, which places them at risk for obesity [6]. This paper describes the key behavioral factors contributing to the development of obesity in older adults, the major pathways through which obesity affects mobility function, and the relationship between important comorbidities related to decreased lifespan and mobility function in obese, older adults. Concurrently, lifestyle interventions, including diet and exercise, targeted toward obese older adults are described as a means to target mobility function and limit the functional limitations associated with increased mortality risk.

2. Behavioral Mechanisms of Obesity in Older Adults

Both obesity and sedentary lifestyle appear to contribute to the body composition changes (i.e., increased body fat, decreased muscle mass) that promote age-related functional decline [7–9]. As such, obese, older adults may be particularly susceptible to the adverse effects of weight gain because of the loss of muscle mass that occurs with aging (i.e., sarcopenia) [10, 11]. Loss in muscle mass by itself is associated with impairments in mobility in older adults [12]. Moreover, the combination of muscle loss and fat gain may act synergistically to lead to further reductions in mobility in older adults [13–15].

Excessive dietary intake, specifically, is a major factor influencing the overall health and body weight of older adults. Epidemiological studies indicate that per capita energy intake has increased by approximately 300 kcal per day from 1985 to 2000 [16]. Unfortunately, the trend of increasing dietary consumption also coincides with a trend to expend less energy with increasing age [17]. Physical inactivity represents another major contributor to the development of obesity and obesity-related morbidity [18]. Currently, the majority of older adults in the US do not engage in even the minimum physical activity recommendations [19]. Moreover, the Centers for Disease Control and Prevention recently reported that 40% of adults engage in no leisure-time physical activity [19]. This is of particular concern in the elderly as older adults are less active with advancing age [17].

3. Impact of Obesity on Declines in Mobility

Obesity poses several threats to mobility during aging. Most notable is the direct effect of excess body weight on movement. As body mass increases, the energy and strength required to move the body increases correspondingly. Specifically, work from the Baltimore Longitudinal Study of Aging showed that total knee generative mechanical work expenditure is higher in older adults with obesity [20]. Similarly, work by Messier et al. [21] demonstrated that the absolute peak vertical ground-reaction forces increase almost directly in proportion with body weight. These factors likely contribute to the slower preferred walking speed consistently found in obese, older adults compared to nonobese, older adults [20].

A growing body of evidence also indicates that relative skeletal muscle mass (i.e., skeletal muscle mass/body mass), compared to absolute body mass, is a strong predictor of impairments in mobility performance, such as walking and stair climbing [22, 23]. For example, Janssen and colleagues [22] found that there is an increased likelihood of functional impairment and disability in older adults if muscle loss progresses to the point where skeletal muscle mass, relative to body weight, reaches 30% below the mean for young adults. Alarmingly, 45% of men and 59% of women in this sample of over 4,500 older adults (age $\geq$ 60 years) were classified as having sarcopenia, as determined by low relative skeletal muscle mass to body weight. Sarcopenia is strongly associated with the development of functional disability and can lead to the loss of independence for afflicted individuals [22, 24]. Typically, older adults lose an average of 1-2% of their muscle mass yearly after the age of 50 [25, 26]. However, recently the loss in muscle strength has been shown to decline at a faster rate (3–5%) than previously thought [27], and it has been argued that muscle weakness is a stronger predictor of these changes than muscle mass [24, 28]. Additionally, the combination of muscle weakness and obesity increases the risk of mobility impairment greater than obesity or muscle weakness alone [29]. The reductions in both muscle size and the function through maximal strength are important factors in the loss of mobility among older adults [30, 31]. Somewhat paradoxically, obese, older adults typically have more absolute total muscle mass and strength than nonobese peers. Despite this fact, obese individuals also have higher amounts of intermuscular adipose tissue, a potential detriment to the muscle's force-generating capacity [32, 33]. Furthermore, the disparity in total muscle mass is typically not sufficient to account for the disparity in body mass, meaning that obese persons have a lower strength to body mass ratio—an important factor in mobility. This paradoxical state of low (relative) muscle mass and high body weight has come to be known as sarcopenic obesity

[34, 35]. These findings are particularly salient because both muscular strength and power, particularly in the weight-bearing lower limb muscle groups [36], have been strongly associated with habitual and maximal gait velocities, as well as other measures of physical function (i.e., chair stand time) in community-dwelling, mobility-limited, older adults [37]. Collectively, these works suggest that a low muscle/body mass ratio directly impairs movement among obese, older adults.

Musculoskeletal pain, often in the form of osteoarthritis, is also associated with obesity and directly impacts the ability of older persons to remain mobile. Complaints of pain in the leg joints related to osteoarthritis, particularly in the knee, are common in older adults. In fact, self-reported joint pain is often cited as the main factor affecting mobility in older adults [38]. Noteworthy, mobility limitations due to joint pain are compounded by obesity [39]. This is significant considering that most adults with knee osteoarthritis have a body mass in the overweight or obese range (body mass index (BMI) $\geq$ 25 kg/m^2) [40]. Additionally, the Women's Health and Aging Study found that obesity was a distinct risk factor for substantially increasing the risk of mobility disability among individuals with chronic pain [41]. In line with this, obesity has been identified as the main preventable risk factor for the onset and progression of osteoarthritis and the associated pain by some experts [42]. Importantly, individuals with chronic pain often modify and lessen their activity by walking and engaging in less physically demanding activities. As such, the age-related degeneration and the associated pain, which is compounded with obesity, can lead to significant reductions in activity.

In addition to the direct and immediate effects that excess body weight has on movement, obesity may have an overlooked, long-term impact on mobility function through the development of comorbid disease conditions. The development of such conditions, including cardiovascular disease and diabetes, may lead to mobility impairments beyond those observed during obesity alone. In fact, accumulating evidence indicates that older adults with diabetes, obese or not, experience more severe losses of muscle mass and strength than those who are not diabetic [43]. Although the mechanisms that cause this accelerated loss of mobility among older adults who have diabetes are unclear, much of the decline may be due to exacerbated changes in body composition typically observed in advanced age and/or obesity.

Collectively, studies to date indicate that excessive body weight, metabolic disease, and sarcopenia all contribute to declines in mobility function (see Figures 1 and 2) [1]. Significantly, comorbidities associated with metabolic disease and sarcopenia are prevalent in older adults, and the health consequences associated with each condition are compounded by obesity (Figure 1). Each of these conditions is directly associated with lifestyle habits. As previously discussed, excessive caloric intake and a sedentary lifestyle promote weight gain and contribute to the progression of age-related sarcopenia and declines in mobility function. Over time, these changes can accelerate the development of mobility impairments and disability (Figure 2). Although aging is the primary risk factor for both of these conditions, healthy dietary and physical activity habits have demonstrated efficacy in attenuating the progression of these conditions. In the next section, we will highlight important research findings over the past few decades demonstrating the beneficial effects of nutritional and exercise interventions on mobility function in obese, older adults.

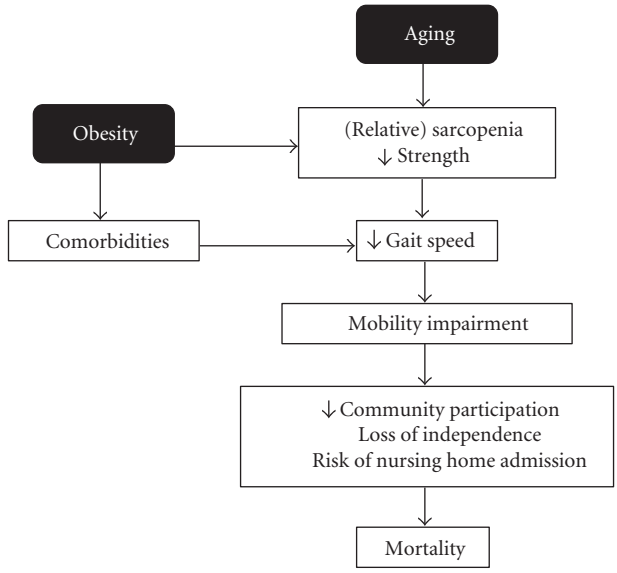

Figure 1: Conceptual model illustrating how obesity potentiates age-related declines in gait speed that might lead to mobility impairment, loss of independence, and mortality.

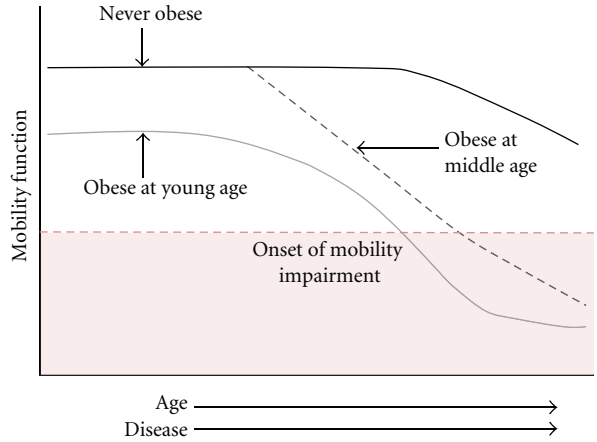

Figure 2: Theoretical illustration demonstrating the relative impact of obesity on mobility function within the context of aging and disease onset.

4. Diet and Exercise Interventions to Improve Mobility Function in Obese Older Adults

A current challenge for clinicians and researchers working with overweight, older adults is to design lifestyle-based interventions that can produce significant weight loss while minimizing the loss of fat-free mass. Concerns about weight

loss in obese, older adults relate to the documented declines in dynamic force production capability during aging, which are most notable in weight-bearing lower limb muscle groups [36, 44]. Caloric restriction is generally required to achieve significant weight loss; typically 1/3rd to 1/4th of the lost weight is fat-free mass, a significant concern for older adults. In contrast, exercise can preserve muscle mass [45, 46] and improve muscle quality [47, 48] in older adults, but does not typically produce significant weight loss by itself [49]. As such, identifying interventions designed to maximize functional improvement while limiting muscle mass loss during weight loss is a current challenge.

Weight loss through diet and exercise interventions may improve mobility through several mechanisms (see Figure 3). First, weight loss through caloric restriction could lessen the mechanical load on weak joints and muscles, thereby improving mobility. For example, Messier et al. found a direct association between weight loss and attenuation of knee joint moments and forces during walking in overweight and obese older adults with knee osteoarthritis following an 18-month, weight-loss intervention [21]. Specifically, this study found that each pound of weight lost was associated with a four times reduction in the load exerted on the knee per step, which would equate to more than 4,800 pounds less in compressive load per mile walked. Messier et al. [50] also looked at a subset of these participants dividing them into high, low, and no weight loss groups (groups lost 10.2%, 2.7%, and 0%, resp.). This study provided evidence that large weight loss in overweight and obese older adults reduces maximum knee compressive forces significantly more than small weight losses. Collectively, these results suggest that weight loss achieved through lifestyle changes can induce biomechanical improvements in knee joints loads during walking, thereby reducing or limiting mobility impairments. Below, we review the effects of lifestyle interventions involving both dietary and exercise modification on changes in mobility function in obese, older adults.

4.1. Calorie Restriction. Negative energy balance can be achieved by reducing energy intake or increasing energy expenditure. Calorie restriction (CR) has consistently been shown to extend lifespan and reduce age-related diseases in numerous species [51]. Emerging findings also suggest that CR can produce health benefits for nonobese humans (BMI range = 23.5–29.9), such as reductions in body weight and whole body fat mass [52] and beneficial effects on "biomarkers of aging" (i.e., fasting insulin level, core body temperature) in overweight individuals (BMI range = 25.0–29.9) [53]. The effects of CR on changes in mobility function in obese, older adults, however, are less well known. Two recent studies described below discuss the effect that diet-alone interventions may have on mobility and physical function in obese, older adults.

A recent study conducted by Avila and colleagues evaluated the effect of a 10-week dietary intervention compared to a diet plus resistance training (RT) intervention on physical

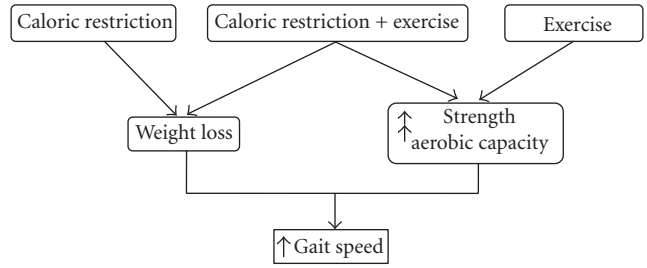

Figure 3: Model illustrating the potential effects that single- and multicomponent interventions have on gait speed.

functioning in obese, older adults [54]. Both groups significantly increased walking speed, documented by reduced time to complete the 400-meter walk test (reduction in time for diet: −36 sec; diet plus RT: −40 sec). These results suggest that diet alone may improve global mobility function in obese, older adults; however, these effects may be heightened when a dietary and exercise intervention are combined.

In an even more recent study, Villareal and colleagues conducted a clinical trial evaluating changes in physical function in obese, older adults who were randomized into a weight management program, exercise training program, weight management plus exercise training program, or control group [55]. The diet-only weight management program consisted of a balanced diet producing an energy deficit of 500 to 750 kcal per day from participants' daily energy requirements. The primary measure of physical performance was the modified Physical Performance Test which included several standardized tasks such as walking 50 feet, standing up from a chair, lifting a book, and climbing one flight of stairs. Compared to the control group, the diet-only weight management group significantly improved their score on the Physical Performance Test, increased peak oxygen consumption, lost more fat mass, and reduced the time needed to complete an obstacle course. It is important to note, however, that, while the weight management program improved physical function in obese, older adults, the combination of diet and exercise resulted in the greatest improvements in physical function. These results are discussed further below.

4.2. Exercise Training. Epidemiological data have clearly demonstrated a dose-response pattern for physical activity to reduce the risk of mobility limitations [56, 57]. Additionally, many small clinical trials have reported beneficial effects of aerobic exercise on physical capacity and gait speed [58, 59]. For example, Brown and Holloszy conducted a series of studies that clearly demonstrate that aerobic exercise alone is effective at increasing walking velocity through improvements from cardiovascular capacity in healthy lean, older adults [60]. Despite this knowledge, there has been little carryover to specific studies on obese, older adults. The studies discussed below provide information regarding general mobility adaptations to resistance and/or aerobic interventions in obese and nonobese older adults.

Resistance exercise is the best method of improving skeletal muscle performance. Accumulating evidence suggests

that resistance training increases muscle strength in older adults, thereby greatly attenuating the losses of strength, power, and muscle mass that occur during aging [61, 62]. Moreover, resistance training has robust effects on the muscle strength and overall physical function of older adults [63, 64]. Further, recent studies have found that resistance-training interventions produce clinically meaningful improvements in gait speed in older adults with mobility limitations and that increased muscular power contributed to these improvements [65].

Numerous studies provide evidence that skeletal muscles, even among frail older adults, adapt vigorously to resistance training with marked myofibre hypertrophy [62]. This is an important finding considering that reductions in muscle power and strength are, at least in part, related to preferential type II myofibre atrophy [66]. For example, a study by Charette and colleagues found that a 12-week resistance training program in older women (mean age 69 years ± 1 year) significantly increased the cross-sectional area of the type II muscle fibers in comparison with an educational control group [61]. In another study, Fiatarone and colleagues examined the effects of an 8-week, high-intensity resistance program in ten frail, institutionalized older adults (90 years ± 1 year). The researchers found that mid-thigh muscle area increased by 9.0% ± 4.5% and that mean tandem gait speed improved by 48% [62]. In addition, participants had an average strength gain of 174%. In another recent study, Ferri and colleagues assessed changes in muscle strength and power following a 16-week resistance program in a population of men between the ages of 65 and 81. The researchers found not only an increase in maximal force production but also a significant increase in muscle power [63]. These studies provide compelling evidence that a progressive resistance training program can reduce muscle atrophy, improve gait speed, and increase muscle power.

Combination exercise training programs are often used to meet the current physical activity recommendations by the ACSM/AHA which state that older adults should take a multifactorial approach to enhancing physical activity by performing aerobic, strength, and flexibility exercise. Findings from recent trials support the efficacy of interventions that incorporate both aerobic and resistance training (with or without dietary intervention) for improving physical function in obese, older adults [55]. Another recent clinical trial sought to determine whether combining aerobic and resistance exercise compared to either modality alone (i.e., combination versus aerobic exercise alone versus resistance exercise alone) would exert greater effects on risk factors for disease and disability [67]. Abdominally obese older men and women completed a six-month intervention consisting of either resistance exercise three times per week, aerobic exercise (treadmill walking) five times per week, resistance and aerobic exercise three times per week, or a nonexercise control. All intervention groups showed improvements in functional limitations; however, this improvement was greater for the combined exercise group compared to the aerobic-only exercise group. Furthermore, the combined and aerobic-only groups lost significantly more total fat and abdominal fat than the control and resistance-only group, while skeletal muscle mass was most improved in the combined and resistance exercise group.

However, there is contradictory information presented by Manini and colleagues that demonstrated that one year of aerobic and resistance exercise performed two times per week was ineffective at improving long-distance walking speed in obese men and women when compared to nonobese [68]. Despite little change in long-distance walking speed (400 meters), obese individuals did manage to gain clinically significant improvements in a short performance battery of physical function that includes chair rises, balance tests, and 4-meter walking test.

The most recent data by Villareal and colleagues help to confirm the independent effects of exercise (aerobic + resistance exercise) on mobility and physical function in obese men and women [55]. In this study, exercise alone (3 times per week) resulted in significant improvements in a battery of functional tasks that included walking 50 feet, climbing one flight of stairs, and performance on a Romberg balance test—a motor coordination test that requires subjects to maintain static balance under progressively more difficult altered base of support conditions. Additional functional tasks that improved in the Villareal study included putting on and removing a coat, picking up a penny, standing up from a chair, and lifting a book. Collectively, these effects were greater than a concurrent control group and exceeded the improvements in a group who completed a diet-only intervention.

Collectively, these studies suggest that exercise alone can have robust effects on physical function in obese, older adults. However, these effects might not exceed those found with nonobese individuals. Therefore, an exercise program combining aerobic and resistance exercise provides the most beneficial health effects in obese older adults.

4.3. Multicomponent Interventions. Many lifestyle interventions designed to improve physical function in obese, older adults have typically included both a dietary component and a physical activity program, and thus can be classified as a *multicomponent intervention*. Because studies that intervene with caloric restriction plus exercise generally demonstrate beneficial effects on long-distance walking ability [4], a multicomponent intervention that includes caloric restriction may be needed to optimize the benefits of moderate intensity physical activity in obese, older adults. A relatively small number of studies have examined the effects of multicomponent interventions that combine dietary restriction plus exercise in obese, older adults.

Findings from recent trials support the effectiveness of multicomponent (i.e., diet plus aerobic plus resistance exercise) interventions for improving mobility function in obese, older adults. For example, the addition of resistance training to a diet plus aerobic exercise program can attenuate the loss of skeletal muscle during weight loss in adults aged 65 and older [69]. Diet plus supervised resistance and aerobic exercise regimens have also been found to substantially improve mobility (i.e., walking speed) and

function in obese, older adults [70, 71]. A recent study examined the effects of a 24-week multicomponent intervention compared to a Successful Aging Educational Control group on changes in mobility function in overweight, older women with moderate functional impairments. Participants in the weight loss plus exercise (WL + E) intervention condition were instructed to reduce their caloric intake by 500 to 1000 kcal/day and to attend weekly group-based counseling sessions, as well as three center-based supervised exercise sessions per week. The exercise regimen included an aerobic phase (15 minutes), a resistance-training phase (15 minutes), a second aerobic phase (15 minutes), and a cool-down phase (15 minutes). This intervention resulted in substantial improvements in mobility (i.e., increase in walking speed = 0.15 meters/second) in this high-risk population [72]. Participants in the Successful Aging Educational Control condition received weekly lectures on a variety of health topics relevant to older adults. Noteworthy, walking speed among participants in this group did not change. In a recent study, Avila et al. [54] examined the effect of a 10-week moderate intensity resistance training program combined with diet-induced weight loss on body and muscle composition and physical function in obese, older adults. The combination of resistance training and diet was found to be more effective than diet alone in producing fat loss, reducing intermuscular adipose tissue, and improving strength. However, no differences were found between groups on measures of physical function.

Another recent trial investigated the independent and combined effects of dietary and exercise interventions [55]. For this 52-week study, participants were assigned to a control group, caloric restriction group, exercise group, or a caloric restriction plus exercise group. The exercise intervention included both aerobic and resistance training components. While physical function (i.e., Physical Performance Test) improved for all the intervention groups compared to the control group, the diet plus exercise group exhibited significantly better physical function compared to the diet-only and exercise-only groups. Additionally, the diet plus exercise group lead to improved peak oxygen consumption, strength, balance, and gait speed compared to all other groups. These findings suggest that multicomponent weight loss interventions can significantly improve mobility and physical function in obese, older adults.

5. Conclusions

Obesity is a major health concern in most developed countries. When combined with advance age, the detrimental health effects of obesity are magnified. These negative effects manifest in poor physical performance that can be captured with a simple measurement of gait speed. The use of gait speed not only provides a marker of physical performance but also represents a strong predictor of longevity. As such, we have presented gait speed as a surrogate endpoint that estimates the effect of lifestyle interventions on longevity. Importantly, there is now extensive evidence demonstrating that lifestyle interventions involving modification of dietary and exercise patterns are effective in producing clinically significant improvements in gait speed. While interventions emphasizing either modification of diet or exercise have beneficial effects on gait speed, the benefits are optimized with multicomponent interventions. Therefore, interventions involving both diet and exercise may hold the greatest potential for improving mobility and potentially increasing longevity in obese, older adults.

References

[1] S. Studenski, S. Perera, K. Patel et al., "Gait speed and survival in older adults," *Journal of the American Medical Association*, vol. 305, no. 1, pp. 50–58, 2011.

[2] M. Cesari, S. B. Kritchevsky, A. B. Newman et al., "Added value of physical performance measures in predicting adverse health-related events: results from the health, aging and body composition study," *Journal of the American Geriatrics Society*, vol. 57, no. 2, pp. 251–259, 2009.

[3] J. Dumurgier, A. Elbaz, P. Ducimetiere et al., "Slow walking speed and cardiovascular death in well functioning older adults: prospective cohort study," *British Medical Journal*, vol. 339, pp. 1–7, 2009.

[4] J. M. Guralnik, L. Ferrucci, E. M. Simonsick, M. E. Salive, and R. B. Wallace, "Lower-extremity function in persons over the age of 70 years as a predictor of subsequent disability," *The New England Journal of Medicine*, vol. 332, no. 9, pp. 556–561, 1995.

[5] M. Cesari, "Role of gait speed in the assessment of older patients," *Journal of the American Medical Association*, vol. 305, no. 1, pp. 93–94, 2011.

[6] K. M. Flegal, M. D. Carroll, C. L. Ogden, and L. R. Curtin, "Prevalence and trends in obesity among US adults, 1999–2008," *Journal of the American Medical Association*, vol. 303, no. 3, pp. 235–241, 2010.

[7] S. D. R. Harridge and A. Young, "Skeletal muscle," in *Principles and Practices of Geriatric Medicine*, pp. 898–905, Wiley, London, UK, 1998.

[8] R. N. Baumgartner, P. M. Stauber, D. McHugh, K. M. Koehler, and P. J. Garry, "Cross-sectional age differences in body composition in persons 60+ years of age," *Journals of Gerontology Series A*, vol. 50, no. 6, pp. M307–M316, 1995.

[9] G. B. Forbes, "Longitudinal changes in adult fat-free mass: influence of body weight," *American Journal of Clinical Nutrition*, vol. 70, no. 6, pp. 1025–1031, 1999.

[10] R. Roubenoff, "Sarcopenic obesity: the confluence of two epidemics," *Obesity Research*, vol. 12, no. 6, pp. 887–888, 2004.

[11] T. W. Buford, S. D. Anton, A. R. Judge et al., "Models of accelerated sarcopenia: critical pieces for solving the puzzle of age-related muscle atrophy," *Ageing Research Reviews*, vol. 9, no. 4, pp. 369–383, 2010.

[12] W. J. Evans, "Skeletal muscle loss: cachexia, sarcopenia, and inactivity," *American Journal of Clinical Nutrition*, vol. 91, no. 4, 2010.

[13] R. N. Baumgartner, "Body composition in healthy aging," *Annals of the New York Academy of Sciences*, vol. 904, pp. 437–448, 2000.

[14] C. S. Blaum, Q. L. Xue, E. Michelon, R. D. Semba, and L. P. Fried, "The association between obesity and the frailty syndrome in older women: the Women's Health and Aging Studies," *Journal of the American Geriatrics Society*, vol. 53, no. 6, pp. 927–934, 2005.

[15] M. Cesari, S. B. Kritchevsky, R. N. Baumgartner et al., "Sarcopenia, obesity, and inflammation–results from the Trial of Angiotensin Converting Enzyme Inhibition and Novel Cardiovascular Risk Factors study," *American Journal of Clinical Nutrition*, vol. 82, no. 2, pp. 428–434, 2005.

[16] E. A. Finkelstein, C. J. Ruhm, and K. M. Kosa, "Economic causes and consequences of obesity," *Annual Review of Public Health*, vol. 26, pp. 239–257, 2005.

[17] J. R. Pleis, J. S. Schiller, and V. Benson, "Summary health statistics for U.S. adults: National Health Interview Survey, 2000," *Vital and Health Statistics Series 10*, no. 215, pp. 1–132, 2003.

[18] J. P. Chaput and A. Tremblay, "Obesity and physical inactivity: the relevance of reconsidering the notion of sedentariness," *Obesity Facts*, vol. 2, no. 4, pp. 249–254, 2009.

[19] S. A. Carlson, J. E. Fulton, D. A. Galuska, J. Kruger, F. Lobelo, and F. V. Loustalot, "Prevalence of self-reported physically active adults—United States, 2007," *Morbidity and Mortality Weekly Report*, vol. 57, no. 48, pp. 1297–1300, 2008.

[20] S. U. Ko, S. Stenholm, and L. Ferrucci, "Characteristic gait patterns in older adults with obesity—results from the Baltimore Longitudinal Study of Aging," *Journal of Biomechanics*, vol. 43, no. 6, pp. 1104–1110, 2010.

[21] S. P. Messier, R. F. Loeser, G. D. Miller et al., "Exercise and dietary weight loss in overweight and obese older adults with knee osteoarthritis: the arthritis, diet, and activity promotion trial," *Arthritis and Rheumatism*, vol. 50, no. 5, pp. 1501–1510, 2004.

[22] I. Janssen, S. B. Heymsfield, and R. Ross, "Low relative skeletal muscle mass (sarcopenia) in older persons is associated with functional impairment and physical disability," *Journal of the American Geriatrics Society*, vol. 50, no. 5, pp. 889–896, 2002.

[23] M. Estrada, A. Kleppinger, J. O. Judge, S. J. Walsh, and G. A. Kuchel, "Functional impact of relative versus absolute sarcopenia in healthy older women," *Journal of the American Geriatrics Society*, vol. 55, no. 11, pp. 1712–1719, 2007.

[24] M. Visser, T. B. Harris, J. Langlois et al., "Body fat and skeletal muscle mass in relation to physical disability in very old men and women of the Framingham heart study," *Journals of Gerontology Series A*, vol. 53, no. 3, pp. M214–M221, 1998.

[25] F. Lauretani, C. R. Russo, S. Bandinelli et al., "Age-associated changes in skeletal muscles and their effect on mobility: an operational diagnosis of sarcopenia," *Journal of Applied Physiology*, vol. 95, no. 5, pp. 1851–1860, 2003.

[26] T. J. Marcell, "Sarcopenia: causes, consequences, and preventions," *Journals of Gerontology Series A* , vol. 58, no. 10, pp. 911–916, 2003.

[27] M. J. Delmonico, T. B. Harris, M. Visser et al., "Longitudinal study of muscle strength, quality, and adipose tissue infiltration," *American Journal of Clinical Nutrition*, vol. 90, no. 6, pp. 1579–1585, 2009.

[28] A. B. Newman, V. Kupelian, M. Visser et al., "Strength, but not muscle mass, is associated with mortality in the health, aging and body composition study cohort," *Journals of Gerontology Series A*, vol. 61, no. 1, pp. 72–77, 2006.

[29] D. R. Bouchard and I. Janssen, "Dynapenic-obesity and physical function in older adults," *Journals of Gerontology Series A*, vol. 65, no. 1, pp. 71–77, 2010.

[30] B. C. Clark and T. M. Manini, "Sarcopenia =/= dynapenia," *Journals of Gerontology Series A*, vol. 63, no. 8, pp. 829–834, 2008.

[31] T. M. Manini and B. C. Clark, "Dynapenia and aging: an update," *Journal of Gerontology Series A*. In press.

[32] B. H. Goodpaster, C. L. Carlson, M. Visser et al., "Attenuation of skeletal muscle and strength in the elderly: the health ABC study," *Journal of Applied Physiology*, vol. 90, no. 6, pp. 2157–2165, 2001.

[33] B. H. Goodpaster, S. W. Park, T. B. Harris et al., "The loss of skeletal muscle strength, mass, and quality in older adults: the Health, Aging and Body Composition Study," *Journals of Gerontology Series A*, vol. 61, no. 10, pp. 1059–1064, 2006.

[34] R. N. Baumgartner, S. J. Wayne, D. L. Waters, I. Janssen, D. Gallagher, and J. E. Morley, "Sarcopenic obesity predicts instrumental activities of daily living disability in the elderly," *Obesity Research*, vol. 12, no. 12, pp. 1995–2004, 2004.

[35] M. Zamboni, G. Mazzali, F. Fantin, A. Rossi, and V. Di Francesco, "Sarcopenic obesity: a new category of obesity in the elderly," *Nutrition, Metabolism and Cardiovascular Diseases*, vol. 18, no. 5, pp. 388–395, 2008.

[36] K. A. Landers, G. R. Hunter, C. J. Wetzstein, M. M. Bamman, and R. L. Weinsier, "The interrelationship among muscle mass, strength, and the ability to perform physical tasks of daily living in younger and older women," *Journals of Gerontology Series A*, vol. 56, no. 10, pp. B443–B448, 2001.

[37] J. F. Bean, D. K. Kiely, S. Herman et al., "The relationship between leg power and physical performance in mobility-limited older people," *Journal of the American Geriatrics Society*, vol. 50, no. 3, pp. 461–467, 2002.

[38] W. H. Ettinger, R. Burns, S. P. Messier et al., "A randomized trial comparing aerobic exercise and resistance exercise with a health education program in older adults with knee osteoarthritis: the Fitness Arthritis and Seniors Trial (FAST)," *Journal of the American Medical Association*, vol. 277, no. 1, pp. 25–31, 1997.

[39] L. M. Verbrugge, D. M. Gates, and R. W. Ike, "Risk factors for disability among U.S. adults with arthritis," *Journal of Clinical Epidemiology*, vol. 44, no. 2, pp. 167–182, 1991.

[40] R. Marks, "Obesity profiles with knee osteoarthritis: correlation with pain, disability, disease progression," *Obesity*, vol. 15, no. 7, pp. 1867–1874, 2007.

[41] S. E. Lamb, J. M. Guralnik, D. M. Buchner et al., "Factors that modify the association between knee pain and mobility limitation in older women: the women's health and aging study," *Annals of the Rheumatic Diseases*, vol. 59, no. 5, pp. 331–337, 2000.

[42] A. Powell, A. J. Teichtahl, A. E. Wluka, and F. M. Cicuttini, "Obesity: a preventable risk factor for large joint osteoarthritis which may act through biomechanical factors," *British Journal of Sports Medicine*, vol. 39, no. 1, pp. 4–5, 2005.

[43] T. N. Kim, M. S. Park, S. J. Yang et al., "Prevalence and determinant factors of sarcopenia in patients with type 2 diabetes: the Korean Sarcopenic Obesity Study (KSOS)," *Diabetes Care*, vol. 33, no. 7, pp. 1497–1499, 2010.

[44] I. S. Raj, S. R. Bird, and A. J. Shield, "Aging and the force-velocity relationship of muscles," *Experimental Gerontology*, vol. 45, no. 2, pp. 81–90, 2010.

[45] M. A. Fiatarone, E. F. O'Neill, N. D. Ryan et al., "Exercise training and nutritional supplementation for physical frailty in very elderly people," *The New England Journal of Medicine*, vol. 330, no. 25, pp. 1769–1775, 1994.

[46] R. A. Fielding, N. K. LeBrasseur, A. Cuoco, J. Bean, K. Mizer, and M. A. Fiatarone Singh, "High-velocity resistance training increases skeletal muscle peak power in older women," *Journal of the American Geriatrics Society*, vol. 50, no. 4, pp. 655–662, 2002.

[47] W. R. Frontera, C. N. Meredith, K. P. O'Reilly, H. G. Knuttgen, and W. J. Evans, "Strength conditioning in older men:

skeletal muscle hypertrophy and improved function," *Journal of Applied Physiology*, vol. 64, no. 3, pp. 1038–1044, 1988.

[48] F. C. Hagerman, S. J. Walsh, R. S. Staron et al., "Effects of high-intensity resistance training on untrained older men. I. Strength, cardiovascular, and metabolic responses," *Journals of Gerontology Series A*, vol. 55, no. 7, pp. B336–B346, 2000.

[49] S. B. Votruba, M. A. Horvitz, and D. A. Schoeller, "The role of exercise in the treatment of obesity," *Nutrition*, vol. 16, no. 3, pp. 179–188, 2000.

[50] S. P. Messier, C. Legault, R. F. Loeser et al., "Does high weight loss in older adults with knee osteoarthritis affect bone-on-bone joint loads and muscle forces during walking," *Osteoarthritis and Cartilage*, vol. 19, pp. 272–280, 2011.

[51] L. Fontana and S. Klein, "Aging, adiposity, and calorie restriction," *Journal of the American Medical Association*, vol. 297, no. 9, pp. 986–994, 2007.

[52] S. B. Racette, E. P. Weiss, D. T. Villareal et al., "One year of caloric restriction in humans: feasibility and effects on body composition and abdominal adipose tissue," *Journals of Gerontology Series A*, vol. 61, no. 9, pp. 943–950, 2006.

[53] L. K. Heilbronn, L. De Jonge, M. I. Frisard et al., "Effect of 6-month calorie restriction on biomarkers of longevity, metabolic adaptation, and oxidative stress in overweight individuals: a randomized controlled trial," *Journal of the American Medical Association*, vol. 295, no. 13, pp. 1539–1548, 2006.

[54] J. J. Avila, J. A. Gutierres, M. E. Sheehy, I. E. Lofgren, and M. J. Delmonico, "Effect of moderate intensity resistance training during weight loss on body composition and physical performance in overweight older adults," *European Journal of Applied Physiology*, vol. 109, no. 3, pp. 517–525, 2010.

[55] D. T. Villareal, S. Chode, N. Parimi et al., "Weight loss, exercise, or both and physical function in obese older adults," *The New England Journal of Medicine*, vol. 364, no. 13, pp. 1218–1229, 2011.

[56] X. Z. He and D. W. Baker, "Body mass index, physical activity, and the risk of decline in overall health and physical functioning in late middle age," *American Journal of Public Health*, vol. 94, no. 9, pp. 1567–1573, 2004.

[57] M. M. Hillsdon, E. J. Brunner, J. M. Guralnik, and M. G. Marmot, "Prospective study of physical activity and physical function in early old age," *American Journal of Preventive Medicine*, vol. 28, no. 3, pp. 245–250, 2005.

[58] T. Manini, M. Marko, T. VanArnam et al., "Efficacy of resistance and task-specific exercise in older adults who modify tasks of everyday life," *Journals of Gerontology Series A*, vol. 62, no. 6, pp. 616–623, 2007.

[59] K. R. Vincent, R. W. Braith, R. A. Feldman, H. E. Kallas, and D. T. Lowenthal, "Improved cardiorespiratory endurance following 6 months of resistance exercise in elderly men and women," *Archives of Internal Medicine*, vol. 162, no. 6, pp. 673–678, 2002.

[60] M. Brown and J. O. Holloszy, "Effects of walking, jogging and cycling on strength, flexibility, speed and balance in 60- to 72-year olds," *Aging*, vol. 5, no. 6, pp. 427–434, 1993.

[61] S. L. Charette, L. McEvoy, G. Pyka et al., "Muscle hypertrophy response to resistance training in older women," *Journal of Applied Physiology*, vol. 70, no. 5, pp. 1912–1916, 1991.

[62] M. A. Fiatarone, E. C. Marks, N. D. Ryan, C. N. Meredith, L. A. Lipsitz, and W. J. Evans, "High-intensity strength training in nonagenarians. Effects on skeletal muscle," *Journal of the American Medical Association*, vol. 263, no. 22, pp. 3029–3034, 1990.

[63] A. Ferri, G. Scaglioni, M. Pousson, P. Capodaglio, J. Van Hoecke, and M. V. Narici, "Strength and power changes of the human plantar flexors and knee extensors in response to resistance training in old age," *Acta Physiologica Scandinavica*, vol. 177, no. 1, pp. 69–78, 2003.

[64] R. U. Newton, K. Häkkinen, A. Häkkinen, M. McCormick, J. Volek, and W. J. Kraemer, "Mixed-methods resistance training increases power and strength of young and older men," *Medicine and Science in Sports and Exercise*, vol. 34, no. 8, pp. 1367–1375, 2002.

[65] J. F. Bean, D. K. Kiely, S. Larose, R. Goldstein, W. R. Frontera, and S. G. Leveille, "Are changes in leg power responsible for clinically meaningful improvements in mobility in older adults?" *Journal of the American Geriatrics Society*, vol. 58, no. 12, pp. 2363–2368, 2010.

[66] D. N. Proctor, W. E. Sinning, J. M. Walro, G. C. Sieck, and P. W. R. Lemon, "Oxidative capacity of human muscle fiber types: effects of age and training status," *Journal of Applied Physiology*, vol. 78, no. 6, pp. 2033–2038, 1995.

[67] L. E. Davidson, R. Hudson, K. Kilpatrick et al., "Effects of exercise modality on insulin resistance and functional limitation in older adults: a randomized controlled trial," *Archives of Internal Medicine*, vol. 169, no. 2, pp. 122–131, 2009.

[68] T. M. Manini, A. B. Newman, R. Fielding et al., "Effects of exercise on mobility in obese and nonobese older adults," *Obesity*, vol. 18, no. 6, pp. 1168–1175, 2010.

[69] T. N. Frimel, D. R. Sinacore, and D. T. Villareal, "Exercise attenuates the weight-loss-induced reduction in muscle mass in frail obese older adults," *Medicine and Science in Sports and Exercise*, vol. 40, no. 7, pp. 1213–1219, 2008.

[70] P. Chomentowski, J. J. Dubé, F. Amati et al., "Moderate exercise attenuates the loss of skeletal muscle mass that occurs with intentional caloric restriction-induced weight loss in older, overweight to obese adults," *Journals of Gerontology Series A*, vol. 64, no. 5, pp. 575–580, 2009.

[71] D. T. Villareal, M. Banks, D. R. Sinacore, C. Siener, and S. Klein, "Effect of weight loss and exercise on frailty in obese older adults," *Archives of Internal Medicine*, vol. 166, no. 8, pp. 860–866, 2006.

[72] S. D. Anton, T. M. Manini, V. A. Milsom et al., "Effects of a weight loss plus exercise program on physical function in overweight, older women: a randomized controlled trial," *Clinical Interventions in Aging*, vol. 6, pp. 141–149, 2011.

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Research Article

Cognitive Beliefs and Future Time Perspectives: Predictors of Mortality and Longevity

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On the basis of postulates derived from cognitive-behavioral theory, research and therapy, the authors explored the extent to which older adults' cognitive beliefs of a just world and their perspectives on future time and similarity or self-continuity with the future self are predictors of long-term survival. After baseline assessment of health and cognitive beliefs and future perspectives of time and self-continuity as predictors of mortality, 440 participants (ages 65 to 87) were followed longitudinally for 6.5 years. Consistent with our hypotheses, findings demonstrated that a significantly higher percentage of survivors were individuals who showed higher scores on beliefs in a just world and on both the future time perspective and the future self-continuity perspective at the time of baseline assessments. Conversely, mortality risk was much higher for individuals who scored low on these predictor variables, and high on distrust. Implications for health and longevity are discussed.

1. Introduction

To date, one of the most understudied etiologies of older adults' survival and longevity has been the role of their cognitive beliefs and worldviews that possibly interact with their functional and mental capacities to endure, challenge, overcome, and survive in the face of the numerous struggles and obstacles in advanced old age.

In earlier research on predictors of mortality, the focus has been exclusively on variations in physical health and sociodemographic variables to explain and predict differences in longevity and mortality rates across a wide age range. More recently, studies have explored the relationship between the 5-factor personality traits [1–4] and other stress-inducing traits of perfectionism and dysfunctional dependency traits to predict greater longevity or increased risk of mortality [5]. The present study presents a clear departure from earlier studies that have focused on sociodemographic and personality factors to explain and predict differences in all-cause mortality rates in later life. The goal of the present research is to move outside the personality and traits model to other second-order cognitive-behavioral factors to predict differences in all-cause mortality rates in later life. Cognitive-behavioral theorists argue on both theoretical and empirical grounds that individuals' cognitive beliefs exert a great deal of influence on their health, resilience, and longevity and may logically be assumed to be robust predictors of impending mortality or conversely of longevity. However, the ability of cognitive belief systems to predict important health outcomes of survival and longevity has traditionally been questioned because of the putative effects of individuals' earlier life experiences such as parental loss and divorce [6]. More recent explorations into individuals' cognitive beliefs have been drawing attention to a cluster of beliefs systems that may counter the effects of earlier negative experiences and may serve as strengthening factors toward enhancing longevity. Recent research using more modern concepts of evaluating dominant cognitive beliefs and cognitive perspectives of individuals (e.g., beliefs about a just world (BJW) for self and others, beliefs about one's future time, beliefs about one's self-continuity with the future, and beliefs about social, political, and interpersonal trust)

has provided growing evidence that individuals' cognitive beliefs and perspectives are indeed related to health-related processes [7, 8] leading to longer survival and longevity as a final health outcome. Also, in recent years, the availability of reliable and valid measures of cognitive beliefs of justice and fairness [9, 10], future time perspectives [11], and future self-continuity perspectives [12, 13] has increased our understanding of the predictive value of individuals' cognitive beliefs as strengthening or debilitating factors in health-related outcomes of resilience and longevity.

The present study addresses the relationship between newly emerging sets of important cognitive belief systems and the potential for increased longevity. For example, a number of researchers [10, 14] have demonstrated empirically that individuals' tenacious cognitive beliefs in a just world (BJW) society are not only predictive of their subjective well being and resilience, but more importantly drive them toward investment in long-term goals and a commitment to better self-care of health and a longer life. As a result, life-span scholars are now more keenly exploring the proactive role that individuals' beliefs about a just world (BJW) may play in their future well being and healthy physical survival (see [15] Tomaka and Blascovitch, 1994), low levels of depression [16], and less loneliness [17]. Other dominant cognitive beliefs which have been seen to be related to healthy survival processes or which present increased risks of mortality include beliefs about interpersonal trust and trust in key institutions [18, 19]. Individuals who have strong positive beliefs of trust in the interpersonal and institutional domains are commonly expected to live lives that are more organized and planned, as distinguished from lives of instability, anxiety, and caution [19]. Other perspectives and belief systems that are predictive of planned healthy survival include "Future Time Perspective (FTP)" and "Self-Continuity with the Future Perspective" (FSC). The FTP perspective is a measure of individuals' perceived belief about how much time participants had left in life. According to Carstensen [11], the subjective sense of remaining time has profound effects on basic human processes, including motivation, cognition, and behaviors. With increasing age, constraints on time left shift individuals' priorities about how remaining time can be protected. Along somewhat similar lines, the future self-continuity perspective (FSC) indicates that participants' beliefs about their similarity and connection as well as caring and liking for their future self 10-, 15-, or 20-years from now [13] determine and shift their motivation to protect the potential future person. For purposes of the present study, our underlying conceptual assumption in both these futuristic perspectives is that individuals who perceive their time horizons and their self-continuity with the future as more limited would be more likely to discount the future, and thus more unlikely to plan for self-care and self-management of the future, whereas those who feel the future horizons are more open-ended and expansive are more likely to plan and organize for a secure future. Implicit in these perspectives is the prediction that individual differences in the experience of self-continuity could have positive pragmatic consequences for future health care and healthy survival. One logical assumption is that people who experience little or no continuity with the future self may not aspire to control future health-related processes whereas people who experience much similarity or self-continuity with the future self are likely more motivated to work toward shaping a better survival.

While previous research has demonstrated empirically the predictive value of the preceding sets of cognitive beliefs and perspectives in regard to well-being and physical and mental health-related processes and outcomes, findings have been drawn from the study of a wide range of ages. Thus, there is the question of whether the prognostic value of these belief systems continues into advanced old age. To date, the number of longitudinal studies of cognitive beliefs as predictors of longevity or as risk to mortality in old age is limited. To address this issue, longitudinal data obtained exclusively from samples of adults in advanced age are required. Accordingly, the purpose of our current research was to examine longitudinally the extent to which specific and select sets of cognitive beliefs are enabling, strengthening, or disabling with respect to long-term health, resilience, and longevity of older adults and have predictive value for all-cause mortality in advanced old age. The question is of increasing interest to health professionals and gerontologists for both practical and conceptual reasons.

In the section which follows, we review briefly the research literature that both explains and extends the assumptions underlying the theory and goals of concepts of BJW, FTP, and FSC and their potential for predicting longer survival/or reduced risk for mortality.

2. Conceptual and Theoretical Framework for the Research

The just world hypothesis and how it may relate to health and longevity is easily stated. Individuals have a strong need to believe that they live in a world where people generally get what they deserve. The belief that the world is just enables the individual to confront his/her physical and social environment as though they were stable and orderly. Without such a belief, it would be difficult for the individual to commit himself/herself to the pursuit of long-range goals or even to the socially regulated behavior of day-to-day life. Since the belief that the world is just serves such an important adaptive function for the individual, people are very reluctant to give up this belief, and they can be greatly troubled if they encounter evidence that suggests that the world is not really just or orderly after all [20]. According to Lerner and Miller's just-world theory, people who believe that the world treats them fairly may plan confidently for their future, expecting their lives to be orderly, meaningful, and controllable, foreseeing a positive future or viewing one's living situation as justly deserved and hence fair [21]. In turn, this expectation promotes mental health, meaning that the belief in a just world (BJW) can be seen as a "positive illusion" [22]. Indeed research links BJW to many indices of subjective well being including a greater purpose in life and commitment to planned healthy survival [23]. There is

empirical evidence showing that individuals who strongly believe in a just world have been seen to experience less stress and more positive affect than individuals with a weaker BJW (e.g., [16, 24]).

The preceding conceptual underpinnings and the recent theory and research related to cognitive beliefs of a just world (BJW) lend weight to the proposed hypothesis of our present study that strong BJW beliefs about justice for the self portend positive social consequences and health-related benefits for the future. As such, individuals' strong cognitive beliefs about a just world (BJW) may be early predictors of their continued physical and mental well being at later stages of life and would serve to protect them against the stress associated with the challenges of later life.

In the current study, we reason that the predictive power of BJW to enhance longevity derives uniquely from perceived justice and BJW beliefs. In light of the preceding discussion, our leading hypotheses for the current study were (1) that individuals' beliefs that the world is just to themselves (BJW-self) are particularly predictive of their longer term survival and longevity, and (2) that the associations between cognitive beliefs of a just world would be observed more powerfully among measures of *BJW for self only* (as distinguished from *BJW for others*). In essence, we reason that it is the perception of one's own, more so than other individuals', outcomes that would most powerfully predict longevity or possible risks of mortality; (3) that individuals' stronger levels of interpersonal trust and trust in the major communal institutions (as associated with their perceptions of justice in the BJW beliefs) are early predictors of their longevity, or conversely stronger levels of distrust in the major communal institutions would be associated with increased risk of mortality.

A related second goal of our current study was to examine the predictive value of other related cognitive perspectives such as future time perspectives not previously studied as predictors of mortality and longevity. On the basis of postulates derived from Carstensen's [11] theory on the influence of a sense of time on human development (also see [13, 25]) theory on individual differences in future self-continuity, we reason that while time eventually runs out for all individuals, individuals who hold more expansive and open-ended future time horizons or who foresee stronger self-continuity with the future self (compared to those who hold more limited future time horizons and less self-continuity with the future self) are less likely to discount the significance of future time and are more invested in self-preservation for the future. Accordingly, we hypothesize (4) that individuals' varying beliefs about future time left (FTP) and their beliefs about their ability to maintain self-continuity with the future (FSC) are critical markers or early predictors of longevity and of the risks of mortality.

While we acknowledge that the preceding associations between mortality and cognitive beliefs may not have been apparent in early and middle-age adulthood, our expectation is that the associations will be especially observable and relevant in late life functioning and will emerge as early markers or predictors of mortality or longevity.

3. Sampling Frame for the Study and Recruitment of Participants

Participants for the study were randomly recruited from the registry listings of four branch offices of community services and community organizations for seniors (Ministry of Health Services and Health Policy 1992), a governmental organization responsible at the time for social services and health policy in Southern Alberta. A sampling strategy with proportional stratification in function of geographical zone (metropolitan, urban, or rural) was used to ensure that the sample was representative of the general population of older adults living in three big cities and various rural areas in Southern Alberta. Participants came from three mid-sized cities (populations ranging from 170,000 to 300,000 individuals) and surrounding suburban and rural areas in Southern Alberta (Canada). It should be noted that various levels of community dwellings ranging from upper middle class private houses to low income apartment housing, and assisted living homes were included in the final recruitment.

Initially 760 brochures briefly describing the research were mailed on a staggered basis to seniors' households requesting individuals' participation with a one-time offer of a $30 gift certificate to compensate for their time. The purpose of the study was explained as an attempt to understand older individuals' beliefs, hopes, expectancies, and planned goals for the foreseeable future. By the end of eight weeks, responses were received from 132 individuals who volunteered their participation. The remaining 628 individuals were subsequently recontacted by mail, and of these 137 individuals who wanted additional information about the research were contacted by phone. Another 333 individuals agreed to participate in response to further advertising and a to a *second* and *third* "Call for Participants," made 3 and 4 months later, bringing the total number of willing participants to 470. Eligibility criteria for the selection of participants included (1) being 65 years or older; (2) being able to understand, speak, and write English; (3) not having a diagnosis of cognitive dysfunction registered in the medical files; (4) being available for baseline assessments; (5) able to specify by name, address, phone number, and other relevant details, one or more family members or care-givers willing to serve as informants to the research team; (6) willing to sign a consent form.

3.1. Procedures. The initial interview with each of the 470 participants and their family members took approximately one-and-a-half hours, with interviews staggered over a period of 16 weeks. The majority of participants were interviewed in their homes in order to obtain baseline information about health status in relation to the chronic condition presented, to obtain participants' formal consent to participate and to arrange for their family member or primary care giver to contact us periodically concerning the participant's general progress. Typically, two family members for each participant contracted to be the informants. There were 62 husband-wife couple participants. We provided informants with postage-paid envelopes to contact us at regular intervals, as stated in the contact form, concerning

the participant's health status (improving, stable, declining somewhat, seriously declining), and in the event of the participant's death, to provide the exact date of death.

Within the first four months of the completion of baseline assessments for the study, a total of 30 participants withdrew their participation for a number of reasons, mostly because of the care-givers' reluctance to cooperate. Data are presented for the remaining 440 participants who, following baseline assessments, stayed the entire 6.5 years' course of the study. We wrote "thank you notes" and sent gift coupons to care givers at every wave of the study, as a token of appreciation of their continuing participation. There was no further attrition of participants during the course of the next 6.5 years. Ascertainment of mortality (date of death) was done solely on the basis of report of the informants. Family members preferred that we used this follow-up and contact procedure because it was more personal and private. However, as far as possible, we double checked dates of death against easily accessible local/provincial mortuary listings.

3.1.1. Time Line for the Study. The study comprised 11 waves of data collection. Following a small pilot study, we conducted in 1995 on study procedures and assessment scales, baseline measures (wave 1) were obtained between September and December 2000 in a staggered way, followed by 10 subsequent waves of contacts with informants and/or participants to obtain "summary progress" data on participants. Contacts were made at approximately eight-month intervals (240 days apart) till early December 2007, approximately 6.5 years after baseline.

3.1.2. Assessment of Cognitive Beliefs about a Just World, Interpersonal Trust and Control, and Perspectives on Future Time and Self-Continuity with the Future. Participants agreed to complete paper-and-pencil tests at their own pace, at home or in their place of study, and approximately 10 percent sought the help of research assistants to record their responses.

At baseline, participants completed the following.

Measure of Beliefs of a Just World (BJW). BJW were assessed with a scale originally developed by Lipkus and Siegler [9] but further improved by Bègue and Bastounis [23], in order to separate items of BJW pertaining to self from items of BJW pertaining to others. Participants rated on a scale of 1 (*strongly disagree*) to 6 (*strongly agree*) 8 items of BJW beliefs pertaining to the self. Sample items are "I feel that the world treats me fairly in life," I feel that I get what I deserve," "I feel that my efforts are noticed and rewarded," I feel that people treat me with the respect I deserve," "I feel that I earn the rewards and punishments I get." Sample items from the BJW pertaining to others include "I feel that the world treats other people fairly," "I feel that people get what they deserve," "I feel that people get what they are entitled to get," "I feel that when people meet with misfortune, they have brought it upon themselves." Scores for BJW (self) and scores for the BJW (others) ranged from 8 to 48. We used the option of scoring the BJW-self and BWJ-others as continuous scales. Higher scores denote stronger BJW beliefs for self and others rated separately. Cronbach's alpha were .84 and .74 for the BJW-Self and BWJ-Others, respectively.

Measure of Future Time Perspective (FTP). FTP was assessed with the future time perspective scale developed by Carstensen [26]. Participants rated on a scale from 1 (very untrue for me) to 7 (very true for me) the degree with which they agreed with each of 10 items. Sample items are "Many opportunities await me in the future," "Most of my life still lies ahead of me," "I expect that I will set many new goals in the future," "My future seems infinite to me," "There is plenty of time left in my life to make new plans," "I have the sense that time is running out," "As I get older, I begin to experience time as limited." We used the option of scoring the FTP as a continuous scale in the first instance. Scores on this measure ranged from 10 to 70. The high scores represented a more expansive and open-ended future time perspective. Cronbach's alpha for the FTP scale was .82.

Measure of Future Self-Continuity (FSC). FSC was assessed by means of an adapted psychometric measure of future self-continuity originally devised by Frederick [12] that used a single-item measure (i.e., how similar/connected are you to your past and future self for 5-, 10-, 15-, and 20-year intervals on a 1–100 scale?). To facilitate the comprehension of the concept of continuity, the individual's endorsement of similarity between present and future selves was presented pictorially by a range of five circles with *no overlap* to circles with *complete overlap*. The index of future self-continuity featured two questions on 7-point scale marked at each point by two circles that ranged from showing no overlap at one end of the scale to depicting almost complete overlap at the other end (see [27] for circles depicting no overlap to complete overlap), thus, making the measuring devise more concretely comprehensible to older adults. Participants first selected the pair of circles that best described how similar/connected they felt to the future self 15 years from now, and how much they cared about the future self. Subsequently, their responses were invited to two questions: "How connected do you feel to your future self 15 years from now?" (*not at all* = 0 to *very much* = 7). "How much do you care for your future self?" (*not at all* = 0 to *very much* = 7). We used the option of scoring the FSC scale (similarity/caring) as a continuous scale in the first instance. The index score of future self-continuity ranged from 7 to 35, and caring for the future self ranged from 7 to 35, assessed in terms of one continuous total index score ranging from 14 to 70. The high scores represented higher levels of future self-continuity/caring for future self. Cronbach's alpha for the FSC scale was .74.

Interpersonal and Society Trust Measure. Individuals' degree of interpersonal trust and trust in the surrounding public institutions that are perceived to represent justice and fairness were assessed by means of scale items adapted from the *The Rotter Trust Scale* [19]. As a first step in adapting the scale, a number of items were written using a 5-point

Likert format, (1) strongly agree to (5) strongly disagree. An attempt was made to sample a wide variety of social objects so that a subject would be called upon to express his/her *trust* of outside agents, friends, and family members. Sample items include "In dealing with strangers one is better off to be cautious until they have provided evidence that they are trustworthy," "Most elected public officials are not really sincere in their campaign promises"; "Most friends can be trusted to support you for life"; "I am able to share my innermost thoughts and feelings with family because of my trust in them"; "I do not like to reveal personal information to outside agents even when they claim to be helping me"; "I am wary of other people's motives" "I believe that most people are basically good and trustworthy"; "I am a private person and find it hard to trust people I do not know well." In the final form of this scale, the 24 items selected were similarly balanced. Twelve items indicated *trust for agreeing,* and 12 items indicated *distrust for agreeing* (with the range of scores for both the trust items and distrust items being 12 to 60). A few filler items were included to disguise the true purpose of the scale. Cronbach's alpha for the trust scale and distrust scale were .77 and .72, respectively.

Spheres of Control [18]. The scale has little or no conceptual overlap with the BJW scale and, hence, was selected to provide an independent measure of control. The scale is comprised of 30 items with the three spheres of control (personal efficacy, interpersonal control, and sociopolitical control) each represented by 10 items each rated on a 5-point Likert scale ranging from "disagree" to "agree". In the present research, we collapsed the scores across these three spheres of control because the results of our hypothesis tests were not affected by distinguishing between them. Specimen items for the personal efficacy scale, interpersonal scale, and sociopolitical scale, respectively, include "It's pointless to keep working on something that is too difficult for me"; "When I make plans I am almost certain to make them work"; "I have no trouble making and keeping friends"; "I find it easy to play an important part in most group or individual situations". "In the long run, we as voters are responsible for bad government on a local or national level"; "It is difficult for people to have much control over things politicians do in offices." The scoring of some items is reversed before summing the subset. A total index score for the 30 items was obtained with scores ranging from 30 to 150. Higher scores represent a more internal locus of control. Cronbach's alpha for the Control scale was .75.

Measure of Self-Esteem (SEI: [28]). This inventory was used to measure self-esteem as a global and stable disposition. The inventory has 10 items, 5 positively keyed and 5 negatively keyed. Each item is rated on a 4-point Likert type scale. Cronbach's alpha for the SEI scale was .88.

Physical Function. Physical Function was assessed by means of a single item taken from the physical function mobility index inquiring about one's ability (*yes/no*) to climb one flight of stairs without help. This one-item question was intended to seek information on physical fitness.

It should be noted that all measures administered to the participants were formatted in terms of language and structure appropriate for adults having a ninth-grade education. All paper-and-pencil tests and self-report measures used in the study were previously piloted on a volunteer group of 20 men and women aged 60 to 80 years. Subsequent modifications were made in the instructions and illustrations given for responding to the five-point ratings of test items. This procedure was undertaken to ensure that even those participants who were elderly and had relatively limited education could validly complete the measures.

3.1.3. Assessment of Health Variables and Family Relationship Measures. Advanced old age is commonly associated with an increase in disabilities, higher rates of health care use, and higher need for social support. Thus, we felt the need to examine further fluctuations in risk of mortality after controlling for health-related covariates, which were assessed as follows:

Survey of Number of Visits to Health Providers. As a part of a separate survey, respondents listed the number of visits that they had paid in the previous year to health providers, including visits to family physicians, community health clinics, and emergency health units, including hospital visits.

IADL Index of Disability. We based this index on the self-reported ability for 12 daily living activities included in the IADL. Respondents were asked to indicate whether they had experienced limitations in 12 areas of functioning with responses coded (1) for *yes* and (0) for *no,* for example, able to use a telephone; boarding a bus without assistance; lifting or carrying groceries; personal grooming and personal hygiene care; doing light house work; taking medications; walking one block. All the items were summed to form one index of IADL, with scores ranging from 0 to 12. Cronbach's alpha for the IADL scale was .81.

Measure of Satisfaction with Family and Social Support. Social satisfaction was assessed with 10 items adapted from Zimet et al. [29]. Participants rated how satisfied they were with their social partners in general, and how satisfied they were with their family and relatives on a rating scale ranging from 1 (*very dissatisfied*) to 3 (*very satisfied*). Scores ranged from 10 to 30. Cronbach's alpha for the social support satisfaction scale was .89.

3.2. Data Analysis. We conducted all analyses using the *statistical package for social sciences* (*Version10*). We assessed the internal consistency of each trait scale with Cronbach's coefficient alpha and the association of the scales with each other and with other covariates by means of Pearson correlation coefficients. For all analyses, differences between survivors and decedents on the date of final censoring were assessed with t-tests and chi-square tests of association.

Cox proportional hazard models [30] were fitted to estimate the importance of each predictor of mortality. It is important to note that Cox regression, or the proportional

hazards model, is a well-recognized statistical technique for survival analysis which is concerned with studying the time between entry to a study and a subsequent event such as death, as is the case in the present research. Cox regression has the advantage that it allows for the simultaneous exploration of the relationship between survival of persons and several explanatory variables (in our case variables such as cognitive beliefs, future time perspectives, control and trust, etc.). Of particular relevance and interest to our research is that the technique allows for age adjustment of the calculated hazard ratios across a wide range of ages, as was the case in our study. (See "What is a Cox model?," by Stephen J. Walters, a Haywood Group plc publication, May 2003, accessible online at http://www.whatisseries.co.uk/whatis/pdfs/What_is_Cox_model.pdf); see also G.D. Garson, Statnotes, North Carolina State University, online at http://faculty.chass.ncsu.edu/garson/PA765/cox.htm) for details of statistical procedures for achieving adjustment for the effect of age as a covariate. The Cox regression model assumes that the death rate of the population depends on a continuous time variable, which in the present study was the interval between the Wave 1 assessment and date of death, calculated in terms of total number of days.

In the present study, predictor variables included beliefs in a just society (BJW: self and others), future time perspective (FTP), future self-continuity (similarity/caring) perspective (FSC), demographic variables, and health and physical functioning variables. The initial risk ratios (IRRs) (frequently referred to as hazard ratios) are calculated by exponentiating the beta weight for the predictor [31], and regressions are expressed in risk ratios and 95 percent confidence intervals. All Cox regression hazard ratios (IRRs) presented in the present analyses were first adjusted for age. In subsequent analyses, we controlled for three health-related covariates (see [3, 5, 32, 33] for significance of studying health variables).

4. Results

Results are reported in two sections corresponding to our two main research questions. In the first section, we examine key differences between survivors and decedents in baseline characteristics of participants. In specific, we conducted Cox regression analysis of risk rations of mortality to estimate the relative risk of death and the importance of each predictor of mortality for the whole sample. All variables entered in the Cox regression analysis were entered as continuous variables for these analyses. In the second section, we present additional analysis of data where we were unable to derive easily interpretable results from using Cox regressions of continuous variable data. Subsequently, we analyzed the data using categorical variables that we considered were appropriate for the otherwise continuous variables.

4.1. Key Differences and Associations between Survivors and Nonsurvivors (Decedents). Four hundred and forty participants were followed starting in December 2000 when baseline assessments were completed. As of December 2007, after an average of 6.5 years of observation one hundred and forty (32%) deaths had occurred, and 300 (68%) survived. Table 1 provides crude data at baseline on the two subgroups of decedents and survivors. Those who died during the study period were somewhat older. Among health-related variables, the respondents in the two groups were quite different with respect to physical function scores, but not with respect to number of medical visits reported at time of baseline assessment, or in respect of number of disabilities reported on the IADL index (see Table 1). Survivors compared to decedents had significantly higher scores on measures of beliefs in just world (BJW-Self), on the future time perspective (FTP) and future self-continuity/caring for the future self (FSC) perspective scale. By contrast, decedents compared to survivors had significantly higher scores on the measure of distrust on the interpersonal and society scale and on the control scale.

First, we examined the intercorrelations of measures of beliefs in a just world (BJW; self and others), future time perspective (FTP), future self-continuity perspective (continuity/caring for future self), trust (agreement with trust items and agreement with distrust items) and control with one another, and with demographic variables (age and education), and baseline indicators of number of medical visits, IADL disability, and satisfaction with social support. As expected, the two scales of BJW were positively related to each other, with correlations ranging from .41 to .64; $P < .001$. Overall, the BJW-self scale, the FTP scale, and FSC scale were positively correlated with one another, with correlations ranging from .34 to .68; $P < .001$. In general, the BJW, FTP, FSC, and trust measures were not correlated significantly with demographic variables of age and education. The one notable exception, however, was that the distrust measure was significantly correlated with age and education, with correlations ranging from .34 to .39; $P < .001$ (Note: the detailed table of correlations is available from the authors on request).

4.1.1. Psychometric Information on Two Dimensions of BJW (Self/Others), Two Dimensions of Future Time Perspectives (Time Left/Self-Connected with Future), Trust/Distrust and Measures of Self-Esteem and Control. Table 2 provides psychometric information on the predictor variables for mortality risks selected for the research.

Examination of the mean scores for the various scales for which baseline data were obtained shows that the mean score of the control scale (80.1) was quite elevated when considered within the context of validity norms outlined in Paulhus [18]. However, the mean scores for the BJW, FTP, FSC. Trust and Distrust scales were consistent with means reported for these scales elsewhere. The internal consistencies of the various trait measures as reported in Table 2 are consistent with and converge appropriately with internal consistencies reported for the various scales elsewhere (see [10, 12, 19, 25]).

When Cox proportional hazard models *adjusted for age* [30] were fitted to estimate the relative risk of death and the importance of each predictor of mortality for the whole

Table 1: Baseline characteristics of participants who survived (survivors) or died (decedents) Total $N = 440$.

Characteristics (range of scores)	Survivors $N = 300$ (68%)	Decedents = $N = 140$ (32%)	P Value
Age, years	74.4 (6.3)	78.6 (6.5)	<.001
Education, years	14.1 (3.7)	13.9 (3.0)	.339
Women 66.0% (291) Men 34.0% (149)	195; 67% 102; 69%	96; 33% 47; 31%	<.001*
Beliefs in just world (BJW: self) (8–48)	32.8 (4.1)	20.4 (4.7)	<.001
Beliefs in just world (BJW: others) (8–48)	22.7 (2.1)	21.8 (3.0)	.461
Future time perspective (FTP) (10–70)	36.8 (6.1)	29.9 (7.6)	<.001
Future self-continuity (FSC)—15 yrs (14–70)	32.0 (3.5)	24.0 (2.1)	<.001
Trust: interpersonal and society (12–60)	33.1 (5.1)	29.5 (6.1)	<.05
Distrust: interpersonal and society (12–60)	27.5 (5.1)	39.9 (6.2)	<.001
Control (30–150)	69.6 (4.8)	67.0 (5.9)	.239
High satisfaction with social support (percentage)	61%	48%	<.001
Number of medical visits in preceding year, at baseline:	5.9 (2.1)	6.1 (2.8)	.139
Self-esteem (10–30)	16.8 (5.2)	15.9 (6.9)	.239
High physical functions score (percentage)	31%	22%	<.001
IADL disabilities (12 items)	5.00 (2.0)	6.00 (2.7)	.330

Note: All data are presented as mean ratings and (standard deviations) unless otherwise indicated. P values are based on t-tests; *denotes variables where P values are based on χ^2 tests of association.

Table 2: Psychometric information on beliefs in just world (self and others) and other related future time perspective measures and trust measures.

Measures	Mean	(SD)	α
Beliefs in just world (BJW: self) (8–48)	36.6	(6.1)	.84
Beliefs in just world (BJW: others) (8–48)	25.7	(5.8)	.74
Future time perspective (FTP) (10–70)	39.9	(5.2)	.82
Future self-continuity (FSC)—15 yrs (14–70)	34.8	(6.1)	.74
Agreement with trust items: interpersonal and society (12–60)	37.6	(5.3)	.77
Agreement with distrust items: interpersonal and society (12–60)	50.2	(6.9)	.72
Control (30–150)	80.1	(10.6)	.75
Social support satisfaction (10–30)	22.4	(3.82)	.89
Self-esteem (10–40)	23.9	(3.1)	.88

Note: The alpha denotes the coefficient alpha, a measure of internal consistency.

sample of 440 participants, the findings showed that BJW (self), FTP, and FSC were positively and significantly related to *reduced risk of mortality*. In other words, scores on these measures were *inversely* related to risk of mortality, and associated with a significantly *reduced* risk of mortality. As seen in Table 3, BJW (others), trust, self-esteem, and social support satisfaction were associated with a rather weak or marginal reduction in risk of mortality. By contrast, distrust was related to a significantly *increased risk of mortality.*

4.2. Results of Additional Analyses. We conducted a hierarchical regression analysis ($N = 440$) in order to examine further the relative contribution of the variables to risk of mortality (Table 4).

This analysis revealed the ability of BJW-self to predict risks of mortality over and above other predictor variables. As seen in Table 4 (Model 1 Model 2), results clearly show that adding the cognitive beliefs of BJW (self) variable separately contributed to the explanation of variance in mortality risks significantly and uniquely, over and above that offered by the three variables of FTP, FSC, and distrust. In other words, the results in Table 4 confirm that BJW (self) predicts an association with significant reduction in mortality risks independent of the other predictor variables. This rules out the sceptical view that the ability of BJW-Self to predict outcomes is an artifact of correlations with other factors previously associated with outcomes of BJW, for example, control or trust.

4.3. Comparisons of Extreme High Scorers and Extreme Low Scorers Using Trichotomization. Additionally, we conducted a preliminary examination of how deaths were distributed across low, average, and high trait standard scores. Our inquiry suggested possible violations of the Cox proportional

Table 3: Relative risk of death associated with beliefs in just world and other related future time perspective measures and trust measures ($N = 440$) (Range of scores).

Measures	Relative risk	95% Confidence intervals
Beliefs in just world (BJW: self) (8–48)	.798	.706, .892
Beliefs in just world (BJW: others) (8–48)	1.003	.996, 1.014
Future time perspective (FTP) (10–70)	.842	.817, .867
Future self-continuity (FSC)—15 yrs (14–70)	.865	.819, .913
Agreement with trust items: interpersonal and society (12–60)	.970	.940, 1.011
Agreement with distrust items: interpersonal and society (12–60)	1.049	.940, 1.158
Control (30–150)	1.005	.996, 1.034
Social support satisfaction (10–30)	.945	.929, .969
Self-esteem (10–40)	.970	.940, 1.011

Table 4: Hierarchical regression analysis ($N = 440$) showing ability of BJW-self to predict mortality over and above control, interpersonal and society distrust, future time perspective, and future self-continuity perspective.

Variable	Model 1		Model 2	
	β	t	β	t
Control	.061	.75	.065	1.05
Overall distrust	.459	6.18**	.399	4.44*
Future time perspective	.559	6.32**	.456	5.88**
Future self-continuity	.479	4.99*	.407	4.69*
BJW-self			.579	6.92**
Adjusted R^2	.475		.523	
F change	41.86**		18.14**	

* $P < .05$.
** $P < .001$.

hazards assumption. The proportionality assumption is violated when the relative risk of the outcome does not change in the same manner for equivalent changes in the levels of a risk factor or covariate [34]. Categorical variables may then be appropriate for an otherwise continuous variable [34] and predictors may be trichotomized based on broad cut-off points [35]. Although trichotomization may reduce statistical power, it has the possible advantage that trichotomized domain scores will be more readily interpretable [35]. Accordingly, following scores on each of the predictor variables (converted to standard scores), tertiles were computed for each of the predictor variable subscale measures. We plotted survival curves showing specifically and with greater focus on the relationship of predictor variables to mortality, with survival rates plotted for *extreme high scorers* (*at 67th percentile and above*) and *extreme low scorers* (*at 33rd percentile and below*).

Four survival curves of particular interest and relevance to our research hypotheses are portrayed in Figures 1 to 3.

Figure 1 showing the survival curve for beliefs in a just world (BJW-self) suggests that individuals with *extreme high scores* (at 67th percentile and above) on this dimension are at significantly *reduced* risk for mortality compared to individuals with *extreme low scores* (at 33rd percentile and below). The percentages for those who survived in the two groups are 79.6% and 70.7% percent, respectively, indicating a 9% difference in mortality. The ratio of fractions of decedents to survivors $(1 - 0.707)/(1 - 0.796) = 1.433$ (see Figure 1) suggests a 43.3% *lower* risk of death for *extreme* high scorers compared to *extreme* low scorers. The findings from the survival curve presented in Figure 1 further confirm the predictive ability of this measure seen earlier in Table 3

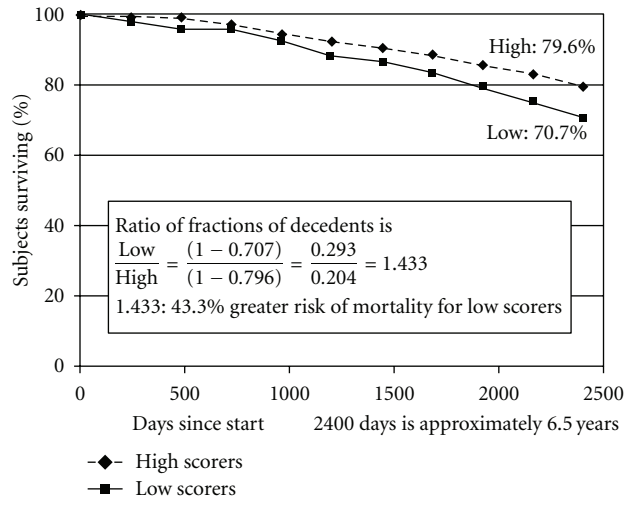
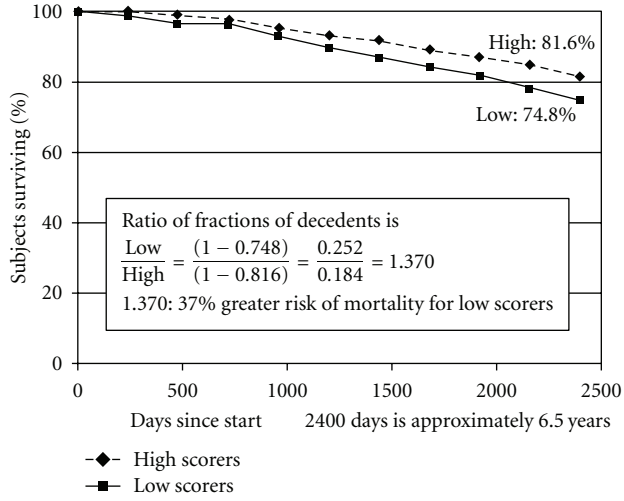
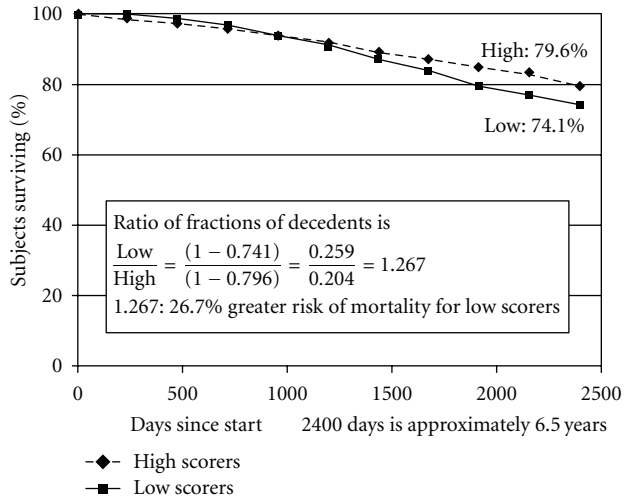


Figure 1: Survival in the groups with high and low scores on *BJW* (*self*) over approximately 6.5 years.



(a)



(b)

Figure 2: (a) Survival in the groups with high and low scores on *future time perspective* over approximately 6.5 years. (b) Survival in the groups with high and low scores on *future self-continuity* over approximately 6.5 years.

where BJW-self was used as continuous variable in the Cox regression analyses.

Figure 2(a) showing the survival curve for future time perspectives (FTPs) suggests that individuals with *extreme high scores* on this dimension (at 67th percentile and above) are at a significantly *reduced* risk *for mortality* compared to individuals with *extreme low scores* (at 33rd percentile and below). The percentages for those who survived in the two groups are 81.6% and 74.8% percent, respectively, indicating a 7% difference in mortality. The ratio of fractions of decedents to survivors $(1 - 0.748)/(1 - 0.816) = 1.370$; (see Figure 2(a)) suggests a 37.0% *reduced risk of death* for *extreme high scorers* compared to *extreme low scorers*. The findings from the survival curve presented in Figure 2(a) further confirm the predictive ability of this measure seen earlier in Table 3 where future time perspective was used as continuous variable in the Cox regression analyses.

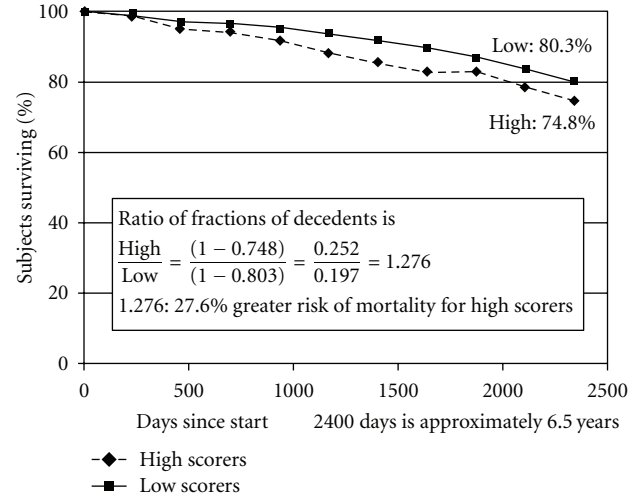


Figure 3: Survival in the groups with high and low scores on *Distrust* over approximately 6.5 years.

Figure 2(b) showing the survival curve for future self-continuity perspectives (FSCs) suggests that individuals with *extreme high scores* (at 67th percentile and above) on this dimension are at a significantly *reduced* risk for mortality compared to individuals with *extreme low scores* (at 33rd percentile and below). The percentages for those who survived in the two groups are 79.6 and 74.1 percent, respectively, indicating a 5.5% difference in mortality. The ratio of fractions of decedents to survivors $(1 - 0.741)/(1\text{-}0.796) = 1.267$ (see Figure 2(b)) suggests a 26.7% *reduced risk of death* for *extreme high scorers* compared to *extreme low scorers.* The findings from the survival curve presented in Figure 2(b) further confirm the predictive ability of this measure seen earlier in Table 3 where the future self-continuity perspective measure was used as continuous variable in the Cox regression analyses.

Conversely, the survival curve for distrust (Figure 3) suggests that individuals with *extreme high scores* (at 67th percentile and above) on this dimension, compared with those with *extreme low scores* (at 33rd percentile and below), are at a significantly *increased risk for mortality.* The percentages for those who survived in the two groups are 80.3% and 74.8%, respectively, indicating a 5.5% difference in mortality. The ratio of fractions of decedents to survivors (see Figure 3) $(1 - 0.748)/(1 - 0.803) = 1.276$ suggests a 27.6% *higher risk of death* for *extreme high scorers* on distrust, compared to *extreme* low scorers. The findings from the survival curve presented in Figure 3 further confirm the predictive ability of this measure seen earlier in Table 3 where the distrust measure was used as continuous variable in the Cox regression analyses.

To sum up the results, when the tertile split analysis was done, BJW (self), future time perspectives (FTPs) and future self-continuity perspectives (FSCs) were found to be differentially associated with mortality risks. When BJW (self) scores, future time perspective scores, and self-continuity or similarity with the future self scores were

Table 5: Relative risk of death associated with cognitive beliefs, future perspectives, and related traits after adjusting for three covariates entered in the multiple regression analysis.

Trait	β	F	Multiple R	R^2	Risk ratio model	
					RR	95% CI
Covariates combined	.54	6.55**				
BJW (Self)	−.48	5.91**	.51	.26	.832	.809, .855
BJW (Others)	.15	<1.00	.24	.06	1.004	.990, 1.018
FTP (Future time)	−.41	5.21**	.34	.12	.852	.809, .895
FSC (Future similarity)	−.31	4.27**	.28	.08	.842	.816, .868
Trust: agreement	−.18	2.02*	.20	.04	.976	.962, .990
Distrust: agreement	.37	4.57**	.26	.07	1.048	1.024, 1.072
Self-esteem	−.16	<1.00	.20	.04	.998	.964, 1.032
Social support satisfaction	−.29	2.01*	.17	.03	.935	.907, .963
Control	.21	2.11*	.14	.02	1.005	.996, 1.034
Total				.72		

* $P < 05$; ** $P < .001$; Degrees of freedom for $F = 12, 424$.
Covariates include: participants' (1) number of visits to health providers at baseline, (2) IADL index score for disability at baseline, and (3) satisfaction with family and friends support. Note: covariates were entered simultaneously. Relative risk ratio (RR) and confidence intervals (CIs) were determined after means were adjusted for covariates.

extremely low (33rd percentile and below), *mortality risk was significantly greater*, compared to *extremely high* scores on these measures (67th percentile and above). In other words, high scores on these measures represented reduced risk to mortality. However, the variable of distrust revealed a somewhat contrasting pattern in the tertile analysis. When distrust scores were *extremely low* (33rd percentile and below), *mortality risk was significantly lower*, compared to *extremely high distrust scores* (67th percentile and above) which were associated with *an increase in mortality risk*.

4.4. Controlling for Health-Related Variables. In a separate set of regression analyses, we explored the influence of three health-related covariates (Table 5). These analyses were intended mainly to see whether differences in cognitive predictors of mortality would be maintained after controlling for three health-related covariates assessed at baseline: (1) number of visits in the previous year to medical practitioners and health providers, (2) total index score for the measure of disability (IADL) in daily life activities, and (3) total score for self-rated satisfaction with family and social support. Initially, these health-related covariates were controlled for one at a time, using a Cox regression analysis procedure. However, the results of the Cox regression analyses were ambiguous. We then re-ran the analysis using multiple regression, adjusting for the covariates (Table 5).

As seen in Table 5, the negative beta values for beliefs in a just world, future time perspective, future self-continuity, and social support satisfaction, and the significant F values observed in the multiple regression analysis are consistent with the *direction of reduced risk ratios* shown in the relative risk (RR) and 95% confidence interval (CI) model (Table 3). Similarly, after adjusting for the covariates, the positive direction of the beta values and the significant F values for distrust in the multiple regression analyses is consistent with the *direction of increased risk ratios* for this variable in the relative risk (RR) and 95% CI model. Thus, overall, the relationship between cognitive beliefs about a just world, future time perspectives, and risks for mortality were maintained after adjusting for the health-related covariates. In other words, the direction of the association we saw in the earlier Cox regression analyses (Table 3) between scores on cognitive beliefs, future perspectives, distrust, and risks for mortality remained unchanged after adjusting for the health-related covariates.

In further analyses, we found no statistically significant interaction effects among the sociodemographic variables and health variables relating to the prediction of mortality.

5. Discussion and Interpretation of Results

5.1. Beliefs in Just World (BJW-Self). Consistent with our hypothesis, our findings from the Cox regression analysis of the predictor variable of BJW revealed that BJW is positively related to survival. Additionally, our findings based on a hierarchical regression analysis of a two-model design show that the variable of BJW (self) had stronger ability than all other cognitive variables explored in this research to predict mortality risk. These findings lend weight to recent theory and research suggesting that the predictive power of BJW-self seems to derive uniquely from perceived justice and that most persons have a strong need to perceive the world to be just, a belief system that conceivably serves to protect their health and survival [10, 14, 23]. Our findings that high levels of beliefs about justice and fairness are early predictors of longer survival are consistent with our hypothesis. These findings advance and extend the assumptions of earlier researchers' work on perceived justice and fairness [9, 10] as linked to better health and well being. Our findings show that individuals' concern that the world is just to themselves, as distinguished from others, is particularly predictive of their healthy survival. Along with earlier researchers, we speculate that BJW for self contributes to a larger extent to individuals' psychosocial adjustment,

providing them with useful resources in times of stress, and correspondingly becomes linked with reduced risks of mortality. In our study, BJW (others) was not differentially linked with mortality risks. However, we cannot conclude that BJW-Self alone generates the link with reduced risk of mortality. Consequently, the mortality correlates with BJW in the spheres of self and others remain to be determined more fully.

5.2. Future Time Perspective. Overall we predicted that while time is finite and limited for all, an increased or more expansive or open-ended future time perspective would reduce the risk of mortality, based on a logical assumption that a more expansive view on time left may promote valuation of future plans to safeguard the future self and protect it against risks of mortality. Consistent with our hypothesis, our findings from the Cox regression analysis (adjusted for age) show that an expansive future time perspective is associated with a reduced risk of mortality for our older sample. Our findings lend support to earlier postulates that the subjective sense of time plays an essential role in human motivation, and gradually time left becomes a better predictor than a range of other cognitive variables that contribute to behavioral and psychological processes at work in old age [11]. The present direction of findings advances and extends assumptions of future time perspectives proposed by previous researchers (e.g., [12]) according to whom individuals' perceptions of remaining time to live determines their potential for evaluating and shaping future time and future prospects concerning identity and health. Extrapolating from our findings, we speculate that incorporating an expansive time horizon influences individuals to pay relatively greater attention in the time left to make pragmatic and more practical decisions concerning a healthier future life. Additionally, we speculate that individuals in our sample who showed more expansive or open-ended time horizons at the time of baseline assessment would have already maximized attempts to preserve and protect future personal health and survival. However, our findings suggest that older adults' perception of time left is linked rather nonspecifically with risks of mortality. It remains for future researchers to disentangle the specific psychological processes that link limited horizons of time left with increased risk of mortality.

5.3. Future Self-Continuity. Overall, we predicted that increased future self-continuity and caring for future self would reduce the risk of mortality, based on a logical assumption that increased future self-continuity perceptions may promote the valuation of future plans to safeguard the future self and to protect it against risks of mortality. Consistent with our hypothesis, our findings from the Cox regression analysis (adjusted for age) show that future self-continuity as a variable is associated with reduced risk of mortality. *Extreme high scorers* on future self-continuity and caring for the future (i.e., individuals who perceived greater similarity between their present and future self), compared with *extreme low scorers* (i.e., individuals who perceived minimum similarity between their present and future self) had a significantly reduced risk of mortality (37%). Although the present results cannot specify the causal direction of the association between future self-continuity and reduced risk of mortality nor specify the health-related processes at work to explain this association, the research findings provide initial evidence that high similarity and high self-continuity with future self is a significant predictor of longevity. Overall, our findings fit well with the philosophical analysis by Parfit [36] that individuals who feel similar to their future selves are more likely to make more prudent decisions for the future, including health-related decisions, because of their perceived close connectedness to the future self. Alternatively, we speculate that individuals who perceived their current physical and mental health to be stable at the time of baseline assessments were also able to identify better with their future self as being one of healthy survival and, therefore, worth protecting. Many questions remain. Of particular interest, in the context of a longitudinal study, is the initial evidence that behavioral differences in longevity are driven differentially by perceived future self-continuity.

5.4. Trust/Distrust. Our findings from the Cox regression analysis, showing that high distrust predicted a significantly increased risk of mortality, are consistent with our hypothesis. These findings are not at variance with earlier theoretical proposals suggesting that historically older adults have had difficulty in accepting the status quo as defined by younger authorities in the social system and may tend to be more distrusting of those authorities. Our findings suggest that extreme high levels of distrust which typified some individuals in our sample, compared with extreme low levels of trust, increased the risk of mortality by 27.6 percent. Overall, the Cox regression analysis revealed that distrust is empirically predictive of an increased risk of mortality. We speculate that high levels of distrust may be analogous to a form of pathological social isolation or breakdown in communications that leads to increased risk for physical and psychological disease [37, 38]. Conceivably distrust becomes linked with a shorter life span.

5.5. Study Limitations. Despite the novelty of our data, and thus the importance of their contribution to cognitive etiology of health-related processes such as survival, mortality, or longevity, our study was subject to the limitation that the sample was a volunteer one. Our cognitive scale measures of beliefs in a just world, and other measures exploring individuals' perspectives on future self-continuity and future time, trust and distrust were self-report measures and subject to the biases associated with self-report measures. The preceding sources of unreliability of measurement could potentially both reduce or increase the proportion of variance in the outcome variables for which our models were able to account. However, we are confident that we had a sufficiently large and representative sample of urban and rural older adults, and the measures we used showed high internal consistency.

5.6. Conclusions. Our study provided novel indications that cognitive belief systems and future time perspectives of

older adults may have much to contribute to increased or reduced mortality risks in late life. Thus, we suggest that basic cognitive beliefs and future time perspectives that were examined in the present research for their association with increased or reduced risk for mortality are an important direction for future research. It remains for future researchers to try to integrate these various cognitive factors into some kind of theoretical frameworks to gain a precise appreciation of how these factors may be causally linked with longevity.

5.7. Practical Implications for Clinical Practice. Extrapolating from the findings of the study concerning the association between beliefs in a just world and the potentials for a longer survival, it may be reasonable to postulate that the findings have possible implications for clinical practice with older adults who may be dealing with traumatic life events such as life threatening illness, loss of loved ones, and other natural disasters. It is conceivable that these individuals may, as a result of adverse and stressful experiences, feel an abrupt decline in their beliefs in a just world or a decline in their faith or trust in interpersonal connectedness. It is suggested that clinicians be particularly mindful of clients who score extremely low on measures of BJW and future time perspectives. Conceivably, they are at greater risk of mortality. Similarly, those individuals scoring extremely high on measures of distrust and control may be at greater risk of mortality. By exploring with older clients their beliefs about a just world and their trust or faith in interpersonal connectedness, geriatric service providers and psychologists may be able to assist clients to develop a realistic self-profile of their sources of life strengths in relation to self-growth, resilience, and control over every day life situations. Thus, clinicians may be able to assist elderly clients to draw on internal sources of strengths, such as reflecting on their beliefs about a just world and their beliefs about trusting others, as a means to strengthening and enhancing future self-continuity. Based on our findings suggesting that individuals' strong beliefs in a just world and positive perspectives regarding future time may possibly protect them against risk to mortality, geriatric service providers are encouraged to engage in a dialogue with their clients on these themes. Such dialogues may not only serve to act as buffers for clients against stress and anxiety about the future, but may also assist clients in building up greater motivation for living.

From a pragmatic standpoint, we postulate that interventions aimed at strengthening individuals' beliefs in a just world at an earlier age, and encouraging future-self continuity in the earlier adulthood years, might motivate people to care more for their potential person in the later years, and conceivably keep people healthier longer and thereby reduce risk of mortality. Similarly, communication programs that foster interpersonal and institutional trust or help older adults combat high levels of distrust (which in our research were associated with significantly increased risk of mortality) may be of value to a longer survival.

Conflict of Interests

The authors declare that they have no conflict of interests.

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References

[1] B. W. Roberts, N. R. Kuncel, R. Shiner, A. Caspi, and L. R. Goldberg, "The power of personality: the comparative validity of personality traits, socioeconomic status, and cognitive ability for predicting important life outcomes," *Perspectives on Psychological Science*, vol. 2, no. 4, pp. 313–345, 2007.

[2] H. S. Friedman, J. S. Tucker, J. E. Schwartz et al., "Psychosocial and behavioral predictors of longevity: the aging and death of the "termites"," *American Psychologist*, vol. 50, no. 2, pp. 69–78, 1995.

[3] P. S. Fry and D. L. Debats, "Perfectionism and the five-factor personality traits as predictors of mortality in older adults," *Journal of Health Psychology*, vol. 14, no. 4, pp. 513–524, 2009.

[4] R. D. Goodwin and H. S. Friedman, "Health status and the five-factor personality traits in a nationally representative sample," *Journal of Health Psychology*, vol. 11, no. 5, pp. 643–654, 2006.

[5] P. S. Fry and D. L. Debats, "Perfectionism and other related trait measures as predictors of mortality in diabetic older adults: a six-and-a half-year longitudinal study," *Journal of Health Psychology*. In press. The online version of this article can be found at http://hpq.sagepub.com/content/early/2011/03/26/1359105311398684.

[6] L. R. Martin, H. S. Friedman, J. S. Tucker et al., "An archival prospective study of mental health and longevity," *Health Psychology*, vol. 14, no. 5, pp. 381–387, 1995.

[7] C. Dalbert, *The Justice Motive as a Personal Resource: Dealing with Challenges and Critical Life Events*, Plenum Press/ Kluwer Academic Publishers, New York, NY, USA, 2001.

[8] N. T. Feather, "Human values, global self-esteem, and belief in a just world," *Journal of Personality*, vol. 59, no. 1, pp. 83–107, 1991.

[9] I. M. Lipkus and I. C. Siegler, "The belief in a just world and perceptions of discrimination," *The Journal of psychology*, vol. 127, no. 4, pp. 465–474, 1993.

[10] I. M. Lipkus, C. Dalbert, and I. C. Siegler, "The importance of distinguishing the belief in a just world for self versus for others: implications for psychological well-being," *Personality and Social Psychology Bulletin*, vol. 22, no. 7, pp. 666–677, 1996.

[11] L. L. Carstensen, "The influence of a sense of time on human development," *Science*, vol. 312, no. 5782, pp. 1913–1915, 2006.

[12] S. Frederick, *Discounting, time preference, and identity*, dissertation, Department of Social and Decision Sciences. Carnegie-Mellon University, 1999.

[13] H. Ersner-Hershfield, M. T. Garton, K. Ballard, G. R. Samanez-Larkin, and B. Knutson, "Don't stop thinking about tomorrow: individual differences in future self-continuity account for saving," *Judgment and Decision Making*, vol. 4, no. 4, pp. 280–286, 2009.

[14] C. L. Hafer and L. Bègue, "Experimental research on just-world theory: problems, developments, and future challenges," *Psychological Bulletin*, vol. 131, no. 1, pp. 128–167, 2005.

[15] J. Tomaka and J. Blascovich, "Effects of justice beliefs on cognitive appraisals of subjective, physiological, and behavioral responses to potential stress," *Journal of Personality and Social Psychology*, vol. 67, no. 4, pp. 732–740, 1994.

[16] C. Ritter, D. E. Bensen, and C. Snyder, "Belief in a just world and depression," *Sociological Perspectives*, vol. 33, pp. 235–252, 1990.

[17] W. H. Jones, J. E. Freemon, and R. A. Goswick, "The persistence of loneliness: self and other determinants," *Journal of Personality*, vol. 49, pp. 27–48, 1981.

[18] D. Paulhus, "Sphere-specific measures of perceived control," *Journal of Personality and Social Psychology*, vol. 44, no. 6, pp. 1253–1265, 1983.

[19] J. B. Rotter, "A new scale for the measurement of interpersonal trust," *Journal of personality*, vol. 35, no. 4, pp. 651–665, 1967.

[20] M. J. Lerner and D. T. Miller, "Just world research and the attribution process: looking back and ahead," *Psychological Bulletin*, vol. 85, no. 5, pp. 1030–1051, 1978.

[21] C. L. Hafer and J. M. Olson, "Beliefs in a just world and reactions to personal deprivation," *Journal of Personality*, vol. 57, pp. 799–823, 1989.

[22] S. E. Taylor and J. D. Brown, "Illusion and well-being: a social psychological perspective on mental health," *Psychological Bulletin*, vol. 103, no. 2, pp. 193–210, 1988.

[23] L. Bègue and M. Bastounis, "Two spheres of belief in justice: extensive support for the bi-dimensional model of belief in a just world," *Journal of Personality*, vol. 71, no. 3, pp. 435–463, 2003.

[24] R. J. Bulman and C. B. Wortman, "Attributions of blame and coping in the "real world": severe accident victims react to their lot," *Journal of Personality and Social Psychology*, vol. 35, no. 5, pp. 351–363, 1977.

[25] F. R. Lang and L. L. Carstensen, "Time counts: future time perspective, goals, and social relationships," *Psychology and Aging*, vol. 17, no. 1, pp. 125–139, 2002.

[26] L. L. Carstensen, *Future Time Perspective Scale*, Stanford University, Stanford, Calif, USA, 1996.

[27] A. Aron, E. N. Aron, and D. Smollan, "Inclusion of other in the self scale and the structure of interpersonal closeness," *Journal of Personality and Social Psychology*, vol. 63, no. 4, pp. 596–612, 1992.

[28] M. Rosenberg, C. Schooler, C. Schoenbach, and F. Rosenberg, "Global self-esteem and specific self-esteem: different concepts, different outcomes," *American Sociological Review*, vol. 60, pp. 141–156, 1995.

[29] G. D. Zimet, N. W. Dahlem, S. G. Zimet, and G. K. Farley, "The multidimensional scale of perceived social support," *Journal of Personality Assessment*, vol. 52, pp. 30–41, 1988.

[30] D. R. Cox, "Regression models and life tables," *Journal of the Royal Statistical Society Series B*, vol. 74, pp. 187–220, 1972.

[31] J. L. Kelsey, A. S. Whittmore, A. Evans, and W. D. Thompson, *Methods in Observational Epidemiology*, Oxford University Press, New York, NY, USA, 1996.

[32] P. S. Fry and D. L. Debats, "Psychosocial resources as predictors of resilience and healthy longevity of older widows," in *New Frontiers in Resilient Aging: Life-Strengths and Well Being in Later Life*, P. S. Fry and C. L. M. Keyes, Eds., pp. 185–212, Cambridge University Press, Cambridge, UK, 2010.

[33] A. E. Korten, A. F. Jorm, Z. Jiao et al., "Health, cognitive, and psychosocial factors as predictors of mortality in an elderly community sample," *Journal of Epidemiology and Community Health*, vol. 53, no. 2, pp. 83–88, 1999.

[34] J. D. Kalbfleisch and R. L. Prentice, *Statistical analysis of failure time data*, Wiley, New York, NY, USA, 1980.

[35] A. Weiss and P. T. Costa, "Domain and facet personality predictors of all-cause mortality among medicare patients aged 65 to 100," *Psychosomatic Medicine*, vol. 67, no. 5, pp. 724–733, 2005.

[36] D. Parfit, "Personal identity," *Philosophical Review*, vol. 80, pp. 3–27, 1971.

[37] G. D. Mellinger, "Interpersonal trust as a factor in communication," *Journal of Abnormal and Social Psychology*, vol. 52, no. 3, pp. 304–309, 1956.

[38] P. S. Fry and D. L. Debats, "Sources of life strengths as predictors of late-life mortality and survivorship," *International Journal of Aging and Human Development*, vol. 62, no. 4, pp. 303–334, 2006.

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Research Article

Why Are Native Hawaiians Underrepresented in Hawai'i's Older Adult Population? Exploring Social and Behavioral Factors of Longevity

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Native Hawaiians comprise 24.3% of Hawai'i's population, but only 12.6% of the state's older adults. Few published studies have compared health indicators across ethnicities for the state's older adult population or focused on disparities of Native Hawaiian elders. The current study examines data from two state surveillance programs, with attention to cause of death and social-behavioral factors relevant to elders. Findings reveal that Native Hawaiians have the largest years of productive life lost and the lowest life expectancy, when compared to the state's other major ethnic groups. Heart disease and cancer are leading causes of premature mortality. Native Hawaiian elders are more likely to report behavioral health risks such as smoking and obesity, live within/below 100–199% of the poverty level, and find cost a barrier to seeking care. Indicated is the need for affordable care across the lifespan and health services continuum. Future research might explain behavioral factors as influenced by social determinants, including historical trauma on Native Hawaiian longevity.

1. Introduction

Native Hawaiians are descendents of the aboriginal peoples inhabiting the Hawaiian archipelago prior to western contact in 1778 and exercising sovereign governance prior to the 1892 overthrown of the Hawaiian Kingdom by the United States (USA) [1, 2]. The 2000 US census enumerated 401,000 Americans (0.1% of the total population) of full or part-Hawaiian ethnicity, about 60% of whom reside in the State of Hawai'i. Native Hawaiians comprise about 24.3% of the state's current population [3].

As in other states, the population of Hawai'i is aging, with an increasing number of residents living into old age. Although life expectancy in Hawai'i exceeds that of other US states, studies conducted within the state reveal continuing ethnic differences in life expectancy [3]. As depicted in Figure 1, over six decades (1950–2000) Native Hawaiians have consistently had the lowest life expectancy when compared to the state's three other largest groups, namely, Caucasians, Filipinos (Americans), and Japanese (Americans). Notably, the magnitude of this disparity—about 10 years lower than the longest lived group—has not changed over time. About 16% of deaths among Native Hawaiians in 2005 occurred before 45 years, which is at least two times higher than for any other ethnic group living in the state [4]. Mortality disparities are particularly significant when comparing Native Hawaiians with Japanese; in 2005, 60% of deaths among Japanese occurred at age 80+ years, compared to only 25% of Native Hawaiians.

As a result, Native Hawaiians are underrepresented in the older age groups. In 2008, 21.4% of the state's overall population was 60+ years of age but only 11.1% of Native Hawaiians are in this age group. To look at it another way, Native Hawaiians comprised 24.3% of the total state

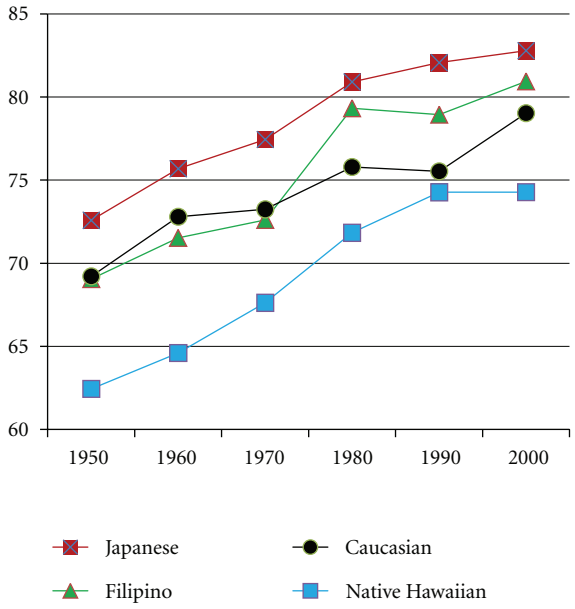

Figure 1: Life expectancy by ethnicity in Hawai'i, 1950 through 2000.

population in 2008, but only 12.6% of residents age 60+ are Native Hawaiians. Table 1 displays population totals and age distributions of the state's four largest ethnic groups.

Other investigators have examined data for the Native Hawaiian population in general and have found that Native Hawaiians have a higher prevalence of obesity, numerous chronic conditions, and greater impoverishment than other ethnic groups living in the state [5–7]. The relatively poor health status of the Native Hawaiian population overall has been of significant interest since the publication of the seminal *E Ola Mau: The Native Hawaiian Health Needs Study Report* [5]. Historic difficulty in Native Hawaiians' use of western mainstream healthcare services due to socioeconomic disadvantage, discrimination, and cultural misunderstanding are highlighted. Subsequently, social welfare researchers in gerontology and Native Hawaiian health have documented a number of socioeconomic disparities (e.g., higher rates of poverty, lower educational levels, homelessness, and incarceration) as well as health disparities (e.g., higher rates of diabetes and certain types of cancer, lower utilization of health services). To improve Native Hawaiian health and well-being, these researchers have underscored the need to consider more distal factors such as historical trauma or the cumulative effect of negative physical, sociocultural, political, and economic changes on current health disparities [7–12]. Concomitantly, they articulate the strengths of traditional Native Hawaiian culture, including those cultural values and practices on health, care giving, and social support that might be integrated into interventions which offer the prospect of increased longevity and enhanced quality of life [8, 10, 11].

Importance of this research notwithstanding, no recent studies have compared health indicators by ethnicity for older adults in Hawai'i with a specific focus on Native Hawaiian elders [8]. Research described here attempts to address this gap in the current knowledge and was conducted by researchers associated with *Hā Kūpuna*: National Resource Center for Native Hawaiian Elders. Funded by the US Administration on Aging (AoA), *Hā Kūpuna* is one of three resource centers for native elders in the USA. The name "*Hā Kūpuna*" derives from the Native Hawaiian cultural belief that one's life essence (spiritual energy, ancestral knowledge) is transmitted to others through sharing of the *hā* (breath of life). It is believed that such sharing allows *kūpuna* (elders) to pass on vital knowledge and wisdom to subsequent generations, thus perpetuating valued cultural traditions and a positive sense of identity. Grounded in these and other traditional Native Hawaiian cultural values, *Hā Kūpuna* seeks to assure the transmission of *hā* from *kūpuna* (elders) to younger generations by achieving parity in life expectancy and good health among Native Hawaiian older adults comparable to that of other older Americans [13]. As a center dedicated to the health of Native Hawaiian elders, *Hā Kūpuna* develops and disseminates knowledge to inform policy and service innovations.

In this paper, we examine proximal influences to the health and longevity of Native Hawaiian elders, specifically describe the causes of premature mortality among Native Hawaiians, and identify the ways in which sociodemographic and behavioral factors of Hawai'i's elders vary by ethnicity. Our research was guided by these questions: (1) what are causes of premature mortality? (2) How does this vary by ethnicity? and (3) How do sociodemographic and health behavioral indicators vary by ethnicity? To address these questions we reviewed relevant statistical data collected by two Hawai'i Department of Health surveillance programs.

2. Materials and Methods

Written requests for data were submitted to two surveillance programs of the Hawai'i Department of Health (DOH): Vital Records and the Hawai'i Behavioral Risk Factor Surveillance System (HBRFSS). Data sources are briefly described, as well as methods used to arrive at population-based statistics relevant for examining the underrepresentation of Native Hawaiian elders in the state's older (≥60 years) adult population.

2.1. Causes of Premature Mortality. We requested data on the state's largest ethnic groups from the Vital Records program, which routinely gathers information on births, deaths, and marriages that take place in the state. Based on death record data, we calculated the Years of Productive Life Lost (YPLL) for the state's largest ethnic groups. YPLL is an index that measures the extent of premature mortality by giving a weight to each premature death from the predetermined cutoff age, with proportionally higher weights for younger deaths [14]. Any death before the cutoff age is defined as a premature death. Because YPLL is especially sensitive to the age distribution of the population, it cannot be used in cross-ethnic-group comparisons [15]. However, YPLL can be converted to a rate that is independent of sample size and

Table 1: Population totals and distributions of young people, adults, older adults by four largest ethnic groups, Hawaii, 2008.

	Native Hawaiian	Caucasian	Filipino	Japanese	Total
	305,838	259,851	200,042	273,016	1,257,607
	24.3%	20.7%	15.9%	21.7%	100%
column %					
<20 years	38.0%	13.1%	29.2%	17.2%	25.4%
Age 20–59	50.9%	55.3%	55.1%	51.0%	53.3%
Age 60+	11.1%	31.6%	15.7%	31.8%	21.4%%
Total	100.0%	100.0%	100.0%	100.0%	100.0%%
row %					
<20 years	36.4%	10.6%	18.3%	14.7%	100.0%
Age 20–59	23.3%	21.5%	16.4%	20.8%	100.0%
Age 60+	12.6%	30.6%	11.7%	32.3%	100.0%

population distribution following a method proposed by Lee [16]. These rates, herein called Total Population Life Lost per person in a lifetime (TPLL), can be compared across ethnic groups.

To construct TPLL, we obtained resident death data from Hawai'i for the 3-year period, 1999–2001, for each age group by ethnicity, sex, and underlying cause of death of the deceased. Averaging 3 years of death data was done to smooth annual fluctuations in death. Cause of death is coded following the International Classification of Disease 10th Revision (ICD-10) [17]. We analyzed six causes of death common among older adults—cancer, heart disease, cerebrovascular disease, unintentional injuries, suicide, and diabetes—in addition to total death.

Both population and death data were classified into age groups, <1, 1–4 years, and then in 5-year intervals. All deaths in a given age group are assumed to have occurred at the midpoint age of the interval. We set age 70 as the cutoff for premature mortality in conformance with recommendations of the National Center for Health Statistics [18]. With the upper limit for the premature death at 70, the YPLL owing to premature mortality is given by

$$\text{YPLL} = \sum (70 - c_i) d_i, \tag{1}$$

where, c_i is the midpoint of the ith age interval and d_i is the number of deaths in the ith age interval. Thus (70-c_i) is the weight given to the deaths in the ith age group. The summation runs from age 0 to 70. For the cause-specific YPLL, d_i is the number of deaths from that particular cause of death. YPLL, as a function of d_i, is to a large extent determined by the size and age distribution of the population as well as premature mortality.

To convert YPLL to a rate, we applied the index proposed by Lee [16], which is independent of population size and age distribution. In Lee's method, the age-specific weight of YPLL is multiplied by the age-specific death rate of the population, that is,

$$\sum \frac{\left(70 - c_j\right) d_j}{p_j}, \tag{2}$$

where p_j is the population of age j. The resultant is the annual *number* of YPLL expected to occur *per person* in age j. Originally, Lee named this measure as the cumulative rate of potential life lost (CRPLL). But here we call it the total potential life lost per person in lifetime (TPLL), since it also presents the number of premature deaths. When the age is grouped, TPLL is expressed by

$$\text{TPLL} = \sum \frac{n_i (70 - c_i) d_i}{p_i}, \tag{3}$$

where n_i is the length of the interval of the age group. This is the total number of YPLL expected per person before age 70 in the study population. To establish 95% confidence intervals (CIs), we estimated the standard deviation of TPLL in each ethnic group

$$\sum \frac{[n_i (70 - c_i)]^2}{p_i} \frac{d_i}{p_i} \frac{p_i - d_i}{p_i}. \tag{4}$$

Further, as the 3-year average of death was used for d_i, the variance of our TPLL is the above quantity divided by 3.

2.2. Variations in Socioeconomic, Clinical, and Behavioral Factors. We requested special data runs from the Hawai'i Behavioral Risk Factor Surveillance System (HBRFSS) to examine ethnic variation in sociodemographic, clinical, and behavioral factors in the state's elders. Part of the Behavioral Risk Factor Surveillance System (BRFSS) of the Centers for Disease Control and Prevention (CDC), the HBRFSS gathers data by telephone from about 6,000 randomly selected adults (≥18 years of age). Respondents are queried about behaviors that directly or indirectly affect health and health-related topics. For example, BRFSS solicits information on height and weight to calculate body mass index (BMI), health behaviors (e.g., smoking), health screening (e.g., use of breast, cervical and colorectal cancer early detection screening), and chronic disease (e.g., diabetes, hypertension). Hawai'i sample data are adjusted and weighted based on ethnicity distributions, and estimates are produced for Native Hawaiians, Caucasians, Filipinos, and Japanese. We requested a special tabulation that averaged responses from three years of data (2005, 2006, and 2007) relevant to older

Table 2: Years of Total Potential Life Lost (TPLL) and standard errors for the total population and 4 ethnic groups by cause of death, 1991–2001.

Cause	Native Hawaiian	Caucasian	Filipino	Japanese	Total population
All Causes	5.28 (0.12)	3.38 (0.10)	3.03 (0.11)	2.57 (0.11)	3.30 (0.05)
Cancer	1.16 (0.05)	0.68 (0.03)	0.70 (0.04)	0.64 (0.04)	0.74 (0.02)
Heart Disease	1.35 (0.06)	0.48 (0.03)	0.52 (0.04)	0.37(0.03)	0.59 (0.02)
Injuries	0.59 (0.05)	0.52 (0.04)	0.31 (0,04)	0.22 (0.04)	0.40 (0.02)
Suicide	0.29 (0.03)	0.24 (0.03)	0.19 (0.03)	0.18 (0.03)	0.21 (0.01)
Stroke	0.22 (0.02)	0.07 (0.01)	0.15 (0.02)	0.16 (0.02)	0.13 (0.01)
Diabetes	0.16 (0.02)	0.05 (0.01)	0.06 (0.01)	0.04 (0.01)	0.06 (0.01)

adults (≥60 years of age) residing in Hawai'i. After years of data were combined, HBRFSS provided data tables for older adults as a whole, and for the four largest ethnic groups. The sample size of older adults over three years included 658 Native Hawaiians, 2,652 Caucasians, 561 Filipinos, and 1,840 Japanese, for a total of 6,346 elders.

3. Results and Discussion

3.1. Results: Causes of Premature Mortality. For Hawai'i, the overall TPLL before age 70 per person from premature mortality was estimated at 3.3 years in 2000, but TPLL varied by ethnic group (Table 2). Among the groups we analyzed, the smallest number of years lost was observed for Japanese at 2.6 years and, as expected, the largest was for Native Hawaiians, at 5.3 years. The difference in TPLL between Japanese and Native Hawaiians is twofold, meaning that, on average, Native Hawaiians are losing twice as many years of potential life as Japanese. Among the six causes of death, cancer caused the largest number of potential years lost before age 70, with 0.74 years per person, accounting for more than 22% of overall TPLL. Heart disease, ranking second (0.59 years), accounted for 18% of overall TPLL. Unintentional injuries ranked third (0.40 years), accounting for 12% of overall TPLL. Death from suicide (0.21 years), stroke (0.13 years), and diabetes (0.06 years) ranked fourth, fifth, and sixth, respectively.

As illustrated in Figure 2, the importance of cause of death varied considerably by ethnic group. Cancer was the most important cause of TPLL in all populations, except Native Hawaiians, while heart disease was top-ranked for Native Hawaiians. While cancer and heart disease were the top-leading causes of TPLL in most ethnic groups, accidents ranked second for Caucasians, while accidents ranked third for other groups. Suicide, cerebrovascular disease, and diabetes ranked fourth, fifth, and sixth in each ethnic group. Native Hawaiians had the highest TPLL for each of the six causes of death, almost twofold higher for cancer than the other groups, two-to-four times higher for heart disease, and two-to-three times higher for diabetes.

3.2. Results: Sociodemographic and Behavioral Indicators. Sociodemographic data from HBRFSS indicate that Native Hawaiian older adults have the largest proportion of females to males; women account for 61.5% of Hawaiian elders

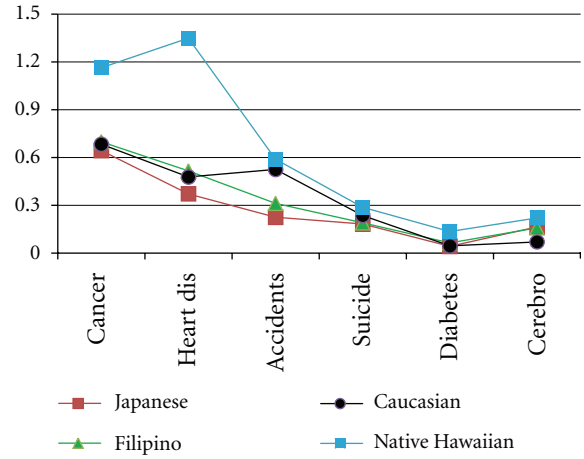

Figure 2: Total years of potential life loss by ethnic group and disease, 1999–2001.

compared to Caucasian females who account for 50.9% of Caucasian elders. This suggests that a disproportionate amount of Native Hawaiian men are dying before the age of 60 years. About 38.2% of Native Hawaiian elders attended at least one year of college. This percent is similar to that of Filipino elders (39.7%), but much lower than for Japanese (56.1%) and Caucasians (72.7%). Further, about 24% of Native Hawaiian elders are employed (perhaps indicating that they have had to delay retirement due to higher impoverishment rates), compared to only 18% of Japanese elders.

According to HBRFSS, the percentage of elders with health insurance was relatively high overall (97%) although the percentage of Native Hawaiian elders reporting access to health insurance was 95%. Approximately 7.2% of Native Hawaiian elders reported not being able to see a physician in the past year due to cost, compared to less than 1% of Japanese elders. Native Hawaiian elders reported the highest prevalence of asthma (20% versus 11% overall) and diabetes (25% versus 17% overall). However, Native Hawaiians reported prevalence of heart attached, angina/CHD, and stroke similar to Caucasians.

HBRFSS data indicate a relatively high prevalence of behaviors associated with increased disease risk among Native Hawaiian elders. For example, Native Hawaiian elders were most likely of the four ethnic groups to smoke every

Table 3: Health indicators in 60+ population by ethnicity, HBRFSS, 2005–07.

		All people age 60+	Native Hawaiian	Caucasian	Filipino	Japanese
Self-rated helath, insurance, checkups, MD visits	Rate health as fair/poor	22.7	26.3	19.0	28.0	22.5
	Need adaptive equipment	12.8	18.0	12.8	14.0	11.2
	Health insurance	97.2%	95.3%	96.8%	97.5%	98.4%
	Could not see MD in past 12 months due to cost	3.2%	7.2%	3.8%	3.8%	0.6%
	No PCP	4.9%	6.3%	7.0%	3.8%	2.8%
	Routine checkup in past year	82.9%	81.0%	79.4%	81.3%	86.4%
Chronic conditions	Told by MD with asthma	10.8%	19.7%	11.8%	10.2%	7.6%
	Told by MD have diabetes	16.5%	24.9%	11.9%	23.8%	14.9%
	Told by MD had heart attack	9.6%	11.3%	12.0%	8.7%	7.2%
	Told by MD have angina/CHD	8.6%	11.4%	10.1%	6.4%	6.5%
	Told by MD had stroke	6.8%	7.2%	7.5%	4.4%	6.7%
Lifestyle risks	Smoked 100+ cigarette in lifetime	48.1%	56.8%	57.0%	37.2%	43.1%
	Current smokers (daily/occasional)	8.9%	14.6%	9.8%	9.7%	6.9%
	Binge drinkers (4-5 + drinks at once)	7.0%	8.9%	9.0%	4.0%	5.9%
	Obese	16.5%	36.2%	19.2%	16.1%	7.7%
	Participated in physical activity outside of regular job in past 30 days	77.0%	72.9%	81.4%	72.1%	75.6%
Screening practices	For women, had mammogram in last 2 years	81.3%	77.7%	76.2%	82.3%	85.8%
	For men, had PSA test in past 12 months	54.0%	37.7%	58.8%	33.7%	60.5%
	For men, had DRE in past 12 months	45.0%	34.2%	48.6%	28.2%	49.7%
	Ever had sigmoidoscopy or colonoscopy	66.0%	52.7%	70.6%	47.3%	73.0%

day or occasionally, about 14.6% compared to only 6.9% of Japanese elders (although Native Hawaiian elders with a history of smoking were most likely to state that they were trying to quit or have stopped smoking at least for one day in the past year). Native Hawaiian and Caucasian elders were most likely to report drinking more than four or five alcoholic beverages on one occasion (about 9% in each group). About one-third of all ethnic groups reported that they were overweight, however 36% of Native Hawaiian elders were obese compared to only 8% of Japanese elders.

Data from HBRFSS indicate that 28% of Filipinos, 26% of Native Hawaiians, 23% of Japanese, and 19% of Caucasian elders rated their personal health as fair or poor. About 18% of Hawaiian elders responded that they were most likely to need special equipment, in comparison to 13% of all elders. About 80% of female elders reported having a mammogram within the last two years, including 78% of Native Hawaiian female elders. Among male elders, Native Hawaiians and Filipinos were the least likely to have had a Prostate-Specific Antigen (PSA) test for prostate cancer in the past 12 months (all male elders = 54%, Native Hawaiians = 37.7%, Filipinos = 33.7%). About 47% of Filipino elders and about 53% of Hawaiian elders have ever had a sigmoidoscopy or colonscopy to check for colorectal cancer, compared to 71% of Caucasians and 73% of Japanese.

4. Discussion

Data from two state surveillance programs highlight ethnic differences in cause of death, health, and behavioral indicators that may help to explain some of the ethnic differences in life expectancy. To summarize, the data demonstrate clear disadvantages for Native Hawaiian elders. For example, Native Hawaiian elders are least likely to have attended college and most likely to have incomes below 200% of poverty. They are least likely to have access to a Primary Care Physician, have routine checkups, or to participate in early detection cancer screening. Further, they are most likely to smoke and be obese. In comparison to other major ethnic groups, Native Hawaiian elders have the highest or second highest prevalence of a number of chronic diseases, including asthma and diabetes. Heart disease is the leading cause of premature death for Native Hawaiians, and Native Hawaiians lose significantly more years of productive life to heart disease, cancer, injuries, suicide, stroke, and diabetes than other ethnic groups. These findings for older adults are consistent with earlier research on the general Hawaiian population that documents serious health and social disparities [3–6, 9].

There were four major limitations to our research; these limitations point to a number of remaining and critical gaps in knowledge about Native Hawaiian elders. First,

the study describes, but does not explain, the relationship of ethnicity and health indicators. Thus, caution must be exercised in using findings to directly inform policy or practice innovations. Continued research is needed to explain the relationship of Native Hawaiian ethnicity and proximal health indicators. Second, the study captured ethnic variation in health outcomes; the role of gender as it interacts with ethnicity was not explored. Research across the life course consistently exposes disparities by gender, and this is especially so in later life [19, 20]. For example, poverty rates for older women are nearly twice as high as for men. Our understanding of the lives of Native Hawaiian elders will be strengthened by the inclusion of gender in analyses of social and health disparities. Third, this study used surveillance data collected from Hawai'i residents only; thus, findings cannot be generalized to populations outside the state, including Native Hawaiians living on the North American continent. At present, our knowledge of Hawaiian elders residing in the continental USA is very limited. This is an important omission since nearly 40% of Native Hawaiians live in the contiguous states, primarily California, Washington, and Oregon [21]. Extending life expectancy and improving quality of life requires that we understand life expectancies, health status, health care needs, preferences for care, and utilization patterns of all Native Hawaiian elders. Future research is needed to determine if Native Hawaiians on the continent experience similar disparities, such as shorter life expectancies than other ethnic groups in their new communities.

Fourth and finally, the current study relied on surveillance data that did not address distal factors, such as the influence of historical trauma and systemic discrimination on current health disparities. Native Hawaiian and other indigenous health researchers consistently emphasize the influence of historical trauma and intergenerational marginalization on current health disparities [1, 5, 8–12, 22–26]. Sotero's Conceptual Model of Historical Trauma [25] posits that subjugation of a population by a dominant group has a cumulative effect on the physical, sociocultural, political, and economic well-being of the oppressed group. The trauma is felt by first-generation survivors (i.e., those who directly experienced the traumatic events), as well as by successive generations of their descendents. Across succeeding generations, the traumatic impact may be mitigated to some degree by cultural resiliency and other group protective factors. However, the result is an excess of social and physical ills that ultimately lead to population-specific health disparities.

In the case of Native Hawaiians, historical records document the precipitous decline in population numbers and health status of Native Hawaiians following contact with the West [26]. At the earliest known point of contact in 1778, members of the Cooke exploratory expedition describe natives of the islands as hardy, robust, and capable of great physical activity. However, during the first 150 years of western contact, Native Hawaiians suffered disability and premature death from foreign diseases such as influenza, measles, small pox, syphilis, and mumps [1, 23]. Depopulation was severe; in the first century of contact the native population declined from an estimated 800,000 to 50,000 [26]. Depopulation was accompanied by cultural degradation with the native language and many traditional practices outlawed and/or subordinated as inferior to the English language and western practices, respectively [23, 24].

Among the most devastating changes were those related to shifts in the land tenure system from one of collective stewardship to one of private ownership and monopoly capitalism [24]. This shift assured the rise of westerner-owned plantations and eventually, led to the overthrow of the Hawaiian Kingdom in 1892 and US annexation in 1898. The exponential growth of the plantation economy and subsequent loss of Native Hawaiian sovereignty coupled with severe depopulation caused a collective grief among Native Hawaiian survivors [9, 23, 24]. *Na maka'ainana* (those who tend the land, common people) were alienated from their *'aina* (land), the source of their spiritual, social, and economic well-being. As natives lost access to their land, there was a mass exodus to port cities where they became wage laborers and in some cases, debtors, paupers, and *na pa'ahao* (prisoners, convicts) [12, 24].

In line with Sotero [25], Native Hawaiian health researchers are linking the poor health of Native Hawaiians in contemporary times to this cascade of adverse historic events and intergenerational social marginalization [9, 10, 12]. Despite the relative success of Native Hawaiian organizations and groups to build cultural pride, positive identity, and holistic health in communities, the social marginalization of Native Hawaiians persists as reflected in social indicators spanning the breadth of the life cycle. For example, Native Hawaiian children are overrepresented in the state's child welfare population, Native Hawaiian youth are disproportionately represented in the state's juvenile justice system, and Native Hawaiian adult men and women are overrepresented at every stage of Hawai'i's criminal justice system [2, 27].

The overall picture remains one of a population with more social and health disparities in comparison to the other large ethnic groups in the state. In the last few decades some social policies have been enacted to redress past wrongs and support programs aimed at reducing the disparate health outcomes affecting Native Hawaiians (e.g., Public Law 100–579, the Native Hawaiian Health Care Act of 1988). However, it is clear that more must be done to increase the longevity of Native Hawaiians and enhance the quality of life among Native Hawaiian elders.

Our findings underscore the ongoing need to target behavioral risks affecting Native Hawaiian longevity across the life span. Health promotions grounded in evidence-based strategies and tailored on Native Hawaiian cultural preferences, as well as socio-economic circumstances have been recommended to reduce smoking and sedentary lifestyle, improve dietary practices, and increase participation in early detection screening [9–13]. In the past, health promotions that disregard relevant cultural, socio-economic factors, and systemic barriers have been received with distrust and even resentment by Native Hawaiian consumers who have experienced such efforts as cultural impositions [1, 12]. Thus, in targeting behavioral risks affecting longevity,

it is crucial to develop interventions that adhere to principles of Community-Based Participatory Research [28] and meaningfully involve Native Hawaiian communities in identification of barriers and assets salient to intervention development and delivery.

5. Conclusions

Based on results from the current study, we conclude with three service and policy implications for Native Hawaiian elders. First, all Hawai'i residents need primary health care that includes health education to promote optimal health practices, as well as chronic disease prevention and control. The inclusion of health promotion and disease prevention and control in primary care is especially important for Native Hawaiians who as a group have shorter life expectancies than other ethnic groups and who enter their senior years with more chronic conditions and poorer health habits than most other ethnic groups. Health promotion and disease management programs are needed to both inform Native Hawaiians of their risk for heart disease, and other chronic conditions, as well as to reduce the adverse impact of these conditions on their overall well-being. Contributors to heart disease, such as hypertension and high cholesterol, can be controlled by diet, exercise, and medications. Although many cancers cannot be prevented, they can be cured and/or controlled if diagnosed and treated early. Thus, enrollment in evidence-based health promotion and disease management programs should be encouraged financially and programmatically.

Second, all Hawai'i residents need health care to be affordable and this seems especially important for Native Hawaiian elders. Unfortunately, the high costs of deductibles, copayments, and noninsured treatments lead to delays in seeking necessary health care and may discourage elders from completing treatment or taking medications as recommended. Compounding the problem is the fragmented system of long-term care, both in the provision and funding of services. This issue has critical implications for Native Hawaiians, who may require long-term care services early in life due to earlier onset of disability. Nationally, long-term care services and financing are undergoing major programmatic changes because of the demand for cost-effective and efficient practices for improving quality of life of individuals in need of long-term care. Improvements to the long-term care system include Aging and Disability Resource Centers (ADRCs), which offer one-stop shopping for individuals in need of long-term care services, cash, and counseling programs (through which elders and caregivers are provided vouchers to pay for long-term care services and providers of their choice), expansion of community residential care models (such as assisted living, small group homes, and geriatric foster care), and the culture change movement (i.e., to make nursing homes more "homelike" and less institutional). The adaptation of these or other new models that aim to streamline and humanize long-term care, while reducing costs, should be balanced and sensitive to the health profile and needs of each *kūpuna* or Native Hawaiian elder.

Third, services need to reflect older adult preferences [29]. For Native Hawaiian elders this may include preferences for culturally based programs and services. As all professionals will find themselves working with increasing numbers and proportions of diverse older adults with chronic disease, this becomes an increasingly important care component. Similar to all older adults, quality care for Native Hawaiian elders acknowledges their desire to remain in their own homes with an array of assistance from families, friends, and home and community-based services that honor and reflect their culture [9]. Not surprisingly, quality of life is influenced by the education and training of professionals and other service workers who provide the care. Successful interventions for Native Hawaiian elders are predicated on practitioners having an understanding of cultural values and traditions that influence elder's health practices, with cultural preferences that may include involvement of the elder's extended family and use of health promotion approaches reflecting holistic wellness [9–11, 13]. Continued effort is needed to develop an affordable and culturally responsive health care system that supports acute care, as well as health education and promotion, disease prevention, early disease management and treatment, and community-based long-term care. Further, services must be offered to people across the life span, thereby offering the opportunity for parity in life expectancy and the hopeful prospect of good health to Native Hawaiian elders comparable to that of other older adults.

Disclosure

The information in this paper does not reflect the option of the US Administration on Aging.

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References

[1] R. K. Blaisdell, "Historical and cultural aspects of Native Hawaiian health," in *Social Process in Hawai'i, The Health of Native Native Hawaiians: A Selective Report on Health Status and Health Care in the 1980*, E. L. Wegner, Ed., vol. 32, pp. 1–21, University of Hawai'i, Honolulu, Hawaii, USA, 1989.

[2] Office of Hawaiian Affairs, *The Disparate Treatment of Native Native Hawaiians in the Criminal Justice System*, Office of Hawaiian Affairs, Honolulu, Hawaii, USA, 2010.

[3] Hawai'i State Department of Health, "Hawai'i Health Survey 2008," 2009, http://hawaii.gov/health/statistics/vital-statistics/hhs/hhs_08/hhs08t11.pdf.

[4] C. B. Park, K. L. Braun, B. Y. Horiuchi, C. Tottori, and A. T. Onaka, "Longevity disparities in multiethnic Hawaii: an analysis of 2000 life tables," *Public Health Reports*, vol. 124, no. 4, pp. 579–584, 2009.

[5] Hawai'i Health Information Corporation. (n.d.), "Health trends in Hawai'i," http://www.healthtrends.org/demo_overview.aspx.

[6] Alu Like, Inc., *E ola mau: The Native Hawaiian Health Needs Study Report*, Native Hawaiian Health Research Consortium, Honolulu, Hawaii, USA, 1985.

[7] D. B. Johnson, N. Oyama, L. LeMarchand, and L. Wilkens, "Native Hawaiians mortality, morbidity, and lifestyle: comparing data from 1982, 1990, and 2000," *Pacific Health Dialog*, vol. 11, no. 2, pp. 120–130, 2004.

[8] N. Mokuau, J. Garlock-Tuialiʻi, and P. Lee, "Has social work met its commitment to native Hawaiians and other Pacific Islanders? A review of the periodical literature," *Social Work*, vol. 53, no. 2, pp. 115–121, 2008.

[9] C. V. Browne, N. Mokuau, and K. L. Braun, "Adversity and resiliency in the lives of Native Hawaiian elders," *Social Work*, vol. 54, no. 3, pp. 253–261, 2009.

[10] J. K. Kaholokula, A. H. Nacapoy, and K. Dang, "Social justice as a public health imperative for Kanaka Maoli," *AlterNative*, vol. 5, pp. 117–137, 2009.

[11] L. S. Kaʻopua, "Developing a culturally responsive breast cancer screening promotion with native Hawaiian women in churches," *Health & Social Work*, vol. 33, no. 3, pp. 169–177, 2008.

[12] L. S. Kaʻopua, S. H. Park, M. E. Ward, and K. L. Braun, "Testing the feasibility of a culturally tailored breast cancer screening intervention with Native Hawaiian women in rural churches," *Health & Social Work*, vol. 36, no. 1, pp. 55–65, 2011.

[13] L. Choy, N. Mokuau, C. Browne, and K. L. Braun, "Integration of cultural concepts in establishing Ha Kupuna: the national resource center for Native Hawaiian elders," *Journal of Native Aging and Health*, vol. 3, no. 1, pp. 5–12, 2008.

[14] S. McDonnell, K. Vossberg, R. S. Hopkins, and B. Mittan, "Using YPLL in health planning," *Public Health Reports*, vol. 113, no. 1, pp. 55–61, 1998.

[15] J. M. Romeder and J. R. McWhinnie, "Potential years of life lost between ages 1 and 70: an indicator of premature mortality for health planning," *International Journal of Epidemiology*, vol. 6, no. 2, pp. 143–151, 1977.

[16] W. C. Lee, "The meaning and use of the cumulative rate of potential life lost," *International Journal of Epidemiology*, vol. 27, no. 6, pp. 1053–1056, 1998.

[17] World Health Organization, "International Classification of Disease, 10th Revision," 2007, http://apps.who.int/classfication/apps/icd/icd10online/.

[18] J. C. Kleinman, "Mortality," in *Statistical Notes for Health Planners*, National Center for Health Statistics, Ed., pp. 1–16, U.S. Department of Health, Education, and Welfare, Rockville, Md, USA, 1977.

[19] J. G. Gonyea and N. R. Hooyman, "Reducing poverty among older women: social security reform and gender equity," *Families in Society*, vol. 86, no. 3, pp. 338–346, 2005.

[20] P. Herd, "Crediting care or marriage? Reforming social security family benefits," *The Journals of Gerontology Series B*, vol. 61, no. 1, pp. S24–S34, 2006.

[21] C. DeNavas-Walt, D. Proctor, J. Smith, and U.S. Census Bureau, *Current Population Reports, P60-233, Income, Poverty, and Health Insurance Coverage in the United States: 2006*, U.S. Government Printing Office, Washington, DC, USA, 2007, http://www.census.gov/prod/2007pubs/p60-233.pdf.

[22] M. Y. Brave Heart and L. M. DeBruyn, "The American Indian Holocaust: healing historical unresolved grief," *American Indian and Alaska Native Mental Health Research*, vol. 8, no. 2, pp. 56–78, 1998.

[23] O. Bushnell, *The Gifts of Civilization: Germs and Genocide in Hawaiʻi*, University of Hawaiʻi, Honolulu, Hawaii, USA, 1993.

[24] L. S. Kaʻopua, "Training community practitioners in a research intervention: practice examples at the intersection of cancer, Western science, and native Hawaiian healing," *Cancer Control*, vol. 10, no. 5, pp. 5–12, 2003.

[25] M. M. Sotero, "A conceptual model of historical trauma: implications for public health practice and research," *Journal of Health Disparities Research and Practice*, vol. 1, no. 1, pp. 93–108, 2006.

[26] D. E. Stannard, *Before the Horror: The Population of Hawaiʻi on the Eve of Western Contact*, University of Hawaiʻi, Social Science Research Institute, Honolulu, Hawaii, USA, 1989.

[27] Child Welfare Information Gateway, "Child Welfare Information Gateway," 2011, http://www.childwelfare.gov/pubs/issue_briefs/racial_disproportionality/racial_disproportionality.pdf.

[28] M. Minkler and N. Wallerstein, "Introduction to community based participatory research," in *Community Based Participatory Research for Health*, M. Minkler and N. Wallerstein, Eds., pp. 3–26, Jossey-Bass, San Francisco, Calif, USA, 2003.

[29] A. J. Lehning and M. J. Austin, "Long-term care in the United States: policy themes and promising practices," *Journal of Gerontological Social Work*, vol. 53, no. 1, pp. 43–63, 2010.

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Research Article

Is There a Reversal in the Effect of Obesity on Mortality in Old Age?

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Studies of obesity and its relationship with mortality risk in older persons have yielded conflicting results. We aimed to examine the age-related associations between obesity and mortality in older persons. Data were drawn from the Cross-Sectional and Longitudinal Aging Study (CALAS), a national survey of a random sample of older Jewish persons in Israel conducted during 1989–1992. Analyses included 1369 self-respondent participants aged 75–94 from the Cross-Sectional and Longitudinal Aging Study (CALAS). Mortality data at 20-year followup were recorded from the Israeli National Population Registry. Obesity was significantly predictive of higher mortality for persons aged 75–84, but from age 85 onwards, obesity had a protective effect on mortality albeit at a nonsignificant level. Being underweight was consistently predictive of mortality. Findings suggest that the common emphasis on avoiding obesity may not apply to those advancing towards old-old age, at least as far as mortality is concerned.

1. Introduction

Increased obesity rates comprise a major public health concern over the world [1, 2]. While obesity was associated with increased mortality risk for young and middle-aged adults [3, 4], studies of older persons have yielded conflicting results. Some indicate higher mortality rates for obese older persons [4–6], while others found no such associations [7–9] or evidence for a reversed relationship, that is, linking decreased mortality with higher Body Mass Index (BMI) values [9–13].

Studies of the relationship between obesity and mortality among older persons suggest the impact of obesity varies according to age. In a study of persons aged 44–101 with 23 years of followup, obesity increased mortality only among persons under 75 years of age [5]. Similarly, a study of adults aged 30 and over with a 12-year followup found higher mortality rates for obese younger persons, but not for those aged 75 and over [14]. In line with that, higher BMI was associated with lower mortality among persons aged 70–88 [9], among older persons aged 70 and older [12], and among persons aged 75–89 [10].

This paper addresses the following question: what are the age-related associations between obesity and mortality in older persons? Accordingly, we examine the impact of obesity on mortality in persons who survived to old-old age, that is, persons over 75 years of age.

2. Methods and Procedures

2.1. Participants and Procedure. The sample was part of the Cross-Sectional and Longitudinal Aging Study (CALAS). The CALAS conducted a multidimensional assessment of a random sample of the older Jewish population in Israel, stratified by age group (75–79, 80–84, 85–89, and 90–94), gender, and place of birth (Asia-Africa, Europe-America, Israel). Data collection took place during 1989–1992. The inclusion criteria were being self-respondent, community

dwelling or a nursing home resident, and between 75–94 years of age. More information regarding the CALAS appears in various publications [15–17]. The CALAS was approved for ethical treatment of human participants by the Institutional Review Board of the Chaim Sheba Medical Center in Israel. The present analyses include 1369 self-respondent participants aged 75–94. The mean age was 83.52 (SD = 5.42).

3. Measures

Sociodemographics include age, gender, place of birth (Israel, Middle East/North Africa, Europe/America), having children (number of children alive and number of children deceased), education (number of years of education), and financial situation (having additional income beyond social security, 0 = no, 1 = yes). Smoking status was measured by two items, based on the EPESE questionnaire [18]: Do you smoke (yes, no); Did you smoke in the past (yes, no).

3.1. BMI. The interviewer measured the participant's weight and height, and Body Mass Index was calculated (<22 = underweight, 22–30, >30 = obese) [19].

3.2. Mortality Followup. Mortality data within 20 years from the date of sampling were recorded from the Israeli National Population Registry (NPR). Of the original sample, 59 participants were still alive. Hence we have complete mortality data on 95.1% of the sample.

4. Statistical Analysis

The impact of obesity on 20-year mortality was tested using Cox regression models. In order to examine whether obesity had an effect on mortality, we examined the impact of obesity on mortality after controlling for stratification variables (age, gender, origin), as well as for education, financial status, and having children. The impact of obesity as well as underweight was examined. The same model was examined separately for males and females and separately for ages 75–84 and for ages 85–94. Each analysis was conducted twice, once with community-dwelling participants and once with the full sample, including nursing home residents.

5. Results

Descriptive information on background variables and BMI for both the full sample and community dwellers is presented in Table 1.

While underweight was significantly predictive of mortality under all conditions examined (total population, for each gender, and each age group), obesity was not significantly predictive of mortality when the whole population was examined (Table 2). However, when participants were divided by age, obese persons aged 75–84 had a significantly higher mortality Hazard Ratio (HR) than those with normal weight for the total sample (HR = 1.296, Confidence Interval [CI] = 1.026–1.659, $P = .030$) and for community dwellers (HR = 1.320, CI = 1.036–1.683, $P = .025$). In contrast, obese persons aged 85–94 had a lower, albeit nonsignificant risk ratio for the total sample (HR = .944, CI = .703–1.268, $P > .05$) and for community dwellers (HR = .854, CI = .616–1.185, $P > .05$), indicating that at this age obesity no longer posed a greater risk of mortality. When dividing participants by gender, no significant impact for obesity on mortality was found. Upon adding smoking status (ever versus never) as a covariate to the analysis, the results were nearly identical (see Table 3); smoking status was not significant when included with all the other covariates, but BMI was. The findings concerning the relationship between BMI and 20-year mortality among community dwellers aged 75–84 and 85–94 are illustrated in the survival curves in Figure 1.

Table 1: Sample characteristics.

	Total (N = 1369)	Community dwellers (n = 1200)
	M (SD)/%	M (SD)/%
Age	83.52 (5.42)	83.10 (5.32)
Gender (female)	46.3	44.9
Place of birth		
Europe/America	37.5	37
Middle East/N. Africa	30.6	32.7
Israel	31.8	30.3
Education (n = 1310/1149)	7.79 (5.54)	7.63 (5.51)
Financial status		
(no additional income)	39.4	40.3
(n = 1312/1155)		
Having children (yes)	90.5	91.6
BMI		
Obese (>30)	11.8	11.9
Underweight (<22)	15.8	15.7
(missing)	16	14.7

6. Discussion

We investigated the relationship between obesity and mortality in a random sample of older Israelis, with all participants having survived to at least 75 years old at baseline. Obesity was predictive of mortality for those aged 75–84 years, but from age 85 onwards, obesity had a protective effect on mortality albeit at a nonsignificant level. When examining the entire sample (ages 75–94), obesity was not predictive of mortality. Being underweight was consistently predictive of mortality.

Our findings linking obesity to increased mortality rates among older persons aged 75–84 support those of Ajani et al. [4], reporting greater mortality risk as BMI increased among men aged 70–84 and those of De Gonzalez et al. [20], reporting increased all-cause mortality rates in white never-smokers aged 70–84 with BMI over 30 in a median 10-year followup. Because a variety of age groups within old age have

Table 2: Cox Regression Models Predicting Mortality at 20-year followup by BMI, and age.

	Full sample				Age groups							
					75–84 year olds				85–94 years old			
	CD and NH residents		CD		CD and NH residents		CD		CD and NH residents		CD	
	$N = 1369$		$n = 1200$		$n = 809$		$n = 752$		$n = 560$		$n = 448$	
	HR	CI	HR	CI	HR	CI	HR	CI	HR	CI	HR	CI
Background												
Age	1.068[c]	1.055–1.081	1.066[c]	1.052–1.080	1.074[c]	1.044–1.105	1.073[c]	1.043–1.105	1.062[b]	1.025–1.100	1.065[b]	1.025–1.107
Gender (male=1)	1.31[c]	1.151–1.492	1.307[c]	1.138–1.502	1.50[c]	1.266–1.777	1.518[c]	1.272–1.813	1.093	.887–1.346	1.049	.834–1.318
Origin												
East	.857	.722–1.017	.874	.729–1.048	.972	.777–1.216	1.006	.799–1.268	.731[a]	.559–.955	.715[a]	.533–.958
West	1.032	.889–1.199	1.043	.888–1.225	1.027	.849–1.244	1.038	.848–1.270	1.028	.805–1.313	1.034	.786–1.359
Years of education	.995	.983–1.008	.996	.983–1.010	.997	.980–1.015	.999	.981–1.017	.992	.974–1.011	.993	.972–1.015
Additional income	.940	.824–1.071	.933	.812–1.072	.845[d]	.711–1.003	.858	.718–1.026	1.040	.845–1.279	.993	.791–1.247
Had children	.702[b]	.568–.868	.680[b]	.535–.863	.657[b]	.501–.861	.622[b]	.464–.835	.791	.554–1.129	.780	.506–1.204
BMI												
Obese (>30)	1.117	.930–1.342	1.091	.898–1.326	1.297[a]	1.026–1.639	1.320[a]	1.036–1.683	.944	.703–1.268	.854	.616–1.185
Underweight (<22)	1.414[c]	1.202–1.665	1.409[c]	1.183–1.677	1.278[a]	1.014–1.611	1.317[a]	1.036–1.684	1.474[b]	1.162–1.870	1.400[a]	1.075–1.824
χ^2 (df = 2)	16.511[c]		13.95[b]		7.374[a]		8.178[a]		10.855[b]		8.188[a]	

[a]$P < .05$, [b]$P < .01$, [c]$P < .001$.
[d]$.1 > P > .05$.
CD= Community Dwellers; NH= Nursing Home; HR= Hazard Ratio= Exp (b); CI= Confidence Interval.

Table 3: Cox Regression Models Predicting Mortality at 20-year followup by BMI, and age, controlling for smoking status (Ever versus Never).

	Full sample				Age groups							
					75–84 year olds				85–94 years old			
	CD and NH residents		CD		CD and NH residents		CD		CD and NH residents		CD	
	$N = 1369$		$n = 1200$		$n = 809$		$n = 752$		$n = 560$		$n = 448$	
	HR	CI	HR	CI	HR	CI	HR	CI	HR	CI	HR	CI
Background												
Age	1.068[c]	1.055–1.082	1.067[c]	1.053–1.081	1.077[c]	1.047–1.108	1.076[c]	1.045–1.108	1.062[b]	1.025–1.100	1.065[b]	1.025–1.107
Gender (male = 1)	1.267[b]	1.104–1.453	1.265[b]	1.092–1.465	1.429[c]	1.195–1.709	1.449[c]	1.202–1.746	1.087	.871–1.357	1.051	0.823–1.342
Origin												
East	.849	0.715–1.008	0.867	0.723–1.040	0.960	0.767–1.201	0.993	.788–1.252	0.730[a]	0.559–0.955	0.715[a]	0.533–0.958
West	1.030	0.887–1.197	1.042	0.887–1.224	1.024	0.846–1.240	1.033	.844–1.264	1.028	0.805–1.313	1.033	0.786–1.358
Years of education	0.994	0.982–1.007	0.996	0.982–1.009	0.998	0.981–1.015	0.999	.981–1.017	0.992	0.974–1.011	0.993	0.972–1.015
Additional income	0.940	0.829–1.070	0.932	0.811–1.071	0.844[d]	0.711–1.003	0.858[d]	0.717–1.026	1.040	0.845–1.279	0.993	0.791–1.247
Had children	0.705[b]	0.570–0.872	0.684[b]	0.538–0.869	0.661[b]	.504–.867	0.629[b]	0.468–0.844	0.791	0.554–1.130	0.780	0.506–1.204
Smoking status (1= ever)	1.106	0.969–1.262	1.103	0.959–1.269	1.153[d]	0.974–1.364	1.151	0.966–1.371	1.015	0.816–1.263	0.993	0.781–1.263
BMI												
Obese (>30)	1.122	0.934–1.347	1.096	.902–1.332	1.291[a]	1.022–1.631	1.315[a]	1.032–1.676	0.946	0.704–1.273	0.853	0.614–1.187
Underweight (<22)	1.411[c]	1.199–1.661	1.404[c]	1.179–1.671	1.267[a]	1.005–1.597	1.304[a]	1.025-1.657	1.475[b]	1.163–1.871	1.400[a]	1.075–1.824
χ^2 (df = 2)	16.346[c]		13.699[b]		7.003[a]		7.789[a]		10.852[b]		8.185[a]	

[a]$P < .05$, [b]$P < .01$, [c]$P < .001$. [d].1 > P > .05.
HR= Hazard Ratio= Exp (b); CI= Confidence Interval.

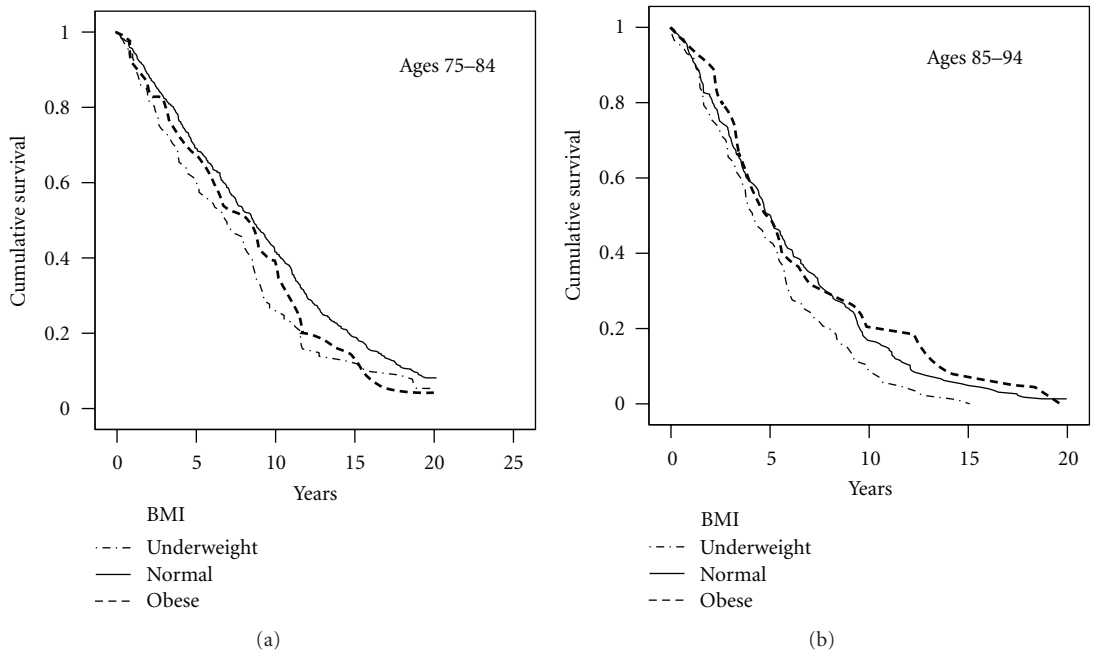

Figure 1: Kaplan-Meier Survival Curves according to Body Mass Index (BMI) in community dwellers. Obesity was significantly predictive of higher mortality for persons aged 75–84, but not for 85–94. Being underweight was consistently predictive of mortality. BMI < 22 = underweight, 22–30 = normal, >30 = obese.

been previously studied, comparing findings is somewhat problematic. Yet, current findings are in discord with some previous evidence. For example, no excess mortality was associated with obesity for persons aged 75–84 [14] in a convenience sample of 62,116 men and 262,019 women from the American Cancer Society's Cancer Prevention Study I. Generalizability of these results is limited due to numerous exclusions (e.g., including only never smokers, only white ethnicity). In a slightly older age group, a protective effect of obesity was found, linking it to lower mortality in a sample of 470 in-patients hospitalized for acute illness aged 75–89 [10]. Similarly, decreased mortality rates were associated with higher BMI among persons of 70–88 years of age [9]. Considering the age range in these studies, it may be that a shift in the relationship between obesity and mortality takes place between age 80 and 90.

Supporting the latter notion, obese persons aged 85–94 in the current study had a lower, though not statistically significant, mortality hazard ratio, in contrast with the significantly higher mortality risk of those 75–84 years old. Previous investigations of mortality and obesity among persons over 80 [5] and over 85 years of age [14] yielded no significant associations between the two.

The relationship between obesity and mortality in older persons is controversial (see [21]). In a review of that relationship [22], obesity was associated with elevated mortality risk in most studies. In line with that, studies on the effect of weight loss in older persons support its favorable health outcomes, even regarding minor amounts of weight loss [21]. These benefits include reducing blood pressure and the severity of diseases such as osteoarthritis and non-insulin-dependent diabetes mellitus [23]. In contrast, inverse associations between BMI and long-term mortality in persons with Chronic Heart Failure (CHF) have been reported in a number of studies [24–29], in what was termed "the obesity paradox" [28]. Following that notion, increased BMI was linked to decreased mortality in hospitalized persons aged 75–89 [10]. Current findings suggest a possible direction for disentangling the obesity paradox, in that the impact of obesity on mortality in old age was age-dependent, shifting from detrimental to favorable between age 80 and 90. In support of that, the concepts of frailty and disability in old age have been proposed to be age-sensitive [30]. It may be that as one gets older, the protective effects of obesity become more pronounced. Lower rates of osteoporosis in heavier persons, possibly due to greater weight-bearing bone formation [31], may reduce their risk of falls and subsequent potential trauma. Obesity may also provide energy reserves in times of stress, illness, and trauma [32, 33]. In addition, obesity may prolong the period of predeath weight loss, as aging is associated with decreased food intake [34]. A progressive drop in BMI was found in a longitudinal study following a small sample of healthy Okinawan centenarians (aged at least 99 years at baseline) until death between ages 110–112, thus suggesting that BMI decline at very old ages may signal sarcopenia, frailty, and/or underlying subclinical pathology [35]. Decreased mortality among persons with obesity in very old ages may reflect a selective survival effect [36, 37] whereby persons who are more prone to the adverse health outcomes of obesity due to the effect of genetic or environmental factors suffer from higher mortality in

middle age, which leaves a more resilient overweight older population [11]. Another possible explanation is that of a ceiling effect, as absolute mortality long-term risk increases with age and eventually converges, regardless of any health-associated risk levels [11, 30].

Evidence regarding the most effective means for achieving weight loss in older persons is controversial, with modest positive outcomes reported for interventions involving diet, physical exercise, and a combined approach (see [38, 39]). For example, a high saturated fat and no-starch diet yielded weight loss without adverse effects on lipids in persons aged 53–73 after 6 weeks [40]. In another study, regular physical exercise for a period of 12 months successfully reduced body weight and body fat in overweight and obese women aged 50–75 [41]. However, in a study of obese and overweight persons with knee osteoarthritis aged 60 and over, the combination of diet and exercise interventions over a period of 18 months provided better overall improvements in measures of pain, function, and mobility, compared with each modality alone [42]. Nonetheless, current knowledge on interventions aimed at weight reductions in the older population is limited, particularly with regard to mortality outcomes [38]. Future studies of that population which assess interventions' effect on mortality are needed.

One limitation of the study involves the use of BMI as a single indicator of body fat. In adults with BMI within the range of 25 to 34.9, BMI fails to provide an adequate biomarker of body fat [43]. In addition, increased mortality due to overweight in older persons is largely affected by a high rate of chronic disease and diminished lean mass [44]. Previous investigations support the value of examining additional body habitus indices. Specifically, lean mass and Lean Mass Index (LMI) predicted all-cause mortality in older Asian persons over 65 years of age, while BMI did not [45]. Similarly, Waist Circumference (WC), as opposed to BMI, predicted mortality at 12-year followup in older persons with CHF and more modestly so among those without CHF [46]. Indeed, public health guidelines indicate the consideration of WC when assessing health risks in overweight adults [43]. Finally, the reported adverse effects associated with visceral fat on health conditions including cardiovascular disease, insulin resistance, and metabolic syndrome [47, 48] support the use of anthropometric measures of abdominal obesity in assessing obesity in older persons [46]. Due to our use of an existing dataset, we were unable to examine the associations between mortality and other measures such as chronic disease, lean mass parameters, and WC. Future studies may benefit from the inclusion of anthropometric measures.

In sum, with the increasing numbers of old-old persons and of their life expectancy, extra attention is often given to avoiding obesity. Current findings suggest that such an emphasis may not apply to those advancing towards old-old age, at least as far as mortality is concerned. As, unlike in some previous investigations of obesity in old age, the current study involved a national representative sample, the results may be more generalizable. While our data included participants as old as 94 at baseline, future research could examine the impact of obesity on mortality when following even older ages.

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Conflict of Interests

The authors have no conflict of interests to declare.

References

[1] World Health Organization, *Obesity: Preventing and Managing the Global Epidemic- Report of a WHO Consultation on Obesity, 3-5 June, 1997*, World Health Organization, Geneva, Switzerland, 1998.

[2] C. L. Ogden, M. D. Carroll, L. R. Curtin, M. A. McDowell, C. J. Tabak, and K. M. Flegal, "Prevalence of overweight and obesity in the United States, 1999-2004," *Journal of the American Medical Association*, vol. 295, no. 13, pp. 1549–1555, 2006.

[3] K. M. Flegal, B. I. Graubard, D. F. Williamson, and M. H. Gail, "Excess deaths associated with underweight, overweight, and obesity," *Journal of the American Medical Association*, vol. 293, no. 15, pp. 1861–1867, 2005.

[4] U. A. Ajani, P. A. Lotufo, J. M. Gaziano et al., "Body mass index and mortality among US male physicians," *Annals of Epidemiology*, vol. 14, no. 10, pp. 731–739, 2004.

[5] M. M. Corrada, C. H. Kawas, F. Mozaffar, and A. Paganini-Hill, "Association of body mass index and weight change with all-cause mortality in the elderly," *American Journal of Epidemiology*, vol. 163, no. 10, pp. 938–949, 2006.

[6] R. J. Thorpe and K. F. Ferraro, "Aging, obesity, and mortality: misplaced concern about obese older people?" *Research on Aging*, vol. 26, no. 1, pp. 108–129, 2004.

[7] J. L. Locher, D. L. Roth, C. S. Ritchie et al., "Body mass index, weight loss, and mortality in community-dwelling older adults," *Journals of Gerontology A*, vol. 62, no. 12, pp. 1389–1392, 2007.

[8] I. A. Lang, D. J. Llewellyn, K. Alexander, and D. Melzer, "Obesity, physical function, and mortality in older adults," *Journal of the American Geriatrics Society*, vol. 56, no. 8, pp. 1474–1478, 2008.

[9] J. Stessman, J. M. Jacobs, E. Ein-Mor, and M. Bursztyn, "Normal body mass index rather than obesity predicts greater mortality in elderly people: the Jerusalem longitudinal study," *Journal of the American Geriatrics Society*, vol. 57, no. 12, pp. 2232–2238, 2009.

[10] A. Weiss, Y. Beloosesky, M. Boaz, A. Yalov, R. Kornowski, and E. Grossman, "Body mass index is inversely related to mortality in elderly subjects," *Journal of General Internal Medicine*, vol. 23, no. 1, pp. 19–24, 2008.

[11] J. P. Reis, C. A. MacEra, M. R. Araneta, S. P. Lindsay, S. J. Marshall, and D. L. Wingard, "Comparison of overall obesity and body fat distribution in predicting risk of mortality," *Obesity*, vol. 17, no. 6, pp. 1232–1239, 2009.

[12] D. C. Grabowski and J. E. Ellis, "High body mass index does not predict mortality in older people: analysis of the longitudinal study of aging," *Journal of the American Geriatrics Society*, vol. 49, no. 7, pp. 968–979, 2001.

[13] I. Janssen, P. T. Katzmarzyk, and R. Ross, "Body mass index is inversely related to mortality in older people after adjustment for waist circumference," *Journal of the American Geriatrics Society*, vol. 53, no. 12, pp. 2112–2118, 2005.

[14] J. Stevens, J. Cai, E. R. Pamuk, D. F. Williamson, M. J. Thun, and J. L. Wood, "The effect of age on the association between body-mass index and mortality," *New England Journal of Medicine*, vol. 338, no. 1, pp. 1–7, 1998.

[15] M. Ben-Ezra and D. Shmotkin, "Predictors of mortality in the old-old in Israel: the cross-sectional and longitudinal aging study," *Journal of the American Geriatrics Society*, vol. 54, no. 6, pp. 906–911, 2006.

[16] A. Walter-Ginzburg, D. Shmotkin, T. Blumstein, and A. Shorek, "A gender-based dynamic multidimensional longitudinal analysis of resilience and mortality in the old-old in Israel: the cross-sectional and longitudinal aging study (CALAS)," *Social Science and Medicine*, vol. 60, no. 8, pp. 1705–1715, 2005.

[17] D. Shmotkin, T. Blumstein, and B. Modan, "Tracing long-term effects of early trauma: a broad-scope view of Holocaust survivors in late life," *Journal of Consulting and Clinical Psychology*, vol. 71, no. 2, pp. 223–234, 2003.

[18] J. Cornoni-Huntley, D. B. Brock, A. M. Ostfeld et al., *Established Populations for Epidemiological Studies of the Elderly: Resource Data Book*, NIH Publication no. 86-2443, U.S. Public Health Service, Washington, DC, USA, 1980.

[19] A. Alfaro-Acha, G. V. Ostir, K. S. Markides, and K. J. Ottenbacher, "Cognitive status, body mass index, and hip fracture in older Hispanic adults," *Journal of the American Geriatrics Society*, vol. 54, no. 8, pp. 1251–1255, 2006.

[20] A. B. De Gonzalez, P. Hartge, J. R. Cerhan et al., "Body-mass index and mortality among 1.46 million white adults," *New England Journal of Medicine*, vol. 363, no. 23, pp. 2211–2219, 2010.

[21] M. Zamboni, G. Mazzali, E. Zoico et al., "Health consequences of obesity in the elderly: a review of four unresolved questions," *International Journal of Obesity*, vol. 29, no. 9, pp. 1011–1029, 2005.

[22] A. Heiat, V. Vaccarino, and H. M. Krumholz, "An evidence-based assessment of federal guidelines for overweight and obesity as they apply to elderly persons," *Archives of Internal Medicine*, vol. 161, no. 9, pp. 1194–1203, 2001.

[23] F. X. Pi-Sunyer, "A review of long-term studies evaluating the efficacy of weight loss in ameliorating disorders associated with obesity," *Clinical Therapeutics*, vol. 18, no. 6, pp. 1006–1035, 1996.

[24] G. C. Fonarow, P. Srikanthan, M. R. Costanzo, G. B. Cintron, and M. Lopatin, "An obesity paradox in acute heart failure: analysis of body mass index and inhospital mortality for 108 927 patients in the Acute Decompensated Heart Failure National Registry," *American Heart Journal*, vol. 153, no. 1, pp. 74–81, 2007.

[25] J. P. Curtis, J. G. Selter, Y. Wang et al., "The obesity paradox: body mass index and outcomes in patients with heart failure," *Archives of Internal Medicine*, vol. 165, no. 1, pp. 55–61, 2005.

[26] C. H. Davos, W. Doehner, M. Rauchhaus et al., "Body mass and survival in patients with chronic heart failure without cachexia: the importance of obesity," *Journal of Cardiac Failure*, vol. 9, no. 1, pp. 29–35, 2003.

[27] L. W. Lissin, A. J. Gauri, V. F. Froelicher, A. Ghayoumi, J. Myers, and J. Giacommini, "The prognostic value of body mass index and standard exercise testing in male veterans with congestive heart failure," *Journal of Cardiac Failure*, vol. 8, no. 4, pp. 206–215, 2002.

[28] C. J. Lavie, A. F. Osman, R. V. Milani, and M. R. Mehra, "Body composition and prognosis in chronic systolic heart failure: the obesity paradox," *American Journal of Cardiology*, vol. 91, no. 7, pp. 891–894, 2003.

[29] T. B. Horwich, G. C. Fonarow, M. A. Hamilton, W. R. MacLellan, M. A. Woo, and J. H. Tillisch, "The relationship between obesity and mortality in patients with heart failure," *Journal of the American College of Cardiology*, vol. 38, no. 3, pp. 789–795, 2001.

[30] A. Kulminski, A. Yashin, S. Ukraintseva et al., "Accumulation of health disorders as a systemic measure of aging: findings from the NLTCS data," *Mechanisms of Ageing and Development*, vol. 127, no. 11, pp. 840–848, 2006.

[31] S. L. Edelstein and E. Barrett-Connor, "Relation between body size and bone mineral density in elderly men and women," *American Journal of Epidemiology*, vol. 138, no. 3, pp. 160–169, 1993.

[32] J. Potter, K. Klipstein, J. J. Reilly, and M. Roberts, "The nutritional status and clinical course of acute admissions to a geriatric unit," *Age and Ageing*, vol. 24, no. 2, pp. 131–136, 1995.

[33] J. Hedlund, "Community-acquired pneumonia requiring hospitalisation. Factors of importance for the short- and long term prognosis," *Scandinavian Journal of Infectious Diseases*, supplement, no. 97, pp. 1–60, 1995.

[34] J. E. Morley, "Decreased food intake with aging," *Journals of Gerontology A*, vol. 56, no. 2, pp. 81–88, 2001.

[35] D. C. Willcox, B. J. Willcox, N. C. Wang, Q. He, M. Rosenbaum, and M. Suzuki, "Life at the extreme limit: phenotypic characteristics of supercentenarians in Okinawa," *Journals of Gerontology A*, vol. 63, no. 11, pp. 1201–1208, 2008.

[36] E. M. Inelmen, G. Sergi, A. Coin, F. Miotto, S. Peruzza, and G. Enzi, "Can obesity be a risk factor in elderly people?" *Obesity Reviews*, vol. 4, no. 3, pp. 147–155, 2003.

[37] S. Rössner, "Obesity in the elderly—a future matter of concern?" *Obesity Reviews*, vol. 2, no. 3, pp. 183–188, 2001.

[38] M. D. Witham and A. Avenell, "Interventions to achieve long-term weight loss in obese older people," *Age and Ageing*, vol. 39, no. 2, Article ID afp251, pp. 176–184, 2010.

[39] C. W. Bales and G. Buhr, "Is obesity bad for older persons? a systematic review of the pros and cons of weight reduction in later life," *Journal of the American Medical Directors Association*, vol. 9, no. 5, pp. 302–312, 2008.

[40] J. H. Hays, A. DiSabatino, R. T. Gorman, S. Vincent, and M. E. Stillabower, "Effect of a high saturated fat and no-starch diet on serum lipid subfractions in patients with documented atherosclerotic cardiovascular disease," *Mayo Clinic Proceedings*, vol. 78, no. 11, pp. 1331–1336, 2003.

[41] M. L. Irwin, Y. Yasui, C. M. Ulrich et al., "Effect of exercise on total and intra-abdominal body fat in postmenopausal women," *Journal of the American Medical Association*, vol. 289, no. 3, pp. 323–330, 2003.

[42] S. P. Messier, R. F. Loeser, G. D. Miller et al., "Exercise and dietary weight loss in overweight and obese older sdults with knee osteoarthritis: the arthritis, diet, and activity promotion trial," *Arthritis and Rheumatism*, vol. 50, no. 5, pp. 1501–1510, 2004.

[43] National Heart, Lung, and Blood Institute Expert Panel, *Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults of Work*, National Institutes of Health, Bethesda, Md, USA, 1998.

[44] W. C. Willett, F. B. Hu, G. A. Colditz et al., "Underweight, overweight, obesity, and excess deaths," *Journal of the American Medical Association*, vol. 294, no. 5, pp. 551–553, 2005.

[45] S. S. Han, K. W. Kim, K. I. Kim et al., "Lean mass index: a better predictor of mortality than body mass index in elderly Asians," *Journal of the American Geriatrics Society*, vol. 58, no. 2, pp. 312–317, 2010.

[46] G. Testa, F. Cacciatore, G. Galizia et al., "Waist circumference but not body mass index predicts long-term mortality in elderly subjects with chronic heart failure," *Journal of the American Geriatrics Society*, vol. 58, no. 8, pp. 1433–1440, 2010.

[47] D. B. Carr, K. M. Utzschneider, R. L. Hull et al., "Intra-abdominal fat is a major determinant of the national cholesterol education program adult treatment panel III criteria for the metabolic syndrome," *Diabetes*, vol. 53, no. 8, pp. 2087–2094, 2004.

[48] P. Bjorntorp, ""Portal" adipose tissue as a generator of risk factors for cardiovascular disease and diabetes," *Arteriosclerosis*, vol. 10, no. 4, pp. 493–496, 1990.

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Research Article

Do Stress Trajectories Predict Mortality in Older Men? Longitudinal Findings from the VA Normative Aging Study

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We examined long-term patterns of stressful life events (SLE) and their impact on mortality contrasting two theoretical models: allostatic load (linear relationship) and hormesis (inverted U relationship) in 1443 NAS men (aged 41–87 in 1985; $M = 60.30$, SD = 7.3) with at least two reports of SLEs over 18 years (total observations = 7,634). Using a zero-inflated Poisson growth mixture model, we identified four patterns of SLE trajectories, three showing linear decreases over time with low, medium, and high intercepts, respectively, and one an inverted U, peaking at age 70. Repeating the analysis omitting two health-related SLEs yielded only the first three linear patterns. Compared to the low-stress group, both the moderate and the high-stress groups showed excess mortality, controlling for demographics and health behavior habits, HRs = 1.42 and 1.37, $ps < .01$ and $< .05$. The relationship between stress trajectories and mortality was complex and not easily explained by either theoretical model.

1. Introduction

A lifespan developmental approach to stress and health is based on two premises. First, stressors are not isolated occurrences but can have lifelong effects on health through setting up different patterns of vulnerability and resilience [1, 2]. The second premise is that there are increasing individual differences in stress and health trajectories with age [3]. However, there are surprisingly few longitudinal studies of changes in stress, especially in later life. Inconsistent results in the literature on the effects of stress on health and mortality may reflect the aggregation of individuals who may have had very different experiences over their life.

The study had two purposes. First, using 18 years of data in a large sample of middle-aged and older men, we sought to identify patterns of change in stressful life events across the lifespan. The second was to examine the effects of different types of stress trajectories on mortality. Specifically, we contrasted an allostatic load model [4] which assumes that higher levels of chronic stress will have the greatest adverse effects on health, with a hormesis model [5–7], which suggests that moderate amount of stress may have protective effects on health.

1.1. Aging and Stressful Life Events. Early correlational studies found that the number of life events is negatively associated with age (for reviews see [8, 9]). However, most life events inventories sample events more common in young adulthood, such as graduations, new jobs, marriages, having children, and divorces. Newer life event measures that targeted middle-aged and older adults show little or slightly negative correlations with age [8] although health-related events show increased frequency in late life [9], as do loss events [10, 11]. A few longitudinal studies have examined age-related changes in the number and type of stressful life events.

Chiriboga [12] found that young adults reported more life events (both negative and positive) than older adults, but there were no differences between individuals in mid-life and late life. However, the longitudinal analyses spanning a 12-year period did not find a linear decrease with age. Rather, the number of events fluctuated over time, suggesting both cohort and period effects. In a preliminary six-year longitudinal study, Yancura et al. [13] found suggestions of nonlinear change over time, with the number of self-reported life events increasing until about age 65 and then decreasing into later life. Ensel and Lin [14, 15] used data spanning 15 years, but the 12-year gap between the last two assessments did not permit an examination of trajectories over time, although they did find that the more distal events predicted the more recent ones A few studies examined growth curve stress trajectories in adults [10, 11, 16] but over short periods of time (e.g., three to seven years). The results varied by type of stressor, the context, and respondent characteristics. For example, there was little change in stress over a three-year period among married women in their thirties, but recently divorced women had higher levels of stress that decreased over three years. Divorced women with antisocial behavior patterns decreased less, however. In older adults, especially in African Americans, loss-related events tended to increase [10, 11].

1.2. Stressful Life Events and Mortality. The adverse effects of stress on both mental and physical health are now well documented [17–20]. However, there are inconsistent results in the literature on the relationship between stress and mortality. While some studies have found that high levels of stressful life events do predict mortality [21, 22], this may be restricted primarily to health-related stressful life events [23]. Other studies have found no relationships between life events and mortality [24], and one found an inverse association [25].

Others have suggested that chronic stress may be a better predictor of health outcomes than the simple occurrence of a life event [26, 27]. However, even in this area, there are mixed results. For example, while Schulz and Beach [28] showed an increased risk of mortality among caregivers, Fredman et al. [29] found a decreased risk—perhaps because those who are healthier are more likely to take on the arduous task of caregiving.

Allostatic load (AL; [30]) is a construct assessed across multiple biosystems, including cardiovascular, metabolic, neuroendocrine, and inflammatory markers that are presumed to reflect chronic psychosocial stress. A number of studies have shown that AL is associated with higher mortality levels (for a review, see [31]). However, few studies have examined psychosocial stress measures in the context of allostatic load, and the ones that have generally find little or no relationship between various components of AL and psychosocial stress [32]. Yancura et al. [33] found that stress was unrelated to a similar measure, the metabolic syndrome, although positive coping strategies were protective.

There are a number of possible reasons for the difficulties in relating stress to mortality. In addition to individual differences in vulnerability to stress, such as personality, coping, and differential access to social resources, very few studies actually examine stress over a long period of time. Given that the types of chronic illnesses related to mortality often take decades to develop, it would be surprising if a single assessment of life events were strongly associated with mortality. Even the chronic stress measures are seldom longitudinal. Thus, examining long-term stress patterns may be a more promising way to link stress with mortality.

However, there is another possible explanation. While most stress theories implicitly assume that there is a linear relationship between stress and adverse outcomes, there is some evidence to suggest that there is a nonlinear relationship. Biogerontologists have recently become interested in *hormesis,* which is defined as "the phenomenon in which adaptive responses to low doses of otherwise harmful conditions improve the functional ability of cells and organisms" ([34], page 1). Most researchers assume that there is a linear, dose-response relationship between stress and adverse health outcomes, but hormesis suggests that there is a nonlinear or inverted U relationship, such that low or even moderate doses of an otherwise toxic substance provide protective benefits against future exposure to stresses. Although still controversial, there is a growing body of evidence for hormesis in organisms such as flatworms, plants, and mice [6, 7, 35], as well as in cardiac and neuronal cells [36]. Presumably, the mechanism is the activation of stress repair mechanisms at the cellular level, such as heat shock proteins. While hormesis has been studied most frequently in plant and animal models, it has been linked to longevity in humans [35, 37]. However, the study of hormesis typically involves physical stressors such as radiation, and little work has been done with psychosocial stressors.

Dienstbier's [38] construct of "physiological toughening"—in which intermittent exposure to stress results in better functioning—is consistent with the hormesis model. The field of stress-related growth [39] has also challenged the assumption of linear effects of stress on health psychological outcomes. Applying the models to human stress and health leads to some interesting and somewhat counterintuitive hypotheses, namely, individuals who experience intermittent or moderate amounts of stress may develop better coping resources which allow them to be more resilient to stress in later life. For example, in a longitudinal study, Schnurr et al. [40] found that individuals who experienced moderate combat exposure had improved MMPI profiles 20 years later, but individuals with no or high combat exposure had worsened MMPI profiles.

People who have chronically high stress levels may experience resource depletion [41], leading to impaired ability to cope with stress. However, individuals who, for whatever reasons, avoid stressors, that is, who are extremely risk averse, may not develop the resources which allow them to adequately cope with problems that arise in late life. In a qualitative study, Vaillant [42], described one such person who managed to adopt an extremely self-limiting lifestyle in that she managed to avoid employment, marriage, and even friends, but was poorly equipped to deal with problems in later life.

A possible explanation for the inconsistent relations between stress and mortality is that hormesis may apply to psychosocial as well as physiological stressors. Thus, the possibility exists that there is a nonlinear relationship between stress and mortality—that moderate stress levels may be associated with better longevity, while both chronic high and low levels may be associated with poorer longevity.

1.3. Present Study. The primary aim of this study was to compare longitudinal change in life events and its consequences on mortality, using 18 years of data from the VA Normative Aging Study (NAS), a longitudinal panel study which has followed a large sample of men since the mid-1960s. The NAS has collected up to nine assessments of stressful life events. To our knowledge, this constitutes the longest extant longitudinal study of stress and coping using quantitative measures and, as such, provides a unique resource to model changes in stress patterns from mid- to late life and their relationships to mortality.

First, we examined the developmental trajectories of stressful life events. Only a few studies have examined trajectories of life events in adulthood [10, 11, 16]. We hypothesized that life events would increase until approximately age 65 and decrease thereafter [13]. Further, we hypothesized that by using latent class growth analyses (LCGAs; [43]), we would identify at least three different patterns of trajectories, with some individuals exhibiting chronically elevated levels of life events, others showing stable low levels, and others showing moderate or intermittent levels of life events. We estimated two sets of LCGA models, one for stressful life events measure including health items, and one for a measure excluding health items. The purpose of the first set of analyses was to examine how stressful life events, including health problems, changed with age; the second omitted health problems in order to avoid a potential confound between stress and health when predicting mortality.

The second aim was to understand how these developmental trajectories of stress related to all-cause mortality, contrasting an allostatic load model with a hormetic one. If the allostatic load model is correct, there should be a linear relationship between stress and mortality. However, if the hormesis model is correct, then the relationship should be nonlinear (U-shaped, with higher mortality at either end of stress distribution). We hypothesized that individuals in the chronic high- or low-stress categories would be more likely to have higher rates of mortality than those in the intermediate stress categories.

2. Methods

2.1. Sample. The VA Normative Aging Study (NAS) screened over six thousand men between 1961 and 1968 and enrolled 2,280, aged 21–81, who had good health, defined as the absence of chronic illness and blood pressure below 140/90 as well as likely geographic stability, which was indexed by extensive ties in the Boston area (see [44]). The men are equally divided between blue and white collar workers, and reflected the racial profile of Boston in the late 1950s, that is, primarily white. As of August 2010, 852 (37.4%) were participating, 1,305 (57.2%) were deceased, 89 (3.9%) were lost or dropped out, and 34 (1.5%) were too sick to participate. The current mean age is 79 (SD = 6.4, range = 63–98).

In this study, we began with 1,565 men who completed the initial stress inventory in 1985. We computed stress trajectories from 1985 to 2002 (an 18-year period) on the 1443 men who had at least three assessments of stressful life events. Mean age of this sample in 1985 was 60.30 (SD = 7.73, range = 41–87). We then predicted subsequent mortality, using these stress trajectories as a predictor, and including covariates from various surveys and medical examinations, which decreased the n to 977. This subsample did not differ from the larger sample in health ratings, smoking, alcohol consumption, or education although they were more likely to be married, $X^2(1; N = 1222) = 4.30$, $p < .05$.

2.2. Procedures. NAS men complete biomedical examinations every three years as well as periodic mail surveys assessing psychosocial variables, which typically have response rates exceeding 80%. The NAS began collecting stressful life events in 1985, using three mail surveys over six years, which also included information on mental and physical health. Response rates for these surveys were typically over 80%. In 1987, the Health and Social Behavior (HSB) survey was developed to obtain psychosocial information at the time of the biomedical exam. The HSB was included in a packet of materials sent to the men before they reported for their triennial physical exam. The HSB included measures assessing stress and mental and physical health; response rates typically exceed 95%. Data collected between 1985 and 2002 were selected to develop the stressful life event trajectories. Data from the triennial medical exams and several surveys were used for the covariates.

2.3. Measures

2.3.1. Stressful Life Events. In the NAS, the Elders Life Stress Inventory (ELSI; [8]) was included in both the Social Support and the Health and Social Behavior (HSB) surveys. The ELSI is a 30 item measure that assesses events likely to occur in middle-aged and older adults during the past year. These items include institutionalization of parent or spouse, child's divorce and retirement. The ELSI uses a nested construction technique; that is, where possible, there are several items in each domain such that the more items that are reported, the more serious are the problems in that domain. For example, the disruptions in marital relationships domain contains three items (e.g., marital problems, separation, and divorce). Thus, the stress ratings correlate .96 with simple counts of items. For those analyses with health outcomes, scoring for the ELSI excludes the items related to worsening health. The ELSI has good criterion validity, correlating about .2 to .3 with physical and psychological health outcomes, comparable to other stressful life event measures. Although this life events measure is correlated with other types of stress measures, such as hassles and perceived stress, as well

as personality measures such as neuroticism, it contributes independent variance to health outcomes [45].

We scored the ELSI in two ways: a total sum which includes all 30 items and one which omits the two items which tap health events. As mentioned earlier, we used stressful life events from 1985 to 2002. We used a total of 7,634 observations for 1443 men (M = 5.29, SD = 1.58, range = 3–9).

2.3.2. Covariates. In the models predicting mortality as a function of stress trajectory, we included a number of covariates that are known to affect mortality, including a self-reported health rating, demographics, and health behavior habits.

Educational attainment was derived from an item in the original Social Screening Survey which was collected at study enrollment. This was recorded into less than high school (9.31%), high school (24.97%) least some college (51.18%), and graduate or professional education (14.53%). Marital status was derived from a survey in 1986 and dichotomized into married (88.46%) versus not married (11.54%).

For self-reported health, we used a single item assessing general health from the first Social Survey in 1985 taken from the SF-36 [46]. Health was rated from 1–5, where 1 = poor and 5 = excellent (M = 4.2, SD = 0.72). Note that 90% rated their health as good or excellent. Drinking status was derived from a survey item administered in 1986 and was coded to identify nondrinker (12.93%), light or moderate (71.69%), and heavy drinkers (15.38%). Current smokers (13.03%) were identified based on an interview at the physical exam closest to 1985.

2.3.3. Mortality. Vital status of NAS participants was monitored by periodic mailings, and when notified of a death, official death certificates were obtained. Death certificates were reviewed by a physician, and cause of death coded by an experienced RN using ICD-9.

2.4. Analyses. To identify different trajectory classes of stressful life events, we used a semi-parametric mixture-model approach for Nagin [47] and Jones et al. [43]. The method is implemented in the TRAJ procedure in SAS [43] and uses a group-modeling approach that employs a mixture of probability distributions that are specified to match the data being analyzed. The mixture models used deal with heterogeneity in the population by modeling subpopulations which differ in regard to their parameter values. The marginal density for the outcome y is

$$f(y) = \sum_{k=1}^{K} \Pr(C = k) \Pr(\mathbf{Y} = y \mid C = k) = \sum_{k=1}^{K} p_k f(y, \lambda_k), \tag{1}$$

where p_k the probability of belonging to class k corresponding to parameters λ_k, which depend on time, thus creating longitudinally based latent trajectories.

The TRAJ procedure allows one to model $\Pr(\mathbf{Y} = y \mid C = k)$ in a number of ways, including a standard Poisson model

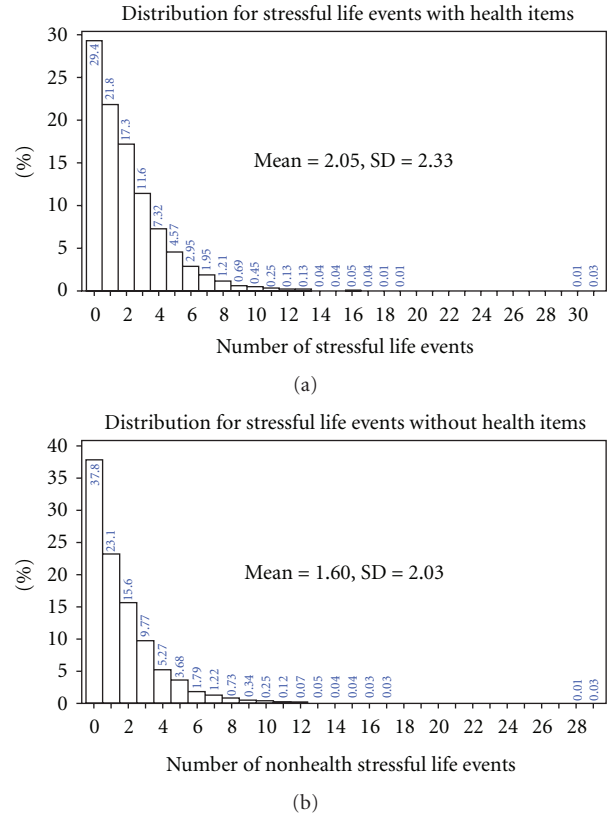

Figure 1: Distribution of the stressful life event measures.

appropriate for count data. However, because approximately 30% of NAS men did not report stressful life events (see Figure 1), it was necessary to use a zero-inflated Poisson (ZIP) model to account for the fact that there are more zeros than would be expect under the Poisson assumption [48]. As with standard Poisson models, covariates such as age are related linearly to the log of the Poisson mean. We used the Voung [49] test to compare the zero-inflated model with an ordinary Poisson regression model for both the SLE sum and the SLE sum omitting the health items. In both instances, the Vuong Non-Nested Hypothesis test statistics suggested the ZIP model was superior to the standard Poisson model ($p < .001$ for both outcomes).

We computed trajectories of life events against age centered at 63. In addition, we tested a quadratic term for age, based on preliminary analyses, which suggested that there might be nonlinear trajectories [13]. The number of classes present in the data is determined by comparing models with the same class structure but varying numbers of classes. In order to identify the correct number of classes, we used the change in Bayesian information criterion (BIC, [50]) between models as an approximation to the log of Bayes factor [51], $2\log_e(B_{10}) \approx 2(\Delta\text{BIC})$, where ΔBIC is the BIC of the more complex (alternative) model minus the BIC of the simpler (null) model [52]. Finally, we considered the model's interpretability.

In order to graph the predicted trajectories for each group, c, we used the following model, written with no

covariates adjustments, which is based on the growth mixture-model described earlier in this paper:

$$\varphi_{cj} = p_{0cj} + \left(1 - p_{0cj}\right) * p_{1cj},$$

$$p_{0cj} = \frac{\exp\left(\alpha_{0c} + \alpha_{1c} * \text{age}_j\right)}{1 + \exp\left(\alpha_{0c} + \alpha_{1c} * \text{age}_j\right)}, \quad (2)$$

$$p_{1cj} = \exp\left(\beta_{0c} + \beta_{1c} * \text{age}_j + \beta_{2c} * \text{age}_j^2\right),$$

where φ_{cj} indicates the predicted probability for the group c at time j and φ_{cj} was composed using both predicted probabilities p_{0cj} and p_{1cj} which were obtained from the zero-inflation and the Poisson parts of the model. Note that parameters α_{0c} and α_{1c} are associated with the zero-inflation part of the model, while β_{0c}, β_{1c}, and β_{2c} are coefficients associated with the Poisson part of the model. Here, the estimate β_{2c} will be zero for groups where the nonlinear pattern is not best described with the group trajectory.

2.4.1. Proportional Hazards Models. A Cox [53] proportional hazard model was used to investigate the effect of stressful life event trajectory classes on mortality. The general equation is

$$\lambda(t_i \mid C_i, X_i) = \lambda_0(t_i) e^{\beta_1 C_i + \beta_2 X_i}, \quad (3)$$

where $\lambda_0(t_i)$ is individual i's risk of dying with baseline hazard rate (λ_0) at time t, C is the class indicator estimate obtained from the growth mixture model in the first stage, and X represents the vectors of demographics and health behaviors. Parameter β_1 represents the effect of the class of stressful life event trajectories on risk of dying, and parameter β_2 represents the effect of demographics (i.e., education) and health behaviors (i.e, drinking) on risk of dying.

Following Mroczek et al. [54], we used two different proportional hazard models, where the first model used only class assignments, and the second model utilized both class estimates and covariates to determine whether any of the effects of life events class on mortality were affected by the covariates.

3. Results

3.1. Types of Life Event Trajectories. An iterative procedure was used to determine the number of classes, with the number of classes determined by change in the model fit criterion, BIC [43]. We first examined the total life events measure, which included the two health items. As can be seen in Table 1, the fit improved as classes were added to the model, starting with a single-class model and moving up to a model with four classes. However, beyond four classes, the fit became worse. Thus, we determined that the four-class model provided the best estimate of the true number of classes.

Table 2 provides the intercept and linear slope estimates for the four classes. While we tested the quadratic term for all of the classes, it was only significant for the first class. We compared a simpler model in which only the first class had a quadratic term to a more complex model which estimated quadratic terms for all classes. The model comparison test for the more complex model did not significantly improve over the simpler model, $2\Delta\text{BIC} = -14.23$, so we retained the simpler model.

Table 1: Determining the number of classes for stressful life event trajectories including health items.

Classes	log likelihood	BIC1	AIC	2ΔBIC
1	−15417.42324	−15435.60944	−15422.42324	—
2	−14741.18617	−14773.92133	−14750.18617	1323.38
3	−14634.89249	−14682.17661	−14647.89249	183.49
4	−14602.43102	−14664.26409	−14619.43102	35.83
5	−14590.9149	−14667.29693	−14611.9149	−6.07

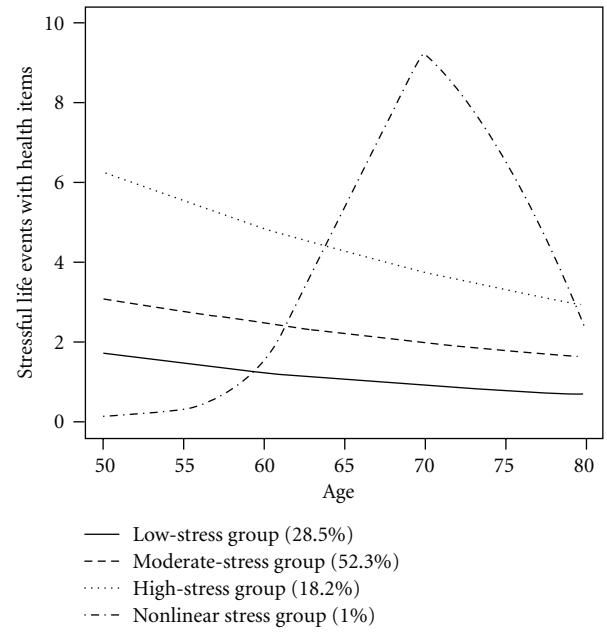

Figure 2: Graphs of the predicted trajectories for the different stressful life event classes (including health items).

Figure 2 presents the estimated trajectories for each of the four classes. Three of the classes showed decreasing slopes, with low, medium, and high intercept values. Class 1 (low stress), Class 2 (moderate stress) and Class 3 (high stress) contained 28.5%, 52.3%, and 18.2% of the sample. However, Class 4 (nonlinear) shows an inverted U-shaped relationship between life events and age, with life events increasing from age 50 to about 70, and decreasing thereafter. However, this group only contained about 1% of the sample.

We repeated the procedure for the life events measure that omitted the two health items. As can be seen in Table 3, only three classes were found for this scoring of the life events measure. Further, none of the quadratic terms were significant, so the final model included only linear terms for the slopes. As can be seen in Figure 3 and Table 4, all of the groups had negative slopes, indicating decreases over time, with low, medium, and high intercepts (Classes 1, 2, and 3 had 32.36%, 55.09%, and 12.54% of the sample, resp.).

Table 2: Latent class growth analysis results for stressful life events including health items.

Classes	Parameter	Estimate	Error	Parameter = 0	Prob > |T|
1	Intercept	0.21144	0.05578	3.79	.00
	Linear	−0.03782	0.00476	−7.941	.001
2	Intercept	0.98264	0.03549	27.691	.001
	Linear	−0.02215	0.00221	−10.024	.001
3	Intercept	1.69093	0.02611	64.765	.001
	Linear	−0.02336	0.00202	−11.563	.001
4	Intercept	0.4921	0.31959	1.54	0.1237
	Linear	0.36437	0.06986	5.216	.001
	Quadratic	−0.01735	0.00331	−5.248	.001
	Alpha0	−1.80433	0.05954	−30.302	.001
	Alpha1	0.02563	0.00558	4.598	.001
Group membership					
1	(%)	28.53623	3.29379	8.664	.001
2	(%)	52.28231	2.93452	17.816	.001
3	(%)	18.15619	1.80972	10.033	.001
4	(%)	1.02527	0.37458	2.737	.001

Table 3: Determining the number of classes for stressful life events excluding health items.

Classes	log likelihood	BIC1	AIC	2ΔBIC
1	−13698.23027	−13716.41647	−13703.2	—
2	−13147.60309	−13180.33825	−13156.6	1072.16
3	−13061.26944	−13108.55355	−13074.3	143.57
4	−13051.09206	−13112.92514	−13068.1	−8.74

These class assignments were used in the subsequent analyses predicting mortality, as they omitted the potential confounds of the health items.

3.2. Classes of Life Event Trajectories and Mortality. By 2010, 693 (48.02%) of the sample was deceased. The deaths were not equally distributed across the three trajectory classes of stressful life events (no health items), $X^2(2, N = 1443) = 15.10$, $p < .01$. Only 40.69% of the low-stress group was deceased, compared to 51.19% and 53.04% of the moderate- and high-stress groups, respectively.

As mentioned earlier, we implemented two different proportional hazards models. In the first model, we investigated the effect of classes of stress trajectories on mortality. Here, we were using the low-stress group as the comparison group. In the second model, we included covariates assessing marital status, education, self-rated health and alcohol and tobacco use. As can be seen in Table 5, both the moderate- and high-stress groups were almost 50% more likely to die than the low-stress group (hazard ratios (95% CI) = 1.43 (1.16, 1.76) and 1.49 (1.10, 2.02), ps <.001, and .01, resp.). Adding the covariates reduced these slightly, but they remained significant (hazard ratios = 1.42 (1.14, 1.76) and 1.37 (1.01, 1.87), $p = 001$ and $p < .05$, resp.). Being married and having better self-reported health were protective factors, while being a nondrinker and being a smoker were risk factors for mortality.

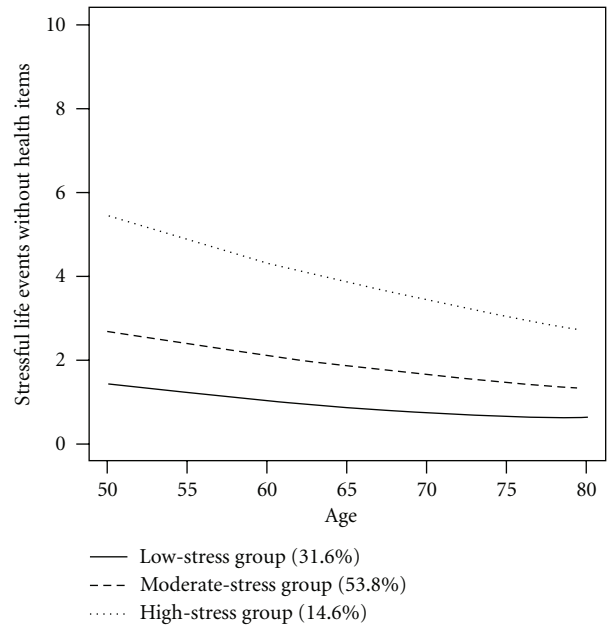

Figure 3: Graphs of the predicted trajectories for the different life event classes (excluding health items).

4. Discussion

We examined two major issues in the study of aging and stress. First, we examined patterns of change in two measures of stressful life events over 18 years, one a summary score which included health events, and a second that excluded the two health events. Using growth mixture models, we found four patterns for the stressful life event measure which

Table 4: Latent class growth analysis results for stressful life events including health items.

Classes	Parameter	Estimate	Error	Parameter = 0	Prob > |T|
1	Intercept	−0.01395	0.06973	−0.2	0.8415
	Linear	−0.04067	0.0054	−7.539	.001
2	Intercept	0.838	0.04367	19.187	.001
	Linear	−0.02431	0.0026	−9.335	.001
3	Intercept	1.61165	0.03105	51.905	.001
	Linear	−0.0199	0.00238	−8.363	.001
	Alpha0	−1.52872	0.05863	−26.075	.001
	Alpha1	0.03221	0.00565	5.706	.001
Group membership					
1	(%)	31.61516	3.98985	7.924	.001
2	(%)	53.83532	3.46901	15.519	.001
3	(%)	14.54953	1.66608	8.733	.001

Table 5: Proportional hazard models predicting mortality ($N = 977$).

Parameter	Estimate	StdErr	ChiSq	prob	Hazard ratio	HRLower CL	HRUpper CL
Equation 1							
Moderate-stress group	0.356	0.107	10.947	.001	1.428	1.156	1.764
High-stress group	0.398	0.155	6.638	.010	1.490	1.100	2.018
Equation 2							
Moderate-stress group	0.352	0.109	10.440	.001	1.422	1.149	1.761
High-stress group	0.315	0.157	4.023	.045	1.371	1.007	1.866
Health rating	− 0.288	0.064	20.497	.001	0.749	0.661	0.849
Married	− 0.331	0.131	6.458	.011	0.718	0.556	0.927
Less than high school	− 0.231	0.184	1.596	.207	0.793	0.553	1.136
College	− 0.121	0.111	1.174	.279	0.886	0.712	1.103
Postgrad	0.038	0.150	0.065	0.799	1.039	0.774	1.394
Nondrinker	0.314	0.123	6.496	0.011	1.369	1.075	1.743
Heavy drinker	− 0.009	0.125	0.005	0.944	0.991	0.776	1.267
Smoker	0.215	0.127	2.859	0.091	1.240	0.966	1.591

included the health events. Three showed linear decreases over time and were distinguished primarily by the initial number of events (low, moderate, and high stress). The fourth pattern, however, demonstrated a nonlinear inverted U, which peaked at about age 70. In the analysis of the life events measure which excluded health events, the first three patterns were replicated but the fourth pattern was not found, suggesting that the health events accounted for the nonlinearity.

To our knowledge, this study is unique in two respects: first in examining longitudinal change in self-reported stressors over nearly two decades and second in identifying different patterns of change and relating these patterns to mortality. Earlier studies either spanned much shorter periods of time [10, 11, 16] or used stressful life events coded from interviews [12]. While this latter study found cross-sectional differences between young, middle-aged, and older adults, with younger adults having higher levels of stressful life events, they did not find longitudinal decreases over time. However, our study focused primarily on middle-aged and older men and did find decreases even using a measure which assesses events more likely to occur to older adults. It is possible that a different pattern of results might have occurred had we included young adults or women in the sample.

Second, we considered whether different patterns of stressful life events were associated with mortality. As reviewed earlier, research has shown a highly variable pattern of results, with different studies showing positive, negative, and no effects on mortality [21, 22, 24, 25]. We hypothesized that multiple assessments of life events to tap stress chronicity might be presumed to show greater effects on health outcomes than a single measure. Indeed, this has long been hypothesized by the allostatic load model [30]. However, studies of allostatic load have been limited by omitting psychosocial measures of chronic stress in favor of clinical measures. Further, some more recent studies failed to find associations between chronic stress and some of the allostatic load biomarkers (see [32], for a review).

Thus, we sought to conduct a more stringent test of the impact of stress on mortality by examining different patterns of psychosocial stress, which included specific indicators

of chronic stress over longer periods of time. Further, we excluded health events, given that some have suggested that the relationship between stress and mortality is limited to health-related events [23]. Thus, we utilized the stress classes generated from the growth mixture models of stressful life events, excluding the ones related to health. Moreover, while most theories of stress and health implicitly assume a linear relationship, we hypothesized that the pattern of results would support a nonlinear model. Specifically, we tested the hormesis model currently being advocated by some biogerontologists, in which moderate stress levels are thought to be protective for health outcomes [6, 7, 34].

Using a proportional hazards model, in which the different stress patterns were entered first, and then covariates added (see [54]), we found that both the moderate- and high-stress groups showed significantly higher risk of mortality than the low-stress group. This relationship was only slightly attenuated by the addition of standard health behavior risk factors (e.g., smoking and drinking) as covariates. Thus, our hypotheses were partially supported. Chronic stress did predict mortality even when health-related events were excluded. However, contrary to the prediction from hormesis theory, those with moderate-stress levels did not show enhanced longevity. Rather, the low-stress group showed the lowest levels of mortality. However, it is noteworthy that both the moderate- and high-stress groups showed similar mortality risks, which is also not predicted by the allostatic load model.

There are a couple of possible explanations for this finding. The first is that the allostatic load model may be largely correct but needs to acknowledge an asymptotic relationship in which higher levels of stress do not confer much additional risk over moderate levels (see [55]). The second possibility reflects the relative youth of the field of hormesis—there are currently few guidelines for what level of stress might be considered hormetic. The low-stress group did have some stress—averaging nearly 2 SLEs during their 50s, dropping down to about 1 in their 80s. It is possible that this low level was actually hormetic and provided protection over the higher levels. In other words, low (but not zero) stress, rather than moderate stress, is protective. One or two stressful life events a year may be manageable, allowing individuals to cope in a fashion which increases mastery, theoretically increasing hormesis. However, anything over two stressful life events might prove overwhelming. To adequately test this, we would need to find populations which experienced no stress, which would be difficult.

Nonetheless, this nonlinear relationship is quite interesting, and might account for the inconsistency in the findings in the literature. Much as the relationship between age and mental health is muddied by its nonlinear (J-shaped) relationship [56–58], so too the nonlinear, asymptotic relationship between stress and mortality might have created similar inconsistencies. Further research is needed to replicate and test these findings.

The covariates also showed an interesting pattern of results. Those with better self-rated health showed lower levels of mortality, supporting many other studies [59]. Likewise, married men had lower mortality, as previously found [60]. While education had no significant effect, men who were teetotalers showed higher risk of mortality [61]. Smoking was only marginally related to mortality, undoubtedly because so few of the NAS men have continued to smoke. Nonetheless, stressful life event levels clearly had independent effects on mortality.

4.1. Limitations and Future Studies. A major limitation of this study was that the sample consisted primarily of white, middle-class men, and other patterns of results would likely be found in more diverse samples which include women and minorities. It is also possible that other types of stress measures, such as daily stressors, would show a different pattern of results. It is surprising that the effects of life events on mortality did not appear to be mediated through health behavior habits such as drinking and smoking but rather had independent effects. Future studies should examine whether the types of biomarkers used to measure allostatic load mediate the effects of psychosocial stress on mortality. Also, it is possible that the use of longitudinal trajectories may have obscured any hormesis-like relationships. Growth mixture models "smooth out" the curve, and it is possible that an analytical technique which better addresses the intermittent nature of stress might show physiological toughening effects [38].

Nonetheless, this study shows that longitudinal assessments of stressful life events significantly predict mortality independent of standard risk factors and that high stressful life event levels did not convey appreciably greater risk than moderate ones. Future studies should investigate both possible nonlinear relationships between stress and health as well as potential mediating pathways.

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References

[1] C. Aldwin and D. Stokols, "The effects of environmental change on individuals and groups: some neglected issues in stress research," *Journal of Environmental Psychology*, vol. 8, no. 1, pp. 57–75, 1988.

[2] G. Elder and M. Shanahan, "The life course and human development," in *Handbook of Child Psychology*, R. Lerner and W. Damon, Eds., vol. 1 of *Theoretical Models of Human Development*, pp. 665–715, John Wiley & Sons, Hoboken, NJ, USA, 6th edition, 2006.

[3] D. Dannefer, "What's in a name? An account of the neglect of variability in the study of aging," in *Emergent Theories of Aging*, J. E. Birren and V. L. Bengtson, Eds., pp. 356–384, Springer, New York, NY, USA, 1988.

[4] B. S. McEwen, "Glucocorticoids, depression, and mood disorders: structural remodeling in the brain," *Metabolism: Clinical and Experimental*, vol. 54, no. 5, pp. 20–23, 2005.

[5] E. J. Calabrese, "Paradigm lost, paradigm found: the re-emergence of hormesis as a fundamental dose response model in the toxicological sciences," *Environmental Pollution*, vol. 138, no. 3, pp. 379–411, 2005.

[6] E. J. Calabrese, "What is hormesis?" in *Mild Stress and Healthy Aging: Applying Hormesis in Aging Research and Interventions*, E. Le Bourg and S. I. S. Rattan, Eds., pp. 5–20, Springer, New York, NY, USA, 2008.

[7] G. J. Lithgow, T. M. White, A. Melov, and T. E. Johnson, "Thermotolerance and extended life-span conferred by single-gene mutations and induced by thermal stress," *Proceedings of the National Academy of Sciences of the United States of America*, vol. 92, no. 16, pp. 7540–7544, 1995.

[8] C. Aldwin, "The elders life stress inventory (ELSI): egocentric and nonegocentric stress," in *Stress and Coping in Late Life Families*, M. A. P. Stephens, J. H. Crowther, S. E. Hobfoll, and D. L. Tennenbaum, Eds., Applied Psychology: Social Issues and Questions, pp. 49–69, Hemisphere, New York, NY, USA, 1990.

[9] C. Aldwin, "Does age affect the stress and coping process? The implications of age differences in perceived locus of control," *The Journals of Gerontology B*, vol. 46, pp. 174–180, 1991.

[10] L. K. George and S. M. Lynch, "Race differences in depressive symptoms: a dynamic perspective on stress exposure and vulnerability," *Journal of Health and Social Behavior*, vol. 44, no. 3, pp. 353–369, 2003.

[11] S. M. Lynch and L. K. George, "Interlocking trajectories of loss-related events and depressive symptoms among elders," *Journals of Gerontology B*, vol. 57, no. 2, pp. S117–S125, 2002.

[12] D. A. Chiriboga, "Crisis, challenge, and stability in the middle years," in *Multiple Paths of Midlife Development*, M. E. Lachman and J. B. James, Eds., pp. 293–343, University of Chicago Press, Chicago, Ill, USA, 1997.

[13] L. A. Yancura, C. M. Aldwin, and A. Spiro III, "Does stress decrease with age? A longitudinal examination of stress in the normative aging study," *The Gerontologist*, vol. 39, article 212, 1999.

[14] W. M. Ensel and N. Lin, "Distal stressors and the life stress process," *Journal of Community Psychology*, vol. 24, no. 1, pp. 66–82, 1996.

[15] W. M. Ensel and N. Lin, "Age, the stress process, and physical distress: the role of distal stressors," *Journal of Aging and Health*, vol. 12, no. 2, pp. 139–168, 2000.

[16] F. O. Lorenz, R. L. Simons, R. D. Conger, G. H. Elder Jr., C. Johnson, and W. Chao, "Married and recently divorced mothers' stressful events and distress: tracing change across time," *Journal of Marriage and Family*, vol. 59, no. 1, pp. 219–232, 1997.

[17] S. Cohen, D. Janicki-Deverts, and G. Miller, "Psychological stress and disease," *Journal of the American Medical Association*, vol. 298, no. 14, pp. 1685–1687, 2007.

[18] D. S. Krantz and M. K. McCeney, "Effects of psychological and social factors on organic disease: a critical assessment of research on coronary heart disease," *Annual Review of Psychology*, vol. 53, pp. 341–369, 2002.

[19] S. Ramachandruni, E. Handberg, and D. S. Sheps, "Acute and chronic psychological stress in coronary disease," *Current Opinion in Cardiology*, vol. 19, no. 5, pp. 494–499, 2004.

[20] B. H. Singer and C. D. Ryff, Eds., *New Horizons in Health: An Integrative Approach*, National Academy Press, Washington, DC, USA, 2001.

[21] P. M. Lantz, J. S. House, R. P. Mero, and D. R. Williams, "Stress, life events, and socioeconomic disparities in health: results from the Americans' changing lives study," *Journal of Health and Social Behavior*, vol. 46, no. 3, pp. 274–288, 2005.

[22] A. Rosengren, K. Orth-Gomer, H. Wedel, and L. Wilhelmsen, "Stressful life events, social support, and mortality in men born in 1933," *British Medical Journal*, vol. 307, no. 6912, pp. 1102–1105, 1993.

[23] A. C. Phillips, G. Der, and D. Carroll, "Stressful life-events exposure is associated with 17-year mortality, but it is health-related events that prove predictive," *British Journal of Health Psychology*, vol. 13, no. 4, pp. 647–657, 2008.

[24] E. Maunsell, J. Brisson, M. Mondor, R. Verreault, and L. Deschênes, "Stressful life events and survival after breast cancer," *Psychosomatic Medicine*, vol. 63, no. 2, pp. 306–315, 2001.

[25] J. F. Hollis, J. E. Connett, V. J. Stevens, and M. R. Greenlick, "Stressful life events, type A behavior, and the prediction of cardiovascular and total mortality over six years," *Journal of Behavioral Medicine*, vol. 13, no. 3, pp. 263–280, 1990.

[26] M. S. Kopp and J. Rethelyi, "Where psychology meets physiology: chronic stress and premature mortality—the Central-Eastern European health paradox," *Brain Research Bulletin*, vol. 62, no. 5, pp. 351–367, 2004.

[27] L. I. Pearlin, C. S. Aneshensel, J. T. Mullan, and C. J. Whitlatch, "Caregiving and its social support," in *Handbook of Aging and the Social Science*, R. H. Binstock and L. K. George, Eds., pp. 283–302, Academic Press, San Diego, Calif, USA, 4th edition, 1996.

[28] R. Schulz and S. R. Beach, "Caregiving as a risk factor for mortality: the caregiver health effects study," *Journal of the American Medical Association*, vol. 282, no. 23, pp. 2215–2219, 1999.

[29] L. Fredman, J. A. Cauley, M. Hochberg, K. E. Ensrud, and G. Doros, "Mortality associated with caregiving, general stress, and caregiving-related stress in elderly women: Results of caregiver-study of osteoporotic fractures," in *Journal of The American Geriatrics Society*, vol. 58, pp. 937–943, 2010.

[30] B. S. McEwen and T. Seeman, "Protective and damaging effects of mediators of stress: elaborating and testing the concepts of allostasis and allostatic load," *Annals of the New York Academy of Sciences*, vol. 896, pp. 30–47, 1999.

[31] R. Juster, B. S. McEwen, and S. J. Lupien, "Allostatic load biomarkers of chronic stress and impact on health and cognition," *Neuroscience and Biobehavioral Reviews*, vol. 35, no. 1, pp. 2–16, 2010.

[32] O. Gersten, W. D. Dow, and L. Rosero-Bixby, "Stressors over the life course and neuroendocrine system dysregulation in Costa Rica," *Journal of Aging and Health*, vol. 22, no. 6, pp. 748–771, 2010.

[33] L. A. Yancura, C. M. Aldwin, M. R. Levenson, and A. Spiro III, "Coping, affect, and the metabolic syndrome in older men: how does coping get under the skin?" *Journals of Gerontology B*, vol. 61, no. 5, pp. P295–P303, 2006.

[34] E. Le Bourg and S. I. S. Rattan, "Hormesis and aging: what's the deal?" in *Mild Stress and Healthy Aging: Applying Hormesis in Aging Research and Interventions*, E. Le Bourg and S. I. S. Rattan, Eds., pp. 1–4, Springer, New York, NY, USA, 2008.

[35] A. M. Vaiserman, "Irradiation and hormesis," in *Mild stress and Healthy Aging: Applying Hormesis in Aging Research and Interventions*, E. Le Bourg and S. I. S. Rattan, Eds., pp. 21–42, Springer, New York, NY, USA, 2008.

[36] C. Chiueh, T. Andoh, and P. B. Chock, "Induction of thioredoxin and mitochondrial survival proteins mediates

preconditioning-induced cardioprotection and neuroprotection," *Annals of the New York Academy of Sciences*, vol. 1042, pp. 403–418, 2005.

[37] D. Holzman, "Hormesis: fact or fiction?" *Journal of Nuclear Medicine*, vol. 36, no. 12, pp. 13–16, 1995.

[38] R. A. Dienstbier, "Arousal and physiological toughness: implications for mental and physical health," *Psychological Review*, vol. 96, no. 1, pp. 84–100, 1989.

[39] L. Calhoun and R. Tedeschi, "The foundations of posttraumatic growth," in *The Handbook of Posttraumatic Growth: Research and Practice*, L. Calhoun and R. Tedeschi, Eds., Lawrence Erlbaum, Mahwah, NJ, USA, 2006.

[40] P. Schnurr, S. Rosenberg, and M. Friedman, "Change in MMPI scores from college to adulthood as a function of military service," *Journal of Abnormal Psychology*, vol. 102, no. 2, pp. 288–296, 1993.

[41] S. E. Hobfoll, "Social and psychological resources and adaptation," *Review of General Psychology*, vol. 6, no. 4, pp. 307–324, 2002.

[42] G. Vaillant, *Aging Well: Surprising Guideposts to a Happier Life*, Little-Brown, Boston, Mass, USA, 2002.

[43] B. L. Jones, D. S. Nagin, and K. Roeder, "A SAS procedure based on mixture models for estimating developmental trajectories," *Sociological Methods and Research*, vol. 29, no. 3, pp. 374–393, 2001.

[44] A. Spiro III and R. Bosse', "The Normative Aging Study," in *Encyclopedia of Aging*, G. Maddox, Ed., pp. 744–746, Springer, New York, NY, USA, 3rd edition, 2001.

[45] C. Aldwin, M. R. Levenson, A. Spiro III, and R. Bossé, "Does emotionality predict stress? Findings from the normative aging study," *Journal of Personality and Social Psychology*, vol. 56, no. 4, pp. 618–624, 1989.

[46] J. E. Ware and C. D. Sherbourne, "The MOS 36-item short-form health survey (SF-36). I. Conceptual framework and item selection," *Medical Care*, vol. 30, no. 6, pp. 473–483, 1992.

[47] D. S. Nagin, "Analyzing developmental trajectories: a semi-parametric, group-based approach," *Psychological Methods*, vol. 4, no. 2, pp. 139–157, 1999.

[48] D. Lambert, "Zero-inflated poisson regression, with an application to defects in manufacturing," *Technometrics*, vol. 34, no. 1, pp. 1–13, 1992.

[49] Q. Vuong, "Likelihood ratio tests for model selection and non-nested hypotheses," *Econometrica*, vol. 57, pp. 307–333, 1989.

[50] G. Schwartz, "Estimating the dimension of a model," *The Annals of Statistics*, vol. 6, pp. 461–464, 1978.

[51] R. E. Kass and L. Wasserman, "A reference Bayesian test for nested hypotheses and its relationship to the Schwarz criterion," *Journal of the American Statistical Association*, vol. 90, pp. 928–934, 1995.

[52] A. V. D'Unger, K. C. Land, P. L. McCall, and D. S. Nagin, "How many latent classes of delinquent/criminal careers? Results from mixed Poisson regression analyses of the London, Philadelphia, and Racine Cohorts studies," *The American Journal of Sociology*, vol. 103, pp. 1593–1630, 1998.

[53] D. R. Cox, "Regression models and life tables (with discussion)," *Journal of the Royal Statistical Society B*, vol. 74, pp. 187–220, 1972.

[54] D. K. Mroczek, A. Spiro III, and N. A. Turiano, "Do health behaviors explain the effect of neuroticism on mortality? Longitudinal findings from the VA normative aging study," *Journal of Research in Personality*, vol. 43, no. 4, pp. 653–659, 2009.

[55] M. Rutter, "Pathways from childhood to adult life," *Journal of Child Psychology and Psychiatry and Allied Disciplines*, vol. 30, no. 1, pp. 23–51, 1989.

[56] C. Aldwin, A. Spiro III, M. R. Levenson, and R. Bossé, "Longitudinal findings from the normative aging study: I. Does mental health change with age?" *Psychology and Aging*, vol. 4, no. 3, pp. 295–306, 1989.

[57] J. Newman, "Aging and depression," *Psychology and Aging*, vol. 4, pp. 150–165, 1989.

[58] R. C. Kessler, C. Foster, P. S. Webster, and J. S. House, "The relationship between age and depressive symptoms in two national surveys," *Psychology and Aging*, vol. 7, no. 1, pp. 119–126, 1992.

[59] E. L. Idler and Y. Benyamini, "Self-rated health and mortality: a review of twenty-seven community studies," *Journal of Health and Social Behavior*, vol. 38, no. 1, pp. 21–37, 1997.

[60] J. S. Tucker, H. S. Friedman, D. L. Wingard, and J. E. Schwartz, "Marital history at midlife as a predictor of longevity: alternative explanations to the protective effect of marriage," *Health Psychology*, vol. 15, no. 2, pp. 94–101, 1996.

[61] C. J. Holahan, K. K. Schutte, P. L. Brennan, C. K. Holahan, B. S. Moos, and R. H. Moos, "Late-life alcohol consumption and 20-year mortality," *Alcoholism: Clinical & Experimental Research*, vol. 34, no. 11, pp. 1961–1971, 2010.

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Review Article

Pathways to Aging: The Mitochondrion at the Intersection of Biological and Psychosocial Sciences

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Compelling evidence suggests that both biological and psychosocial factors impact the process of aging. However, our understanding of the dynamic interplay among biological and psychosocial factors across the life course is still fragmentary. For example, it needs to be established how the interaction of individual factors (e.g., genetic and epigenetic endowment and personality), behavioral factors (e.g., physical activity, diet, and stress management), and psychosocial experiences (e.g., social support, well-being, socioeconomic status, and marriage) in perinatal, childhood, and adulthood influence health across the aging continuum. This paper aims to outline potential intersection points serving as an interface between biological and psychosocial factors, with an emphasis on the mitochondrion. Mitochondria are cellular organelles which play a critical role in cellular senescence. Both chronic exposure to psychosocial stress and genetic-based mitochondrial dysfunction have strikingly similar biological consequences; both predispose individuals to adverse age-related health disorders and early mortality. Exploring the interactive nature of the factors resulting in pathways to normal healthy aging, as well as those leading to morbidity and early mortality, will continue to enhance our ability to translate research into effective practices that can be implemented throughout the life course to optimise the aging process.

1. Introduction

Aging is the inescapable process by which individuals, from the age of about 30 years old onwards, gradually lose maximal functional capacity [1]. Some resilient individuals experience a slow decline lasting several decades, attaining ages past one hundred years old and more [2]. These are exceptional centenarians who experience minimal physical impairment [3] along with healthy minds and bodies [4, 5]. However, many individuals experience more rapid functional declines in their 60's or 70's, sometimes afflicted with the "frailty syndrome"—defined as a lack in general strength and unusual susceptibility to disease or to other infirmity [6]—and these individuals often suffer from multiple age-related morbidities such as cardiovascular disease, neurodegenerative diseases, diabetes, and cancer [7]. The majority of individuals lie between these two extreme scenarios, with an average life expectancy of 81 years old in North America [8].

In the past century, we have witnessed significant increases in life expectancy as more individuals live longer [9, 10]. This increase in average life expectancy has undoubtedly resulted from advances in medical technologies and preventive medicine that prevent most (>80%) early deaths due to acute illnesses (e.g., infections and injuries) and prolong life of individuals afflicted with chronic life-threatening conditions (e.g., HIV/AIDS and cardiovascular disease) [10]. The outcome of effectively delaying mortality is that morbidity is postponed—or "compressed"—to older ages, as described in Fries' *compression of morbidity hypothesis* [11]. The incidence of age-related diseases has remained stable over the last decades, or even increased, which signifi-cantly contributes to health care costs [10]. Given that an unprecedentedly large proportion of the population is expected to reach 60–80 years of age in the next two decades [12], changes in political and social health policy will be necessary to face societal challenges [10, 13]. Comprehensive frameworks including the panoply of factors capable of potently modulating the human aging process may be essential to address the impending social imperative of implementing health-enhancing strategies for the elderly, at minimal costs.

A central question concerning longevity remains: Why do some people live long whereas others die early? Another equally critical question concerns morbidity: Why is aging associated with a greater incidence of almost every categorized disease—including degenerative, metabolic, and malignant disorders? Since disease incidence, mortality, and longevity are all associated terms in the same aging equation, a more general question may be posed: What are the pathways that impact individuals' rate of aging?

While it is well understood that both biological and psychosocial factors impact the aging process, it is still unclear *how* psychosocial factors influence cellular aging and translate into aging of the whole organism [4]. Below is a selective review focusing on physiological systems susceptible to constitute critical convergence points, acting as integrators of the interactive forces imposed by both biological and psychosocial factors. Understanding how this physiological integration takes place will improve researchers' means to develop multilevel interventions that optimize the decline in physical function associated with aging.

Physical health and function depends on the coordinated functioning of several organs and physiological systems that allow the organism in dynamic balance to adapt to perpetual environmental challenges. Failure to adapt to challenges (e.g., healing wounds, increasing energy expenditure, and replenishing dying postmitotic cells) may occur in aging. Thus, senescence-induced loss of cell numbers and/or optimal functioning can result in suboptimal organ function [14]. For this reason, markers of cellular aging, such as nuclear DNA telomere length, is occasionally used as an indicator of aging. These are the protective caps at the end of chromosomes, whose reduction in length is often used as a reliable and proximal indicator of cellular senescence [15].

2. Biological Determinants of Aging—A Role for the Mitochondrion?

Biological factors influence the aging process. An important constituent of mammalian cells are mitochondria. These dynamic subcellular organelles contain their own circular DNA, are the principal site of cellular adenosine triphosphate (cellular energy currency) synthesis, regulate cell death through apoptotic signalling, and are the major source of reactive oxygen species (ROS) within the cell [16, 17]. One of the most scrutinized hypothesis in aging research is the mitochondrial theory of aging which stipulates that, over time, mitochondrial DNA accumulates oxidative damage from ROS, which negatively impacts mitochondrial function, leading to cellular dysfunction, organ failure, and ultimately results in age-related disease [14]. Data supporting this theory has been obtained from transgenic animals with enhanced protection against mitochondrial oxidative damage [18, 19]. These mice, which over-express a mitochondrial-targeted catalase, are resistant to age-related insulin resistance [18] and have slightly increased lifespan [19]. However, existing data render this theory imperfect [20–23] and evidence supporting a direct role of ROS in aging has largely been correlative [24]. Furthermore, examples exist in vertebrates (e.g., naked mole rat [25]) and invertebrates (*c. elegans* [26, 27]) where the typical negative correlation between ROS production and lifespan is uncoupled. Although ROS-induced damage has not consistently been causally linked to aging, a general shift in intra- and extracellular redox state towards more oxidized levels occurs in aging cells and in the blood of aged individuals, which could have important implications for redox-sensitive signalling pathways and their influence on the aging process [28, 29].

In contrast, a general role of mitochondria in the aging process is supported by abounding experimental evidence [30–37]. For example, animals with a deficient proof-reading version of the mitochondrial DNA polymerase gamma (PolG mutator mice), a defect which leads to an abnormally rapid accumulation of mitochondrial DNA mutations, exhibit several characteristics reminiscent of an accelerated aging phenotype (e.g., graying of fur, loss of muscle and brain mass, and kyphosis) [38, 39]. This indicates that mitochondrial DNA damage is capable of causing aging-like symptoms such as organ dysfunction and early mortality. It must be noted that whether this model actually mimics natural human aging is uncertain [40, 41]. Similarly, whether ROS dictates the aging process [23] remains a contentious issue. Nevertheless, although its exact cellular and physiological impact remain unclear, the integrity of mitochondrial DNA is challenged during aging [42, 43] and may contribute to cellular senescence and consequently, to the progressive functional changes in organs that characterize the aging process.

Further evidence supporting a role of mitochondria in the aging process comes from interventions that influence mitochondrial function. The only intervention capable of extending life span in animals—lifelong caloric restriction [44]—diminishes damage to mitochondrial DNA and concomitantly decreases the age-related decline in muscle aerobic capacity [45, 46]. Of note, caloric restriction has also been reported to decrease the incidence of age-related illnesses (e.g., cancer) in rodents [47], providing an interesting empirical link between mitochondrial integrity and age-related morbidity. Moreover, evolutionarily inherited single nucleotide polymorphisms yielding genetic variants of mitochondrial DNA, called haplogroups, may influence mitochondrial function and health outcomes in humans (reviewed in [14]). Indeed, mitochondrial haplogroups have been associated with mitochondrial ROS production and cellular oxidative capacity [48–50], resting metabolic rate and energy expenditure in humans [51], and disease incidence, progression, and longevity [14, 52–54]. Collectively, this suggests that intrinsic mitochondrial factors (e.g., related to mitochondrial DNA) can indeed influence the aging process.

Like caloric restriction, physical activity and exercise are potent stimuli that increase mitochondrial content and function [55–57]. Physical activity reduces the age-related decline in function of different organ systems including brain and muscles. Indeed, it is established that individuals who are more physically active exhibit lower incidences of age-related diseases and mortality [58–60] as well as better control of

existing chronic diseases [60]. Furthermore, endurance exercise prevents the premature aging-like characteristics of the PolG mutator mice including mitochondrial abnormalities, skeletal muscle, and brain atrophy [61]. The converse is also true. Physical *inactivity* leads to a reduction in mitochondrial content and function [56, 62] and contributes towards insulin resistance (i.e., prediabetic state) [63] and enhanced metabolic risk [64].

The aforementioned physiological dysregulations are more commonly observed in old age. For example, older individuals (63–70 years old) who are sedentary, but not those who are active, have lower mitochondrial content than young individuals [65]. Finally, dietary lipid supply (e.g., virgin olive oil) has been shown to impact membrane composition in brain mitochondria and to reduce oxidative damage to these organelles with aging in rats [66]. Thus, factors that impact mitochondrial function (i.e., levels of physical activity, caloric restriction, and diet) can consequently impact age-related disease incidence, progression, and survival.

The findings outlined in this section are consistent with the notion that biological mechanisms determine the aging process. Additional arguments supporting this notion also exist. They notably include the loss of molecular fidelity with time as the major cause of aging [67] and the recently discovered link between mitochondrial function, telomere length, and cellular senescence [68, 69]. Because each aspect outlined above appears to modulate the aging process in small yet sizeable ways, we must acknowledge that evidence suggests that the rate of aging is not solely determined by single biological factors, such as how many calories are ingested, which genetic polymorphism an individual has inherited, and how much physical activity is performed. Rather, in real-life situations, the rate of aging for a given individual must ultimately be determined by the dynamic and reciprocal interplay of these and many other factors, as discussed below.

3. Psychosocial Determinants of Aging

Despite the fact that aging research has generally been dissected using the biological scalpel, psychological and social variables are also important modulators of the aging process associated with mortality [70–73]. For example, personality and lifestyle may influence longevity in humans [4]. In a prospective study of patients with coronary heart disease, the authors found that pessimism and anxious personality traits were associated with adverse age-related health outcomes such as greater cancer incidence [74] and all-cause mortality [75]. Degradation in negative affect (i.e., more negative emotions) was also a strong prognostic indicator of long-term mortality in coronary heart disease patients [76], suggesting that negative emotions can adversely influence survival and resilience.

On the other hand, centenarians with engaged lifestyle and certain personality traits (e.g., emotional stability, extraversion, and openness) tend to have higher mental health status, a healthy sign of aging, when compared to those who do not possess these traits [77]. A twenty-year prospective population study showed that individuals with more positive self-perceptions of aging tended to live about seven years longer than those with less positive perceptions of aging [78]. Likewise, self-rated health—SRH, how an individual subjectively rates his/her health—is one of the most powerful statistical predictor of morbidity and mortality [79, 80]. Of note, SRH is often a more powerful statistical predictor of mortality than clinical and biological assessments of health. Similarly, high socioeconomic status is associated with more positive multisystemic physiological profiles (i.e., allostatic load), which predict lower morbidity and mortality rates with aging [81, 82]. There is also evidence that "protective" psychosocial factors such as control beliefs and quality of social support (i.e., emotional links with family and friends) contribute to better maintenance of functional capacity with aging [83]. Although not directly supporting a causal link between psychosocial factors and longevity, these data strongly suggest that several psychosocial factors impact physiological pathways to aging and distal outcomes such as mortality and longevity.

Psychosocial factors also have similar effects on more proximal biological indices of aging. Not living with a partner (i.e., being unmarried) is associated with accelerated cellular aging, as evidenced by shorter telomere length in blood leukocytes of unmarried middle-aged men and women [84]. Similar reports by Epel and colleagues demonstrate that psychological stress is associated with accelerated telomere shortening [85]. Likewise, depression has been linked with accelerated rates of aging and cellular senescence [86] and mortality [75], demonstrating that psychosocial forces may accelerate cellular aging [72]. Collectively, these findings indicate that psychosocial forces can exert both negative and positive influences on the aging process, affecting both morbidity and mortality.

4. Mitochondria: Interfacing Two Worlds

As mentioned above, mitochondria influence cellular function [16, 17] and impairments in mitochondrial function due to genetic variations/mutations [14] or other stresses such as physical inactivity [56] may accelerate the aging process. Interestingly, several hormones including those involved in the body' stress responses to psychosocial stressors modulate the synthesis of new mitochondria (mitochondrial biogenesis) and can modify important parameters of mitochondrial function [87]. Indeed, mitochondrial DNA transcription and mitochondrial biogenesis are modulated by the glucocorticoid hormone cortisol downstream from the hypothalamic-pituitary-adrenal (HPA) axis, by catecholamines secreted by the sympathetic-innervated adrenal medulla (epinephrine and norepinephrine), thyroid hormones, and by the steroid hormone estrogen, as well as by several cytokines (e.g., IL-1α, IL-1β, and TNFα) [88].

In fact, the mitochondrial DNA sequence contains putative response elements for several hormonal receptors (e.g., thyroid and glucocorticoid hormones, and insulin) [89] and some receptors for glucocorticoids, thyroid hormones,

and estrogen have even been found in mitochondria of different cell types [90, 91]. Acutely, these "stress" hormones increase mitochondrial biogenesis and function [87]. However, chronic exposure to elevated levels of these hormones, which can be induced by psychosocial stressors (e.g., social isolation, depression, and violent or abusive social environment) [93], can lead to reductions in mitochondrial mass (see Figure 1) and concomitant increases in mitochondria-derived ROS production [87, 92]. These mitochondrial outputs synergistically damage cellular components and contribute to cellular senescence when chronically produced at high levels.

Beyond the direct effects that psychosocial stresses exert on mitochondrial function, psychosocial factors can also influence individual's lifestyles, such as levels of physical activity and inactivity (i.e., sedentariness) [93]. For example, negative perceptions of one's body image and negative influence from family and friends are associated with lower levels of physical activity [95–97]. Similarly, people who are depressed or suffer from mental illnesses also tend to be more physically inactive [98, 99]. Physical inactivity can in turn undermine physical and mental health [100], predisposing inactive young individuals to depression later in life [101].

Physical activity has positive effects on mitochondrial function and counteracts inflammatory processes and age-related chronic diseases [55]. It can even buffer against the negative effects of chronic stress on telomere shortening [102]. In fact, the beneficial effects of physical activity and exercise on the hormonal system (e.g., increases in interleukin 6, growth hormone, brain-derived growth factor—BDNF) [103, 104], on psychological and cognitive aspects (e.g., decrease in stress levels and reactivity to stress, depression, improved well-being) [105], as well as on metabolic regulation (e.g., increased mitochondrial mass and improved insulin sensitivity) [65, 106], suggest that exercise and physical activity exert multisystemic protective effects which can prevent the deleterious consequences of chronic stress [107]. Indeed, improving physical fitness has been shown to decrease hormonal, physiological and psychological markers of chronic stress [72]. Psychosocial factors and physical activity can therefore interact to influence mitochondrial function and modulate the impact of chronic stress on the body.

Because mitochondria influence cellular aging and are responsive to stress hormone levels, they are especially well equipped to act as key integrators that synergistically influence biological and psychosocial factors (Figure 1). As described above, work in the psychosocial sciences has unravelled important links between how individuals feel, their social contexts, and the effects of these factors on mortality and longevity. However, how these factors influence and interact with biological factors remains to be explored in more depth. The findings described above and many others bring new evidence coaxing researchers to focus on the *interactions* of biological and psychosocial forces that influence the aging process [70]. Conclusions derived from research *not* adopting an integrative approach risk being uni-dimensional and thus difficult to apply towards different real-life contexts, where individuals age under the collective influence of factors of different nature.

5. Interdisciplinarity: A Necessary Framework for Aging Research?

Interdisciplinarity and even transdisciplinarity [108] have emerged as key necessities in the field of aging and others [108–110]. Both the National Institutes of Health (NIH) in the USA and the Canadian Institutes of Health Research (CIHR) funding agencies have opened institutes on/of aging promoting broad mandates that necessarily reach across traditional disciplinary boundaries. Although some problems are best addressed with the approach of a single discipline, other issues require the integration of several disciplines to fully comprehend the complexity of the processes at play [111]. This is particularly true for aging [109]. Likewise, the discipline of developmental psychopathology, driven by the impetus to grasp and impact the complexities of mental health at different stages of development, has evolved to become a deeply "interdisciplinary field that seeks to elucidate the interplay among the biological, psychological, and social-contextual aspects of normal and abnormal development across the life course" [112, p.16]. The writings of Cicchetti and Toth [112] are particularly enlightening regarding the conceptual, theoretical, and practical directions to take to achieve such a degree of integration within a discipline. They particularly stress the importance of cross-disciplinary dialogue [112], along with the need for an emphasis on the process of development naturally resulting from the interdependence over time of multiple biological and psychosocial factors [113].

In this spirit, the aging process is influenced by several interactive forces inherent to the individual (e.g., genetic endowment, physical activity, diet, lifestyle, and personality) and forces inherent to the psychosocial environment (e.g., sociocultural context, family, and life stress) that continuously and progressively interact over long periods. Aging must therefore be approached from an ontogenic perspective not solely focused on end outcomes (i.e., mortality, comparison of aged and young individuals) or on the molecular factors predicting these outcomes. Instead, aging research would benefit from being guided by a perspective focused on the changes and interactions among biological and psychosocial processes, which take place across stages of human development throughout the lifespan. Table 1 lists five conceptual propositions elaborated by Ryff and Singer [70] to advance hypothesis-testing research focused on healthy aging as an interdisciplinary process. In a similar way that mental health and disease emerge from cumulative life experiences in infancy, childhood, and adulthood [112–114], aging must also be a deeply experience-dependent process where the biology influences how a person responds to their experiences, but where the biology is also shaped by those experiences.

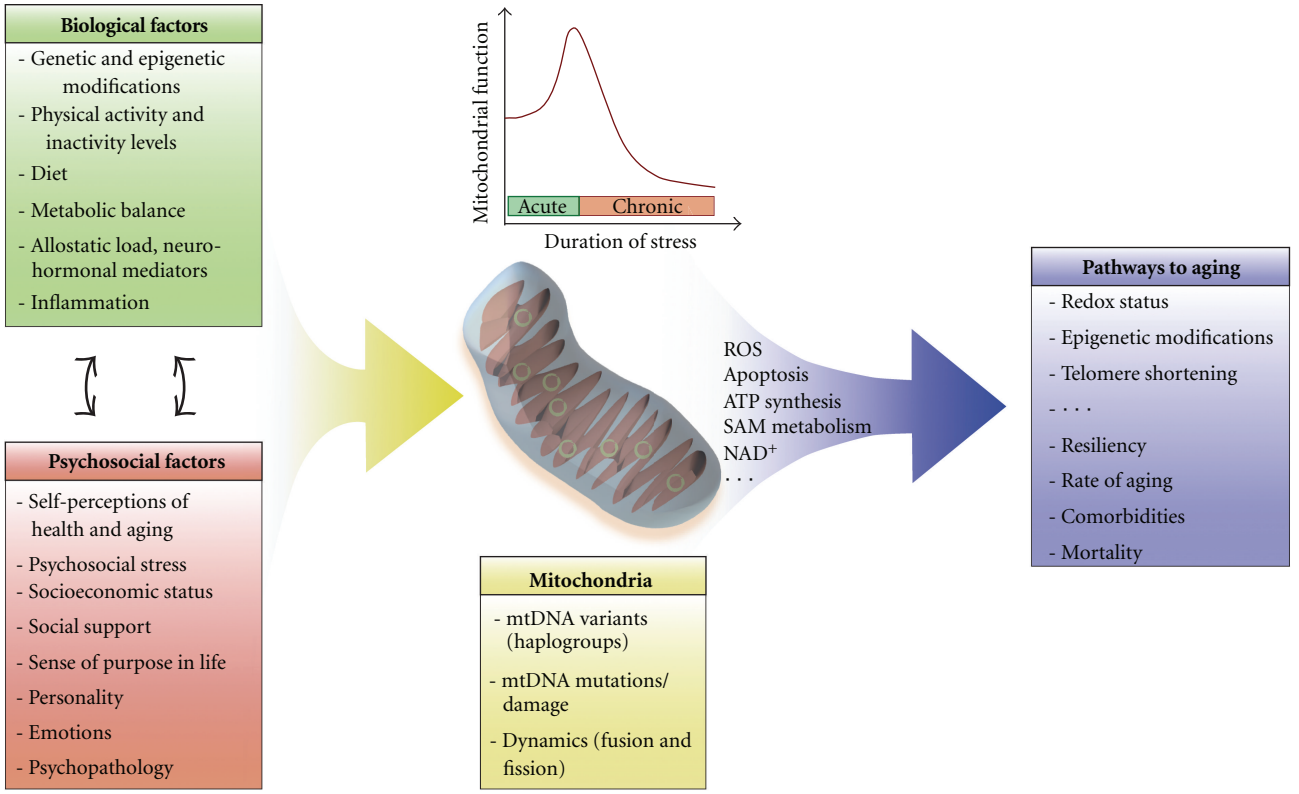

Figure 1: Mitochondria influence pathways to aging by operating at the intersection of biological and psychosocial factors. Biological and psychosocial factors dynamically/bidirectionally interact to influence mitochondrial content and function in the body's tissues. The same factors can exert opposite effects on mitochondrial function, depending on the duration of exposure. For instance, acute stressors tend to upregulate mitochondrial biogenesis and function, whereas chronic stressors tend to downregulate mitochondrial biogenesis and function (top center graph). Mitochondrial-level factors influence mitochondrial function and may determine mitochondrial responsivity to upstream biological and psychosocial influences. In response to multiple individual and environmental factors, mitochondria produce outputs influencing cellular function, gene expression, and cellular senescence. As a result, pathways to aging are ultimately determined by the integrated and synergistic influence of multiple biological and psychosocial factors.

Table 1: Research propositions to advance knowledge of healthy aging—Adapted from Ryff and Singer [70].

Proposition 1	*Health promotion processes:* positive psychosocial factors predict better biological regulation *Premise:* positive health and high levels of well-being are associated with lower morbidity, decreased physical symptoms and pain, increased longevity, increased resistance to illness, decreased stroke incidence, and better glycemic control.
Proposition 2	*Resilience Processes:* positive psychosocial factors protect against the damaging effects of external adversity *Premise:* psychological strengths (e.g., personality traits and coping ability) and favourable social situations (e.g., social/family support and high socioeconomic status) are associated with "physiological toughness" and an enhanced ability to maintain a high-level of functioning in the face of adversity.
Proposition 3	*Recovery and repair processes:* Positive psychosocial factors facilitate the regaining of functional and/or biological capacities *Premise:* hopeful individuals with optimistic beliefs and positive expectations about their health have better prognosis from heart surgeries, some cancers and HIV/AIDS, and possibly better DNA repair mechanisms.
Proposition 4	*Compensation processes:* psychological or biological strengths can offset the negative health consequences of psychological or biological weaknesses *Premise:* psychological distress and adversity can be moderated by positive psychological traits (e.g., coping strategies and affective styles).
Proposition 5	*Gene expression processes:* psychosocial factors as mitigating against the negative and promoting the positive *Premise:* many people with genetic susceptibilities to certain diseases never develop them; psychosocial and other environmentallydriven epigenetic factors may modulate genetic susceptibility to disease and gene expression patterns that impact health in aging.

6. Experience-Dependent Modulation of Aging: A Role for Epigenetics?

The experience-dependent nature of aging has important implications for the research questions that are posed (e.g., How do specific events/factors at different life stages interact to modulate the rate of aging?). This suggests that cumulative prenatal, earlylife, young adulthood, and later life circumstances impact pathways to aging. This could be mediated in part by alterations in the stress system across the life course: induced alterations in neurological substrates that signal stressful information [114, 115] as well as neurobiological and allostatic processes involving inflammation and oxidative stress [72, 116]. Thus, as suggested by Epel [72], psychological and metabolic stress may constitute a potent recipe for accelerated cellular aging.

Epigenetics, which involves the laying of relatively stable imprints on the genome that impact gene expression and cellular function over time [117, 118], is increasingly revealed as a candidate intersection point between biological and psychosocial processes in several age-related chronic diseases [119, 120]. It is known that epigenetic marks are altered in aging [121, 122] and in several age-related disease states such as cancer, neurodegenerative, and autoimmune diseases [123], as well as type 2 diabetes [124, 125]. The altered epigenome could therefore mediate the experience-dependent modulation of the aging rate and age-related morbidities across the life span. Further to that point, mitochondria themselves possess a plastic mtDNA epigenome [126] and have the potential to generate powerful signals capable of affecting the nuclear epigenome [127, 128], making these organelles well equipped to play a critical interfacing role between the environment and the genome [127]. Although this remains to be empirically supported, it is an hypothesis that integrates knowledge about the health consequences attributable to genetic variations, calorie intake, physical activity/inactivity, and neurobiological substrates of psychosocial stress into a unified framework for aging research.

Taken together, the reviewed literature indicates that an increasing number of biological factors (e.g., mtDNA haplotypes, hormones, genetic polymorphisms affecting cellular signalling pathways, factors epigenetic imprints), behavioral and (e.g., diet/calorie intake, and exercise), and psychosocial (e.g., psychological stress, depression, personality, and marital status) factors influence the aging process. The challenge lying ahead of researchers in this field lies in the exploration of the intersection points linking these multiple levels of analysis spanning several disciplines. For example, what physiological processes interact with the psychosocial effects of being married, of meditating regularly, or of experiencing psychological well-being and sense of purpose in life, which ultimately culminate in reduced telomere shortening [129]? What combination of elements lead to resilience and successful adaptation to aging? And what are the combinations leading to age-related risk and ill-health? Interdisciplinary initiatives aimed at describing the interactive biopsychosocial processes that link these multiple levels will yield new knowledge of the pathways to aging, which in turn will inform effective prevention and intervention strategies. Network perspectives inspired from systems biology [130, 131] allowing modeling of complex nonlinear interactions among the studied variables may prove useful in this endeavour. Similarly, building comprehensive theories of aging will require the combined efforts of researchers from different disciplines contributing diverse complementary expertise, perspectives, and approaches to study aging.

7. Individualized Aging Trajectories

Effective strategies for promoting healthy aging will need to be individualized. The perfect individualization of treatment and prevention of age-related disorders may appear as an unattainable utopia at this point in time. This is particularly the case because up until now, our knowledge of the dynamic interplay between the different biological and psychosocial levels of analysis is still fragmentary, which impedes discoveries about the complex processes from which individual-specific pathways of aging emerge. A well-known principle in biology and developmental psychopathology is that of "equifinality," whereby multiple distinct pathways lead to the same outcome. The reciprocal principle is that of "multifinality," whereby the same set of pathways lead to different outcomes [112]. Likewise, the source of interindividual differences in aging trajectories undoubtedly lies in the interplay of several interdependent pathways of which there is no single universal "right" combination that can be prescribed.

Means must be developed to distinguish between optimal (i.e., living to your full biological potential) and suboptimal (i.e., dying or having disease sooner than your constitution should permit) rates/trajectories of aging. From the onset, it can be established that optimal aging is characterized by a slow progressive decline in physiological functions, maintenance of well-being for the majority of the lifespan, and only a short period of very poor physical health leading to death. But what are the biomolecular (i.e., gene expression, mitochondrial function, and biomarkers) sings of optimal adaptation to the passing decades? What are the normative ontogenic trajectories, or healthy biological and physiological signatures of successful aging? Having answers to these questions will enable researchers to more accurately distinguish dysfunction from normal function in different aged organ systems. Ryff, Singer and colleagues have established biological correlates and a conceptual framework aimed at deciphering the biological and psychosocial underpinnings of resilience, positive health, and successful aging [71, 132–134]. Building such a knowledge base of normal molecular, cellular, physiological, and psychosocial signatures of aging may also translate into more refined means to detect predisease or preclinical deviations from normal adaptation and to prevent age-related diseases.

Thus far, despite the fact that more resources are being invested to study specific aspects of the normal pathways leading to healthy aging [71, 132–134], relatively little data is available to address pressing questions about healthy aging. A noteworthy exception is the MacArthur Studies of Successful

Aging, which have collected a rich dataset spanning multiple biological and psychosocial levels over several years, thus providing an exceptional design for longitudinal evaluation of the biological-psychosocial interactions for a large cohort of elderly individuals [135]. Future smaller-scale (i.e., intervention trials and animal-based models) integrative research initiatives should build from the strengths and experience of this and other such longitudinal endeavours [136].

8. Conclusions

In conclusion, as a rejoinder to the question "What are the pathways that impact individuals rate of aging?", we ought to answer that there are surely several different pathways to healthy aging. These pathways must depend not on singular factors acting independently, but on interactive forces among multiple levels of function operating in synergy [108], including biological, behavioural, psychosocial and spiritual factors [108]. Identifying the developmental nature of "pathways to aging" is an interdisciplinary task inviting researchers in aging to join forces to discover and refine our comprehension of the intersections between our respective disciplines. Biomedical scientists need to appreciate the complexity of biological-psychosocial interactions involved in health processes; and psychosocial researchers need to appreciate the underlying biological factors susceptible to modulate individual responses to psychosocial challenges. This can be achieved, along with the ensuing collaborative interdisciplinary successes in research, by defining and empirically testing potential intersection points among biological and psychosocial disciplines. A deeper understanding of these intersections, and of the ensuing mind-body crosstalk [92], will enhance our appreciation of the multiple interacting facets that collectively determine optimal and suboptimal rates of aging for individuals. Testing and defining inter- and transdisciplinary intersection points should also enhance our ability to translate health discoveries into applicable interventions to promote the health and quality of life of an increasingly old population.

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References

[1] G. M. Martin, "Help wanted: phenomenological models for research on aging," *Science of Aging Knowledge Environment*, vol. 2002, p. VP2, 2002.

[2] P. Martin, M. MacDonald, J. Margrett, and L. W. Poon, "Resilience and longevity: expert survivorship of centenarians," in *New Frontiers in Resilient Aging: Life-Strengths and Well-Being in Late Life*, P. Fry and C. Keyes, Eds., pp. 213–238, Cambridge University Press, New York, NY, USA, 2010.

[3] J. Evert, E. Lawler, H. Bogan, and T. Perls, "Morbidity profiles of centenarians: survivors, delayers, and escapers," *Journals of Gerontology. Series A*, vol. 58, no. 3, pp. 232–237, 2003.

[4] L. W. Poon, P. Martin, A. Bishop et al., "Understanding centenarians' psychosocial dynamics and their contributions to health and quality of life," *Current Gerontology and Geriatrics Research*, vol. 2010, Article ID 680657, 13 pages, 2010.

[5] L. W. Poon, P. Martin, G. M. Clayton, S. Messner, C. A. Noble, and M. A. Johnson, "The influences of cognitive resources on adaptation and old age," *International Journal of Aging and Human Development*, vol. 34, no. 1, pp. 31–46, 1992.

[6] H. Bergman, L. Ferrucci, J. Guralnik et al., "Frailty: an emerging research and clinical paradigm—issues and controversies," *Journals of Gerontology. Series A*, vol. 62, no. 7, pp. 731–737, 2007.

[7] Q. L. Xue, "The frailty syndrome: definition and natural history," *Clinics in Geriatric Medicine*, vol. 27, no. 1, pp. 1–15, 2011.

[8] United Nations, "World Population Prospects, The 2006 Revisions," 2006, http://www.un.org/esa/population/publications/wpp2006/WPP2006_Highlights_rev.pdf

[9] J. W. Vaupel, "Biodemography of human ageing," *Nature*, vol. 464, no. 7288, pp. 536–542, 2010.

[10] S. J. Olshansky, D. P. Goldman, Y. Zheng, and J. W. Rowe, "Aging in America in the twenty-first century: demographic forecasts from the MacArthur foundation research network on an aging society," *Milbank Quarterly*, vol. 87, no. 4, pp. 842–862, 2009.

[11] J. F. Fries, "Aging, natural death, and the compression of morbidity," *New England Journal of Medicine*, vol. 303, no. 3, pp. 130–135, 1980.

[12] S. Wild, G. Roglic, A. Green, R. Sicree, and H. King, "Global prevalence of diabetes: estimates for the year 2000 and projections for 2030," *Diabetes Care*, vol. 27, no. 5, pp. 1047–1053, 2004.

[13] V. Mor, "The compression of morbidity hypothesis: a review of research and prospects for the future," *Journal of the American Geriatrics Society*, vol. 53, no. 9, pp. S308–S309, 2005.

[14] D. C. Wallace, "A mitochondrial paradigm of metabolic and degenerative diseases, aging, and cancer: a dawn for evolutionary medicine," *Annual Review of Genetics*, vol. 39, pp. 359–407, 2005.

[15] S. R. W. L. Chan and E. H. Blackburn, "Telomeres and telomerase," *Philosophical Transactions of the Royal Society B*, vol. 359, no. 1441, pp. 109–121, 2004.

[16] P. S. Brookes, Y. Yoon, J. L. Robotham, M. W. Anders, and S. S. Sheu, "Calcium, ATP, and ROS: a mitochondrial love-hate triangle," *American Journal of Physiology*, vol. 287, no. 4, pp. C817–C833, 2004.

[17] M. R. Duchen, "Mitochondria in health and disease: perspectives on a new mitochondrial biology," *Molecular Aspects of Medicine*, vol. 25, no. 4, pp. 365–451, 2004.

[18] H. Y. Lee, C. S. Choi, A. L. Birkenfeld et al., "Targeted expression of catalase to mitochondria prevents age-associated reductions in mitochondrial function and insulin resistance," *Cell Metabolism*, vol. 12, no. 6, pp. 668–674, 2010.

[19] S. E. Schriner, N. J. Linford, G. M. Martin et al., "Medecine: extension of murine life span by overexpression of catalase

targeted to mitochondria," *Science*, vol. 308, no. 5730, pp. 1909–1911, 2005.

[20] Y. C. Jang and H. V. Remmen, "The mitochondrial theory of aging: insight from transgenic and knockout mouse models," *Experimental Gerontology*, vol. 44, no. 4, pp. 256–260, 2009.

[21] R. T. Hepple, "Alterations in mitochondria and their impact in aging skeletal muscle," in *Sarcopenia—Age-Related Muscle Wasting and Weakness: Mechanisms and Treatments*, G. Lynch, Ed., pp. 135–158, Springer, New York, NY, USA, 2011.

[22] M. Picard, D. Ritchie, K. J. Wright et al., "Mitochondrial functional impairment with aging is exaggerated in isolated mitochondria compared to permeabilized myofibers," *Aging Cell*, vol. 9, no. 6, pp. 1032–1046, 2010.

[23] A. B. Salmon, A. Richardson, and V. I. Pérez, "Update on the oxidative stress theory of aging: does oxidative stress play a role in aging or healthy aging?" *Free Radical Biology and Medicine*, vol. 48, no. 5, pp. 642–655, 2010.

[24] H. Van Remmen and D. P. Jones, "Current thoughts on the role of mitochondria and free radicals in the biology of aging," *Journals of Gerontology. Series A*, vol. 64, no. 2, pp. 171–174, 2009.

[25] B. Andziak, T. P. O'Connor, W. Qi et al., "High oxidative damage levels in the longest-living rodent, the naked mole-rat," *Aging Cell*, vol. 5, no. 6, pp. 463–471, 2006.

[26] J. M. Van Raamsdonk and S. Hekimi, "Reactive oxygen species and aging in caenorhabditis elegans: causal or casual relationship?" *Antioxidants and Redox Signaling*, vol. 13, no. 12, pp. 1911–1953, 2010.

[27] W. Yang and S. Hekimi, "A mitochondrial superoxide signal triggers increased longevity in caenorhabditis elegans," *PLoS Biology*, vol. 8, no. 12, Article ID e1000556, 2010.

[28] D. P. Jones, "Extracellular redox state: refining the definition of oxidative stress in aging," *Rejuvenation Research*, vol. 9, no. 2, pp. 169–181, 2006.

[29] G. J. Brewer, "Epigenetic oxidative redox shift (EORS) theory of aging unifies the free radical and insulin signaling theories," *Experimental Gerontology*, vol. 45, no. 3, pp. 173–179, 2010.

[30] L. P. Guarente, L. Patridge, and D. C. Wallace, *Molecular Biology of Aging*, Cold Springs Harbor, New York, NY, USA, 2008.

[31] H. V. Remmen and A. Richardson, "Oxidative damage to mitochondria and aging," *Experimental Gerontology*, vol. 36, no. 7, pp. 957–968, 2001.

[32] L. A. Loeb, D. C. Wallace, and G. M. Martin, "The mitochondrial theory of aging and its relationship to reactive oxygen species damage and somatic mtDNA mutations," *Proceedings of the National Academy of Sciences of the United States of America*, vol. 102, no. 52, pp. 18769–18770, 2005.

[33] A. Terman, T. Kurz, M. Navratil, E. A. Arriaga, and U. T. Brunk, "Mitochondrial Turnover and aging of long-lived postmitotic cells: the mitochondrial-lysosomal axis theory of aging," *Antioxidants and Redox Signaling*, vol. 12, no. 4, pp. 503–535, 2010.

[34] G. Lenaz, A. Baracca, R. Fato, M. L. Genova, and G. Solaini, "New insights into structure and function of mitochondria and their role in aging and disease," *Antioxidants and Redox Signaling*, vol. 8, no. 3-4, pp. 417–437, 2006.

[35] I. Bratic and A. Trifunovic, "Mitochondrial energy metabolism and ageing," *Biochimica et Biophysica Acta*, vol. 1797, no. 6-7, pp. 961–967, 2010.

[36] S. Y. Park, B. Choi, H. Cheon et al., "Cellular aging of mitochondrial DNA-depleted cells," *Biochemical and Biophysical Research Communications*, vol. 325, no. 4, pp. 1399–1405, 2004.

[37] T. Wenz, "Mitochondria and PGC-1α in aging and age-associated diseases," *Journal of Aging Research*, vol. 2011, Article ID 810619, 12 pages, 2011.

[38] A. Trifunovic, A. Wredenberg, M. Falkenberg et al., "Premature ageing in mice expressing defective mitochondrial DNA polymerase," *Nature*, vol. 429, no. 6990, pp. 417–423, 2004.

[39] C. C. Kujoth, A. Hiona, T. D. Pugh et al., "Medicine: mitochondrial DNA mutations, oxidative stress, and apoptosis in mammalian aging," *Science*, vol. 309, no. 5733, pp. 481–484, 2005.

[40] R. A. Miller, D. Gershon, T. A. Prolla, and R. H. Weindruch, "Evaluating evidence for aging," *Science*, vol. 310, no. 5747, pp. 441–443, 2005.

[41] K. Khrapko, Y. Kraytsberg, A. D. N. J. de Grey, J. Vijg, and E. A. Schon, "Does premature aging of the mtDNA mutator mouse prove that mtDNA mutations are involved in natural aging?" *Aging Cell*, vol. 5, no. 3, pp. 279–282, 2006.

[42] C. Desler, M. L. Marcker, K. K. Singh, and L. J. Rasmussen, "The importance of mitochondrial DNA in aging and cancer," *Journal of Aging Research*, vol. 2011, Article ID 407536, 9 pages, 2011.

[43] R. Gredilla, "DNA damage and base excision repair in mitochondria and their role in aging," *Journal of Aging Research*, vol. 2011, Article ID 257093, 9 pages, 2011.

[44] S. L. Hlelfand, J. H. Beuer, and J. G. Wood, "Calorie restriction in lower organisms," in *Molecular Biology of Aging*, L. P. Guarente, L. Patridge, and D. C. Wallace, Eds., Cold Springs Harbor, New York, NY, USA, 2008.

[45] R. T. Hepple, D. J. Baker, J. J. Kaczor, and D. J. Krause, "Long-term caloric restriction abrogates the age-related decline in skeletal muscle aerobic function," *FASEB Journal*, vol. 19, no. 10, pp. 1320–1322, 2005.

[46] R. T. Hepple, "Why eating less keeps mitochondria working in aged skeletal muscle," *Exercise and Sport Sciences Reviews*, vol. 37, no. 1, pp. 23–28, 2009.

[47] E. J. Masoro, "Caloric restriction and aging: an update," *Experimental Gerontology*, vol. 35, no. 3, pp. 299–305, 2000.

[48] R. Moreno-Loshuertos, R. Acín-Pérez, P. Fernández-Silva et al., "Differences in reactive oxygen species production explain the phenotypes associated with common mouse mitochondrial DNA variants," *Nature Genetics*, vol. 38, no. 11, pp. 1261–1268, 2006.

[49] E. Ruiz-Pesini, A. C. Lapena, C. Diez-Sanchez et al., "Human mtDNA haplogroups associated with high or reduced spermatozoa motility," *American Journal of Human Genetics*, vol. 67, no. 3, pp. 682–696, 2000.

[50] A. Marcuello, D. Martínez-Redondo, Y. Dahmani et al., "Human mitochondrial variants influence on oxygen consumption," *Mitochondrion*, vol. 9, no. 1, pp. 27–30, 2009.

[51] G. J. Tranah, T. M. Manini, K. K. Lohman et al., "Mitochondrial DNA variation in human metabolic rate and energy expenditure," *Mitochondrion*. In press.

[52] Y. Nishigaki, N. Fuku, and M. Tanaka, "Mitochondrial haplogroups associated with lifestyle-related diseases and longevity in the Japanese population," *Geriatrics and Gerontology International*, vol. 10, no. 1, pp. S221–S235, 2010.

[53] S. L. Hendrickson, H. B. Hutcheson, E. Ruiz-Pesini et al., "Mitochondrial DNA haplogroups influence AIDS progression," *AIDS*, vol. 22, no. 18, pp. 2429–2439, 2008.

[54] E. Khusnutdinova, I. Gilyazova, E. Ruiz-Pesini et al., "A mitochondrial etiology of neurodegenerative diseases: evidence from Parkinson's disease," *Annals of the New York Academy of Sciences*, vol. 1147, pp. 1–20, 2008.

[55] C. Handschin and B. M. Spiegelman, "The role of exercise and PGC1α in inflammation and chronic disease," *Nature*, vol. 454, no. 7203, pp. 463–469, 2008.

[56] I. R. Lanza and K. Sreekumaran Nair, "Regulation of skeletal muscle mitochondrial function: genes to proteins," *Acta Physiologica*, vol. 199, no. 4, pp. 529–547, 2010.

[57] J. R. Sattelmair, J. H. Pertman, and D. E. Forman, "Effects of physical activity on cardiovascular and noncardiovascular outcomes in older adults," *Clinics in Geriatric Medicine*, vol. 25, no. 4, pp. 677–702, 2009.

[58] T. M. Manini, J. E. Everhart, K. V. Patel et al., "Daily activity energy expenditure and mortality among older adults," *Journal of the American Medical Association*, vol. 296, no. 2, pp. 171–179, 2006.

[59] T. M. Manini, "Energy expenditure and aging," *Ageing Research Reviews*, vol. 9, no. 1, pp. 1–11, 2010.

[60] US Department of Health and Human Services, *Physical activity and health: a report of the Surgeon General*, US Department of Health and Human Services, Public Health Service, CDC, National Center for Chronic Disease Prevention and Health Promotion, Atlanta, Ga, USA, 1996.

[61] A. Safdar, J. M. Bourgeois, D. I. Ogborn et al., "Endurance exercise rescues progeroid aging and induces systemic mitochondrial rejuvenation in mtDNA mutator mice," *Proceedings of the National Academy of Sciences of the United States of America*, vol. 108, no. 10, pp. 4135–4140, 2011.

[62] P. A. Figueiredo, S. K. Powers, R. M. Ferreira, F. Amado, H. J. Appell, and J. A. Duarte, "Impact of lifelong sedentary behavior on mitochondrial function of mice skeletal muscle," *Journals of Gerontology. Series A*, vol. 64, no. 9, pp. 927–939, 2009.

[63] R. Krogh-Madsen, J. P. Thyfault, C. Broholm et al., "A 2-wk reduction of ambulatory activity attenuates peripheral insulin sensitivity," *Journal of Applied Physiology*, vol. 108, no. 5, pp. 1034–1040, 2010.

[64] J. P. Thyfault and F. W. Booth, "Lack of regular physical exercise or too much inactivity," *Current Opinion in Clinical Nutrition and Metabolic Care*, vol. 14, no. 4, pp. 374–378, 2011.

[65] A. Safdar, M. J. Hamadeh, J. J. Kaczor, S. Raha, J. deBeer, and M. A. Tarnopolsky, "Aberrant mitochondrial homeostasis in the skeletal muscle of sedentary older adults," *PLoS One*, vol. 5, no. 5, Article ID e10778, 2010.

[66] J. J. Ochoa, R. Pamplona, M. C. Ramirez-Tortosa et al., "Age-related changes in brain mitochondrial DNA deletion and oxidative stress are differentially modulated by dietary fat type and coenzyme Q 10," *Free Radical Biology and Medicine*, vol. 50, no. 9, pp. 1053–1064, 2011.

[67] L. Hayflick, "Biological aging is no longer an unsolved problem," *Annals of the New York Academy of Sciences*, vol. 1100, pp. 1–13, 2007.

[68] E. Sahin, S. Colla, M. Liesa et al., "Telomere dysfunction induces metabolic and mitochondrial compromise," *Nature*, vol. 470, no. 7334, pp. 359–365, 2011.

[69] E. Sahin and R. A. Depinho, "Linking functional decline of telomeres, mitochondria and stem cells during ageing," *Nature*, vol. 464, no. 7288, pp. 520–528, 2010.

[70] C. D. Ryff and B. Singer, "Understanding healthy aging: key components and their integration," in *Handbook of Theories of Aging*, V. L. Bengtson, D. Gans, N. Putney, and M. Silverstein, Eds., Springer, New York, NY, USA, 2009.

[71] B. Singer and C. D. Ryff, "Positive health: resilience, recovery, primary prevention, and health promotion," in *New Horizons in Health: An Integrative Approach*, National Academy Press, Washington, DC, USA, 2001.

[72] E. S. Epel, "Psychological and metabolic stress: a recipe for accelerated cellular aging?" *Hormones*, vol. 8, no. 1, pp. 7–22, 2009.

[73] M. Marmot, "Social determinants of health inequalities," *Lancet*, vol. 365, no. 9464, pp. 1099–1104, 2005.

[74] J. Denollet, "Personality and risk of cancer in men with coronary heart disease," *Psychological Medicine*, vol. 28, no. 4, pp. 991–995, 1998.

[75] J. Denollet, S. U. Sys, N. Stroobant, H. Rombouts, T. C. Gillebert, and D. L. Brutsaert, "Personality as independent predictor of long-term mortality in patients with coronary heart disease," *Lancet*, vol. 347, no. 8999, pp. 417–421, 1996.

[76] J. Denollet and D. L. Brutsaert, "Reducing emotional distress improves prognosis in coronary heart disease: 9-year mortality in a clinical trial of rehabilitation," *Circulation*, vol. 104, no. 17, pp. 2018–2023, 2001.

[77] P. Martin, J. Baenziger, M. MacDonald, I. C. Siegler, and L. W. Poon, "Engaged lifestyle, personality, and mental status among centenarians," *Journal of Adult Development*, vol. 16, no. 4, pp. 199–208, 2009.

[78] B. R. Levy, M. D. Slade, S. R. Kunkel, and S. V. Kasl, "Longevity increased by positive self-perceptions of aging," *Journal of Personality and Social Psychology*, vol. 83, no. 2, pp. 261–270, 2002.

[79] M. Jylhä, "What is self-rated health and why does it predict mortality? Towards a unified conceptual model," *Social Science and Medicine*, vol. 69, no. 3, pp. 307–316, 2009.

[80] E. L. Idler and Y. Benyamini, "Self-rated health and mortality: a review of twenty-seven community studies," *Journal of Health and Social Behavior*, vol. 38, no. 1, pp. 21–37, 1997.

[81] T. Seeman, E. Epel, T. Gruenewald, A. Karlamangla, and B. S. Mcewen, "Socio-economic differentials in peripheral biology: cumulative allostatic load," *Annals of the New York Academy of Sciences*, vol. 1186, pp. 223–239, 2010.

[82] T. E. Seeman, B. S. McEwen, J. W. Rowe, and B. H. Singer, "Allostatic load as a marker of cumulative biological risk: MacArthur studies of successful aging," *Proceedings of the National Academy of Sciences of the United States of America*, vol. 98, no. 8, pp. 4770–4775, 2001.

[83] M. E. Lachman and S. Agrigoroaei, "Promoting functional health in midlife and old age: long-term protective effects of control beliefs, social support, and physical exercise," *PLoS One*, vol. 5, no. 10, Article ID e13297, 2010.

[84] A. G. Mainous, C. J. Everett, V. A. Diaz et al., "Leukocyte telomere length and marital status among middle-aged adults," *Age and Ageing*, vol. 40, no. 1, pp. 73–78, 2011.

[85] E. S. Epel, E. H. Blackburn, J. Lin et al., "Accelerated telomere shortening in response to life stress," *Proceedings of the National Academy of Sciences of the United States of America*, vol. 101, no. 49, pp. 17312–17315, 2004.

[86] O. W. Wolkowitz, E. S. Epel, V. I. Reus, and S. H. Mellon, "Depression gets old fast: do stress and depression accelerate cell aging?" *Depression and Anxiety*, vol. 27, no. 4, pp. 327–338, 2010.

[87] I. Manoli, S. Alesci, M. R. Blackman, Y. A. Su, O. M. Rennert, and G. P. Chrousos, "Mitochondria as key components of the stress response," *Trends in Endocrinology and Metabolism*, vol. 18, no. 5, pp. 190–198, 2007.

[88] P. Puigserver and B. M. Spiegelman, "Peroxisome proliferator-activated receptor-γ coactivator 1α (PGC-1α): transcriptional coactivator and metabolic regulator," *Endocrine Reviews*, vol. 24, no. 1, pp. 78–90, 2003.

[89] C. D. Berdanier, "Mitochondrial gene expression: Influence of nutrients and hormones," *Experimental Biology and Medicine*, vol. 231, no. 10, pp. 1593–1601, 2006.

[90] A. M. G. Psarra and C. E. Sekeris, "Glucocorticoid receptors and other nuclear transcription factors in mitochondria and possible functions," *Biochimica et Biophysica Acta*, vol. 1787, no. 5, pp. 431–436, 2009.

[91] A. M. G. Psarra, S. Solakidi, and C. E. Sekeris, "The mitochondrion as a primary site of action of steroid and thyroid hormones: presence and action of steroid and thyroid hormone receptors in mitochondria of animal cells," *Molecular and Cellular Endocrinology*, vol. 246, no. 1-2, pp. 21–33, 2006.

[92] V. Brower, "Mind-body research moves towards the mainstream. Mounting evidence for the role of the mind in disease and healing is leading to a greater acceptance of mind-body medicine," *EMBO Reports*, vol. 7, no. 4, pp. 358–361, 2006.

[93] S. J. H. Biddle, T. Gorely, and D. J. Stensel, "Health-enhancing physical activity and sedentary behaviour in children and adolescents," *Journal of Sports Sciences*, vol. 22, no. 8, pp. 679–701, 2004.

[94] J. Du, Y. Wang, R. Hunter et al., "Dynamic regulation of mitochondrial function by glucocorticoids," *Proceedings of the National Academy of Sciences of the United States of America*, vol. 106, no. 9, pp. 3543–3548, 2009.

[95] C. M. Sabiston, J. Brunet, K. C. Kowalski, P. M. Wilson, D. E. Mack, and P. R. E. Crocker, "The role of body-related self-conscious emotions in motivating women's physical activity," *Journal of Sport and Exercise Psychology*, vol. 32, no. 4, pp. 417–437, 2010.

[96] B. Wold and L. Hendry, "Social and environmental factors associated with physical activity in young people," in *Young and Active? Young People and Health-enhancing Physical Activity: Evidence and Implications*, S. J. H. Biddle, N. Cavill, and J. F. Sallis, Eds., pp. 119–132, Health Education Author-ity, London, UK, 1998.

[97] C. M. Sablston and P. R.E. Crocker, "Exploring self-perceptions and social influences as correlates of adolescent leisure-time physical activity," *Journal of Sport and Exercise Psychology*, vol. 30, no. 1, pp. 3–22, 2008.

[98] F. Bonnet, K. Irving, J. L. Terra, P. Nony, F. Berthezène, and P. Moulin, "Depressive symptoms are associated with unhealthy lifestyles in hypertensive patients with the metabolic syndrome," *Journal of Hypertension*, vol. 23, no. 3, pp. 611–617, 2005.

[99] B. Roshanaei-Moghaddam, W. J. Katon, and J. Russo, "The longitudinal effects of depression on physical activity," *General Hospital Psychiatry*, vol. 31, no. 4, pp. 306–315, 2009.

[100] J. A. Pasco, L. J. Williams, F. N. Jacka et al., "Habitual physical activity and the risk for depressive and anxiety disorders among older men and women," *International Psychogeriatrics*, vol. 23, pp. 292–298, 2011.

[101] F. N. Jacka, J. A. Pasco, L. J. Williams et al., "Lower levels of physical activity in childhood associated with adult depression," *Journal of Science and Medicine in Sport*, vol. 14, pp. 222–226, 2011.

[102] E. Puterman, J. Lin, E. Blackburn, A. O'Donovan, N. Adler, and E. Epel, "The power of exercise: buffering the effect of chronic stress on telomere length," *PLoS One*, vol. 5, no. 5, Article ID e10837, 2010.

[103] F. C. Mooren, K. Volker, B. K. Pedersen, A. Schulz, and H. Teschemacher, "Inter- and intracellular signaling," in *Molecular and Cellular Exercise Physiology*, F. C. Mooren and K. Volker, Eds., Human Kinetics, Windsor, Canada, 2005.

[104] B. K. Pedersen, "Exercise-induced myokines and their role in chronic diseases," *Brain, Behavior, and Immunity*, vol. 25, no. 5, pp. 811–816, 2011.

[105] D. Scully, "Physical exercise and psychological well being: a critical review," *British Journal of Sports Medicine*, vol. 32, no. 2, pp. 111–120, 1998.

[106] I. R. Lanza, D. K. Short, K. R. Short et al., "Endurance exercise as a countermeasure for aging," *Diabetes*, vol. 57, no. 11, pp. 2933–2942, 2008.

[107] A. Tsatsoulis and S. Fountoulakis, "The protective role of exercise on stress system dysregulation and comorbidities," *Annals of the New York Academy of Sciences*, vol. 1083, pp. 196–213, 2006.

[108] M. Picard, C. M. Sabiston, and J. K. McNamara, "The need for a trans-disciplinary, global health framework," *Journal of Alternative and Complementary Medicine*, vol. 17, pp. 179–184, 2011.

[109] V. L. Bengtson, D. Gans, N. Putney, and M. Silverstein, *Handbook of Theories of Aging*, Springer, New York, NY, USA, 2nd edition, 2009.

[110] F. Kessel, P. L. Rosenfield, and N. B. Anderson, *Interdisciplinary Research: Case Studies from Health and Social Science*, Oxford University Press, New York, NY, USA, 2008.

[111] B. Singer and C. D. Ryff, *New Horizons in Health: An Integrative Approach*, National Academy Press, Washington, DC, USA, 2001.

[112] D. Cicchetti and S. L. Toth, "The past achievements and future promises of developmental psychopathology: the coming of age of a discipline," *Journal of Child Psychology and Psychiatry*, vol. 50, no. 1-2, pp. 16–25, 2009.

[113] L. A. Sroufe, "The concept of development in developmental psychopathology," *Child Development Perspectives*, vol. 3, no. 3, pp. 178–183, 2009.

[114] S. J. Lupien, B. S. McEwen, M. R. Gunnar, and C. Heim, "Effects of stress throughout the lifespan on the brain, behaviour and cognition," *Nature Reviews Neuroscience*, vol. 10, no. 6, pp. 434–445, 2009.

[115] B. S. McEwen, "Seminars in medicine of the Beth Israel Deaconess Medical Center: protective and damaging effects of stress mediators," *New England Journal of Medicine*, vol. 338, no. 3, pp. 171–179, 1998.

[116] R. P. Juster, B. S. McEwen, and S. J. Lupien, "Allostatic load biomarkers of chronic stress and impact on health and cognition," *Neuroscience and Biobehavioral Reviews*, vol. 35, no. 1, pp. 2–16, 2010.

[117] M. J. Meaney and A. C. Ferguson-Smith, "Epigenetic regulation of the neural transcriptome: the meaning of the marks," *Nature Neuroscience*, vol. 13, no. 11, pp. 1313–1318, 2010.

[118] T.-Y. Zhang and M. J. Meaney, "Epigenetics and the environmental regulation of the genome and its function," *Annual Review of Psychology*, vol. 61, pp. 439–466, 2010.

[119] T. G. Dinan, J. Cryan, F. Shanahan, P. W.N. Keeling, and E. M.M. Quigley, "IBS: an epigenetic perspective," *Nature Reviews Gastroenterology and Hepatology*, vol. 7, no. 8, pp. 465–471, 2010.

[120] J. M. Ordovás and C. E. Smith, "Epigenetics and cardiovascular disease," *Nature Reviews Cardiology*, vol. 7, no. 9, pp. 510–519, 2010.

[121] U. Muñoz-Najar and J. M. Sedivy, "Epigenetic control of aging," *Antioxidants and Redox Signaling*, vol. 14, no. 2, pp. 241–259, 2011.

[122] A. Grolleau-Julius, D. Ray, and R. L. Yung, "The role of epigenetics in aging and autoimmunity," *Clinical Reviews in Allergy and Immunology*, vol. 39, no. 1, pp. 42–50, 2010.

[123] A. Portela and M. Esteller, "Epigenetic modifications and human disease," *Nature Biotechnology*, vol. 28, no. 10, pp. 1057–1068, 2010.

[124] R. Barrès, M. E. Osler, J. Yan et al., "Non-CpG methylation of the PGC-1α promoter through DNMT3B controls mitochondrial density," *Cell Metabolism*, vol. 10, no. 3, pp. 189–198, 2009.

[125] R. Barres and J. R. Zierath, "DNA methylation in metabolic disorders," *American Journal of Clinical Nutrition*, vol. 93, no. 4, pp. 897S–900S, 2011.

[126] L. S. Shock, P. V. Thakkar, E. J. Peterson, R. G. Moran, and S. M. Taylor, "DNA methyltransferase 1, cytosine methylation, and cytosine hydroxymethylation in mammalian mitochondria," *Proceedings of the National Academy of Sciences of the United States of America*, vol. 108, no. 9, pp. 3630–3635, 2011.

[127] D. C. Wallace and W. Fan, "Energetics, epigenetics, mitochondrial genetics," *Mitochondrion*, vol. 10, no. 1, pp. 12–31, 2010.

[128] R. K. Naviaux, "Mitochondrial control of epigenetics," *Cancer Biology and Therapy*, vol. 7, no. 8, pp. 1191–1193, 2008.

[129] T. L. Jacobs, E. S. Epel, J. Lin et al., "Intensive meditation training, immune cell telomerase activity, and psychological mediators," *Psychoneuroendocrinology*, vol. 36, no. 5, pp. 664–681, 2011.

[130] Q. A. Soltow, D. P. Jones, and D. E. L. Promislow, "A network perspective on metabolism and aging," *Integrative and Comparative Biology*, vol. 50, no. 5, pp. 844–854, 2010.

[131] S. S. Knox, "From 'omics' to complex disease: a systems biology approach to gene-environment interactions in cancer," *Cancer Cell International*, vol. 10, article no. 11, 2010.

[132] B. Singer, E. Friedman, T. Seeman, G. A. Fava, and C. D. Ryff, "Protective environments and health status: Cross-talk between human and animal studies," *Neurobiology of Aging*, vol. 26, no. 1, supplement, pp. S113–S118, 2005.

[133] C. D. Ryff, G. Dienberg Love, H. L. Urry et al., "Psychological well-being and ill-being: do they have distinct or mirrored biological correlates?" *Psychotherapy and Psychosomatics*, vol. 75, no. 2, pp. 85–95, 2006.

[134] C. D. Ryff and B. H. Singer, "Social environments and the genetics of aging: Advancing knowledge of protective health mechanisms," *Journals of Gerontology. Series B*, vol. 60, pp. 12–23, 2005.

[135] T. E. Seeman, E. Crimmins, M. H. Huang et al., "Cumulative biological risk and socio-economic differences in mortality: MacArthur Studies of Successful Aging," *Social Science and Medicine*, vol. 58, no. 10, pp. 1985–1997, 2004.

[136] D. C. Stanziano, M. Whitehurst, P. Graham, and B. A. Roos, "A review of selected longitudinal studies on aging: past findings and future directions," *Journal of the American Geriatrics Society*, vol. 58, no. 2, pp. S292–S297, 2010.

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Research Article

Exploring Positive and Negative Affect as Key Indicators of Life Satisfaction among Centenarians: Does Cognitive Performance Matter?

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The aim of this investigation was to determine how cognitive performance was associated with positive and negative affect and life satisfaction over time. This study involved a secondary longitudinal analysis of cross-section data collected at Phase I (1988–1992) and during an 18-month longitudinal followup at Phase II (1992–1998) of the Georgia Centenarian Study. Participants included $N = 137$ centenarians at Time 1 and $N = 68$ survivors at Time 2. Significant stability in cognitive impairment existed at Time 1 and Time 2 for positive ($\beta = .55, P < .01$) and negative affect ($\beta = .54, P < .01$) models. Negative affect at Time 1 was associated with lower life satisfaction at Time 1 ($\beta = -.42, P < .01$). In addition, cognitive impairment at Time 2 was associated with decreased positive emotionality at Time 2 ($\beta = -.39, P > .01$). Furthermore, greater positive affect at Time 2 was associated with greater satisfaction with life at Time 2 ($\beta = .35, P < .01$). It appears that positive emotionality contemporaneously influences the association between cognitive impairment and life satisfaction among centenarians. Implications relative to improving life satisfaction among centenarians are discussed.

1. Introduction

Late adulthood represents a developmental period of contentment in life. Persons surviving to advanced old age are reported to be happier and more satisfied with life than any other age group [1]. This may be due to the fact that old-old adults are effective at diminishing negative affective conditions but optimizing emotionally meaningful life situations [2]. Long-lived persons who have achieved a greater sense of happiness and satisfaction in living have done so precisely for the reason that they have developed the necessary emotional resources to counteract most negative age-associated stressors that may occur near the end of life [3]. For example, old-old adults who have maintained a sense of security in life possessed emotionally gratifying social relations and preserved a favorable view of health positively appraise life [2, 3]. Yet cognitive functioning remains an essential determinant of subjective well-being among persons living 100 years and longer [4–7]. Investigators have acknowledged that normative age-graded cognitive decline suppresses key markers of biopsychosocial well-being including individual autonomy, social involvement, and functional capacity among very old adults [8–11]. This suggests that poor cognitive performance may alter how exceptionally long-lived persons appraise life. However, the extent to which cognitive functioning and emotional affect work in tandem and are associated with life satisfaction among centenarians is not clearly understood. A better understanding of the association between cognition and life satisfaction has implications relative to improving perceived quality of life in extreme longevity.

1.1. Theoretical Considerations. According to Butler [12], reaching advanced old age can create a heightened awareness of impending mortality. Butler [12] theorized that this

influences a "naturally occurring and universal process characterized by the progressive return to consciousness of past experience... to maintain one's sense of personal invulnerability" (p. 66). In other words, life review is an individual act and solitary process. Butler [12] hypothesized that old-old adults have a greater amount of unoccupied time to disengage from normative social involvement in order to gauge their well-being. In effect, contemplation was believed to bring about a sense of failure or fulfillment in life.

However, a disengagement perspective assumes a reduction in ego energy which contributes to subjective well-being in old age. This assumption fails to address the developmental context in which older adults pursue happiness. Erikson [13, 14] recognized late adulthood as the eighth stage of development. During this developmental period, older adults seek some fundamental degree of acceptance for the life, regardless of how fulfilling or challenging it may have been [12]. Erikson [13, 14] argued that sociodevelopmental experiences in later life (e.g., retirement, death of a spouse, family members or close friends, failing health) contribute to a sense of mortality which can precipitate the manifestation of acceptance. In turn, a key developmental task in late adulthood involves active resolution of the life career [14]. Erikson [14] posited that congruence between past failings and accomplishments relative to present life circumstances or conditions of well-being elicits a sense of egointegrity or life satisfaction in later life [14]. Thus, happiness is derived through the positive resolution of the past.

Progression of modern theoretical thinking on subjective well-being over the last three decades has focused on resolving how hedonic qualities contribute to happiness and satisfaction with life [15]. Hedonic well-being is conceptually defined as characteristics that make life satisfying and pleasurable versus dissatisfying and unpleasant [15, 16]. Life satisfaction, the presence of positive affect, and the absence of negative affect are traditionally identified as the core components of hedonism [15, 17] In particular, Diener et al. [18] theorized that positive affect and negative affect influence differential outcomes in life satisfaction relative to individual adaptation to everyday stressors. Based on this theoretical premise, a key assumption is that affective emotions represent adaptive resources that neutralize emotionality and establish a "set-point" of life satisfaction [19].

Recent theoretical and empirical interpretation on subjective well-being has led some investigators to conclude that life satisfaction is derived from cognitive-affective processes that work in tandem to make life pleasurable. According to Socioemotional Selectivity Theory [20, SST], individuals are guided by social (e.g., the feeling of being needed and wanted by others) and emotional (e.g., the need to expand ones horizons or seek meaningful life experiences) goals. As persons reach very late life, they are believed to perceive the remaining years of life as limited.

This motivates them to engage more frequently in reprioritizing present-oriented life goals. In turn, greater activation and use of cognitive resources occur over a shorter period of time. It is assumed that many old-old adults are disadvantaged by a reduced cognitive reserve necessary for managing everyday challenges that require greater personal attention or decision-making skills (e.g., management cognitive impairment). SST assumes that emotional behavior regulation assumes primacy in the lives of very old adults. Therefore, SST posits that long-lived persons seek to avoid negative emotions and maximize positive affect to alleviate aversive physical or mental health-related symptoms that may compromise satisfaction with life [21, 22]. Such behaviors are believed to free remaining cognitive resources that could potentially be used for more emotionally meaningful and satisfying life activities (e.g., interactions with loved ones). Thus, persons derive satisfaction from life not simply because they may encounter age-associated impairment and seek to age disability or disease-free [3, 23]. Rather, they effectively manage limited psychosocial resources for the purpose of regulating negative emotions in order to enhance feelings of happiness and sense of meaning near the end of life [3, 7].

1.2. Happiness and Life Satisfaction. Emotionally meaningful life experiences have a lasting influence on subjective well-being [19]. Past suffering and lifetime achievement contribute to current life satisfaction among old-old survivors [24, 25]. Although many old-old adults view their past as the least satisfying period of life, "right now" is perceived with greater feelings of emotional contentment [25]. This may reflect a cosmic dimension of gerotranscendence in which exceptionally old adults live and rejoice in the moment without dwelling upon past failures [26]. Thus, it is reasonable to assume that old-old adults may be more inclined to savor and recount meaningful life experiences to optimize feelings of happiness.

Life satisfaction has commonly been recognized as an affective-cognitive construct [3, 4, 11, 17, 18]. The developmental past has been reported as a critical "anchor period" and key mediating indicator of positive emotionality [24, 27]. Cognitive appraisal of the past is an adaptive strategy by which old-old adults diminish feelings of unhappiness [27]. Investigators have acknowledged that old-old adults adapt to challenges and threats to personal well-being by maintaining optimistic and meaningful perceptions of ongoing life experiences [24, 27]. In effect, such perspectives have been reported to be emotionally beneficial relative to emotional happiness in life [27]. Therefore, it can be argued that feelings of discontentment compromise subjective well-being, whereas feelings of emotional contentment may bolster satisfaction with life.

1.3. Cognitive Functioning. It is important to note that older adults have a reported preference for recalling positive autobiographical information [28, 29]. Demtsen and Rubin [29] reported that aging adults are twice as likely to report feeling happy than they are to admit to feeling unhappy about life. The tendency to remember the good over the bad is associated with cognitive performance [30, 31]. Better cognitive functioning in advanced old age is associated with greater emotional feelings of meaning and happiness which in turn improves ratings of life satisfaction [3, 11, 29]. In other words, emotional affect can be considered an adaptive

resource that regulates the association between cognition and satisfaction in life [28]. Perhaps, cognition is a key determinant of life satisfaction in exceptionally old age relative to the presence of affective emotions.

Centenarians typically demonstrate great dispersion and variation relative to cognitive performance [3]. Investigators have previously estimated that approximately 20–25% of centenarians can generally be considered as cognitively intact [32, 33]. Another 42–100% are reported to have a considerable degree of dementia [33]. With advancing old age, human beings are more vulnerable to terminal decline or "sudden drop in performance" relative to cognition [34, page 306]. The terminal decline hypothesis specifies that intraindividual change in cognitive performance moves from a preterminal phase of gradual or normative age-associated to a more pronounced and accelerated age-graded decline [34]. Terminal decline in cognitive functioning has been cited as most prevalent among persons who have lived beyond the limits of normal life expectancy [35]. Survival into exceptional old age restricts time left to live. Proximity to death is associated with notable losses in intellectual and cognitive functioning as well as notable changes in subjective well-being believed to remain stable into very old age [36–40]. Variation in associated facets of subjective well-being persists not only over long periods of time but can occur within much shorter time frames [41]. In effect, some investigators have called for greater examination of how associated factors of terminal decline (e.g., cognitive impairment) contribute to perceived subjective well-being as well as emotional-based outcomes of life satisfaction [36–38, 40].

The purpose of this investigation was to determine how cognitive performance is associated with positive and negative affect and satisfaction in life over time. In particular, we sought to answer a key question: how is cognitive performance associated with affect and life satisfaction in exceptional old age? We established two key hypotheses. First, we hypothesized that greater cognitive impairment would diminish feelings of positive affect but increase negative affect over time. Second, we hypothesized that greater cognitive impairment would erode how satisfied centenarians feel about life over time.

2. Method

This investigation involved a secondary analysis of longitudinal data originating from the Phase I and Phase II Georgia Centenarian Study [32, GCS]. Phase I of the GCS was conducted from 1988 to 1992 and involved a baseline cross-sectional investigation at Time 1. Phase II of the GCS was completed from 1992 to1998 and involved an 18 month longitudinal followup of original and surviving Phase 1 participants. Both phases of the study were reviewed and approved by the university Institutional Review Board (IRB).

2.1. Participants Sampling and Procedures. Participants were required to be cognitively intact, community-dwelling, and residing within private residences in the state of Georgia. All participants were cognitively screened and interviewed by a trained interviewer of GCS. Phase I participants consisted of a convenience sample of $N = 137$ during initial assessment at Time 1. Phase 2 participants included a total of $N = 68$ longitudinal survivors who were reassessed and interviewed again 20 months later at Time 2.

All participants were required to be cognitively intact in order to participate. Screening for cognitive status was completed to address two primary considerations: (a) protection of cognitively frail centenarians with advanced cognitive impairment who may not be able to complete a semistructured interview; (b) protection of centenarians from unnecessary stress, fatigue, agitation, or confusion arising during the completion of research involving a *semistructured* interview that would otherwise be beyond their normal daily routine. The Mini-Mental State Examination [42] was the primary instrument used to initially screen all Phase I participants for cognitive status. We considered cognitively intact participants appropriate for study participation to be those with a score of 23 or higher on the Mini-Mental State Examination, as mentioned by Folstein et al. [42]. The Global Deterioration scale [43] served as a secondary cognitive screening instrument. The GDS was used in the event centenarian participants presented with sensory impairments (e.g., vision, hearing) that limited accurate assessment or the ability to fully complete the MMSE. The GDS is a seven-item interviewer rating of subjective memory complaint, orientation, and functional ability. A cut-off score of 2 or less, as mentioned by Reisberg et al. [43], was considered indicative of severe cognitive impairment. All participants were reassessed using the MMSE and the GDS during Phase II 18-month followup.

Demographic characteristics reported by sample participants at Time 1 and longitudinal survivors at Time 2 have been summarized in Table 1. These data were considered for purposes of clarifying whether comparative demographic differences or similarities existed between Time 1 respondents and Time 2 survivors. Special attention was given toward reported marital status, education, and income. Sample variation in these variables has been reported to have underlying and potentially confounding influences in the interpretation of how reported emotionality and life satisfaction may be reported [41]. The majority of participants reported being widowed, having achieved less than a high school education, and earning a low yearly income. Of the follow-up sample, approximately 85.5% were widowed, 64.6% had attained some high school or less, and 72.1% earned an annual income of less than $7,000. To assess differences between respondents in the first and second wave of the study, we computed cross-tabulations. The results suggested that centenarian participants at both waves did not differ relative to marital status χ^2 $(N = 134) = 3.97$, $P = .26$, education χ^2 $(N = 134) = 5.10$, $P = .64$, or annual income χ^2 $(N = 105) = 5.10$, $P = .88$. Therefore, centenarians within this sample were homogeneous relative to marital status, education, and income at Time 1 and Time 2.

Table 1: Frequencies of demographic characteristics among centenarian respondents at Time 1 ($N = 136$) and Time 2 survivors ($N = 68$).

Variable	T1 respondents		T2 survivors	
	N	%	*N*	%
Marital status				
Single	9	6.7%	5	8.1%
Married	6	4.5%	3	4.8%
Widowed	116	86.6%	53	85.5%
Divorced	3	2.2%	1	1.6%
Total	134	100.0%	62	100.0%
Education				
0–4 years	19	14.1%	11	16.1%
4–8 years	39	28.8%	23	33.8%
Some high school	16	11.9%	10	14.7%
High school	13	9.6%	4	5.9%
Business/trade	8	5.9%	4	5.9%
Some college	19	14.1%	7	10.3%
College	12	8.9%	5	7.4%
Graduate school	9	6.7%	4	5.9%
Total	135	100.0%	68	100.0%
Income				
$1,000–1,999	2	1.9%	2	3.7%
$2,000–2,999	4	3.8%	3	5.6%
$3,000–3,999	11	10.5%	9	16.6%
$4,000–4999	20	19.0%	10	18.5%
$5,000–5,999	24	22.9%	11	20.3%
$6,000–6,999	10	9.5%	4	7.4%
$7,000–9,999	15	14.3%	7	13.0%
$10,000–14,999	6	5.7%	3	5.6%
$15,000–19,999	2	1.9%	—	—
$20,000–29,999	6	5.7%	3	5.6%
$40,000 and over	5	4.8%	2	3.7%
Total	105	100.0%	54	100.0%

2.2. Analytical Design. The main objective of this study was to examine how cognitive impairment was associated with positive and negative affect and life satisfaction across two time points. To achieve this goal, we conducted a secondary longitudinal analysis of Phase 1 (Time 1) and Phase 2 (Time 2) GCS data using SPSS 17.0. Path analytic techniques were used to assess stability coefficients, examine path coefficients between variables, and assess cross-lag effects among variables measured across the two time points [44]. In addition, path analytic models were used to identify any key mediating associations [44]. For this study, we assessed variables reflecting cognitive performance, positive and negative affect, and life satisfaction.

2.3. Measures

2.3.1. Cognitive Performance. Cognitive performance was assessed using the Short-Portable Mini-Mental Status Questionnaire [45, SPMSQ]. The SPMSQ was designed as a brief ten-item test of organic brain functioning. The questionnaire is used to determine orientation to time (e.g., what is the date today?) and place (e.g., what is the name of this place?), recall of information (e.g., when were you born?), and numeric ability (e.g., subtract 3 from 20 and keep subtracting backward). A score of 0–2 typically represents normal cognitive functioning, whereas a score of 3-4 errors indicates mild cognitive, a score of 5–7 errors indicates moderate cognitive impairment, and a score of 8 or more errors is suggestive of severe cognitive deficit. The SPMSQ is also adjusted by education. Pfeiffer [45] noted that participants who have a grade school education are allowed one or more errors. However, one less error is allowed if the person received an education beyond high school. For purposes of this study, we used a summary score indicative to total number of errors made on the SPSMQ. A high cumulative number of errors made represented greater cognitive impairment, whereas a low cumulative number of errors represented less cognitive impairment. Cronbach's alpha of this scale was .58 at Time 1 and .73 at Time 2.

2.3.2. Positive/Negative Affect. The Bradburn Affect Balance Scale [46, BABS] served as the primary measure of positive

and negative affect. This scale includes two sets of questions, each consisting of five items. One set of questions is used to measure positive affect (e.g., feeling pleased about having accomplished something; feeling on top of the world), whereas the other set of questions is used to evaluate negative affect (e.g., feeling depressed or very unhappy; feeling vaguely uneasy). Participants were asked to indicate how they had recently felt on a four-point Likert scale (1 = not at all; 4 = often). Positive and negative affective items were coded and summed into a summary of positive and negative affect. A high score on positive affective items indicated high positive emotionality, whereas a low score represented low positive emotionality. Similarly, negative affective items were coded so that a high score reflected high negative emotionality and a low score represented low negative emotionality. Test-retest reliability indicative of Cronbach alpha for positive affect items at Time 1 and Time 2 was $\alpha = .51$ and $\alpha = .66$, respectively, whereas test-retest reliability of negative affect items at Time 1 and Time 2 was $\alpha = .68$ and $\alpha = .60$.

2.3.3. Life Satisfaction. The Life Satisfaction Index-A [47, LSI-A] was used as the primary evaluation of life satisfaction. The LSI-A is a 20-item scale used to assess five characteristics of life satisfaction including zest for living (e.g., the things I do are as interesting to me as they ever were); resolution and fortitude (e.g., I have gotten more breaks in life than most people I know); self-concept (e.g., I feel my age but it does not bother me); congruence (e.g., as I look back on my life, I am fairly well satisfied); mood tone (e.g., I am as happy as when I was younger). Participants were asked to indicate whether they 1 = disagree, 0 = are uncertain, or 3 = agree with each statement. Items were summed to form a cumulative score of life satisfaction. A high score indicated high life satisfaction. A low score reflected low feelings of happiness. Alpha reliability across subscale items was .61 at Time 1 and .61 at Time 2.

3. Results

Means and standard deviations on all study variables were examined to determine whether Time 1 respondent and Time 2 survivors significantly differed relative to responses on all key study variables. These data have been summarized in Table 2. A significant difference in cognitive performance emerged among survivors, $t(41) = -4.25$, $P < .01$. In other words, average number of errors made on the SPMSQ [32] by respondents at T1 ($M = 2.07$, SD = 1.70) was significantly different from the number of errors surviving respondents made at T2 ($M = 3.30$, SD = 2.35). In particular, surviving respondents made a greater average number of errors at T2. This suggests that longitudinal survivors had diminished cognitive abilities during the 18-month follow-up assessment. In addition, there were no significant mean differences in negative affect, $t(41) = .57$, $P = .57$. This suggests that the average negative affect score of respondents at T1 ($M = 8.37$, SD = 3.24) did not significantly differ from the mean negative affect score of survivors at T2 ($M = 8.87$, SD = 3.27). However, there was a significant difference between respondents at Time 1 and those retested at T2 on average scores of positive affect, $t(41) = 2.87$, $P < .05$. This suggests that average positive affect scores among survivors at T2 were greater than at T1. In particular, average positive affect scores between respondents at T1 ($M = 12.42$, SD = 3.10) and T2 survivors ($M = 11.26$, SD = 3.60) were different. Finally, there was no significant difference evident in the average mean scores of respondent life satisfaction at T1 versus T2, $t(41) = -.29$, $P = .77$.

A model of negative affect was then constructed and path relationships were assessed (Figure 1). We first examined stability and cross-lag coefficients. Significant stability existed between cognitive impairment at Time 1 and Time 2 ($\beta = .54$, $P < .01$). Thus, it appears that cognitive impairment remains relative stable over time. Significant stability did not emerge across the negative affect or life satisfaction at Time 1 and Time 2. Slight evidence of a significant cross-lag effect did emerge between negative affect at Time 1 and Time 2 ($\beta = -.29$, $P = .05$). This suggests that greater negative emotions at T1 decrease life satisfaction at T2.

Similarly, we assessed path relationships within a panel-design model of positive affect (Figure 2). Significant stability existed between cognitive impairment at Time 1 and Time 2 ($\beta = .55$, $P < .01$). Thus, it appears that cognitive impairment remains relative stable over time. Significant stability did not emerge across positive affect or life satisfaction at Time 1 and Time 2. Furthermore, there was no evidence of significant cross-lag effects within the positive affect model.

Next, we evaluated path associations between study variables at Time 1 in both models. A significant positive association emerged relative to negative affect and life satisfaction during Time 1 ($\beta = -.42$, $P < .01$, Figure 1). In particular, centenarians possessing greater negative emotions during the initial assessment also felt less satisfied with life. Seventeen percent of variance in life satisfaction at Time 1 was explained by negative affect at Time 1. Relative to positive affect at Time 1, no significant association emerged with life satisfaction at Time 1. Evidence of a nonsignificant association at Time 1 may represent an anomaly of the study design that was used. Exclusion of cognitively impaired participants may have restricted the range of cognitive ability scores among study participants at Time 1. In turn, this may have led to lower associations between cognitive impairment and positive or negative affect. Finally, only 3% of the variance in life satisfaction at Time 1 was explained by positive affect at Time 1.

We then examined path coefficients at Time 2 for both models. No significant path associations were evident between cognitive impairment at Time 2 and negative affect at Time 2, or negative affect at Time 2 and life satisfaction at Time 2. However, greater cognitive impairment at Time 2 was associated with reduced positive emotionality at Time 2 ($\beta = -.39$, $P < .01$). In other words, poor cognitive functioning diminishes the extent to which centenarians experience positive emotions. Nonetheless, greater positive emotions at Time 2 was associated with greater life satisfaction at Time 2 ($\beta = .35$, $P < .01$). It is important to note that approximately 32% of the variance in cognitive impairment at Time 2 was

Table 2: Descriptive statistics of T1 respondent sample ($N = 136$) and Time 2 survivor sample ($N = 68$).

Variable	T1 respondents		T2 survivors	
	M	SD	M	SD
Cognitive impairment	2.07	1.70	3.30	2.35**
Positive affect	12.42	3.10	11.26	3.60*
Negative affect	8.37	3.24	8.87	3.27
Life satisfaction	12.37	3.09	12.41	3.11

Note. *$P < .05$. **$P < .01$

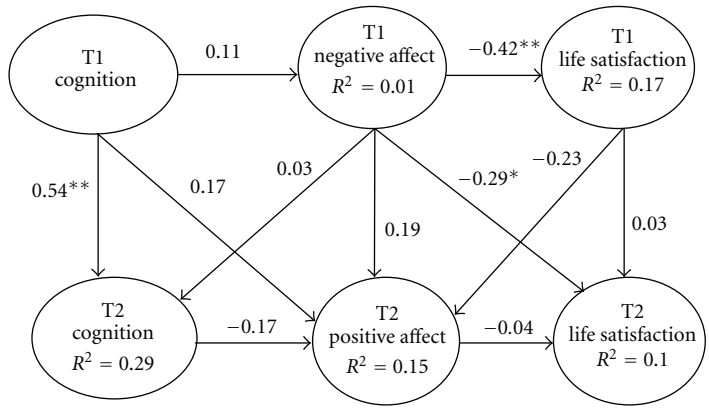

Figure 1: Negative affect model of life satisfaction among centenarians at Time 1 ($N = 136$) to Time 2 ($N = 68$). Note. *$P < .05$, **$P < .01$.

explained by cognition and positive affect at Time 1. Only 18% of the variance in positive affect at Time 2 was explained by cognitive impairment at Time 1 and 2 as well as positive affect at Time 1. Furthermore, 19% of the variance in life satisfaction at Time 2 was explained by positive affect at Time 1 and Time 2 as well as life satisfaction at Time 1.

Finally, we consider potential mediating influences. According to Kenny et al. [48], mediation exits when two conditions are met: (1) the path between a predictor and a mediating variable represents a significant increase, while the path from the mediating variable maintains a significant decrease; (2) the path between a predictor variable and a proximal mediating variable represents a significant decrease, while the path from the mediating variable results in a significant increase.

Based on results from the panel model, two mediating relationships emerged within the positive affect model. First, cognitive impairment at Time 2 represented a key mediating influence between cognitive impairment at Time 1 and positive affect at Time 2. In effect, cognitive impairment at Time 1 continues to erode at Time 2, which in turn diminishes the extent to which centenarians experience positive emotions. Thus, cognition at Time 1 continued to have a negative indirect effect on positive affect in the presence of continued decline in cognitive performance at Time 2 ($.55 \times -.39 = -.21$). Second, positive affect at Time 2 represented a key mediating variable in the association between cognitive impairment and life satisfaction at Time 2. In other words, cognitive impairment at Time 2 continues to compromise current life satisfaction only to the extent centenarians experience decreased positive affect ($-.39 \times .35 = -.14$). Thus, greater cognitive impairment appears to reduce feelings of emotional happiness which in turn further diminishes life satisfaction among centenarians.

4. Discussion

This study provided key evidence to answer the original study question as well as support the original hypotheses. In particular, it appears that cognitive impairment does compromise the extent to which centenarians feel satisfied with life. Two themes emerged based on this finding. First, positive emotions among long-lived persons are associated with continuous decline in cognitive abilities. Second, cognitive impairment has a negative contemporaneous influence on life satisfaction. However, the extent of this association seems to be dependent on whether centenarians are emotionally happy.

In previous work, we have reported that centenarians who feel emotional contentment generally feel satisfied with life [5, 49]. Yet results from this current investigation suggest that this association may depend on cognitive impairment. In particular, it appears that cognitive impairment over time is detrimental to positive emotionality. As lower cognitive changes persist, current positive affective emotions among centenarians decrease.

However, the extent to which cognitive impairment is associated with life satisfaction among centenarians may be due to the degree they feel emotionally happy. In other

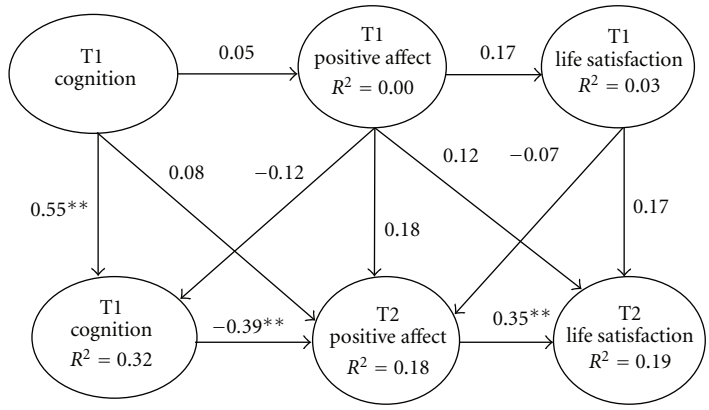

Figure 2: Positive affect model of life satisfaction among centenarians at Time 1 ($N = 136$) to Time 2 ($N = 68$). Note. $^{**}P < .01$.

words, the greater positive emotions appear to diminish the deleterious influence of cognitive impairment on life satisfaction among persons living 100 years and beyond. Such evidence may reflect the developmental patterns in life satisfaction in exceptional old age. Erikson [50] referred to extreme late life as the "ninth stage." In particular, Erikson [50] theorized that living beyond the normative limits of expected human longevity and experiencing ongoing physical or mental deterioration promotes a "shift in meta perspective, from a materialistic and rational vision to a more cosmic and transcendent one, normally followed by an increase in life satisfaction" [50, p. 123]. Investigators have acknowledged the association between positive emotionality and life satisfaction as an adaptive behavior unique to long-lived persons [51]. As centenarians continue to live, they encounter advanced and terminal declines in physical and mental functioning. Such challenges may tax their ability to use psychosocial resources for purposes of adaptation and improved disposition toward life. From the framework of socioemotional selectivity theory [20, 21], it is plausible to argue that centenarians are experts relative to knowing how to selectively dissipate negative emotions and use positive emotional energies to optimize sense of fulfillment in life despite investigators have noted that the interplay between cognition and emotionality is vital source of coping and resilience in old-old age [3]. Thus, the achievement of emotional happiness and life satisfaction among centenarians can be argued as a normative developmental outcome of surviving diminished cognitive abilities.

Gerotranscendence provides an additional explanation of findings. In devising the theory of gerotranscendence, Tornstam [26] posited that the past provides a sense of coherence and gives meaning to the present. This may represent a developmentally normative task of exceptional longevity. Old-old age brings an anticipation of impending loss in cognitive, physical, or functional capacity [35]. Nonetheless, old-old adults remain resilient by adapting new strategies in response to age-associated decline [27]. Contemplation of the accomplishments and traumas of life allows persons in very old age to return with an emancipated comprehension or new appreciation toward self and others [26]. In effect, the joy of appraising and resolving one's life career may serve to lift exceptionally old persons out of despair and regret and into a state of emotional contentment [26, 52, 53]. Lucas [19] has hypothesized that adaptation to alternating life events across the life evokes stability or a "set-point" in levels of happiness. Old-old persons may experience life-altering events which compromise emotional happiness, yet a positive sense of adaptation to change allows them to eventually gain a heightened sense of satisfaction with life. This might help explain the continued impact of cognitive impairment on positive emotionality among centenarians over time. Furthermore, it may provide a plausible explanation for a contemporaneous and indirect association between cognitive impairment and life satisfaction in the presence of positive emotions. Further investigation of the impairment versus emotional well-being dynamic in old-old age is warranted.

Subjective well-being represents an affective-cognitive association [17, 41, 53]. Results from this study appear to support this hypothesis relative to positive affect. Pleasant emotions evolve to the degree that persons possess the cognitive resources to create current satisfying memories in the present as well as to recall meaningful experiences of the past [24, 41]. Some investigators have reported that life satisfaction entails autobiographical recall of essential and meaningful life events [54, 55]. In some cases, high degree of reported life satisfaction in old age represents a deactivated negative affective state [56, 57]. This has been acknowledged to further improve cognitive functioning as well as retrieval of positive emotional experiences across the life-span [57, 58]. The occurrence of positive emotions improves adaptive capacity and allows persons in advanced old age to regain locus of control over unhappy situations or compromising life experiences [59, 60]. Suh et al. [61] proposed that most individuals rely directly on cognitive functioning and affective states to frame judgment of life satisfaction. It can be argued that this may further provide support for the contemporaneous association between positive affect and life satisfaction.

However, poor cognitive performance does reduce positive emotions. Rabbitt et al. [11] reported that decline

in cognitive capacity contributes to heightened feelings of emotional negativity and unhappiness. The trajectory and rate of cognitive decline and memory deficits becomes more pronounced as persons live beyond the expected limits of human life expectancy [6, 10, 30]. This is exacerbated by accumulative everyday hassles (e.g., degree of autonomy in activities of daily living) and personal irritations (e.g., health problems, social isolation) which often require greater use and performance of executive abilities in old-old age [10, 31].

It is important to note that many centenarians possess a limited or finite cognitive reserve to properly counteract age-associated impairment [7, 32]. Impairment in old-old age typically represents noticeable decline in pathological process (e.g., organic brain functioning) which contribute to a simultaneous and terminal drop in overall life satisfaction [36–38, 40]. Yet as persons reach very late life, age-associated impairment becomes secondary, and they perceive their life script more favorably [29, 61]. Investigators have noted that old and very old adults tend to report emotional feelings of contentment twice as often as they endorse feelings of emotional dissatisfaction [29]. Therefore, it is plausible to argue that pleasant emotions remain relatively intact despite increasing cognitive impairment. Perhaps, it is the persistence of positive affective conditions in the face of age-associated impairment that matters most for life satisfaction among long-lived persons.

Nonetheless, our findings do provide support of the detrimental influence poor cognitive functioning has on life satisfaction [11]. In particular, continued cognitive impairment erodes the extent to which centenarians derive favorable impressions of life. Cognitive impairment also appears to have a more contemporaneous association with emotional happiness. In other words, the deleterious influence of cognitive impairment on positive emotions appears to be most salient among those who continue to survive in exceptional old age. Centenarians who have experienced recent cognitive problems feel dissatisfied with life to the extent that they feel happy. Results from this study indicate that impaired cognitive functioning in extreme late life negatively influences a key indicator of life satisfaction, namely, positive affect. This may call into question the potential benefits of life review or reminiscence therapies often used to elicit feeling of emotional happiness and life satisfaction among centenarians [62, 63]. However, this requires further investigation beyond the scope and findings of this study.

Findings from this study provide insight into the subjective well-being of centenarians. Nonetheless, several limitations should be noted. For instance, convenience sampling was used in selecting a participant sample of community-dwelling centenarians residing in the Southern United States. The final sample used for this investigation also represented cognitively intact centenarians. As previously mentioned, researchers have reported an estimated 42–100% of centenarian study samples with moderate to severe degree of dementia [33]. The incorporation of a randomized and population-based sample may have resulted in improved generalizablilty of results. However, caution should still be used in interpreting results across other centenarian populations which may be cognitive impaired, originate from care facilities, or reside in other regions of the United States or world.

Another limitation involved interval of assessment. Although we had two time points of assessments for longitudinal analysis, the selection of different intervals between measurements would have enhanced the study. In particular, this may have led to significant time lag effects which would have improved understanding of how long or after what time cognitive impairment, congruence, or happiness exhibit influences. Additional time points would have also allowed for use of more sophisticated methods and use of growth curve modeling. The assessment of life satisfaction across multiple time points has been reported to advance understanding of the developmental change and temporal mechanisms of subjective well-being [41].

Finally, the measure of life satisfaction used in this study could be improved in future research. We used only one quantitative measure of life satisfaction, the LSI-A [47]. This may have resulted in considerable overlap in constructs representing past satisfaction with life and current happiness. Furthermore, test-retest alpha reliabilities of the LSI-A were low to marginal. Items from the LSI-A [47] may not provide an appropriate assessment of life satisfaction among persons living exceptionally long lives. Life may be conceptualized differently among centenarians than other age groups [62, 63]. Furthermore, life satisfaction in extreme old age may be a more complex and dynamic phenomenon which requires integration of advance quantitative assessments, qualitative evaluations, or greater focus on cross-cultural comparison for improved scientific understanding. For example, the incorporation of a structured life review or a cross-cultural comparison may have presented alternative findings pertaining to longitudinal outcomes of emotional happiness and life satisfaction among exceptionally old adults. Perhaps, such knowledge would improve scientific understanding of whether life satisfaction among centenarians is universally constructed or culturally unique. In effect, qualitative insight could have resulted in more sophisticated interpretation of subjective well-being.

Despite limitations, this study established key insights into how cognitive impairment and affect represent key indicators of life satisfaction. The results also raise potential questions pertaining how to effectively enhance feelings of happiness and life satisfaction among cognitively impaired persons in exceptional old age. Researchers should continue to increase their understanding of the underlying mechanisms of cognition associated with life satisfaction in advanced old age. Particularly, investigators should seek to further explore and model conceptual and theoretical longitudinal models of subjective well-being to examine cognitive change and adaptation in the development of happiness and satisfaction with life.

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References

[1] Y. Yang, "Social inequalities in happiness in the United States, 1972 to 2004: an age-period-cohort analysis," *American Sociological Review*, vol. 73, no. 2, pp. 204–226, 2008.

[2] L. L. Carstensen, H. H. Fung, and S. T. Charles, "Socioemotional selectivity theory and the regulation of emotion in the second half of life," *Motivation and Emotion*, vol. 27, no. 2, pp. 103–123, 2003.

[3] L. W. Poon, P. Martin, and J. Margrett, in *Successful Cognitive and Emotional Aging*, D. V. Depp, Ed., pp. 115–133, American Psychiatric Publishing, Arlington, Va, USA, 2010.

[4] A. I. Berg, L. B. Hassing, G. E. McClearn, and B. Johansson, "What matters for life satisfaction in the oldest-old?" *Aging and Mental Health*, vol. 10, no. 3, pp. 257–264, 2006.

[5] A. J. Bishop, P. Martin, and L. Poon, "Happiness and congruence in older adulthood: a structural model of life satisfaction," *Aging and Mental Health*, vol. 10, no. 5, pp. 445–453, 2006.

[6] L. S. Miller, M. B. Mitchell, J. L. Woodard, A. Davey, P. Martin, and L. W. Poon, "Cognitive performance in centenarians and the oldest old: norms from the georgia centenarian study," *Aging, Neuropsychology, and Cognition*, vol. 17, no. 5, pp. 575–590, 2010.

[7] L. W. Poon, P. Martin, A. Bishop et al., "Understanding centenarians' psychosocial dynamics and their contributions to health and quality of life," *Current Gerontology and Geriatrics Research*, vol. 2010, Article ID 680657, 13 pages, 2010.

[8] S. M. Samuelsson, B. B. Alfredson, B. Hagberg et al., "The Swedish Centenarian study: a multidisciplinary study of five consecutive cohorts at the age of 100," *International Journal of Aging and Human Development*, vol. 45, no. 3, pp. 223–253, 1997.

[9] F. Béland, M. V. Zunzunegui, B. Alvarado, A. Otero, and T. Del Ser, "Trajectories of cognitive decline and social relations," *Journals of Gerontology B*, vol. 60, no. 6, pp. P320–P330, 2005.

[10] M. Motta, L. Ferlito, S. U. Magnolfi et al., "Cognitive and functional status in the extreme longevity," *Archives of Gerontology and Geriatrics*, vol. 46, no. 2, pp. 245–252, 2008.

[11] P. Rabbitt, M. Lunn, S. Ibrahim, M. Cobain, and L. McInnes, "Unhappiness, health and cognitive ability in old age," *Psychological Medicine*, vol. 38, no. 2, pp. 229–236, 2008.

[12] R. N. Butler, "The life review: an interpretation of reminiscence in the aged," *Psychiatry*, vol. 26, pp. 65–76, 1963.

[13] E. H. Erikson, "Identity and the life cycle: selected papers," *Psychological*, no. 1, pp. 50–100, 1959.

[14] E. H. Erikson, *The Life Cycle Completed*, W. W. Norton Company, New York, NY, USA, 1982.

[15] E. Diener, E. M. Suh, R. E. Lucas, and H. L. Smith, "Subjective well-being: three decades of progress," *Psychological Bulletin*, vol. 125, no. 2, pp. 276–302, 1999.

[16] D. Kahneman, E. Diener, and N. Schwarz, Eds., *Well-Being: The Foundations of Hedonic Psychology*, Russell Sage Foundation, New York, NY, USA, 1999.

[17] J. Liang, "A structural integration of the affect balance scale and the Life Satisfaction Index A," *Journals of Gerontology*, vol. 40, no. 5, pp. 552–561, 1985.

[18] E. Diener, R. E. Lucas, and C. N. Scollon, "Beyond the hedonic treadmill: revising the adaptation theory of well-being," *American Psychologist*, vol. 61, no. 4, pp. 305–314, 2006.

[19] R. E. Lucas, "Adaptation and the set-point model of subjective well-being: does happiness change after major life events?" *Current Directions in Psychological Science*, vol. 16, no. 2, pp. 75–79, 2007.

[20] L. L. Carstensen, D. M. Isaacowitz, and S. T. Charles, "Taking time seriously: a theory of socioemotional selectivity," *American Psychologist*, vol. 54, no. 3, pp. 165–181, 1999.

[21] L. L. Carstensen, H. H. Fung, and S. T. Charles, "Socioemotional selectivity theory and the regulation of emotion in the second half of life," *Motivation and Emotion*, vol. 27, no. 2, pp. 103–123, 2003.

[22] C. E. Löckenhoff and L. L. Carstensen, "Socioemotionol selectivity theory, aging, and health: the increasingly delicate balance between regulating emotions and making tough choices," *Journal of Personality*, vol. 72, no. 6, pp. 1395–1424, 2004.

[23] M. A. Cohn, B. L. Fredrickson, S. L. Brown, J. A. Mikels, and A. M. Conway, "Happiness unpacked: positive emotions increase life satisfaction by building resilience," *Emotion*, vol. 9, no. 3, pp. 361–368, 2009.

[24] D. Shmotkin, M. Berkovich, and K. Cohen, "Combining happines's and suffering in a retrospective view of anchor periods in life: a differential approach to subjective well-being," *Social Indicators Research*, vol. 77, no. 1, pp. 139–169, 2006.

[25] M. Mehlsen, M. Platz, and P. Fromholt, "Life satisfaction across the life course: evaluations of the most and least satisfying decades of life," *International Journal of Aging and Human Development*, vol. 57, no. 3, pp. 217–236, 2003.

[26] L. Tornstam, *Gerotranscendence: A Developmental Theory of Positive Aging*, Springer, New York, NY, USA, 2005.

[27] A. C. Åberg, B. Sidenvall, M. Hepworth, K. O'Reilly, and H. Lithell, "On loss of activity and independence, adaptation improves life satisfaction in old age—a qualitative study of patients' perceptions," *Quality of Life Research*, vol. 14, no. 4, pp. 1111–1125, 2005.

[28] M. Mather and L. L. Carstensen, "Aging and attentional biases for emotional faces," *Psychological Science*, vol. 14, no. 5, pp. 409–415, 2003.

[29] D. Bemtsen and D. C. Rubin, "Emotionally charged autobiographical memories across the life span: the recall of happy, sad, traumatic, and involuntary memories," *Psychology and Aging*, vol. 17, no. 4, pp. 636–652, 2002.

[30] E. A. Maguire and C. D. Frith, "Aging affects the engagement of the hippocampus during autobiographical memory retrieval," *Brain*, vol. 126, no. 7, pp. 1511–1523, 2003.

[31] D. D. Vondras, M. R. Powless, A. K. Olson, D. Wheeler, and A. L. Snudden, "Differential effects of everyday stress on the episodic memory test performances of young, mid-life, and older adults," *Aging and Mental Health*, vol. 9, no. 1, pp. 60–70, 2005.

[32] L. W. Poon, A. L. Sweaney, G. M. Clayton et al., "The Georgia Centenarian Study," *International Journal of Aging and Human Development*, vol. 34, no. 1, pp. 1–17, 1992.

[33] Y. Gondo and L. W. Poon, "Cognitive function of centenarians and its influence on longevity," *Annual Review of Gerontology and Geriatrics*, vol. 27, pp. 129–149, 2007.

[34] K. F. Riegel and R. M. Riegel, "Development, drop, and death," *Developmental Psychology*, vol. 6, no. 2, pp. 306–319, 1972.

[35] P. B. Baltes, "On the incomplete architecture of human ontogeny: selection, optimization, and compensation as foundation of developmental theory," *American Psychologist*, vol. 52, no. 4, pp. 366–380, 1997.

[36] D. Gerstorf, N. Ram, R. Estabrook, J. Schupp, G. G. Wagner, and U. Lindenberger, "Life satisfaction shows terminal decline in old age: longitudinal evidence from the German socio-economic panel study (SOEP)," *Developmental Psychology*, vol. 44, no. 4, pp. 1148–1159, 2008.

[37] D. Gerstorf, N. Ram, G. Mayraz et al., "Late-life decline in well-being across adulthood in germany, the united kingdom, and the united states: something is seriously wrong at the end of life," *Psychology and Aging*, vol. 25, no. 2, pp. 477–485, 2010.

[38] D. Gerstorf, N. Ram, C. Röcke, U. Lindenberger, and J. Smith, "Decline in life satisfaction in old age: longitudinal evidence for links to distance-to-death," *Psychology and Aging*, vol. 23, no. 1, pp. 154–168, 2008.

[39] D. K. Mroczek and A. Spiro III, "Change in life satisfaction during adulthood: findings from the veterans affairs normative aging study," *Journal of Personality and Social Psychology*, vol. 88, no. 1, pp. 189–202, 2005.

[40] Y. Palgi, A. Shrira, M. Ben-Ezra, T. Spalter, D. Shmotkin, and G. Kavé, "Delineating terminal change in subjective well-being and subjective health," *Journals of Gerontology B*, vol. 65, no. 1, pp. 61–64, 2010.

[41] M. Eid and R. J. Larsen, Eds., *The Science of Subjective Well-Being*, Guilford Press, New York, NY, USA, 2008.

[42] M. F. Folstein, S. E. Folstein, and P. R. McHugh, "Mini mental state. A practical method for grading the cognitive state of patients for the clinician," *Journal of Psychiatric Research*, vol. 12, no. 3, pp. 189–198, 1975.

[43] B. Reisberg, S. H. Ferris, M. J. de Leon, and T. Crook, "The global deterioration scale for assessment of primary degenerative dementia," *American Journalof Psychiatry*, vol. 139, no. 9, pp. 1136–1139, 1982.

[44] T. D. Little, K. J. Preacher, J. P. Selig, and N. A. Card, "New developments in latent variable panel analyses of longitudinal data," *International Journal of Behavioral Development*, vol. 31, no. 4, pp. 357–365, 2007.

[45] E. Pfeiffer, "A short portable mental status questionnaire for the assessment of organic brain deficit in elderly patients," *Journal of the American Geriatrics Society*, vol. 23, no. 10, pp. 433–441, 1975.

[46] N. M. Bradburn, *The Structure of Psychological Well-Being*, Aldine, Chicago, Ill, USA, 1969.

[47] B. L. Neugarten, R. J. Havighurst, and S. S. Tobin, "The measurement of life satisfaction," *Journal of Gerontology*, vol. 16, pp. 134–143, 1961.

[48] D. A. Kenny, D. A. Kashy, and N. Bolger, "Data analysis in social psychology," in *The Handbook of Social Psychology*, D. Gilbert, S. Fiske, and G. Lindzey, Eds., vol. 1, pp. 233–265, McGraw–Hill, Boston, Mass, USA, 4th edition, 1998.

[49] A. J. Bishop, P. Martin, M. MacDonald, and L. Poon, "Predicting happiness among centenarians," *Gerontology*, vol. 56, no. 1, pp. 88–92, 2010.

[50] J. M. Erikson, "The ninth stage," in *The Life Cycle Completed*, E. H. Erikson, Ed., pp. 105–114, W. W. Norton Company, New York, NY, USA, 1982.

[51] D. Jopp and C. Rott, "Adaptation in very old age: exploring the role of resources, beliefs, and attitudes for centenarians' happiness," *Psychology and Aging*, vol. 21, no. 2, pp. 266–280, 2006.

[52] C. M. Torges, A. J. Stewart, and S. Nolen-Hoeksema, "Regret resolution, aging, and adapting to loss," *Psychology and Aging*, vol. 23, no. 1, pp. 169–180, 2008.

[53] M. T. Davern, R. A. Cummins, and M. A. Stokes, "Subjective wellbeing as an affective-cognitive construct," *Journal of Happiness Studies*, vol. 8, no. 4, pp. 429–449, 2007.

[54] J. P. Serrano, J. M. Latorre, M. Gatz, and J. Montanes, "Life review therapy using autobiographical retrieval practice for older adults with depressive symptomatology," *Psychology and Aging*, vol. 19, no. 2, pp. 272–277, 2004.

[55] C. E. S. Tabourne, "The effects of a life review program on disorientation, social interaction and self-esteem of nursing home residents," *International Journal of Aging and Human Development*, vol. 41, no. 3, pp. 251–266, 1995.

[56] P. A. Allen, K. P. Kaut, R. G. Lord, R. J. Hall, J. W. Grabbe, and T. Bowie, "An emotional mediation theory of differential age effects in episodic and semantic memories," *Experimental Aging Research*, vol. 31, no. 4, pp. 355–391, 2005.

[57] C. R. Hirsch and V. M. Mouratoglous, "Life review of an older adult with memory difficulties," *International Journal of Geriatric Pscyhiatry*, vol. 14, pp. 261–265, 1999.

[58] N. A. Murphy and D. M. Isaacowitz, "Preferences for emotional information in older and younger adults: a meta-analysis of memory and attention tasks," *Psychology and Aging*, vol. 23, no. 2, pp. 263–286, 2008.

[59] C. Wrosch and J. Heckhausen, "Perceived control of life regrets: good for young and bad for old adults," *Psychology and Aging*, vol. 17, no. 2, pp. 340–350, 2002.

[60] C. Wrosch, I. Bauer, and M. F. Scheier, "Regret and quality of life across the adult life span: the influence of disengagement and available future goals," *Psychology and Aging*, vol. 20, no. 4, pp. 657–670, 2005.

[61] E. Suh, E. Diener, S. Oishi, and H. C. Triandis, "The shifting basis of life satisfaction judgments across cultures: emotions versus norms," *Journal of Personality and Social Psychology*, vol. 74, no. 2, pp. 482–493, 1998.

[62] S. B. Merriam, P. Martin, G. Adkins, and L. Poon, "Centenarians: their memories and future ambitions," *International Journal of Aging and Human Development*, vol. 41, no. 2, pp. 117–132, 1995.

[63] S. B. Merriam, "Butler's life review: how universal is it?" *International Journal of Aging and Human Development*, vol. 37, no. 3, pp. 163–175, 1993.

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Research Article

Alcohol Consumption and Risk of All-Cause and Cardiovascular Disease Mortality in Men

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This study examined the association between consumption of alcoholic beverages and all-cause and cardiovascular disease (CVD) mortality in a cohort of men (n = 31,367). In the Cox proportional hazards model adjusted for age, year of examination, body mass index (BMI), smoking, family history of CVD, and aerobic fitness, there were no significant differences in risk of all-cause mortality across alcohol intake groups. Risk of CVD mortality was reduced 29% in quartile 1 (HR = 0.71, 95% confidence interval (CI): 0.53, 0.95) and 25% in quartile 2 (HR = 0.75, 95% CI: 0.58, 0.98). The amount of alcohol consumed to achieve this risk reduction was <6 drinks/week; less than the amount currently recommended. The addition of other potential confounders and effect modifiers including blood pressure, insulin sensitivity, lipid levels, and psychological variables did not affect the magnitude of association. Future research is needed to validate the current public health recommendations for alcohol consumption.

1. Introduction

Light-to-moderate alcohol consumption has been shown in several prospective studies to reduce the risk of all-cause mortality and several cardiac outcomes, including cardiovascular disease (CVD) mortality in men [1–10]. Several hypotheses exist for the mechanism of this protective effect including increased exposure to antioxidants, improved lipid profiles, reduced insulin resistance, decreased blood coagulation, and modified inflammation pathways [11, 12].

Despite a large number of studies examining alcohol consumption and mortality, questions remain about the nature of this relationship. Three primary concerns include the influence of confounding factors, appropriate public health recommendations, and differential effects between types of alcohol [13, 14]. Selection bias is a particular concern when comparing nondrinkers to drinkers. Nondrinkers may abstain from alcohol because of an initial health condition and be more likely to have other conditions that might confound the relationship including obesity, physical inactivity, and overall health problems [14]. In addition, moderate alcohol consumption has been associated with positive psychosocial outcomes including sociability and stress reduction, which may further confound the association with mortality [15, 16]. Confounding is a limitation of nonexperimental studies, yet it is unethical and impractical to conduct long-term randomized controlled trials of alcohol consumption. In an attempt to address this challenge, investigators have used statistical methods to control for possible confounders that may increase the risk of mortality independent of alcohol consumption, but that may be differentially distributed in those who drink alcohol versus those who do not, such as smoking or physical inactivity.

Several problems have hampered the ability to construct an appropriate public health message about alcohol consumption. Methodological variation has led to inconsistent estimates of protective amounts of alcohol and, thus, an inability to quantify safe recommendations [17]. In addition, any protective effect must simultaneously be weighed against

potential negative consequences. While several studies support light-to-moderate alcohol consumption as a protective factor against CVD and all-cause mortality, excessive alcohol intake has been associated with increased triglycerides, high blood pressure, risk of some cancers, liver cirrhosis, heart failure, and all-cause mortality [9, 12, 18]. Also, certain cancers (e.g., breast) may be associated with increased risk at low doses, but no dose response thereafter [19–21]. As a result of these conflicting messages, the public may be confused about the relationship between alcohol and health outcomes.

The purpose of this study was to analyze the relationship between alcohol consumption and all-cause and CVD mortality in a cohort of men while considering a variety of potential confounding and modifying risk factors. Additionally, differential effects of types of alcohol on these health outcomes were investigated.

2. Materials and Methods

2.1. Study Participants. Study participants were from the Aerobics Center Longitudinal Study (ACLS) [22], a prospective epidemiological study of health outcomes in USA adults. The sample for the current analyses was limited to men, the majority of whom were white, middle to upper income, received a baseline examination at the Cooper Clinic in Dallas, Texas between 1974 and 2002 with at least one year of followup, were aged ≥20 years at baseline, were free of known CVD, cancer, or abnormal ECG, and had complete information on alcohol intake and maximal exercise treadmill test. Individuals participating in the ACLS were either referred to the clinic by their employers or physicians or through self-referral. The study was annually reviewed and approved by the Cooper Institute Institutional Review Board, and all participants gave written informed consent for the baseline clinical examination and follow-up.

2.2. Primary Exposure Variable. Alcohol intake in drinks per week among participants was self-reported at baseline. Participants were asked, "How many 12-ounce drinks of beer, 3-ounce drinks of wine (5-ounce drinks of wine during more recent data collection cycles), and/or 1.5-ounce drinks of hard liquor do you consume per week?" Self-report alcohol intake has been previously validated against dietary records in men ($r = 0.86$) [23, 24]. Participants were then classified either as nondrinkers, or in quartiles of drinkers based on baseline alcohol drinking reports.

2.3. Outcome Variables. All participants were followed from the date of their baseline examination until their death or December 31, 2003. Mortality surveillance was primarily through the National Death Index. CVD mortality was defined as the *International Classification of Diseases, Ninth Revision* codes 390 to 449.9 before 1999 and *Tenth Revision* codes I00 to I78 during 1999–2003.

2.4. Covariates. The baseline clinical assessment has been previously described in detail [22, 25, 26]. At the baseline visit patients completed a written survey and anthropometric, blood pressure, blood chemistry, and electrocardiogram measures. Body Mass Index [BMI = weight (kg)/height (m)^2] was calculated from height and weight. Resting blood pressure was assessed via ascultatory method [27]. Blood samples were drawn after a 12-hour overnight fast, and analyzed for fasting glucose and cholesterol using automated bioassays according to the Centers for Disease Control and Prevention Lipid Standardization Program. Hypertension, hypercholesterolemia, and diabetes were defined according to clinical thresholds or self-reported as a previous diagnosis from a physician. Hypertension was defined as resting systolic or diastolic blood pressure ≥140/90 mmHg, hypercholesterolemia was defined as total cholesterol ≥240 mg/dL [28], and diabetes was defined as fasting glucose ≥126 mg/dL [29]. The questionnaire included questions on smoking habits, family history of CVD, and the presence of neuropsychiatric conditions including anxiety, depression, suicidal thoughts, or psychological counseling. Cardiorespiratory fitness (CRF) was assessed using symptom-limited maximal treadmill time during the modified Balke Protocol [22, 30]. Test termination occurred by participant volition or physician determined medical reasons. Test duration has been strongly correlated with maximal oxygen uptake ($r = 0.92$) [31]. Cardiorespiratory fitness is a quantifiable, objective measure with stronger associations to future health outcomes than self-reported physical activity [32]. Treadmill times were classified into fitness quintiles with the first quintile classified as unfit and the remaining four quintiles as fit [33].

2.5. Statistical Analysis. Cox proportional hazards models were used to calculate hazard ratios (HRs) and associated 95% confidence intervals. Alcohol consumption was the primary exposure of interest. All-cause and CVD mortality were fit as the outcomes (i.e., death or not, and time to event or censoring) for survival analysis. Nondrinkers were used as the referent group and those classified as drinkers were categorized into quartiles of exposure. Linear and quadratic trends were tested by fitting the continuous variable of alcohol consumption in the basic model for both all-cause and cardiovascular disease mortality. The basic model was adjusted for age (in years), year of examination, BMI, smoking (current smoker or not), family history of CVD (yes or no), and treadmill time (minutes). Because blood pressure [34], insulin sensitivity [35], and lipid levels [36] have been shown to mediate the relationship between alcohol and CVD mortality, they were added individually and then simultaneously into the basic model. Finally, the full model was adjusted for psychological variables [15, 16].

To examine effect modification due to other health risk factors, multivariate stratified analyses were conducted for dichotomies based on age (<60 and ≥60 years), BMI (<25 and ≥25 kg/m^2), health status (healthy and unhealthy defined as having at least one of the following diseases: hypertension, diabetes, or hyperlipidemia), and fitness (least fit 20% and most fit 80%) to test for effect modification. These models were adjusted for year of examination, smoking, family history of CVD, and age, BMI, health status, and fitness where appropriate.

After analyzing the overall effect of alcohol on mortality, separate analyses were run for each type of alcohol (beer, wine, and liquor), because of the potential differential effects from different types of alcohol. As there were few men who consumed a single beverage type, analysis was conducted separately for quartiles of a single type of alcohol while adjusting for the remaining type of alcohol consumption [37, 38]. Alcohol consumption quartiles were created independently for each alcohol type and hazard ratios adjusted for age, year of examination, BMI, smoking, family history of CVD, maximal treadmill time, and remaining types of alcohol were calculated using nondrinkers as the reference group.

3. Results and Discussion

A total of 31,637 men were included in the sample. The mean age of men at baseline was 42.9 (standard deviation (SD): 9.2) years. Baseline characteristics are presented in Table 1.

Mean follow-up time was 16.0 (8.3) years for a total of 504,735 person-years. There were 1,834 total deaths and 588 CVD deaths. The rate of all-cause and CVD mortality was calculated for the five drinking categories: nondrinkers, quartile 1 (1–3 drinks/week), quartile 2 (4–6 drinks/week), quartile 3 (7–13 drinks/week), and quartile 4 (≥14 drinks/week). Alcohol consumption was linearly associated with both all-cause and CVD mortality rate ($P = 0.002$ and $P = 0.02$).

3.1. Primary Analysis. In the basic model adjusted for age, year of examination, BMI, smoking, family history of CVD, and fitness, there were no significant differences in all-cause mortality risks across alcohol intake groups (Table 2). There was a lower risk of CVD mortality in quartile 1 (HR = 0.71, 95% confidence interval (CI): 0.53, 0.95) and quartile 2 (HR = 0.75, 95% CI: 0.58, 0.98) in the basic model which is less than 6 drinks per week (see Table 3). The addition of potential intermediate variables of glucose, lipids, and blood pressure, or the inclusion of all three, did not attenuate these relationships. The additional adjustment for psychological variables did not alter the observed associations and are not included in the results presented.

3.2. Stratified Analysis. The lower risk of all-cause mortality from light drinking (quartile 1) was attenuated when the results were stratified by BMI, health status, and fitness (see Table 4). However, men younger than 60 years in quartile 1 exhibited a significantly lower risk of all-cause mortality (HR = 0.80, 95% CI: 0.66, 0.96); whereas, men 60 or older did not.

For CVD mortality, there were no significant findings in men who were ≥60 years, had a BMI < 25 kg/m^2, were healthy, or unfit (see Table 5). Younger men had lower risk of CVD mortality in quartile 1 (HR = 0.59, 95% CI: 0.43, 0.83) and quartile 2 (HR = 0.70, 95% CI: 0.52, 0.93). Men with a BMI ≥25 kg/m^2 had lower CVD mortality risk in quartile 1 (HR = 0.67, 95% CI: 0.47, 0.94) and quartile 2 (HR = 0.71, 95% CI: 0.52, 0.97). Unhealthy men had a reduced risk in quartile 3 (HR = 0.74, 95% CI: 0.54, 0.998). When stratified by fitness, fit men exhibited a lower risk in quartile 2 (HR = 0.69, 95% CI: 0.49, 0.96).

3.3. Type of Alcohol. The results of the sensitivity analysis are listed in Table 4. There were a total of 26,767 drinkers, of which 23,728 reported drinking beer, 21,997 reported drinking wine, and 20,416 reported drinking liquor. Therefore, for this analysis, the types of alcohol drinking are not exclusive. For all-cause mortality, there were no significant differences in risk for any three of the alcohol types in comparison to nondrinkers (see Table 6). For CVD mortality, there was a lower risk with beer consumption in quartile 3 (HR = 0.72, 95% CI: 0.52, 0.995), wine consumption in quartile 2 (HR = 0.70, 95% CI: 0.53, 0.93) and liquor consumption in quartile 2 (HR = 0.71, 95% CI: 0.50, 0.93) and Q3 (HR = 0.69, 95% CI: 0.55, 0.93) (see Table 7).

4. Discussion

4.1. All-Cause Mortality. In the current study, the relationship between risk of all-cause mortality with alcohol consumption appeared to evince a flattened J-shaped curve pattern. However, the risk reductions were significant only at the lowest levels of alcohol consumption. Other studies reporting on the association of alcohol consumption and all-cause mortality in men have found protective effects up to three drinks per day [5]. In the Physicians Health Study, Gaziano et al [3], found a reduction in all-cause mortality with 5 to 6 drinks/week and reductions in CVD mortality with all levels of alcohol consumption. Gaziano's results are consistent with a meta-analysis conducted by Di Castelnuovo [39] which found a reduced risk for all-cause mortality up to 4 drinks per day.

4.2. Cardiovascular Disease Mortality. It has been hypothesized that the lower CVD mortality risk associated with limited alcohol intake is due to the cardioprotective effects of alcohol such as improved lipid profiles and decreased blood coagulation [11, 12]. CVD contributes to 34.3% of all deaths in the nation and is an important health outcome [40]; therefore, our results warrant consideration. In this sample, 32% of all deaths were attributed to CVD, similar to the nationwide estimates. There was a 29% and 25% reduction in risk for CVD mortality in the first and second quartiles of alcohol consumption, respectively. This risk reduction was found for 1–6 drinks per week. The lower risk of CVD death may be balanced with an increase in other causes of death potentially due to alcohol consumption, again complicating public health recommendations.

The lower CVD mortality risk we observed was not attenuated when potential intermediate factors of blood pressure, lipids, and glucose were added to the model, suggesting the lower risks were not mediated by these factors. While Suh et al. [7] and Djoussé et al. [41] found that the reduced risk of CVD mortality in women was mediated by lipids and insulin sensitivity, other authors have found the relationship to be independent of these risk factors. Arriola et al. [1] found that blood pressure, cholesterol, and diabetes did not alter

Table 1: Baseline demographics by alcohol consumption level, Aerobics Center for Longitudinal Study (ACLS), 1974–2002.

	Alcohol consumption (drinks/week)				
	Nondrinkers (0)	Quartile 1 (1–3)	Quartile 2 (4–6)	Quartile 3 (7–13)	Quartile 4 (≥14)
n	4870	6733	6150	6841	7043
Age (years)	43.63 (9.65)	42.15 (9.15)	42.34 (9.07)	42.95 (9.23)	43.62 (9.13)
Alcohol (drinks/week)	0	2.01 (0.76)	5.00 (0.87)	9.65 (1.93)	24.62 (13.64)
Body mass index (kg/m^2)	26.65 (4.29)	26.63 (4.02)	26.36 (3.65)	26.28 (3.45)	26.42 (3.35)
Maximal treadmill time (min)	17.24 (5.15)	18.33 (4.89)	18.14 (4.90)	18.50 (4.84)	17.78 (4.83)
Current smokers (%)	10.62	14.18	19.50	22.03	29.09
Systolic blood pressure (mmHg)	121 (13)	121 (13)	121 (13)	121 (13)	122 (13)
Diastolic blood pressure (mmHg)	81 (10)	80.91 (9.47)	80.52 (9.35)	81.17 (9.41)	81.90 (9.53)
Hypertension (%)[a]	30.86	28.32	28.10	30.35	34.49
Fasting glucose (mg/dL)	99.14 (18.92)	99.20 (16.52)	98.80 (14.50)	99.74 (14.50)	101.13 (14.19)
Diabetes (%)[b]	4.58	4.92	4.70	4.37	4.91
Total cholesterol (mg/dL)	205.80 (38.56)	203.21 (39.88)	205.42 (38.29)	209.58 (39.16)	217 (40.42)
Triglycerides (mg/dL)	141.09 (104.59)	137.20 (106.77)	132.02 (94.37)	134.63 (101.78)	144.54 (105.24)
Hyperlipidemia (%)[c]	29.49	26.56	26.03	28.46	35.57
Unhealthy (%)[d]	49.63	46.81	45.69	48.68	55.93
Family history of CVD (%)	26.96	23.91	25.45	24.69	28.44

[a]Defined as systolic or diastolic blood pressure ≥140/90 mmHg or history of physician diagnosis.
[b]Defined as fasting glucose ≥126 mg/dl or history of physician diagnosis.
[c]Defined as fasting total cholesterol ≥240 mg/dl or triglyceride ≥200 mg/dL.
[d]Defined as presence of one or more of following diseases: hypertension, diabetes, or hyperlipid.

Table 2: Hazard ratios of all-cause mortality by alcohol consumption level in men, Aerobics Center for Longitudinal Study (ACLS), 1974–2002.

	Alcohol consumption (drinks/week)				
	Nondrinkers (0)	Quartile 1 (1–3)	Quartile 2 (4–6)	Quartile 3 (7–13)	Quartile 4 (≥14)
Person-years	86,872	91,529	98,303	103,625	124,360
No.	4870	6733	6150	6841	7043
No. of Deaths	343	237	350	349	555
Death rate[a]	43.8	37.2	43.3	41.6	51.2
Basic model[b]	1.00 (referent)	0.86 (0.73–1.01)	0.95 (0.82–1.10)	0.97 (0.83–1.12)	1.09 (0.95–1.25)
Basic model plus					
Blood pressure factors[c]	1.00 (referent)	0.86 (0.73–1.02)	0.94 (0.81–1.09)	0.97 (0.83–1.12)	1.06 (0.92–1.21)
Fasting glucose factors[d]	1.00 (referent)	**0.84 (0.71–0.99)**	0.95 (0.82–1.10)	0.96 (0.82–1.11)	1.07 (0.94–1.23)
Lipid factors[e]	1.00 (referent)	0.86 (0.73–1.01)	0.95 (0.82–1.11)	0.96 (0.83–1.12)	1.09 (0.95–1.25)
All intermediate factors[cde]	1.00 (referent)	**0.84 (0.71–0.99)**	0.94 (0.81–1.09)	0.96 (0.82–1.11)	1.05 (0.91–1.20)

[a]Death rate per 10,000 person-years adjusted for age (in years).
[b]Adjusted for age (in years), year of examination, BMI (kg/m^2), smoking (current smoker or not), family history of CVD (yes or no), and maximal treadmill time (min).
[c]Systolic blood pressure and reported hypertension (yes or no).
[d]Fasting glucose and reported diabetes (yes or no).
[e]Total cholesterol and triglyceride.

their results and, through coronary angiography, Femia et al. [42] found the risk for atherosclerosis and cardiac mortality was independent of cholesterol, hypertension, and diabetes. More research is needed to study the potential mechanisms of alcohol intake on reduced CVD mortality risk.

4.3. Public Health Recommendations. Our results do not support the currently recommended intake of 1-2 drinks per day for men from the American Heart Association [43]. The lower alcohol consumption for risk reduction in our study may result from complete adjustment for confounders including fitness and thorough screening resulting in exclusion of preclinical cases. There are large variations in results from prospective studies examining alcohol consumption and mortality making it difficult to compare findings for universal alcohol recommendations. In Di Castelnuovo et al.'s [39] meta-analysis of all-cause mortality, they found differences among genders, countries of the study, and number of confounders. There has been large variation in methods of reporting and categorizing alcohol consumption.

Table 3: Hazard ratios of CVD mortality by alcohol consumption level in men, Aerobics Center for Longitudinal Study (ACLS), 1974–2002.

	Alcohol consumption (drinks/week)				
	Nondrinkers (0)	Quartile 1 (1–3)	Quartile 2 (4–6)	Quartile 3 (7–13)	Quartile 4 (≥14)
Person-years	86,872	91,529	98,303	103,625	124,360
No.	4742	6663	6049	6735	6860
No. of Deaths	128	70	101	106	183
Death rate[a]	17.0	11.6	13.1	12.9	17.7
Basic model[b]	1.00 (referent)	**0.71 (0.53–0.95)**	**0.75 (0.58–0.98)**	0.83 (0.64–1.08)	1.00 (0.80–1.26)
Basic model plus					
Blood pressure factors[c]	1.00 (referent)	**0.70 (0.53–0.94)**	**0.73 (0.56–0.95)**	0.84 (0.64–1.09)	0.94 (0.75–1.18)
Fasting glucose factors[d]	1.00 (referent)	**0.68 (0.51–0.91)**	**0.75 (0.58–0.98)**	0.82 (0.63–1.07)	0.98 (0.78–1.24)
Lipid factors[e]	1.00 (referent)	**0.71 (0.53–0.95)**	**0.73 (0.56–0.96)**	0.80 (0.62–1.04)	0.95 (0.75–1.19)
All intermediate factors[cde]	1.00 (referent)	**0.68 (0.51–0.91)**	**0.71 (0.55–0.93)**	0.81 (0.62–1.05)	0.88 (0.69–1.11)

[a] Death rate per 10,000 person-years adjusted for age (in years).
[b] Adjusted for age (in years), year of examination, BMI (kg/m^2), smoking (current smoker or not), family history of CVD (yes or no), and maximal treadmill time (min).
[c] Systolic blood pressure and reported hypertension (yes or no).
[d] Fasting glucose and reported diabetes (yes or no).
[e] Total cholesterol and triglyceride.

Table 4: Hazard ratios of all-cause mortality stratified by risk factors[a], Aerobics Center for Longitudinal Study (ACLS), 1974–2002.

	Alcohol consumption (drinks/week)				
	Nondrinkers (0)	Quartile 1 (1–3)	Quartile 2 (4–6)	Quartile 3 (7–13)	Quartile 4 (≥14)
Age					
Age <60 ($n = 30,263$)	1.00 (referent)	**0.80 (0.66–0.96)**	0.92 (0.78–1.09)	1.02 (0.86–1.20)	1.08 (0.93–1.26)
Age ≥60 ($n = 1,374$)	1.00 (referent)	0.86 (0.59–1.25)	0.85 (0.59–1.23)	0.75 (0.52–1.08)	1.04 (0.76–1.44)
BMI					
BMI <25 ($n = 12,037$)	1.00 (referent)	0.85 (0.65–1.11)	1.03 (0.80–1.32)	0.95 (0.74–1.22)	0.97 (0.77–1.23)
BMI ≥25 ($n = 19,600$)	1.00 (referent)	0.86 (0.70–1.06)	0.90 (0.75–1.09)	0.96 (0.79–1.16)	1.12 (0.95–1.33)
Health status[b]					
Healthy ($n = 15,989$)	1.00 (referent)	0.91 (0.69–1.20)	0.92 (0.71–1.19)	1.10 (0.86–1.42)	1.11 (0.87–1.41)
Unhealthy ($n = 15,648$)	1.00 (referent)	0.82 (0.67–1.01)	0.97 (0.81–1.17)	0.88 (0.73–1.07)	1.05 (0.89–1.24)
Fitness level					
Unfit (least fit 20%) ($n = 5,238$)	1.00 (referent)	0.91 (0.67–1.22)	1.02 (0.79–1.32)	1.10 (0.83–1.44)	1.17 (0.92–1.48)
Fit (most fit 80%) ($n = 26,399$)	1.00 (referent)	0.84 (0.69–1.03)	0.91 (0.76–1.10)	0.87 (0.73–1.05)	1.01 (0.85–1.19)

[a] Adjusted for year of examination, smoking (current smoker or not), family history of CVD (yes or no), and each other factor in the table.
[b] Defined as presence or absence of one or more of following diseases: hypertension, diabetes, or hyperlipidemia.

Various authors have used different definitions of drinks ranging from 8 grams of alcohol per drink [10] to 22.8 grams [6]. Therefore, we did not attempt to impose grams of alcohol conversion factors on self-reported drinks per week. There is additional complexity when looking at the myriad of cardiovascular outcomes. Previous studies have used various outcomes including CVD mortality, cardiac mortality, CHD, and myocardial infarction. For example, Beulens et al. [37] found a reduction in risk of myocardial infarction, but not in all-cause or CVD mortality. Due to such variation, it is challenging to establish widespread recommendations for a safe or protective amount of alcohol consumption as related to CV health.

While a decrease in risk of all-cause mortality with alcohol consumption was not found in this sample, there was not a significantly increased risk with the highest amounts of self-reported alcohol consumption. Klatsky [12] defined heavy drinking as greater than 3 drinks per day, which is consistent with the mean 24 drinks per week found in the highest quartile of alcohol consumption in this study. While our sample did not show a higher risk of either all-cause or CVD mortality with the highest alcohol consumption, there are other concerns in recommending drinking alcohol including potential alcohol abuse. Rehm et al. [44] estimated 12.1% of disability-adjusted life years for men in the US were attributed to alcohol-related causes with an estimated cost of $235 billion dollars. Of problem drinkers, 82% report drinking the moderate amount recommended to protect against CVD [17]. Alcohol may be harmful in many ways, even in small amounts (e.g., as a cocarcinogen with tobacco smoke) [45].

Table 5: Hazard ratios of CVD mortality stratified by risk factors[a], Aerobics Center for Longitudinal Study (ACLS), 1974–2002.

	Alcohol consumption (drinks/week)				
	Nondrinkers (0)	Quartile 1 (1–3)	Quartile 2 (4–6)	Quartile 3 (7–13)	Quartile 4 (≥14)
Age					
Age < 60 ($n = 30,263$)	1.00 (referent)	**0.59 (0.43–0.83)**	**0.70 (0.52–0.93)**	0.78 (0.58–1.04)	0.85 (0.66–1.11)
Age ≥ 60 ($n = 1,374$)	1.00 (referent)	1.07 (0.57–2.00)	0.82 (0.43–1.57)	1.06 (0.59–1.91)	1.53 (0.92–2.54)
BMI					
BMI < 25 ($n = 12,037$)	1.00 (referent)	0.80 (0.47–1.37)	0.84 (0.51–1.39)	0.76 (0.46–1.25)	1.02 (0.66–1.59)
BMI ≥ 25 ($n = 19,600$)	1.00 (referent)	**0.67 (0.47–0.94)**	**0.71 (0.52–0.97)**	0.82 (0.60–1.11)	0.91 (0.69–1.19)
Health status[b]					
Healthy ($n = 15,989$)	1.00 (referent)	0.61 (0.32–1.16)	0.73 (0.42–1.28)	1.06 (0.63–1.79)	1.18 (0.72–1.92)
Unhealthy ($n = 15,648$)	1.00 (referent)	0.73 (0.52–1.01)	0.76 (0.56–1.02)	**0.74 (0.54–0.998)**	0.90 (0.69–1.17)
Fitness level					
Unfit (least fit 20%) ($n = 5,238$)	1.00 (referent)	0.65 (0.39–1.08)	0.80 (0.52–1.22)	0.80 (0.50–1.27)	1.00 (0.68–1.44)
Fit (most fit 80%) ($n = 26,399$)	1.00 (referent)	0.72 (0.51–1.04)	**0.69 (0.49–0.96)**	0.73 (0.53–1.00)	0.89 (0.67–1.19)

[a] Adjusted for year of examination, smoking (current smoker or not), family history of CVD (yes or no), and each other factor in the table.
[b] Defined as presence or absence of one or more of following diseases: hypertension, diabetes, or hyperlipidemia.

Table 6: Hazard ratios of all-cause mortality stratified by types of alcohol beverage[a], Aerobics Center for Longitudinal Study (ACLS), 1974–2002.

	Alcohol consumption (drinks/week)				
	Nondrinkers	Quartile 1	Quartile 2	Quartile 3	Quartile 4
Beer ($n = 23,728$)					
No.	4870	3854	4876	5779	4349
No. of Cases	343	147	362	227	237
Hazard ratios	1.00 (referent)	0.87 (0.71, 1.06)	0.99 (0.84, 1.16)	0.90 (0.75, 1.07)	1.15 (0.96, 1.37)
Wine ($n = 21,997$)					
No.	4870	4091	4999	4175	3862
No. of Cases	343	177	370	183	196
Hazard ratios	1.00 (referent)	0.87 (0.72, 1.05)	0.92 (0.79, 1.08)	0.88 (0.73, 1.07)	0.95 (0.78, 1.14)
Liquor ($n = 20,416$)					
No.	4870	3111	5022	3562	3851
No. of Cases	343	126	383	247	423
Hazard ratios	1.00 (referent)	0.93 (0.75, 1.14)	0.96 (0.82, 1.12)	0.89 (0.75, 1.06)	1.13 (0.97, 1.32)

ACLS: Aerobics Center Longitudinal Study.
[a] Adjusted for age (in years), year of examination, BMI (kg/m^2), smoking (current smoker or not), family history of CVD (yes or no), maximal treadmill time (min), and remaining type of alcohol.

4.4. Stratified Analysis. The lower risk for all-cause mortality was attenuated when stratified by BMI and health status, but remained significant in younger men, consistent with stratified results found by Sun et al. [8]. A meta-analysis by Hvidtfeldt et al. [46] found a reduced risk of coronary heart disease in all ages, and our null findings in the older age group may be due to unequal sample size distributions for each level, reducing power in the sparser strata. The lower risk of CVD mortality in the current study remained in younger, overweight, unhealthy, and fit men. Similarly, the protective effect of alcohol consumption has been shown in populations with poor health status including men with hypertension [37], cardiovascular disease [47], and vascular disease including diabetes mellitus [2].

Interestingly, when the data were analyzed by fitness levels, fit men had lower risks of CVD mortality with larger alcohol consumption than did unfit men. This may be due to a synergistic effect as alcohol and fitness affect similar pathways such as lipid, glucose, and cholesterol metabolism. It also may be due to fit nondrinkers having a higher risk due to other latent variables (they may have been high risk and as a result began to exercise and quit drinking). The different risks associated with stratified populations confirm that the relationship between alcohol and mortality is complex and may vary between population subgroups.

4.5. Type of Alcohol. The results of the sensitivity analysis found no significant changes in risk of all-cause mortality when alcohol consumption was stratified by type, consistent with the overall analysis. Although for CVD mortality, our findings suggest that there was a reduction in risk of about 29% for moderate consumption of beer, wine, and liquor. This reduction was most consistent with liquor

Table 7: Hazard ratios of CVD mortality stratified by types of alcohol beverage[a], Aerobics Center for Longitudinal Study (ACLS), 1974–2002.

	Alcohol consumption (drinks/week)				
	Non Drinkers	Quartile 1	Quartile 2	Quartile 3	Quartile 4
Beer ($n = 23,728$)					
No.	4870	3854	4876	5779	4349
No. of Cases	128	43	104	64	75
Hazard ratios	1.00 (referent)	0.71 (0.50, 1.02)	0.76 (0.57, 1.01)	**0.72 (0.52, 0.995)**	1.06 (0.78, 1.45)
Wine ($n = 21,997$)					
No.	4870	4091	4999	4175	3862
No. of Cases	128	55	104	55	64
Hazard ratios	1.00 (referent)	0.76 (0.55, 1.05)	**0.70 (0.53, 0.93)**	0.74 (0.53, 1.04)	0.89 (0.64, 1.23)
Liquor ($n = 20,416$)					
No.	4870	3111	5022	3562	3851
No. of Cases	128	36	112	74	144
Hazard ratios	1.00 (referent)	0.74 (0.51, 1.07)	**0.71 (0.55, 0.93)**	**0.69 (0.50, 0.93)**	0.98 (0.75, 1.27)

[a] Adjusted for age (in years), year of examination, BMI (kg/m^2), smoking (current smoker or not), family history of CVD (yes or no), maximal treadmill time (min), and remaining type of alcohol.

consumption. Beulens et al. [37] also found that liquor drinkers had the strongest association with decreased risk of myocardial infarction. There have been inconsistencies, however, when comparing the types of alcohol on mortality. While several epidemiologic studies, including the current study, have found no differences in risk between types of alcohol consumed [1, 43, 48, 49], others have suggested unique cardioprotection from wine [50]. Experimental studies have shown the ethanol in wine to have additional protection compared to dealcoholized red wine [51]. The differential effects may depend on gender. Compared to dealcoholized beverages, women had increased adiponectin (an anti-inflammatory factor also shown to be associated with improved insulin sensitivity) after drinking red wine, while men had increased levels after drinking liquor and beer [52].

4.6. Strengths and Limitations. Strengths of this study are inclusion of several potential confounders including measures of fitness and psychological variables, lengthy follow-up in a large cohort, exploration of relationships by health risk factors, and stratification by alcohol types. The limitations include that the sample was only men from the Cooper Clinic in Dallas, Tex, USA, predominantly white, and of middle to high incomes, limiting generalizations to entire populations. However, the homogenous population does eliminate potential confounding from gender [4] and race [5, 28], both of which have been shown to influence the relationship between alcohol and mortality. Additionally, this regional population is similar in physiologic variables to other North American populations [53]. The mean age of 43 at baseline with an average follow-up time of 16 years makes these findings particularly relevant for an aging American population. When stratified by age, we did not find a significant association in the older men, but this may have been due to a small sample of men above 60. Future studies are needed to confirm the lower levels of alcohol consumption found to be protective in this study population. Clearly, more work will be required in populations that differ in racial and ethnic composition to that of the ACLS.

While self-reported alcohol consumption has been validated [23], under or overreporting could have influenced the trends in risk reduction in either direction. The format of response did not allow assessment of varied drinking patterns including daily frequency of alcohol consumption. Studies have suggested that alcohol should be consumed in small amounts over multiple occasions for health benefits rather than binge drinking [54]. In the sensitivity analyses, it was not possible to isolate single types of drinkers due to low prevalence of single alcohol-type drinkers.

Despite adjustment for many confounders, confounding may still exist in this and other prospective studies. One potential confounder not adjusted for was diet, particularly fat intake, which plays an established role in cardiovascular outcomes. We did, however, measure and control for blood cholesterol levels which is associated with dietary fat intake [55].

Consistent with previous studies, we used nondrinkers as the referent group [5, 43, 56]. Some researchers have argued against using nondrinkers as the referent group due to confounding predisposing conditions or other latent variables contributing to differential mortality risk independent of alcohol consumption [10]. Mukamal et al. [14] found that nondrinkers are more likely to be in poor health, obese, of low income, or physically inactive; however, they did not exclude or report whether the participants had preexisting vascular disease. In contrast, when adjusted for preexisting disease (excluded or controlled for in our model), Wannamethee and Shaper [10] found ex-drinkers and lifelong nondrinkers had equal CVD mortality and almost equal all-cause mortality. When Hart and Smith [56] examined the health status of nondrinkers to drinkers, they did not differ substantially. Friesema et al. [57] found that the inverse relationship between alcohol consumption and mortality could not be explained by health status alone. Potential confounding would have a less likely effect on CVD mortality

as Klatsky and Udaltsova [5] found that the excess mortality seen in nondrinkers is not related to CVD.

This study attempted to account for any "sick quitters" (former drinkers who stopped due to an adverse health condition) through in-depth baseline assessments to rule out underlying disease and adjustment for confounders. In asymptomatic men, especially with risk factors for CVD, exercise testing has been shown to be a valid predictor of future CVD mortality [58] beyond reported risk factors [59]. In addition, we examined if the risk association varied by levels of these risk factors (health status, age, BMI, and fitness status).

5. Conclusion

We found a trend towards reduction in risk of all-cause mortality with self-reported alcohol consumption and a reduction in risk of CVD mortality with one to six drinks per week, when adjusting for several potential confounders in a large sample of men. The amount of alcohol consumption associated with a reduced risk of CVD mortality was less than the current public health recommendations of 1-2 drinks per day. A lack of consensus on the amount and type of alcohol for protective effects and other potential harmful consequences of alcohol abuse calls for caution when making public health recommendations for alcohol consumption.

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References

[1] L. Arriola, P. Martinez-Camblor, N. Larrañaga et al., "Alcohol intake and the risk of coronary heart disease in the Spanish EPIC cohort study," *Heart*, vol. 96, no. 2, pp. 124–130, 2010.

[2] J. W. J. Beulens, A. Algra, S. S. Soedamah-Muthu, F. L. J. Visseren, D. E. Grobbee, and Y. van der Graaf, "Alcohol consumption and risk of recurrent cardiovascular events and mortality in patients with clinically manifest vascular disease and diabetes mellitus: the Second Manifestations of ARTerial (SMART) disease study," *Atherosclerosis*, vol. 212, no. 1, pp. 281–286, 2010.

[3] J. M. Gaziano, T. A. Gaziano, R. J. Glynn et al., "Light-to-moderate alcohol consumption and mortality in the physicians' health study enrollment cohort," *Journal of the American College of Cardiology*, vol. 35, no. 1, pp. 96–105, 2000.

[4] L. R. Harriss, D. R. English, J. L. Hopper et al., "Alcohol consumption and cardiovascular mortality accounting for possible misclassification of intake: 11-year follow-up of the Melbourne Collaborative Cohort Study," *Addiction*, vol. 102, no. 10, pp. 1574–1585, 2007.

[5] A. L. Klatsky and N. Udaltsova, "Alcohol drinking and total mortality risk," *Annals of Epidemiology*, vol. 17, no. 5, pp. S63–S67, 2007.

[6] A. Sadakane, T. Gotoh, S. Ishikawa, Y. Nakamura, and K. Kayaba, "Amount and frequency of alcohol consumption and all-cause mortality in a Japanese population: the JMS cohort study," *Journal of Epidemiology*, vol. 19, no. 3, pp. 107–115, 2009.

[7] I. Suh, B. J. Shaten, J. A. Cutler, and L. H. Kuller, "Alcohol use and mortality from coronary heart disease: the role of high-density lipoprotein cholesterol," *Annals of Internal Medicine*, vol. 116, no. 11, pp. 881–887, 1992.

[8] W. Sun, C. M. Schooling, W. M. Chan, K. S. Ho, T. H. Lam, and G. M. Leung, "Moderate alcohol use, health status, and mortality in a prospective Chinese elderly cohort," *Annals of Epidemiology*, vol. 19, no. 6, pp. 396–403, 2009.

[9] M. J. Thun, R. Peto, A. D. Lopez et al., "Alcohol consumption and mortality among middle-aged and elderly U.S. adults," *The New England Journal of Medicine*, vol. 337, no. 24, pp. 1705–1714, 1997.

[10] S. G. Wannamethee and A. G. Shaper, "Lifelong teetotallers, ex-drinkers and drinkers: mortality and the incidence of major coronary heart disease events in middle-aged British men," *International Journal of Epidemiology*, vol. 26, no. 3, pp. 523–531, 1997.

[11] M. A. Collins, E. J. Neafsey, K. J. Mukamal et al., "Alcohol in moderation, cardioprotection, and neuroprotection: epidemiological considerations and mechanistic studies," *Alcoholism: Clinical and Experimental Research*, vol. 33, no. 2, pp. 206–219, 2009.

[12] A. L. Klatsky, "Alcohol and cardiovascular health," *Physiology and Behavior*, vol. 100, no. 1, pp. 76–81, 2010.

[13] A. Di Castelnuovo, S. Costanzo, M. B. Donati, L. Iacoviello, and G. de Gaetano, "Prevention of cardiovascular risk by moderate alcohol consumption: epidemiologic evidence and plausible mechanisms," *Internal and Emergency Medicine*, vol. 5, no. 4, pp. 291–297, 2010.

[14] K. J. Mukamal, E. L. Ding, and L. Djoussé, "Alcohol consumption, physical activity, and chronic disease risk factors: a population-based cross-sectional survey," *BMC Public Health*, vol. 6, article no. 118, 2006.

[15] N. El-Guebaly, "Investigating the association between moderate drinking and mental health," *Annals of Epidemiology*, vol. 17, no. 5, pp. S55–S62, 2007.

[16] S. Peele and A. Brodsky, "Exploring psychological benefits associated with moderate alcohol use: a necessary corrective to assessments of drinking outcomes?" *Drug and Alcohol Dependence*, vol. 60, no. 3, pp. 221–247, 2000.

[17] K. A. Ammar, S. Samee, R. Colligan et al., "Is self-reported moderate drinking in the cardiovascular benefit range associated with alcoholic behavior? A population based study," *Journal of Addictive Diseases*, vol. 28, no. 3, pp. 243–249, 2009.

[18] G. Corrao, V. Bagnardi, A. Zambon, and C. La Vecchia, "A meta-analysis of alcohol consumption and the risk of 15 diseases," *Preventive Medicine*, vol. 38, no. 5, pp. 613–619, 2004.

[19] C. Bosetti, A. Altieri, and C. La Vecchia, "Diet and environmental carcinogenesis in breast/gynaecological cancers," *Current Opinion in Obstetrics and Gynecology*, vol. 14, no. 1, pp. 13–18, 2002.

[20] M. C. Pike, L. N. Kolonel, B. E. Henderson et al., "Breast cancer in a multiethnic cohort in Hawaii and Los Angeles: risk

factor-adjusted incidence in Japanese equals and in Hawaiians exceeds that in whites," *Cancer Epidemiology Biomarkers and Prevention*, vol. 11, no. 9, pp. 795–800, 2002.

[21] G. Pöschl, F. Stickel, X. D. Wang, and H. K. Seitz, "Alcohol and cancer: genetic and nutritional aspects," *Proceedings of the Nutrition Society*, vol. 63, no. 1, pp. 65–71, 2004.

[22] S. N. Blair, H. W. Kohl III, R. S. Paffenbarger Jr., D. G. Clark, K. H. Cooper, and L. W. Gibbons, "Physical fitness and all-cause mortality: a prospective study of healthy men and women," *Journal of the American Medical Association*, vol. 262, no. 17, pp. 2395–2401, 1989.

[23] E. Giovannucci, G. Colditz, M. J. Stampfer et al., "The assessment of alcohol consumption by a simple self-administered questionnaire," *American Journal of Epidemiology*, vol. 133, no. 8, pp. 810–817, 1991.

[24] J. R. Hebert, I. S. Ockene, T. G. Hurley et al., "Development and testing of a seven-day dietary recall," *Journal of Clinical Epidemiology*, vol. 50, no. 8, pp. 925–937, 1997.

[25] C. D. Lee, X. Sui, and S. N. Blair, "Combined effects of cardiorespiratory fitness, not smoking, and normal waist girth on morbidity and mortality in men," *Archives of Internal Medicine*, vol. 169, no. 22, pp. 2096–2101, 2009.

[26] W. Byun, J. C. Sieverdes, X. Sui et al., "Effect of positive health factors and all-cause mortality in men," *Medicine and Science in Sports and Exercise*, vol. 42, no. 9, pp. 1632–1638, 2010.

[27] T. G. Pickering, J. E. Hall, L. J. Appel et al., "Recommendations for blood pressure measurement in humans and experimental animals—part 1: blood pressure measurement in humans: a statement for professionals from the subcommittee of professional and public education of the American Heart Association council on high blood pressure research," *Hypertension*, vol. 45, no. 1, pp. 142–161, 2005.

[28] J. I. Cleeman and C. Lenfant, "The National Cholesterol Education Program. Progress and prospects," *Journal of the American Medical Association*, vol. 280, no. 24, pp. 2099–2104, 1998.

[29] J. R. Gavin, K. G. M. M. Alberti, M. B. Davidson et al., "Report of the expert committee on the diagnosis and classification of diabetes mellitus," *Diabetes Care*, vol. 26, supplement 1, pp. S5–S20, 2003.

[30] B. Balke and R. W. Ware, "An experimental study of physical fitness of Air Force personnel," *US Armed Forces Medical Journal*, vol. 10, no. 6, pp. 675–688, 1959.

[31] M. L. Pollock, R. L. Bohannon, and K. H. Cooper, "A comparative analysis of four protocols for maximal treadmill stress testing," *American Heart Journal*, vol. 92, no. 1, pp. 39–46, 1976.

[32] S. N. Blair, Y. Cheng, and J. Scott Holder, "Is physical activity or physical fitness more important in defining health benefits?" *Medicine and Science in Sports and Exercise*, vol. 33, no. 6, pp. S379–S399, 2001.

[33] X. Sui, M. J. LaMonte, and S. N. Blair, "Cardiorespiratory fitness as a predictor of nonfatal cardiovascular events in asymptomatic women and men," *American Journal of Epidemiology*, vol. 165, no. 12, pp. 1413–1423, 2007.

[34] B. Taylor, H. M. Irving, D. Baliunas et al., "Alcohol and hypertension: gender differences in dose-response relationships determined through systematic review and meta-analysis: REVIEW," *Addiction*, vol. 104, no. 12, pp. 1981–1990, 2009.

[35] S. Kiechl, J. Willeit, W. Poewe et al., "Insulin sensitivity and regular alcohol consumption: large, prospective, cross sectional population study (Bruneck study)," *British Medical Journal*, vol. 313, no. 7064, pp. 1040–1044, 1996.

[36] E. B. Rimm, P. Williams, K. Fosher, M. Criqui, and M. J. Stampfer, "Moderate alcohol intake and lower risk of coronary heart disease: meta-analysis of effects on lipids and haemostatic factors," *British Medical Journal*, vol. 319, no. 7224, pp. 1523–1528, 1999.

[37] J. W. J. Beulens, E. B. Rimm, A. Ascherio, D. Spiegelman, H. F. J. Hendriks, and K. J. Mukamal, "Alcohol consumption and risk for coronary heart disease among men with hypertension," *Annals of Internal Medicine*, vol. 146, no. 1, pp. 10–19, 2007.

[38] M. S. Freiberg, H. J. Cabral, T. C. Heeren, R. S. Vasan, and R. C. Ellison, "Alcohol consumption and the prevalence of the metabolic syndrome in the U.S. A cross-sectional analysis of data from the Third National Health and Nutrition Examination Survey," *Diabetes Care*, vol. 27, no. 12, pp. 2954–2959, 2004.

[39] A. Di Castelnuovo, S. Costanzo, V. Bagnardi, M. B. Donati, L. Iacoviello, and G. De Gaetano, "Alcohol dosing and total mortality in men and women: an updated meta-analysis of 34 prospective studies," *Archives of Internal Medicine*, vol. 166, no. 22, pp. 2437–2445, 2006.

[40] D. Lloyd-Jones, R. J. Adams, T. M. Brown et al., "Heart disease and stroke statistics—2010 update: a report from the American heart association," *Circulation*, vol. 121, no. 7, pp. e46–e215, 2010.

[41] L. Djoussé, I. M. Lee, J. E. Buring, and J. M. Gaziano, "Alcohol consumption and risk of cardiovascular disease and death in women: potential mediating mechanisms," *Circulation*, vol. 120, no. 3, pp. 237–244, 2009.

[42] R. Femia, A. Natali, A. L'Abbate, and E. Ferrannini, "Coronary atherosclerosis and alcohol consumption: angiographic and mortality data," *Arteriosclerosis, Thrombosis, and Vascular Biology*, vol. 26, no. 7, pp. 1607–1612, 2006.

[43] A. H. Lichtenstein, L. J. Appel, M. Brands et al., "Diet and lifestyle recommendations revision 2006: a scientific statement from the American heart association nutrition committee," *Circulation*, vol. 114, no. 1, pp. 82–96, 2006.

[44] J. Rehm, C. Mathers, S. Popova, M. Thavorncharoensap, Y. Teerawattananon, and J. Patra, "Global burden of disease and injury and economic cost attributable to alcohol use and alcohol-use disorders," *The Lancet*, vol. 373, no. 9682, pp. 2223–2233, 2009.

[45] A. E. Rogers and M. W. Conner, "Alcohol and cancer," *Advances in Experimental Medicine and Biology*, vol. 206, pp. 473–495, 1986.

[46] U. A. Hvidtfeldt, J. S. Tolstrup, M. U. Jakobsen et al., "Alcohol intake and risk of coronary heart disease in younger, middle-aged, and older adults," *Circulation*, vol. 121, no. 14, pp. 1589–1597, 2010.

[47] S. Costanzo, A. Di Castelnuovo, M. B. Donati, L. Iacoviello, and G. de Gaetano, "Alcohol consumption and mortality in patients with cardiovascular disease. A meta-analysis," *Journal of the American College of Cardiology*, vol. 55, no. 13, pp. 1339–1347, 2010.

[48] R. A. Bell, E. J. Mayer-Davis, M. A. Martin, R. B. D'Agostino, and S. M. Haffner, "Associations between alcohol consumption and insulin sensitivity and cardiovascular disease risk factors: the insulin resistance and atherosclerosis study," *Diabetes Care*, vol. 23, no. 11, pp. 1630–1636, 2000.

[49] C. S. Fuchs, M. J. Stampfer, G. A. Colditz et al., "Alcohol consumption and mortality among women," *The New England Journal of Medicine*, vol. 332, no. 19, pp. 1245–1250, 1995.

[50] A. L. Klatsky, G. D. Friedman, M. A. Armstrong, and H. Kipp, "Wine, liquor, beer, and mortality," *American Journal of Epidemiology*, vol. 158, no. 6, pp. 585–595, 2003.

[51] T. O. Kiviniemi, A. Saraste, J. O. Toikka et al., "A moderate dose of red wine, but not de-alcoholized red wine increases coronary flow reserve," *Atherosclerosis*, vol. 195, no. 2, pp. e176–e181, 2007.
[52] A. Imhof, I. Plamper, S. Maier, G. Trischler, and W. Koenig, "Effect of drinking on adiponectin in healthy men and women: a randomized intervention study of water, ethanol, red wine, and beer with or without alcohol," *Diabetes Care*, vol. 32, no. 6, pp. 1101–1103, 2009.
[53] S. N. Blair, W. B. Kannel, H. W. Kohl, N. Goodyear, and P. W. F. Wilson, "Surrogate measures of physical activity and physical fitness. Evidence for sedentary traits of resting tachycardia, obesity, and low vital capacity," *American Journal of Epidemiology*, vol. 129, no. 6, pp. 1145–1156, 1989.
[54] R. P. Murray, J. E. Connett, S. L. Tyas et al., "Alcohol volume, drinking pattern, and cardiovascular disease morbidity and mortality: is there a u-shaped function?" *American Journal of Epidemiology*, vol. 155, no. 3, pp. 242–248, 2002.
[55] R. Clarke, C. Frost, R. Collins, P. Appleby, and R. Peto, "Dietary lipids and blood cholesterol: quantitative meta-analysis of metabolic ward studies," *British Medical Journal*, vol. 314, no. 7074, pp. 112–117, 1997.
[56] C. L. Hart and G. D. Smith, "Alcohol consumption and mortality and hospital admissions in men from the Midspan Collaborative cohort study," *Addiction*, vol. 103, no. 12, pp. 1979–1986, 2008.
[57] I. H. M. Friesema, P. J. Zwietering, M. Y. Veenstra, J. A. Knottnerus, H. F. L. Garretsen, and P. H. H. M. Lemmens, "Alcohol intake and cardiovascular disease and mortality: the role of pre-existing disease," *Journal of Epidemiology and Community Health*, vol. 61, no. 5, pp. 441–446, 2007.
[58] L. W. Gibbons, T. L. Mitchell, M. Wei, S. N. Blair, and K. H. Cooper, "Maximal exercise test as a predictor of risk for mortality from coronary heart disease in asymptomatic men," *American Journal of Cardiology*, vol. 86, no. 1, pp. 53–58, 2000.
[59] G. J. Balady, M. G. Larson, R. S. Vasan, E. P. Leip, C. J. O'Donnell, and D. Levy, "Usefulness of exercise testing in the prediction of coronary disease risk among asymptomatic persons as a function of the Framingham risk score," *Circulation*, vol. 110, no. 14, pp. 1920–1925, 2004.

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Research Article

Examining the Racial Crossover in Mortality between African American and White Older Adults: A Multilevel Survival Analysis of Race, Individual Socioeconomic Status, and Neighborhood Socioeconomic Context

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We examine whether individual and neighborhood socioeconomic context contributes to black/white disparities in mortality among USA older adults. Using national longitudinal data from the Americans' Changing Lives study, along with census tract information for each respondent, we conduct multilevel survival analyses. Results show that black older adults are disadvantaged in mortality in younger old age, but older black adults have lower mortality risk than whites after about age 80. Both individual SES and neighborhood socioeconomic disadvantage contribute to the mortality risk of older adults but do not completely explain race differences in mortality. The racial mortality crossover persists even after controlling for multilevel SES, suggesting that black older adults experience selective survival at very old ages. Addressing the individual and neighborhood socioeconomic disadvantage of blacks is necessary to reduce mortality disparities that culminate in older adulthood.

1. Introduction

Black older adults (ages 65+) have higher all-cause mortality rates than white older adults in the USA [1, 2]. A growing body of literature attempts to understand the mechanisms explaining these persisting race differences in mortality [3–5] so that we can understand how such race disparities might be reduced or eliminated. Substantial disparities in individual socioeconomic status (SES) by race have been observed in the USA, and research has shown that individual SES (e.g., income and education) partly mediates but does not eliminate the relationship between race and mortality [6–8]. In addition, neighborhood context has been identified as a key factor that contributes to race disparities in morbidity [9–11]. However, few studies have examined how neighborhood context contributes to race difference in mortality [12, 13], particularly at older ages.

The current study employees mixed effect survival analysis to investigate whether SES, measured at both the individual and neighborhood levels, explains black/white differences in mortality among older adults in the USA, using longitudinal national data.

A number of theoretical perspectives, including economic deprivation [14] and social disorganization theory [15, 16], suggest that neighborhood context is associated with health and mortality. Some studies have shown that living in a poor neighborhood is related to increased cause-specific mortality among adults [17–20], after controlling for individual SES variables. However, other studies have shown that there is no significant association between neighborhood context and all-cause mortality for older adults [11, 21, 22].

Very few studies have examined the contribution of neighborhood socioeconomic context to race disparities

in mortality for older adults, although some research has examined this question for younger and middle-aged adults. LeClere and colleagues found that the differential all-cause mortality rates between African American and non-Hispanic white men and women aged 18 and older were partly explained by individual-level SES and were further explained by neighborhood context. The differences in mortality between the black and white adults were completely explained by both individual SES and neighborhood combined [23]. However, some studies indicate that race differences in mortality persist among USA adults even after both individual SES and neighborhood context are controlled. For example, one study demonstrated that mortality for all racial/ethnic groups is related to individual SES and to neighborhood characteristics, among people aged 18–64. The race difference in mortality persisted after controlling for both individual and neighborhood level SES [24].

With regard to older adults, LeClere and colleagues showed that the percentage of female-headed families in the neighborhood was associated with heart disease mortality for women aged 65 and above. Individual SES and neighborhood context fully accounted for the heart disease mortality disparity between white and black older adults [12]. The above research provides some knowledge about the relationships among race, multilevel SES, and all-cause mortality, but the results are not consistent.

Moreover, this body of research has not fully considered the shape of the relationship between race and mortality at older ages. However, research suggests that there is a racial mortality crossover at older ages such that black older adults have higher mortality than white older adults in *young* older ages while white older adults have higher mortality than their black counterparts in *very late* old age [25–30]. It could be that the racial mortality crossover in late old age contributes to the inconsistent conclusions regarding the role of multilevel SES in explaining race difference in mortality among older adults. This is because if crossover effects exist, the effect of race on mortality for a younger subgroup will be positive while the effect of race will be negative for an older age subgroup. Combining age groups together in one "old age" group ages 65+ may result in the positive and negative effects canceling each other out.

There are two primary competing explanations for racial mortality crossover effects [27]. If black older adults are more likely to misreport their age as older than it really is, this would result in more young black older adults being categorized in the oldest old groups. Alternately, black older adults may experience selective mortality. Since black adults are more likely to die at younger ages, the black survivors at very older age should be very robust. Unfortunately, it is difficult to detect age reporting errors in survey data. In any case, the potential existence of the racial mortality crossover effect means that, when studying race differences in mortality among older adults, it is necessary to consider the interaction of race and age.

There have been theoretical debates on the expected patterns of race disparities in mortality over the life course. The double jeopardy hypothesis suggests that blacks are faced with a double burden on health with aging and therefore should experience worse health status and mortality compared to whites at older ages [31]. Another perspective hypothesizes persistent inequality—that race differences in health are set at earlier ages and are then relatively stable throughout the aging process [32]. These two perspectives suggest that crossover effects should not exist. A third perspective describes a different story in which some factors that affect health at younger ages are not as strongly associated with changes in health at older ages. In particular, social factors may be less important than biological factors at older ages. For example, the research by House et al. [33] suggests that the relationship between SES, other risk factors, and health may be buffered by biological robustness (in early adulthood), biological frailty (in later old age), or the existence of social welfare programs, particularly at older ages. This third perspective suggests that the race difference will reduce or even crossover at older ages.

Empirical studies provide some support for the third perspective and have shown that the health disadvantage of blacks not only disappears but crosses over such that black older adults have lower mortality ratios at very old ages (around age 80) [27, 34, 35]. If this crossover effect happens, it may obscure the true relationships among race, multilevel SES and mortality among older adults in different age groups. Given this consideration, we examine the potential for a race crossover effect in mortality among older adults and reexamine the contribution of multilevel SES to mortality and its variation by age among older adults.

Our study extends prior work in a number of ways. The existing literature has contrary findings on the effects of neighborhood context on mortality and the magnitude of the effects of neighborhood context on explaining race differences in mortality among older adults. A number of limitations to prior work may cause this contradiction. First, as described above, most studies examine all older adults combined, rather than examining age-specific changes among older adults. Second, most studies examined *disease-specific* mortality, but it is also important to understand how multilevel SES is associated with *all-cause* mortality. Third, most previous studies used a single indicator or separate indicators of neighborhood context, whereas we combine a number of neighborhood variables to create a more comprehensive neighborhood disadvantage index.

Fourth, our statistical approach represents an improvement on prior work in the USA because we use multilevel survival analyses to account for the multilevel nature of the data while modeling mortality using the greater precision of survival analysis. Most prior work in the USA has not adjusted for the complex survey design effects that result from the multistate sampling method of the data or the nested nature of the individual-level data within neighborhoods, leading to potentially incorrect variance and parameter estimates [36–38]. Some studies considered the correlated data structure and used multilevel logistic regression to estimate the association between multilevel SES and mortality [11, 22, 39, 40]. But in these studies, censoring issues were not addressed leading to potential errors in estimates. Recently, a few studies employed multilevel survival

analysis, which combines the advantages of mixed effects model and Cox proportional hazard model to estimate the effects of multilevel SES on cause-specific mortality [17–19, 41] and all-cause mortality [42]. However, these studies were based in European countries. We utilize this method using a USA sample.

In sum, results regarding the effects of neighborhood SES on mortality and its contribution to race differences in mortality among older adults are mixed. Some national studies provided evidence that multilevel SES is associated with mortality and contributes to explaining race differences in mortality [12, 23, 24, 43]. However, there is still a debate on the magnitude of their contribution to race differences in mortality, particularly at older ages. We build from and attempt to improve upon prior work by introducing an interaction between race and age. We also use multilevel survival models to appropriately estimate the associations between race, the interaction of race and age, individual SES, neighborhood SES, and all-cause mortality over 16 years among older adults in a nationally representative sample of adults in the USA.

2. Materials and Methods

2.1. Data. Wave 1 (W1) of the Americans Changing Lives (ACL) study was conducted in 1986 through face-to-face interviews in the homes of 3,617 adults. The sample was created using a multistage, stratified area probability sample of noninstitutionalized adults aged 25 and older living in the 48 contiguous states, with oversamples of black people and older adults. We limit our analysis to the cohort of people ages 65 and older in order to replicate and extend previous studies that looked at this age cohort [3, 25, 44, 45]. The analytic sample used in this study includes respondents aged 65 or older at W1 (with survivors being aged 81 or older at wave 4) who reported their race as white or black, for a final analytic sample of 1211 respondents aged 65 and older at W1.

Neighborhood variables are taken from the 1990 census at the census tract level, based on each individual's residence at W1 of the interview.

Data were collected by the University of Michigan using approved human subjects protocols. Data analyses were conducted with approval by the University of Wisconsin-Madison social studies institutional review board.

2.2. Variables. Education is measured as years of formal schooling completed by W1 and is included as a continuous variable. Family income at baseline is included as an ordinal variable ranging from 1 to 10 (1 = less than \$5K; 2 = \$5K−\$9999; 3 = \$10K−14,999; 4 = \$15K−19,999; 5 = \$20K−24,999; 6 = \$25K−29,999; 7 = \$30K−39,999; 8 = \$40K−59,999; 9 = \$60K−79,999; 10 = \$80K and above). Regression-based imputations were made for missing income data.

Five neighborhood characteristics (% adults aged 25+ with 16+ years schooling, % households with public assistance, % persons aged 65+ below poverty line, % families that are female-headed, and mean family income) are used to create a neighborhood socioeconomic disadvantage index (Neighborhood SDI). After conducting a factor analysis (see Table 3), each of the five neighborhood measures was standardized, weighted by its factor loading, and summed (% adults aged 25+ with 16+ years schooling and mean family income were reversed) to create the Neighborhood SDI. A higher score means greater neighborhood disadvantage.

Gender is coded 1 for men and 0 for women. Race is a dummy variable (Black = 1; White = 0).

The dependent variable at each wave is all-cause mortality. Mortality was tracked over time and was confirmed with the National Death Index (coded to the year of death) through wave 4 (2001) [33].

2.3. Statistical Analysis. Survival analysis is used to examine predictors of time until death. The basic Cox proportional hazards regression is expressed as [46, 47]

$$h(t \mid Z) = h_0(t) \exp\left(\sum_{k=1}^{p} \beta_k Z_k\right). \quad (1)$$

$h(t \mid Z)$ is the hazard rate at time t for an individual covariates vector Z. $h_0(t)$ is an arbitrary baseline hazard rate. β_k is the estimated parameter vector that represents the direction and magnitude of the association between Z_k and $h(t \mid Z)$ compared to baseline hazard rate. If we assume that censoring time and event for the jth participants are independent given by Z_k, the parameters in (1) could be estimated by maximizing the partial likelihood function in

$$L(\beta) = \prod_{i=1}^{D} \frac{\exp\left[\sum_{k=1}^{p} \beta_k Z_{(i)k}\right]}{\sum_{j \in R(t_i)} \exp\left[\sum_{k=1}^{p} \beta_k Z_{jk}\right]}. \quad (2)$$

In (2), let $t_1 > t_2 \ldots < T_D$ denote the ordered event times. $Z_{(i)k}$ is the kth covariate associated with individual whose death time is t_i. $R(t_i)$ represents the kth covariate associated with individuals who are still in the risk set at a time just prior to t_i. The numerator in (2) represents the information about death where the denominator includes all information about individuals who have not yet died.

When neighborhood socioeconomic context is included in analysis, we model the association between individual survival times within neighborhood. There is an unobservable random effect shared by subjects within a neighborhood. In this case, Cox proportional hazard mixed effects model (PHMM) is appropriate [46, 48, 49]. To simplify the model, one time-invariant neighborhood socioeconomic context variable is included in the analysis. The hazard function for the jth individual in i neighborhood can be expressed as [50, 51]

$$h_{ij}(t \mid Z) = h_0(t) \exp\left(\beta' Z_{ij} + b_i' \omega_{ij}\right). \quad (3)$$

Z_{ij} is the vector covariate and β is the vector of regression coefficient. ω_{ij} is a vector of covariates that have random effects. This equation captures the random effects of the cluster and enables covariate by cluster interactions. Due to the interaction of clustered and individual covariates, ω_{ij} is a subset of Z_{ij} [51, 52].

Table 1: Descriptive statistics (unweighted): percentage distribution or mean (standard deviation in parentheses) at baseline for independent variables (respondents aged 65 and above at baseline, $N = 1211$).

Variables	Range	Percentages or mean
Male (%)	0-1	31.23
Black (%)	0-1	28.98
Age	65–96	73.13 (6.34)
Income	1–10	3.18 (2.16)
Education	0–17	9.99 (3.75)
Neighborhood SDI (standardized)	−3.22–3.26	0 (1)

Neighborhood SDI: Neighborhood Socioeconomic Disadvantage Index.

2.4. Analytic Strategy. A series of Cox proportional hazard models will be presented to examine the association between race, multilevel SES, and mortality. Models 1 to 4 in Table 2 use Cox proportional hazards regression to estimate the contribution of both individual SES and neighborhood SDI to race differences in mortality when neighborhood-level variance is ignored. Mixed effect cox analyses are shown in Models 1b to 4b in Table 2 in order to examine how multilevel SES explains race disparity in mortality when we consider the neighborhood-level variance. Finally, the interaction between race and age is introduced in Models 5 to 7 to investigate whether race differences in mortality differ by different age groups after multilevel SES is controlled.

3. Results

Table 1 presents the descriptive information for the demographic and socioeconomic measures at baseline for 1211 older adults aged 65 and above who reported their race as either white or black. About one third of participants were male, about 45% were married at baseline, and the average age was 73. The mean years of education are about 10. The average family income was in the range of $15,000 to $19,999 in 1986.

The effects of individual SES and neighborhood SDI on mortality are presented in Table 2. Model 1 in Table 2 shows that black older adults had a higher mortality rate (e**0.167 = 1.18) than white older adults. The probability of dying (at an earlier age or by the end of the study period) for black older adults is on average 18% higher than the probability of death for white older adults. When only individual SES measures were added in Model 2, race differences in mortality disappeared and family income is negatively related to mortality. Model 3 examines the effects of neighborhood SDI on mortality. First, we find that neighborhood SDI is positively associated with mortality. This model suggests that, if the neighborhood socioeconomic disadvantage index increases by one standard deviation, the probability of death increases by 10% (e**0.091 = 1.0953). Race differences in mortality persist after controlling for neighborhood SDI. Both individual SES and neighborhood SES were included simultaneously in Model 4. Model 4 indicates that both family income and neighborhood SDI are significantly related to mortality. However, there is no remaining statistically significant race difference in mortality.

Models 1b–4b in Table 2, using mixed effects Cox model, demonstrate similar results. Both individual SES and neighborhood SDI are related to mortality. Race differences in mortality are fully mediated by individual SES. Looking at Models 2–4 and 2b–4b together, we conclude that individual SES is stronger than neighborhood SDI in explaining race differences in mortality.

The interaction of race by age was added in Models 5–7 in Table 2 in order to investigate whether race disparities in mortality vary by age among older adults. Model 5 indicates that there is a significant interaction of race and age, which means that race differences in mortality are not constant across age for older adults. The positive coefficients for race and age and the negative coefficient for the interaction of race and age support theories that suggest a diminishing or crossover effect of race rather than theories suggesting a double jeopardy hypothesis. The positive coefficient of race suggests that black adults experienced a greater risk of death at young old ages. The negative coefficient of the interaction of race and age shows that black older adults' risk of morality increases at a slower rate than white older adults' risk. We compute the turning point age where black and white older adults have an equal risk of mortality after controlling other covariates in Model 5. The formula is 3.028*1 + 0.106*age − 0.038*age = 3.028*0 + 0106*age − 0.038*0 and we get age = 79.68. This means that after around age 80, black older adults had lower risk of dying than whites.

In Model 6, we include individual SES measures to examine their effects on mortality and their contribution to race differences in mortality by age. First, as in Models 1–4 and Models 1b–4b, family income is negatively associated with mortality. Second, including individual SES measures reduced but did not fully explain race differences in mortality by age.

Neighborhood SDI is added in Model 7. We see that living in a neighborhood with greater disadvantage is associated with a greater risk of dying (e**0.086 = 1.09), net of demographic, and individual SES variables, and it further reduces the association between race and mortality by age. Again, there remains a significant racial crossover effect on mortality due to the positive coefficient of race and negative coefficient of the interaction of race and age. The turning point of the crossover is about 74 after controlling for SES at multiple levels.

Finally, all variances of neighborhood tracts are significant in Models 1b to 4b and Models 5 to 7. The standard error presents how much an individual neighborhood varies in its mortality rate compared to the norm [53, 54]. For example, the variance is 0.0051 in Model 4b. Its standard error is 0.07. This suggests that an individual neighborhood has an average 7 percent higher or lower mortality level compared to the norm. This relatively large variation between neighborhoods suggests that it is appropriate to model neighborhood-level variance in mortality analyses among older adults.

Table 2: The effect of individual SES, neighborhood context on mortality over time (1986–2002) for people aged 65+ at baseline: multilevel survival model.

Variables	Model	Model 2	Model 3	Model 4	Model 1b	Model 2b	Model 3b	Model 4b	Model 5	Model 6	Model 7
Black	0.167*	0.013	0.163**	−0.010	0.192**	0.040	0.180*	0.021	3.028***	2.672**	2.529*
Male	0.621***	0.690***	0.586***	0.653***	0.623**	0.668***	0.585***	0.632***	0.609***	0.646***	0.614***
Age	0.087 ***	0.081***	0.087***	0.081***	0.091***	0.086***	0.091***	0.086***	0.106***	0.100***	0.099***
Age*Black									−.038***	−0.035**	−0.034**
Fam. Income		−.092***		−.093***		−.070***		−.071***		−.063***	−0.065*
Education		−0.008		−0.0119		−0.012		−0.0151		−.011***	−0.018
SDI			0.091*	0.107**			0.079*	0.087**			0.086**
Random effect					.0096***	.0069***	.0047***	.0051***	.0051***	.0017***	.0017***

*** $P < 0.001$; ** $P < 0.01$; * $P < 0.05$; + $P < 0.10$.
SDI: Neighborhood Socioeconomic Disadvantage Index; Random effect: random effects of neighborhood; Fam. income: family income.

Table 3: Principal components factor analysis of Neighborhood Socioeconomic Disadvantage Index.

Census item	Factor loading
% 25+ adults with 16+ years schooling	−0.73
% Households with public assistance	0.86
% Persons aged 65+ below poverty line	0.77
% Families female-headed	0.81
Mean family income[a]	−0.79

[a] Reverse-coded when added to index.

In additional analyses, we tested whether race differences in mortality vary by other covariates by testing the interactions of race and other covariates (see Table 4). The results show that no other interactions are significant.

4. Discussion

We examined the role of individual SES and neighborhood SES in explaining race differences in all-cause mortality over time among black and white older adults. We explicitly tested whether race disparities in mortality vary by age and examined the contribution of individual and neighborhood SES to explaining age variations in these race disparities.

One debate in previous studies is whether there are effects of neighborhood SES on mortality for older adults. Some studies found that neighborhood context has no effects on mortality, while other studies showed that there is an association between neighborhood and mortality [22]. We believe that inconsistencies in prior work may be partly due to the fact that most prior work did not take into account the correlated data structure and censoring issues related to examining mortality data over time. We applied a multilevel survival approach (COX PH model with mixed effects) to estimate the effects of neighborhood on mortality. We found that older adults who lived in more disadvantaged neighborhoods had a higher mortality rate, which is consistent with the studies that used COX PH model with mixed effects in other countries [17–19, 39]. Thus, we have more confidence in concluding that there is a significant relationship between neighborhood SDI and all-cause mortality among older adults in the USA.

The second debate is whether individual SES and neighborhood SES help explain race differences in mortality for older adults. Although only very few studies have implicitly examined this issue, the results have been inconsistent. Some studies showed that individual SES fully mediates the relationship between race and mortality, while other studies found that individual SES helped explain the difference with neighborhood context further explaining the difference. However, none of these prior studies considered or modeled the race crossover effect of mortality between black and white older adults.

In this study, we examined the contribution of individual SES and neighborhood SDI to mortality through two sets of analysis—with and without modeling a racial crossover effect. The analysis without modeling a potential crossover effect showed that individual SES fully mediated the relationship between race and mortality. However, the analysis with the interaction term between age and race demonstrated that there is a race crossover effect and further indicated that race differences in mortality persist after both individual SES and neighborhood SDI were controlled. Our analysis suggests that the crossover effect happens around ages 76–80, which is consistent with previous studies [27, 34].

There are a number of implications of this study. First, our study suggests that age reporting bias is probably not the main reason for the racial crossover in mortality. This is because, even if more young black older adults were falsely categorized into the oldest old groups, social factors still exert a large effect on race difference in mortality since significant race differences in mortality exist between blacks and whites at early older age. At the oldest ages, the aging process itself, especially biological factors, may have more weight on mortality and health [33]. Given our data and findings, our results are consistent with a selective mortality explanation for the racial crossover in mortality at later old age for black and white older adults. The selective survival of robust black older adults likely explains the racial crossover.

Another important finding is that our analyses reveal that individual SES explains more of the race differences in mortality than does neighborhood SDI. This is consistent with previous studies [13]. However, we must keep in mind that some of the neighborhood effects on mortality may work *through* their effects on individual-level income

Table 4: The effect of individual SES and neighborhood context on mortality over time (1986–2002) for people aged 65+ at baseline: multilevel survival model (including interactions of race and other covariates).

Variables	Model 1	Model 2	Model 3	Model 4
Black	0.102	0.013	0.222	0.222
Male	0.711***	0.632***	0.634***	0.632***
Age	0.085**	0.085**	0.086***	0.0852***
Black*age				
Black*male	−0.254			
Black*F-income		0.0040		
Black*education			0.0283	
Black*Neigh. SDI				0.0086
F-income	−0.071*	−0.071*	−0.066*	−0.070**
Education	−0.015	−0.015	−0.026	−0.014
Neighborhood SDI	0.075+	0.075+	0.079*	0.077
Random effect	0.0052	0.0051	0.0051	0.0051

*** $P < 0.001$; ** $P < 0.01$; * $P < 0.05$; + $P < .10$.
Neighborhood SDI: Neighborhood Socioeconomic Disadvantage Index; Random effect: random effects of neighborhood; F-income: Family income.

over the life course. Actual neighborhood effects are likely understated when individual-level income is controlled.

There are a number of limitations to our study. First, we only include white and black older adults in our analysis due to sample limitations of racial/ethnic distribution in the ACL data. Future studies should examine how individual and neighborhood socioeconomic context contribute to racial/ethnic disparities in mortality between other racial/ethnic groups. Second, we only include *baseline* individual SES and neighborhood SDI variables in the analysis. Using these static measures may underestimate their effects on mortality and in explaining race disparities in mortality [55]. Examining how dynamic individual SES and neighborhood SDI measures affect mortality over time for older adults is an interesting and challenging research direction for the future.

Third, we limited our analysis to the cohort of older adults aged 65 and above. It may be interesting to compare the results with other age cutoffs and cohorts. Cohort is an important concept in life course theory [56], which expects that people born at a particular time (cohort effect) may experience similar life events that will affect their life path, SES, health, and mortality [57]. For example, the type and degree of racial discrimination experienced may differ by cohort, affecting race differences in the accumulation of individual-level SES and neighborhood segregation. Testing cohort differences in future research could enrich theory and evidence about how multilevel SES contributes to race difference in mortality.

5. Conclusion

Our study extends previous research and contributes to the literature in two major ways. First, we use appropriate statistical methods to estimate the association between multilevel socioeconomic status (at individual and neighborhood levels) and mortality and confirm that older adults living in a disadvantaged neighborhood context experience higher risk of dying at earlier old ages, beyond the impact of individual SES. Second, we demonstrate that there are race crossover effects in mortality at later old age, with black older adults having a mortality advantage at later old age. Moreover, neighborhood SDI helps explain race differences in mortality at older ages. Finally, race differences in mortality did not disappear even after controlling for both individual SES and neighborhood SDI measures once we modeled the racial crossover effect on mortality.

These results help resolve debates in previous studies and help us better understand the association among race, individual SES, and neighborhood context. The socioeconomic contexts that affect black and white Americans into old age affect their mortality risk, leading to selective survival among the most robust black older adults at later old ages. Addressing the individual and neighborhood socioeconomic disadvantage of black people over the life course is necessary to reduce mortality disparities that culminate in early older adulthood.

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References

[1] R. A. Hummer, M. R. Benjamins, and R. G. Rogers, "Racial and ethnic disparities in health and mortality amony the U.S. elderly population," in *Critical Perspectives on Racial and Ethnic Differences in Health in Late Life*, N. B. Anderson, R. A. Bulatao, B. Cohen, and National Research Council (U.S.), Eds., pp. 53–94, National Academies Press, Washington, D.C, USA, 2004.

[2] D. R. Williams and P. B. Jackson, "Social sources of racial disparities in health," *Health Affairs*, vol. 24, no. 2, pp. 325–334, 2005.

[3] L. Berkman, B. Singer, and K. Manton, "Black/white differences in health status and mortality among the elderly," *Demography*, vol. 26, no. 4, pp. 661–678, 1989.

[4] R. A. Hummer, "Black-white differences in health and mortality: a review and conceptual model," *Sociological Quarterly*, vol. 37, no. 1, pp. 105–125, 1996.

[5] J. J. Sudano and D. W. Baker, "Explaining US racial/ethnic disparities in health declines and mortality in late middle age: the roles of socioeconomic status, health behaviors, and health insurance," *Social Science and Medicine*, vol. 62, no. 4, pp. 909–922, 2006.

[6] X. L. Du, S. Fang, S. W. Vernon et al., "Racial disparities and socioeconomic status in association with survival in a large population-based cohort of elderly patients with colon cancer," *Cancer*, vol. 110, no. 3, pp. 660–669, 2007.

[7] L. Franzini, A. Williams, J. Franklin, S. Singletary, and R. Theriault, "Effects of race and socioeconomic status on survival of 1,332 black, hispanic, and white women with breast cancer," *Annals of Surgical Oncology*, vol. 4, no. 2, pp. 111–118, 1997.

[8] P. Sorlie, E. Rogot, R. Anderson, N. J. Johnson, and E. Backlund, "Black-white mortality differences by family income," *The Lancet*, vol. 340, no. 8815, pp. 346–350, 1992.

[9] K. A. Cagney, C. R. Browning, and M. Wen, "Racial disparities in self-rated health at older ages: what difference does the neighborhood make?" *The Journals of Gerontology*, vol. 60, no. 4, pp. S181–S190, 2005.

[10] S. A. Robert and E. Ruel, "Racial segregation and health disparities between black and white older adults," *The Journals of Gerontology*, vol. 61, no. 4, pp. S203–S211, 2006.

[11] Y. Li and S. A. Robert, "The contributions of race, individual socioeconomic status, and neighborhood socioeconomic context on the self-rated health trajectories and mortality of older adults," *Research on Aging*, vol. 30, no. 2, pp. 251–273, 2008.

[12] F. B. LeClere, R. G. Rogers, and K. Peters, "Neighborhood Social Context and Racial Differences in Women's Heart Disease Mortality," *Journal of Health and Social Behavior*, vol. 39, no. 2, pp. 91–107, 1998.

[13] M. A. Winkleby and C. Cubbin, "Influence of individual and neighbourhood socioeconomic status on mortality among black, Mexican-American, and white women and men in the United States," *Journal of Epidemiology and Community Health*, vol. 57, no. 6, pp. 444–452, 2003.

[14] A. T. Geronimus, "To mitigate, resist, or undo: addressing structural influences on the health of urban populations," *American Journal of Public Health*, vol. 90, no. 6, pp. 867–872, 2000.

[15] R. J. Sampson, S. W. Raudenbush, and F. Earls, "Neighborhoods and violent crime: a multilevel study of collective efficacy," *Science*, vol. 277, no. 5328, pp. 918–924, 1997.

[16] R. J. Sampson, J. D. Morenoff, and T. Gannon-Rowley, "Assessing neighborhood effects: social processes and new directions in research," *Annual Review of Sociology*, vol. 28, pp. 443–478, 2002.

[17] B. Chaix, L. Maria, L. John, and M. Juan, "Disentangling contextual effects on cause-specific mortality in a longitudinal 23-year follow-up study: impact of population density or socioeconomic environment?" *International Journal of Epidemiology*, vol. 35, no. 3, pp. 633–643, 2006.

[18] B. Chaix, M. Rosvall, and J. Merlo, "Assessment of the magnitude of geographical variations and socioeconomic contextual effects on ischaemic heart disease mortality: a multilevel survival analysis of a large Swedish cohort," *Journal of Epidemiology and Community Health*, vol. 61, no. 4, pp. 349–355, 2007.

[19] B. Chaix, M. Rosvall, and J. Merlo, "Recent increase of neighborhood socioeconomic effects on ischemic heart disease mortality: a multilevel survival analysis of two large Swedish cohorts," *American Journal of Epidemiology*, vol. 165, no. 1, pp. 22–26, 2007.

[20] M. Wen and N. A. Christakis, "Neighborhood effects on posthospitalization mortality: a population-based cohort study of the elderly in Chicago," *Health Services Research*, vol. 40, no. 4, pp. 1108–1127, 2005.

[21] R. T. Anderson, P. Sorlie, E. Backlund, N. Johnson, and G. A. Kaplan, "Mortality effects of community socioeconomic status," *Epidemiology*, vol. 8, no. 1, pp. 42–47, 1997.

[22] R. G. Wight, J. R. Cummings, A. S. Karlamangla, and C. S. Aneshensel, "Urban neighborhood context and mortality in late life," *Journal of Aging and Health*, vol. 22, no. 2, pp. 197–218, 2010.

[23] F. B. LeClere, R. G. Rogers, and K. D. Peters, "Ethnicity and Mortality in the United States: individual and Community Correlates," *Social Forces*, vol. 76, no. 1, pp. 169–198, 1997.

[24] S. A. Bond Huie, R. A. Hummer, and R. G. Rogers, "Individual and contextual risks of death among race and ethnic groups in the United States," *Journal of Health and Social Behavior*, vol. 43, no. 3, pp. 359–381, 2002.

[25] M. C. Corti, J. M. Guralnik, L. Ferrucci et al., "Evidence for a Black-White crossover in all-cause and coronary heart disease mortality in an older population: the North Carolina EPESE," *American Journal of Public Health*, vol. 89, no. 3, pp. 308–314, 1999.

[26] N. E. Johnson, "The racial crossover in comorbidity, disability, and mortality," *Demography*, vol. 37, no. 3, pp. 267–283, 2000.

[27] S. M. Lynch, J. S. Brown, and K. G. Harmsen, "Black-white differences in mortality compression and deceleration and the mortality crossover reconsidered," *Research on Aging*, vol. 25, no. 5, pp. 456–483, 2003.

[28] E. J. Nan, "The racial crossover in comorbidity, disability, and mortality," *Demography*, vol. 37, no. 3, pp. 267–283, 2000.

[29] K. G. Manton, S. S. Poss, and S. Wing, "The black/white mortality crossover: investigation from the perspective of the components of aging," *The Gerontologist*, vol. 19, no. 3, pp. 291–300, 1979.

[30] S. Wing, K. G. Manton, E. Stallard, C. G. Hames, and H. A. Tryoler, "The black/white mortality crossover: investigation in a community-based study," *The Journals of Gerontology*, vol. 40, no. 1, pp. 78–84, 1985.

[31] A. M. O'Rand and J. Hamil-Luker, "Processes of cumulative adversity: childhood disadvantage and increased risk of heart attack across the life course," *The Journals of Gerontology*, vol. 60, pp. 117–124, 2005.

[32] K. F. Ferraro and M. M. Farmer, "Double jeopardy to health hypothesis for african Americans: analysis and critique," *Journal of Health and Social Behavior*, vol. 37, no. 1, pp. 27–43, 1996.

[33] J. S. House, P. M. Lantz, and P. Herd, "Continuity and change in the social stratification of aging and health over the life course: evidence from a nationally representative longitudinal study from 1986 to 2001/2002 (Americans' Changing Lives Study)," *The Journals of Gerontology*, vol. 60, pp. 15–26, 2005.

[34] M. C. Corti, J. M. Guralnik, L. Ferrucci et al., "Evidence for a black-white crossover in all-cause and coronary heart disease mortality in an older population: the North Carolina EPESE,"

American Journal of Public Health, vol. 89, no. 3, pp. 308–314, 1999.

[35] I. T. Elo and S. H. Preston, "Racial and ethinic differences in mortality at older ages," in *Racial and Ethnic Differences in the Health of Oder Americans*, L. G. Martin and B. J. Soldo, Eds., pp. 10–40, National Academies Press, Washington, D.C, USA, 2004.

[36] L. Xiang, K. K. W. Yau, S. K. Tse, and A. H. Lee, "Influence diagnostics for random effect survival models: application to a recurrent infection study for kidney patients on portable dialysis," *Computational Statistics & Data Analysis*, vol. 51, no. 12, pp. 5977–5993, 2007.

[37] D. A. Freedman, "On the so-called "Huber Sandwich Estimator" and "robust standard errors"," *American Statistician*, vol. 60, no. 4, pp. 299–302, 2006.

[38] P. J. Kelly, "A review of software packages for analyzing correlated survival data," *American Statistician*, vol. 58, no. 4, pp. 337–342, 2004.

[39] H. Bosma, H. Dike van de Mheen, G. J. J. M. Borsboom, and J. P. Mackenbach, "Neighborhood socioeconomic status and all-cause mortality," *American Journal of Epidemiology*, vol. 153, no. 4, pp. 363–371, 2001.

[40] L. L. Roos, J. Magoon, S. Gupta, D. Chateau, and P. J. Veugelers, "Socioeconomic determinants of mortality in two Canadian provinces: multilevel modelling and neighborhood context," *Social Science & Medicine*, vol. 59, no. 7, pp. 1435–1447, 2004.

[41] O. Dejardin, L. Remontet, A. M. Bouvier et al., "Socioeconomic and geographic determinants of survival of patients with digestive cancer in France," *British Journal of Cancer*, vol. 95, no. 7, pp. 944–949, 2006.

[42] C. Marinacci, T. Spadea, A. Biggeri, M. Demaria, A. Caiazzo, and G. Costa, "The role of individual and contextual socioeconomic circumstances on mortality: analysis of time variations in a city of north west Italy," *Journal of Epidemiology and Community Health*, vol. 58, no. 3, pp. 199–207, 2004.

[43] P. M. Krueger, S. A. Bond Huie, R. G. Rogers, and R. A. Hummer, "Neighbourhoods and homicide mortality: an analysis of race/ethnic differences," *Journal of Epidemiology and Community Health*, vol. 58, no. 3, pp. 223–230, 2004.

[44] M. E. Dupre, A. T. Franzese, and E. A. Parrado, "Religious attendance and mortality: implications for the black-white mortality crossover," *Demography*, vol. 43, no. 1, pp. 141–164, 2006.

[45] S. H. Preston, I. T. Elo, I. Rosenwaike, and M. Hill, "African-American mortality at older ages: results of a matching study," *Demography*, vol. 33, no. 2, pp. 193–209, 1996.

[46] J. P. Klein and M. L. Moeschberger, *Survival Analysis: Techniques for Censored and Truncated Data*, Springer, New York, NY, USA, 2nd edition, 2003.

[47] P. D. Allison, *Survival Analysis Using the SAS System: A Practical Guide*, SAS Institute, Cary, NC, USA, 1995.

[48] V. S. Pankratz, M. de Andrade, and T. M. Therneau, "Random-effects cox proportional hazards model: general variance components methods for time-to-event data," *Genetic Epidemiology*, vol. 28, no. 2, pp. 97–109, 2005.

[49] V. S. Pankratz, M. De Andrade, and T. M. Therneau, "Random-effects cox proportional hazards model: general variance components methods for time-to-event data," *Genetic Epidemiology*, vol. 28, no. 2, pp. 97–109, 2005.

[50] F. X. Florin Vaida, "Proportional hazards model with random effects," *Statistics in Medicine*, vol. 19, no. 24, pp. 3309–3324, 2000.

[51] R. H. Xu, "Proportional hazards mixed models: a review with applications to twin models," *Methodoloski Zvezki*, vol. 1, pp. 205–212, 2004.

[52] S. Rabe-Hesketh, S. Yang, and A. Pickles, "Multilevel models for censored and latent responses," *Statistical Methods in Medical Research*, vol. 10, no. 6, pp. 409–427, 2001.

[53] T. M. Therneau and P. M. Grambsch, Penalized Cox Modles and Frailty, 1998.

[54] T. M. Therneau and P. M. Grambsch, *Modeling Survival Data : Extending the Cox Model*, Springer, New York, NY, USA, 2000.

[55] P. McDonough and P. Berglund, "Histories of poverty and self-rated health trajectories," *Journal of Health and Social Behavior*, vol. 44, no. 2, pp. 198–214, 2003.

[56] G. H. Elder, M. K. Johnson, and R. Crosnoe, "The emergence and development of the life course theory," in *Handbook of the Life Course*, J. T. Mortimer and M. J. Shanahan, Eds., Plenum, New York, NY, USA, 2003.

[57] T. R. Holford, "Understanding the effects of age, period, and cohort on incidence and mortality rates," *Annual Review of Public Health*, vol. 12, pp. 425–457, 1991.

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Research Article

Comparing the Support-Efficacy Model among Centenarians Living in Private Homes, Assisted Living Facilities, and Nursing Homes

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We investigated the influence of social relations on health outcomes in very late life by examining the support-efficacy convoy model among older adults who resided in three different residential environments (centenarians in private homes, $n = 126$; centenarians in assisted living facilities, $n = 55$; centenarians in nursing homes, $n = 105$). For each group, path analytic models were employed to test our hypotheses; analyses controlled for sex, mental status, education, perceived economic sufficiency, and activities of daily living. The hypothesized relationships among the models' variables were unique to each of the three groups; three different models fit the data depending upon residential environment. The direct and indirect effects of social relations assessments were positive for the mental and physical health of very old adults, suggesting that participants welcomed the support. However, residential status moderated the associations between the assessments of social relations, self-efficacy, and both outcomes, physical and mental health.

1. Introduction

For older adults, the important relationship between social resources and physical and mental health outcomes is well established [1–5]. However, as the proportion of oldest-old adults in the USA increases, particularly centenarians, and more research attention is given to their study [6, 7], theoretically driven investigations of resources necessary for adaptation to changes associated with advanced age are required [8, 9]. Levitt [10] reviewed work focusing on social development across the life span and recommended the social convoy model as a general or unifying model. In addition, Seeman and Crimmins [11] conducted a review focusing on the effects of the social environment on health and aging from the perspective of both epidemiology and demography. Based on their extensive literature review, including the early work of Antonucci and Jackson [12], they posited a biopsychosocial model of health and aging that included the influence of structural and functional assessments of social relationships on physical and mental health outcomes through psychological characteristics such as self-efficacy.

Recently, Antonucci et al. outlined suggested modifications of the original convoy model of social support based on two decades of empirical and theoretical studies (see Figure 1; [13]). Their social support-efficacy model posits a specific mediator, self-efficacy, through which social relations influence health outcomes. In addition, Antonucci et al., as well as Seeman and Crimmins [11], emphasized that situational or contextual experiences are considered important influences on the association among social relations, self-efficacy, and health outcomes for older adults [13].

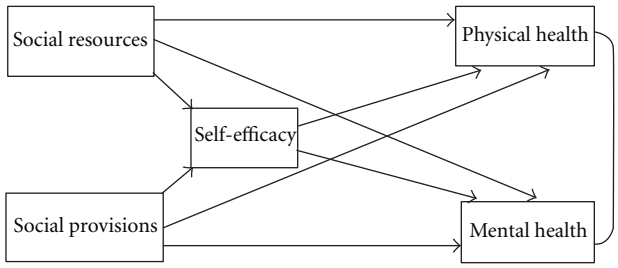

Figure 1: The study's conceptual model was based on the theoretical work of Antonucci et al. [13].

For very old adults, residential setting (e.g., private home, assisted living facility, or nursing home), one assessment of contextual experience, is very salient to the constructs of the support-efficacy model [14–16]. Long-term care is often needed by older adults. It is estimated that by 2050, the total number of individuals subscribing to paid long-term care, whether at home or in some type of residential care, will likely double from 13 million in the year 2000 to 27 million [17]. According to a 2009 report by The National Center for Assisted Living, more than 900,000 adults, whose average age was 86.9 years, resided in assisted living facilities [18]. Regarding nursing home care, researchers found that the number of individuals receiving such care, on any given day, rose from 1.28 million in 1977 to 1.63 million by 1999, a 27% increase [19]. Bowling and Grundy reviewed literature focused on the association between older adults' social resources and mortality in population-based longitudinal studies [20]. They found substantial evidence for the relationship between social support, social network structure, health status, mortality, and likelihood of entry into institutional care.

Regarding the moderating role of residential status, empirical research has demonstrated that measures of social relations, including structural (i.e., social networks) and functional (i.e., social provisions) assessments, differ across residence status [16, 20]. Thus, the current study's purpose was to specify and test a version of Antonucci and colleagues' [13] mediating model (see Figure 1) using cross-sectional data from participants in the Georgia Centenarian Study [21] who resided in three distinct living environments: private homes, assisted living facilities, and nursing homes.

Close social relationships received much attention in the literature over the past decades both from a theoretical [13, 22, 23] and empirical [3, 24–26] perspective resulting in strong evidence that older adults with high levels of social resources enjoy better health (physical and mental) than those lacking in close relationships. Convoy, according to Antonucci et al., depicts the close relationships that surround an individual and promote positive development [13]. Social relations, a term often used to define and identify convoy relationships [24], may be operationalized to encompass social networks (a structural assessment) and social support (a functional assessment) [27]. Structural assessments of convoy members include measures of network size, member type (e.g., friend or family member), network members' geographical propinquity, and frequency and type of contact with network members. Functional assessments address the question of what *type of support* network members provide [28], such as instrumental or emotional support. Krause [29] reviewed over a dozen different types of support from the literature. The current study included both structural and functional assessments of social relations in the tested model.

The literature calls for investigations of mediating mechanisms through which social relationships influence health outcomes, in particular assessments of self-efficacy, human agency, mastery, competence, and control [13, 30]. Conceptually, perceived control over one's life events and conditions is closely related to various measures of competence such as internal locus of control, mastery, and self-efficacy [2, 24, 31]. According to Ross and Sastry [32], perceived control may tap the same underlying construct as self-efficacy. Over time, contact with significant members of one's social network and the different types of support provided may increase an individual's sense of worth and ability to influence the situational environment. This study featured a measure of self-efficacy, competence as measured by the NEO PI-R [33, 34], as the mediating mechanism through which social relations influenced health outcomes. The competence facet, from conscientiousness, addresses how capable or effective an individual feels.

The current study extended the literature by investigating the support-efficacy model with very old adults (i.e., centenarians) and by examining the moderating influence of living environment (e.g., centenarians in private homes, in assisted living facilities, and in nursing homes). Bowling and Grundy challenged researchers to consider influential variables that need to be controlled and intervening variables that might explain such associations [20]. Our analyses controlled for sex, education, perceived economic sufficiency, and instrumental activities of daily living in our analyses [35, 36]. Based on the literature describing the various reasons for residing in a private home, an assisted living facility, and a nursing home (e.g., loss of spouse; decrease in functional, physical, or mental capacity), we hypothesized that the model would fit the data uniquely for each subsample because the living environments and the contextual situations of the participants influence their physical and mental health differentially [37–40]. How the relationships between the models' variables differ by residence status was a research question we investigated.

2. Method

2.1. Participants and Procedures. Data for this study came from a Georgia population-based sample of centenarians and octogenarians collected between 2002 and 2005. The sample, comprised of adults aged 98 years and older, was based on a census from a 44-county northern Georgia region including all skilled nursing and personal care facilities. In addition, registered voter lists and corroborating birth date information was also used to identify participants in that region. A refusal to participate rate was not possible to estimate due to the complexities of the sampling strategy. Of the estimated 1244 eligible centenarians in the population, 19.2% participated, whereas 135 who declined to participate

were found to be age eligible. Thus, an effective response rate has been estimated at 63.9% and nonresponders were very similar in terms of certain demographics (age, gender, and race) to final participants. Also, no substantial differences in cognitive status were expected as the sampling strategy included sufficient participants from institutional settings. The Georgia Centenarian Study, Phase 3 investigated factors related to survival and functioning of centenarians. Further details on the study's sampling, data collection, and design are provided by Poon and colleagues [21].

Thus, 158 cognitively intact, community-dwelling, or institutionalized near-centenarians and centenarians (98 years and older; average age was 99.82 years; range was from 98 to 109) were included in this study. Of the participants, 78.5% were female, 85% were White and 15% Black, and 85% were widowed, whereas only 6% were married.

2.2. Measures

2.2.1. Control Variables. Analyses controlled for participants' sex, mental status, activities of daily living, education, and perceived economic status. For sex, males were coded "0" and females "1." The Mini-Mental Status Examination (MMSE, [41]) was used to control for cognitive ability (mental status); higher scores indicated greater ability. Two commonly used subscales, instrumental activities of daily living (seven items), and physical activities of daily living (six items) were combined to create the self-care capacity assessment, activities of daily living (ADLs, [42]). An example of a question asked included "Can you do your housework?" and was scaled so that 2 = without help (can clean floors, etc.), 1 = with some help (can prepare some things but unable to cook full meals yourself), or 0 = are you completely unable to prepare any meals? Cronbach's alpha for this measure was .88, and it was scaled so that higher scores indicated higher levels of self-care capacity. A single question from the OARS [42] "How well does the amount of money you have take care of your needs?" was used to assess the participant's financial situation (economic sufficiency). This was scaled from 1 (*poorly*) to 3 (*very well*).

2.2.2. Dependent Variables. Research has validated a single-item measure of self-rated physical health as a summary assessment of overall health status, predictive of outcomes such as mortality, BMI, physical activity, and hospitalization among others [43–45]. DeSalvo and colleagues [46] compared the predictive accuracy of a single-item measure of general health with multi-item scales (e.g., mental component summary and physical component summary). They found the single item performed as well as the multi-item measures regarding validity and reliability, in addition to saving time and money over the use of longer instruments. We used an item from the OARS [42] physical health section asking "How would you rate your overall health at the present time?" Responses ranged from 0 (*poor*) to 3 (*excellent*). Similar to physical health, numerous studies have employed a global self-rating of mental health [47, 48]. We assessed self-reported mental health with an item from the OARS [42] asking "How would you rate your mental or emotional health at the present time?" Responses ranged from 0 (*poor*) to 3 (*excellent*).

2.2.3. Predictor Variables. Two commonly used measures for assessing social relations among older adults were included in this study: The Social Provisions Scale (SPS, [49]) and social resources [42]. We employed a 12-item short form of the SPS, a functional assessment that asked questions such as "I have close relationships that provide me with a sense of emotional security and well-being" and "There is no one I can turn to for guidance." Items were scaled from 1 (*strongly disagree)* to 4 (*strongly agree*); Cronbach's alpha for the scale was .75. Higher scores reflected higher levels of social provisions. Social resources, a structural assessment of social relationships, was measured using one question from the OARS [42]. In particular, this question asked "How many times during the past week did you spend some time with someone who does not live with you; that is you went to see them or they came to visit you, or you went out to do things together?" Responses were coded 0 = not at all 1 = once 2 = 2–6 times and 3 = once a day or more. Based upon work previously conducted [16] with this measure and very old adults (i.e., centenarians), we selected a question tapping frequency of network contact because (a) physical limitations (e.g., hearing loss) common with very old adults [50, 51] often limit phone or other communication-only contacts and (b) reduction in network size due to mortality—by definition centenarians have outlived peers, spouses, and often children—limits the numbers of individuals in their network. In addition, due to constraints composed by socioemotional selectivity [23], we did not use questions from the social resources section of the OARS asking about the number of phone conversations or number of network members participants knew well enough to visit as very old adults have likely reduced the number of network contacts to the few most salient. Thus, we selected the question above to tap the *amount or frequency* of contact the participants had with network members in the past week to comprise our structural measure of social relations.

2.2.4. Mediating Variable. The NEO PI-R [34] is a widely used measure that captures participants' impressions of their own personality along the Big Five personality dimensions. Self-efficacy includes an individual's belief in his/her own competence to successfully perform a particular action [31]. In the present study, the competence facet from conscientiousness was used because it taps the degree to which a respondent feels capable and effective. Feeling well prepared to face life and its changes is typical of those who score high on this facet, and the facet is often highly correlated with self-esteem and internal locus of control [34]. Seven items comprised this facet and were scaled from 0 (*disagree)* to 2 (*agree*); Cronbach's alpha for this facet was .70.

Descriptive statistics for each of the study's variables, by residential status, and one-way ANOVA's comparing the means across residential status, are provided in Table 1.

2.3. Data Analytic Procedure. Because previous investigations [16] revealed significant differences in social resources

Table 1: Descriptives and ANOVA results for study variables.

Variables	Private homes M (SD)	Assisted living facilities M (SD)	Nursing homes M (SD)	One-way ANOVA
(1) Age	99.76 (1.71)	100 (2.00)	101 (2.10)	
(2) Sex	76.2% female	86% female	87.6% female	
(3) Mental status (MMSE)	20.43 (7.09)	19.44 (7.87)	11.37 (8.24)	* * *
(4) Years of education	11.65 (3.87)	13 (3.05)	11.15 (3.59)	
(5) Economic sufficiency	2.56 (.63)	2.67 (.48)	2.73 (.47)	
(6) ADLs	37.11 (7.43)	37.42 (7.03)	33.98 (8.88)	**
(7) Physical health	1.87 (.76)	1.85 (.62)	1.82 (.91)	
(8) Mental heath	2.02 (.62)	1.94 (.57)	1.73 (.76)	*
(9) Self-efficacy	5.66 (2.09)	5.64 (2.63)	5.50 (1.98)	
(10) Social resources	1.81 (.78)	1.83 (.75)	1.96 (.84)	
(11) Social provisions	35.19 (2.15)	35.24 (2.55)	34.39 (2.73)	

* Private homes differed from nursing homes, $P < .05$;
** Private homes and assisted living facilities differed from nursing homes, $P < .05$;
*** Each was significantly different from the other, $P < .05$.

and social provisions between octogenarians and centenarians living in private homes and among centenarians residing in private homes, assisted living facilities, and nursing homes, we conducted our analyses by subgroup. Based upon Antonucci and colleagues' model [13], we tested a path analytic model for each subgroup. We used path analysis for a number of reasons. First, because of our limited sample size, we employed manifest rather than latent variables to reduce the number of parameters estimated in our models. Second, if necessary, we also wanted to test for model equivalency across our subgroups. Third, our hypotheses called for tests of directionality with self-efficacy as a mediating mechanism. Working with very old adults presents numerous challenges, one of which is locating and assessing participants who often present with numerous hearing, visual, and other medical conditions, in addition to mortality concerns over time [7], resulting in smaller sample sizes. The literature on sample size and covariance structure modeling has addressed the concern of sample size and the number of parameters estimated [52–54]. Jackson concluded that when small sample sizes (he compared sizes of 50, 100, 200, 400, and 800) are encountered, assessments of model fit such as chi-square goodness-of-fit and RMSEA, for example, are most sensitive to misspecification and therefore, recommended. In addition, Herzog and Boomsma conducted a Monte Carlo study demonstrating that Swain-corrected estimators were robust to small sample sizes and recommended their use for small sample size research [52]. We used their syntax for use with R software for each of our final models presented in the Section 3 below, and no differences in fit were found between what we reported and the Swain-corrected estimations. Thus, a path model allowed us to examine model fit, model equivalency between groups, and indirect effects through the mediating mechanism of self-efficacy.

The literature on multiple-sample testing in structural equation modeling argues for establishing a baseline or best-fitting model for each sample *first* [55]. Kline extends the point and argues that unless an unconstrained model fits well across samples, it makes little sense to test additional constraints [56, page 295]. We first specified and tested the hypothesized support-efficacy model, determining the best model fit for each subgroup (a series of nested model tests examining change in chi-square for each nested model). Analyses were conducted with *Mplus* Version 5.0 [57] using full-information maximum likelihood (FIML) to handle missing data; overall model fit was assessed by employing the Satorra-Bentler chi-square test statistic that is robust to nonnormality of measures, referred to as the MLR χ^2 in *Mplus*. Model evaluation was based on the chi-square goodness of fit test and other fit indices: the comparative fit index—CFI [58], root mean squared error of approximation (RMSEA, [59]), and the standardized root mean squared residual (SRMR). Values close to .95 for CFI, .08 for SRMR, and .06 for RMSEA suggest that good fit between the observed data and the hypothesized model exists [60, 61]. Nested model testing was conducted according to the procedure outlined in *Mplus* [57]. Control variables included sex, mental status, perceived economic sufficiency, and activities of daily living.

3. Results

3.1. Centenarians in Private Homes. A base model with no degrees of freedom (all possible paths were estimated) was tested. The regression of mental health on social resources was not significant, ($\beta = .13$; $P > .05$), neither was the regression of mental health on self-efficacy ($\beta = .05$; $P > .05$). These paths were deleted, and the nested model fit the data well: MLR χ^2 (2, $N = 126$) = 2.36, $P = .31$, CFI = .99; RMSEA = .04; SRMR = .02. In this model, all specified paths were significant (one-tailed tests); however physical health regressed on social provisions ($\beta = .20$; $P > .05$) was the weakest path. Thus, for the sake of parsimony, we specified a model without this path: MLR χ^2 (3, $N = 126$) = 5.14, $P = .16$, CFI =.97; RMSEA =.08; SRMR =.03. We conducted a nested model test according to L. K. Muthén and B. O.

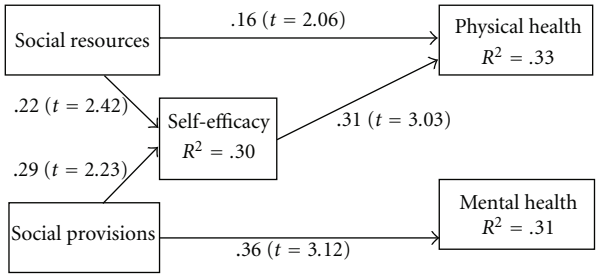

Figure 2: Test results for the support-efficacy model with centenarians residing in private homes; model fit: Satorra-Bentler χ^2 (N = 126; df = 3) =5.14; P = .16; CFI = .97; RMSEA = .08; SRMR = .03*(using F1ML)*. Nonsignificant paths deleted; parameter estimates are from the standardized solution. Test of Indirect Effect for Social Resources on Physical Health (.07; t = 1.93; P = .053; two-tailed). Test of Indirect Effect for Social Provisions on Physical Health (.09; t = 1.66; P = .097; two-tailed). Endogenous variables were controlled for sex, mental status, ADLs, education, and economic sufficiency.

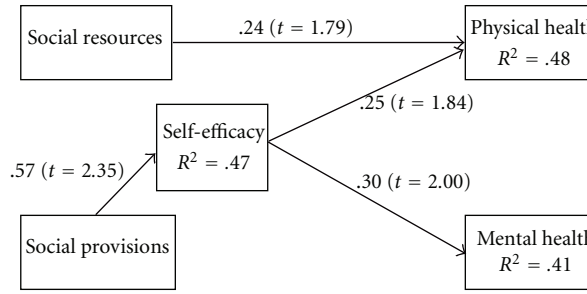

Figure 3: Test results for the support-efficacy model with centenarians residing in assisted living facilities; model fit: Satorra-Bentler χ^2 (N = 55; df = 4) =5.29; P = .26; CFI = .97; RMSEA = .08; SRMR = .04*(using F1ML)*. Parameter estimates are from the standardized solution; endogenous variables were controlled for sex, mental status, ADLs, education, and economic sufficiency.

Muthén [57]; this model's fit was not significantly different from the model with the path regressing physical health on social provisions: $\Delta\chi^2$ (1 df) = 2.50, P = .11.

The final model (see Figure 2) depicts the most parsimonious, best fitting model to our data following the support-efficacy conceptualization for centenarians residing in private dwellings. Direct effects for social resources on physical health (β = .16; P < .05) and social provisions on mental health (β = .36; P < .05) were found in the previous model, whereas in this best-fitting model for centenarians in private homes, no direct effects for social resources on mental health or social provisions on physical health were found. However, consistent with Antonucci and colleagues [13], indirect effects operating through self-efficacy were found both for social resources (.07; P = .053; two-tailed test) and social provisions on physical health (.09; P = .097; two-tailed test), whereas no indirect effects were found for either predictor on the outcome mental health.

3.2. Centenarians in Assisted Living Facilities. In the base model, significant predictors of physical health and self-efficacy were found; no predictors were found for mental health. Physical health was predicted by social resources (β = .34; $P \leq$ .003) and self-efficacy by social provisions (β = .58; $P \leq$ .02). Based on these results we tested a model deleting the path of social provisions predicting physical health, social resources predicting self-efficacy, and the predictors of mental health except controls. This model fit the data adequately: MLR χ^2 (5, N = 55) = 8.93; P = .11; CFI = .91; RMSEA = .12; SRMR = .06. However, in addition to the higher value for RMSEA, one of the modification indices looked promising for the regression of mental health on self-efficacy, so we tested a model including this path. This model fit the data well: MLR χ2 (4, N = 55) = 5.29, P = .26, CFI = .97; RMSEA = .08; SRMR = .04. The nested model chi-square test was significant ($\Delta\chi^2$ = 6.90, 1 df, P = .01). We selected the latter model over the former because the Chi-square and the CFI, RMSEA, and SRMR indices suggested that it was the better fitting model than the nested model with more degrees of freedom (see Figure 3). In addition, we conducted the tests of indirect effects from social provisions to both physical and mental health. The indirect effect on physical health was not significant (P = .18), whereas the indirect effect from social provisions on mental health through self-efficacy reached significance for a hypothesized effect (P = .05; one-tailed test).

3.3. Centenarians in Nursing Homes. The base model used in previous analyses could not be tested because the covariance coverage fell below 10 percent (i.e., all variables and pairs of variables have data for at least ten percent of the sample). Inspection of our individual variables led us to delete our measure for economic sufficiency. In addition, when this model was run, difficulties were encountered estimating appropriate standard errors (nonpositive definite matrix) based on the control variable sex. Of the 105 centenarians living in nursing homes, 92 were female and 13 male. Thus, further models for centenarians in nursing homes excluded economic sufficiency and the control variable, sex. No further estimation difficulties were encountered.

In the tested base model, neither exogenous predictor, social resources (β = .20; P > .05) nor social provisions (β = −.13; P > .05) significantly predicted self-efficacy. However, both self-efficacy (β = .60; P = .01) and social provisions (β = −1.09; P = .008) significantly predicted mental health, whereas only social provisions approached statistical significance predicting physical health (β = .77; P = .14). Regarding the magnitude of the standardized beta for social provisions, Jöreskog [62] noted that it is possible for a standardized coefficient to be greater than one (e.g., 1.04, 1.40, or 2.08) and that it does not necessarily imply error in the model. However, he did point out that such a finding likely points to multicollinearity in the data. Our measure of ADLs was negatively and highly correlated with social provisions (r = −.85) in this model. Thus, we deleted ADLs from the analysis and found that indeed social provisions significantly predicted physical health (β = .43; P = .02), neither measure of social resources predicted self-efficacy, and only self-efficacy predicted mental health

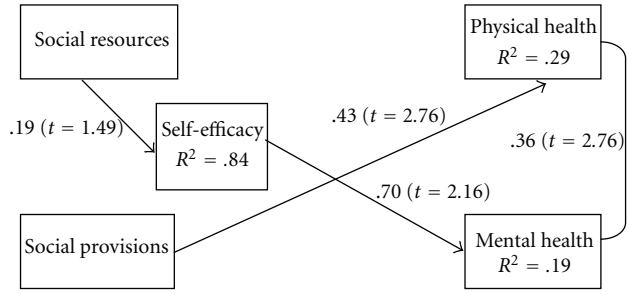

Figure 4: Test results for the support-efficacy model with centenarians residing in nursing homes; model fit: Satorra-Bentler χ^2 (N = 105; df = 5) = 6.33; P = .28; CFI = .94; RMSEA = .05; SRMR = .06 (*using FIML*). Parameter estimates are from the standardized solution; endogenous variables were controlled for mental status and education.

(β = .70; P = .01). In this model, R^2 for physical health was .29; for mental health it was .19, and for self-efficacy it was .84 (see Figure 4). In addition, both mental status and education were significant predictors of self-efficacy. Thus, as a supplemental analysis, we computed a model specifying the indirect effect from these control variables (mental status and education) to mental health through self-efficacy. The standardized indirect effect was .49 (P = .02).

4. Discussion

This study tested the support-efficacy model [13] for each of three centenarian groups participating in the Georgia Centenarian Study [21]: centenarians residing in private homes, centenarians residing in assisted living facilities, and centenarians residing in nursing homes. A focus of the model is the hypothesized indirect effect of social relations on health outcomes of older adults through the mediating mechanism of self-efficacy. Overall, the study's results supported the hypothesized model. The mediator, self-efficacy, completely mediated the influence of social relationships in two of the three models tested on self-report data from centenarians (i.e., those residing in private homes and assisted living facilities). In particular, how the model worked varied by residential status (i.e., private home, assisted living facility, or nursing home); the relationship among the variables for the best fitting model was different for each residential status.

Three findings, consistent across the three models, merit discussion. First, our results supported the hypothesized *positive* influence of social relationships on the physical and mental health of centenarians. These findings are consistent with previous research across the life span [20, 63, 64]. However, ambivalent findings about the valence of social support on psychological outcomes have been reported. For example, perceived support is often beneficial whereas actual received support may be detrimental [65, 66]. We believe our study's positive findings for the influence of social relations on health outcomes for centenarians, regardless of living environment, are best explained by considering the qualitative work of Chen et al. [37]. They developed a grounded theory of elders' decisions to enter assisted living facilities, including the weighing and balancing of gains and losses "to go where the help is" (page 92)—the anticipated outcome of moving. When losses outweighed gains, they went to the help; they moved to a facility. Such a move often includes cognitive, affective, and physical stressors. Often, a move is perceived as loss of independence and is not preferred to the private home, but is preferred relative to entering a nursing home [38, 40, 67]. These decisions and associated stressors also apply when moving from an assisted living facility to a nursing home. We believe that it is possible that centenarians in our study understood the salubrious role social relations provide (whether perceived or received; see [27]) for their independence and ability to live in a private home setting; such support either delays the need to move toward help (maintaining a level of independence for those in private homes) or for those no longer able to remain in a private home, such support could be effective in the transition from private home to assisted living facility. Thus, the direct and indirect effects of social resources (frequency of contact) and social provisions (types of support provided) were positively (i.e., they were welcomed supports) associated with the mental and physical health of our very old participants.

A second finding, for centenarians in private homes and for centenarians in assisted living facilities, consistent with the model and previous research differentiating between the effects of social network and social support variables, is the strong influence of social provisions, compared to the influence of social resources, primarily on outcomes of well being [36, 68]. Our assessment of social relations tapped the frequency of contact between participants and their network of friends and family, whereas Cutrona and Russell's Social Provisions Scale [49] was designed to assess the type of support provided by others. Because social provisions were self-reported by participants, the measure likely included their perceptions of available support [69, 70]; it does not necessarily assess support actually provided. We believe that the Social Provisions Scale, with its breadth of functional supports assessed, tapped the appropriate support needed by our participants for the particular stressor experienced, resulting in a strong relationship between the measure and both physical and mental health.

Consistent with the hypothesized model and the literature regarding mediation through self-efficacy, was a third noteworthy finding: the mediating role of self-efficacy between social relations and the health outcomes for centenarians in private homes and assisted living facilities. Intriguingly, for centenarians in assisted living facilities, the influence of social provisions was *completely* mediated through self-efficacy. The effect on mental health reaches statistical significance for a one-tailed test; the effect on physical health approaches statistical significance for a one-tailed test. Cutrona and Russell [49] provided an explanation about the theoretical underpinnings of their Social Provisions Scale that helps explain this finding. Theoretically, both Bandura [31, 71] and Cobb [72, 73] predicted that social support should lead to an individual's self-assessment of confidence or competency. Such assessments of self-efficacy in turn encourage individuals to attempt difficult tasks, work harder, and persist when facing difficult situations. Thus,

greater levels of self-efficacy could lead to more effective coping and higher levels of physical and mental health. Centenarians in assisted living facilities are surrounded by potential supporters and provisions that are available if needed and called upon [37]. Also, they are not in private homes where they could be isolated or distant from support and they are not in nursing homes where chronic physical and mental health concerns are often experienced. These very old adults, residing in an assisted living facility, may be in the best of situations: they are independent in terms of most if not all activities of daily living and, if needed, social resources in terms of network members' availability and social provisions are close at hand.

Additionally, Holahan et al. [74] proposed and tested a mediation model that posited the indirect effects of social resources on psychological adjustments through personality characteristics such as self-confidence. They found that in situations of high stress the indirect effect model fit the data, whereas in lower stress situations, direct effects were found. It might be that centenarians in our study have been in assisted living facilities for a lengthy period of time and were facing the stressful move to a nursing home. Either explanation of the results fits with the particular effectiveness of the support-efficacy for this group of older adults. In support of this explanation for centenarians in assisted living facilities, the hypothesized mediating model explained the most variance in both outcomes, mental and physical health (41% and 48%, resp.), compared to the models tested for centenarians in other living situations.

Three unique findings of the current study for centenarians in private homes were noted: (a) the influence of social provisions on physical health was *completely* mediated through self-efficacy, whereas it was not for mental health, (b) the influence of social resources on physical health was partially mediated through self-efficacy, and (c) social resources did not influence mental health directly or indirectly; only social provisions directly influenced mental health. Centenarians in private homes are truly expert survivors [75], and while not necessarily healthy as compared to younger adults, they are likely autonomous individuals [76]. The complete mediation of social support or functional helps through self-efficacy hints to the fact that "learned helplessness" has not become the norm for these individuals. Either they are receiving very appropriate support that does not undermine their self-confidence or they perceive support that exists if they need it. Bandura [31] referred to efficacy expectations or beliefs that one can do or perform particular actions, a belief about personal competence [77]. Once again, the literature addressing why older adults transition to assisted living facilities or nursing homes provided insight into the probable reasons for this mediated path. One factor related to relocation is the need for help with activities of daily living and medication management [78]. Perhaps, for this group of centenarians living in a private home and experiencing higher levels of independence than their peers in assisted living facilities or nursing homes, the frequency of contact with social network members (directly and indirectly influencing physical health) and the particular types of support provided by others' assistance enables them to feel competent or capable of accomplishing the tasks necessary to remain in their residential setting, leading to perceptions of better physical health. In addition, these participants could be healthier than those not residing in private homes. Thus, whatever supportive relationships were in place and active helped the centenarians to maintain a level of physical health concomitant with independent living and associated feelings of self-efficacy. As mentioned above, the work of Holohan et al. [74] demonstrated that indirect effects of social support through personality characteristics such as self-efficacy operated primarily under stressful conditions. It may be that centenarians living in private homes will finish their years in a private setting, at least until near the end of life when their physical health begins to precipitously fail. Perhaps private home dwelling is associated with lower stress levels, thus explaining the direct influence of social provisions on their mental health, and why, regarding their physical health, which is necessary for dwelling in a private home, both direct and indirect influences of social relations were operative. In addition, we believe that this line of thinking may be one explanation for the lack of influence on mental health by self-efficacy either directly or as a mediator; the strong, direct influence of social provisions overrode the influence of the participants' personal perspective of their own mastery. Likely, the stress is low in the presence of such supportive help as centenarians living in private dwellings do so with support from others. When that support is perceived to be available or present, it likely contributes more to their mental health than their own perception of self-efficacy.

For centenarians residing in nursing homes, self-efficacy's lack of association with physical health is a unique finding. Once one resides in a nursing facility, it is likely that a strong sense of self-efficacy is not helpful or advantageous over outcomes where one has minimal influence such as physical health [76]. However, instrumental and received social support are concomitant with nursing home residence and likely explain the direct effect of social provisions on physical health. As mentioned in the results section regarding the supplemental analyses, we did find a strong indirect effect of mental status and education on mental health through self-efficacy. In addition, we mentioned the strong association between social provisions and activities of daily living. Consequently, we deleted activities of daily living from the model because of multicollinearity. These findings agree with a recent meta-analysis of 77 reports based on longitudinal data from community-based samples. It found the strongest predictors of nursing home admission included functional disability, cognitive impairment, and prior nursing home use [14]. At this stage of the life span and in this residential setting, there is likely little centenarians can do regarding their physical health, other than utilizing the direct services of others. However, participants who have higher levels of cognitive ability tend to have higher levels of self-efficacy and in turn higher levels of mental health. This discussion may also provide an explanation regarding the influence of self-efficacy on mental health for centenarians residing in nursing homes. Whereas, for those in private homes, perception of self-efficacy was not found to influence mental health—social provisions tended to directly and

significantly influence their mental health—for centenarians in nursing homes, perception of self-efficacy mattered. For participants with higher levels of cognitive ability, what they might have control over is how they view their lives or their mental outlook on life [1, 75, 76].

Our study is not without limitations. First, we examined three distinct groups of centenarians based on residential living status resulting in relatively small sample sizes. Second, our study was cross-sectional in nature; it did not follow very old adults from dwelling in a private home to an assisted living facility, and ultimately to a nursing home, nor was the study able to assess change in the measures over time as proposed by Antonucci et al. [13]. Centenarian studies face difficult design issues because the remaining life expectancy of participants is often less than two years depending upon birth cohort. In addition, we do not know if our participants were in the nursing home for the second or third time with intervals back in private residences or assisted living facilities. Gaugler and colleagues [14] found prior nursing home use to be one of the strongest predictors of nursing home admission. Third, measurement concerns regarding multiple assessments and types of reporting for the constructs studied would reduce possible bias in the results. These limitations provide opportunity for future research to build and expand upon the study and the theoretical model.

The present study added to the existing knowledge base of very old individuals by specifying and testing a version of the support-efficacy model [11, 13]. In addition, we considered a key situational characteristic of very old individuals near the end of the life span as a moderating influence: residential status (e.g., living in a private home, assisted living facility or a nursing home). The study affirmed the specified relationships between variables of the model; however, specific to this study was the finding that the hypothesized model uniquely fit each group of participants. Future work with very old individuals will be aimed to consider and account for the moderating influence of residence status on the constructs examined: social relations, self-efficacy, and both mental and physical health.

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References

[1] D. G. Blazer and C. F. Hybels, "Origins of depression in later life," *Psychological Medicine*, vol. 35, no. 9, pp. 1241–1252, 2005.

[2] N. Krause, "Social support and feelings of personal control in later life," in *Sourcebook of Social Support and Personality*, G. R. Pierce, B. Lakey, I. G. Sarason, and B. R. Sarason, Eds., pp. 335–355, Plenum Press, New York, NY, USA, 1997.

[3] J. L. Moren-Cross and N. Lin, "Social networks and health," in *Handbook of Aging and the Social Sciences*, R. H. Binstock and L. K. George, Eds., pp. 111–128, Academic Press, Burlington, Mass, USA, 6th edition, 2006.

[4] M. Pinquart and S. Sörensen, "Influences of socioeconomic status, social network, and competence on subjective well-being in later life: a meta-analysis," *Psychology and Aging*, vol. 15, no. 2, pp. 187–224, 2000.

[5] M. K. Rohr and F. R. Lang, "Aging well together—a mini-review," *Gerontology*, vol. 55, no. 3, pp. 333–343, 2009.

[6] M. MacDonald, "Social support for centenarians' health, psychological well-being, and longevity," *Annual Review of Gerontology and Geriatrics*, vol. 27, pp. 107–128, 2007.

[7] L. W. Poon and T. T. Pearls, "The trials and tribulations of studying the oldest old," *Annual Review of Gerontology and Geriatrics*, vol. 27, pp. 1–10, 2007.

[8] V. L. Bengston, D. Gans, N. M. Putney, and M. Silverstein, "Theories about age and aging," in *Handbook of Theories of Aging*, V. L. Bengston, D. Gans, N. M. Putney, and M. Silverstein, Eds., pp. 3–24, Springer, New York, NY, USA, 2nd edition, 2009.

[9] V. L. Bengston and K. W. Schaie, Eds., *Handbook of Theories of Aging*, Springer, New York, NY, USA, 1999.

[10] M. J. Levitt, "Social relations across the life span: in search of unified models," *International Journal of Aging and Human Development*, vol. 51, no. 1, pp. 71–84, 2000.

[11] T. E. Seeman and E. Crimmins, "Social environment effects on health and aging: integrating epidemiologic and demographic approaches and perspectives," *Annals of the New York Academy of Sciences*, vol. 954, pp. 88–117, 2001.

[12] T. C. Antonucci and J. S. Jackson, "Social support, interpersonal efficacy, and health: a life course perspective," in *Handbook of Clinical Gerontology*, L. L. Carstensen and B. A. Edelstein, Eds., pp. 291–211, Pergamom Press, New York, NY, USA, 1987.

[13] T. C. Antonucci, K. S. Birditt, and H. Akiyama, "Convoys of social relations: an interdisciplinary approach," in *Handbook of Theories of Aging*, V.L. Bengston, D. Gans, N. M. Putney, and M. Silverstein, Eds., pp. 247–260, Springer, New York, NY, USA, 2nd edition, 2009.

[14] J. E. Gaugler, S. Duval, K. A. Anderson, and R. L. Kane, "Predicting nursing home admission in the U.S: a meta-analysis," *BMC Geriatrics*, vol. 7, article no. 13, 2007.

[15] J. E. Gaugler and R. L. Kane, "Improving practice through research in and about assisted living: implications for a research agenda," *The Gerontologist*, vol. 47, pp. 83–99, 2007.

[16] G. K. Randall, P. Martin, M. McDonald, and L. W. Poon, "Social resources and longevity: Findings from the Georgia Centenarian Study," *Gerontology*, vol. 56, no. 1, pp. 106–111, 2010.

[17] Family Caregiver Alliance, 2005, http://www.caregiver.org/caregiver/jsp/content_node.jsp?nodeid=440 .

[18] National Center for Assisted Living, "Resident profile," December 2009, http://www.ahcancal.org/ncal/resources/Pages/ResidentProfile.aspx .

[19] F. H. Decker, *Nursing Homes, 1977–99: What Has Changed, What Has Not?* National Center for Health Statistics, Hyattsville, Md, USA, 2005, http://www.cdc.gov/nchs/data/nnhsd/NursingHomes1977_99.pdf.

[20] A. Bowling and E. Grundy, "The association between social networks and mortality in later life," *Reviews in Clinical Gerontology*, vol. 8, no. 4, pp. 353–361, 1998.

[21] L. W. Poon, S. M. Jazwinski, and R. C. Green, "Methodological considerations in studying centenarians: lessons learned from the Georgia Centenarian Studies," *Annual Review of Gerontology and Geriatrics*, vol. 27, pp. 231–264, 2007.

[22] F. Blanchard-Fields and A. Kalinauskas, "Theoretical perspectives on social context, cognition, and aging," in *Handbook of Theories of Aging*, V.L. Bengston, D. Gans, N. M. Putney, and M. Silverstein, Eds., pp. 261–276, Springer, New York, NY, USA, 2nd edition, 2009.

[23] L. L. Carstensen, D. M. Isaacowitz, and S. T. Charles, "Taking time seriously: a theory of socioemotional selectivity," *American Psychologist*, vol. 54, no. 3, pp. 165–181, 1999.

[24] T. C. Antonucci, "Social relations: an examination of social networks, social support, and sense of control," in *Handbook of the Psychology of Aging*, J. E. Birren and K. W. Schaie, Eds., pp. 427–453, Academic Press, San Diego, Calif, USA, 2001.

[25] L. K. George, "Perceived quality of life," in *Handbook of Aging and the Social Sciences*, R. H. Binstock and L. K. George, Eds., pp. 320–336, Academic Press, Burlington, Mass, USA, 6th edition, 2006.

[26] N. Krause, "Social relationships in late life," in *Handbook of Aging and the Social Sciences*, R. H. Binstock and L. K. George, Eds., pp. 182–201, Academic Press, Burlington, Mass, USA, 6th edition, 2006.

[27] A. D. Faber and S. Wasserman, "Social support and social networks: synthesis and review," *Advances in Medical Sociology*, vol. 8, pp. 29–72, 2002.

[28] R. Weiss, "The provisions of social relationships," in *Doing Unto Others*, Z. Rubin, Ed., pp. 17–26, Prentice Hall, Englewood Cliffs, NJ, USA, 1974.

[29] N. Krause, "Assessing change in social support during late life," *Research on Aging*, vol. 21, no. 4, pp. 539–569, 1999.

[30] T. L. Bisconti and C. S. Bargeman, "Perceived social control as a mediator of the relationships among social support, psychological well-being, and perceived health," *Gerontologist*, vol. 39, no. 1, pp. 94–103, 1999.

[31] A. Bandura, "Self-efficacy: toward a unifying theory of behavioral change," *Psychological Review*, vol. 84, no. 2, pp. 191–215, 1977.

[32] C. E. Ross and J. Sastry, "The sense of personal control: social-structural causes and emotional consequences," in *Handbook of the Sociology of Mental Health*, C. S. Aneshensel and J. C. Phelan, Eds., pp. 369–394, Plenum, New York, NY, USA, 1999.

[33] P. T. Costa and R. R. McCrae, *Revised NEO Personality Inventory (NEO PI-R) and NEO Five-Factor Inventory (NEO-FFI) Professional Manual*, Psychological Assessment Resources, Odessa, Fla, USA, 1992.

[34] R. L. Piedmont, *The Revised NEO Personality Inventory: Clinical and Research Applications*, Plenum Press, New York, NY, USA, 1998.

[35] T. C. Antonucci, J. E. Lansford, H. Akiyama et al., "Differences between men and women in social relations, resource deficits, and depressive symptomatology during later life in four nations," *Journal of Social Issues*, vol. 58, no. 4, pp. 767–783, 2002.

[36] T. C. Antonucci, R. Fuhrer, and J. F. Dartigues, "Social relations and depressive symptomatology in a sample of community-dwelling French older adults," *Psychology and Aging*, vol. 12, no. 1, pp. 189–195, 1997.

[37] S. L. Chen, J. W. Brown, L. C. Mefford et al., "Elders' decisions to enter assisted living facilities: a grounded theory study," *Journal of Housing for the Elderly*, vol. 22, no. 1-2, pp. 86–103, 2008.

[38] E. K. Rossen, "Assessing older persons' readiness to move to independent congregate living," *Clinical Nurse Specialist*, vol. 21, no. 6, pp. 292–296, 2007.

[39] D. Street, S. Burge, J. Quadagno, and A. Barrett, "The salience of social relationships for resident well-being in assisted living," *Journals of Gerontology. Series B* , vol. 62, no. 2, pp. S129–S134, 2007.

[40] J. P. Tracy and S. DeYoung, "Moving to an assisted living facility: exploring the transitional experience of elderly individuals," *Journal of Gerontological Nursing*, vol. 30, no. 10, pp. 26–33, 2004.

[41] M. F. Folstein, S. E. Folstein, and P. R. McHugh, "'Mini mental state'. A practical method for grading the cognitive state of patients for the clinician," *Journal of Psychiatric Research*, vol. 12, no. 3, pp. 189–198, 1975.

[42] G. G. Fillenbaum, *Multidimensional Functional Assessment of Older Adults: The Duke Older Americans Resources and Services Procedures*, L. Erlbaum Associates, Hillsdale, NJ, USA, 1988.

[43] K. B. DeSalvo, W. P. Fisher, K. Tran, N. Bloser, W. Merrill, and J. Peabody, "Assessing measurement properties of two single-item general health measures," *Quality of Life Research*, vol. 15, no. 2, pp. 191–201, 2006.

[44] M. Jylhä, S. Volpato, and J. M. Guralnik, "Self-rated health showed a graded association with frequently used biomarkers in a large population sample," *Journal of Clinical Epidemiology*, vol. 59, no. 5, pp. 465–471, 2006.

[45] J. E. Rohrer, D. C. Herman, S. P. Merry, J. M. Naessens, and M. S. Houston, "Validity of overall self-rated health as an outcome measure in small samples: a pilot study involving a case series," *Journal of Evaluation in Clinical Practice*, vol. 15, no. 2, pp. 366–369, 2009.

[46] K. B. DeSalvo, N. Bloser, K. Reynolds, J. He, and P. Muntner, "Mortality prediction with a single general self-rated health question: a meta-analysis," *Journal of General Internal Medicine*, vol. 21, no. 3, pp. 267–275, 2006.

[47] M. Olfson, S. C. Marcus, M. Tedeschi, and G. J. Wan, "Continuity of antidepressant treatment for adults with depression in the United States," *American Journal of Psychiatry*, vol. 163, no. 1, pp. 101–108, 2006.

[48] S. H. Zuvekas and J. A. Fleishman, "Self-rated mental health and racial/ethnic disparities in mental health service use," *Medical Care*, vol. 46, no. 9, pp. 915–923, 2008.

[49] C. Cutrona and D. Russell, "The provisions of social relationships and adaptation to stress," in *Advances in Personal Relationships*, J. H. Jones and D. Perlman, Eds., vol. 1, pp. 37–67, JAI Press, Greenwich, Conn, USA, 1987.

[50] Y. Gondo, N. Hirose, Y. Arai et al., "Functional status of centenarians in Tokyo, Japan: developing better phenotypes of exceptional longevity," *Journals of Gerontology. Series A*, vol. 61, no. 3, pp. 305–310, 2006.

[51] S. M. Samuelsson, B. Bauer Alfredson, B. Hagberg et al., "The Swedish Centenarian Study: a multidisciplinary study of five consecutive cohorts at the age of 100," *International Journal of Aging and Human Development*, vol. 45, no. 3, pp. 223–253, 1997.

[52] W. Herzog and A. Boomsma, "Small-sample robust estimators of noncentrality-based and incremental model fit," *Structural Equation Modeling*, vol. 16, no. 1, pp. 1–27, 2009.

[53] D. L. Jackson, "Revisiting sample size and number of parameter estimates: some support for the N:q hypothesis," *Structural Equation Modeling*, vol. 10, no. 1, pp. 128–141, 2003.

[54] D. L. Jackson, "The effect of the number of observations per parameter in misspecified confirmatory factor analytic models," *Structural Equation Modeling*, vol. 14, no. 1, pp. 48–76, 2007.

[55] B. M. Byrne, *Structural Equation Modeling with Amos: Basic Concepts, Applications, and Programming*, Lawrence Erlbaum Associates, Mahwah, NJ, USA, 2001.

[56] R. B. Kline, *Principles and Practice of Structural Equation Modeling*, Guilford Press, New York, NY, USA, 2nd edition, 2005.

[57] L. K. Muthén and B. O. Muthén, *Mplus User's Guide*, Muthén and Muthén, Los Angeles, Calif, USA, 5th edition, 1998–2007.

[58] P. M. Bentler, "Comparative fit indexes in structural models," *Psychological Bulletin*, vol. 107, no. 2, pp. 238–246, 1990.

[59] M. W. Browne and R. Cudeck, "Single sample cross-validation indices for covariance structures," *Multivariate Behavioral Research*, vol. 24, pp. 445–455, 1989.

[60] L. T. Hu and P. M. Bentler, "Cutoff criteria for fit indexes in covariance structure analysis: conventional criteria versus new alternatives," *Structural Equation Modeling*, vol. 6, no. 1, pp. 1–55, 1999.

[61] R. C. MacCallum and J. T. Austin, "Applications of structural equation modeling in psychological research," *Annual Review of Psychology*, vol. 51, pp. 201–226, 2000.

[62] K. G. Jöreskog, "How large can a standardized coefficient be?" 1999, http://www.ssicentral.com/lisrel/techdocs/HowLargeCanaStandardizedCoefficientbe.pdf .

[63] K. Ell, "Social networks, social support, and health status: a review," *The Social Service Review*, vol. 58, no. 1, pp. 133–149, 1984.

[64] E. Rafaeli and M. E. J. Gleason, "Skilled support within intimate relationships," *Journal of Family Theory & Review*, vol. 1, pp. 20–37, 2009.

[65] T. C. Antonucci, J. Akiyama, and J. E. Lansford, "Negative effects of close social relations," *Family Relations*, vol. 47, no. 4, pp. 379–384, 1998.

[66] T. E. Oxman, L. F. Berkman, S. Kasl, D. H. Freeman, and J. Barrett, "Social support and depressive symptoms in the elderly," *American Journal of Epidemiology*, vol. 135, no. 4, pp. 356–368, 1992.

[67] K. B. Wilson, "Historical evolution of assisted living in the United States, 1979 to the present," *The Gerontologist*, vol. 47 Spec No 3, pp. 8–22, 2007.

[68] T. E. Oxman, L. F. Berkman, S. Kasl, D. H. Freeman Jr., and J. Barrett, "Social support and depressive symptoms in the elderly," *American Journal of Epidemiology*, vol. 135, no. 4, pp. 356–368, 1992.

[69] B. Lakey and J. B. Drew, "A social-cognitive perspective on social support," in *Sourcebook of Social Support and Personality*, G. R. Pierce, B. Lakey, I. G. Sarason, and B. R. Sarason, Eds., pp. 107–140, Plenum Press, New York, NY, USA, 1997.

[70] E. S. Mankowski and R. S. Wyer, "Cognitive causes and consequences of perceived social support," in *Sourcebook of Social Support and Personality*, G. R. Pierce, B. Lakey, I. G. Sarason, and B. R. Sarason, Eds., pp. 141–165, Plenum Press, New York, NY, USA, 1997.

[71] A. Bandura, *Social Foundations of Thought and Actions: A Social-Cognitive Theory*, Prentice Hall, Englewood Cliffs, NJ, USA, 1986.

[72] S. Cobb, "Social support as a moderator of life stress," *Psychosomatic Medicine*, vol. 38, no. 5, pp. 300–314, 1976.

[73] S. Cobb, "Social support and health through the life course," in *Aging from Birth to Death: Interdisciplinary Perspectives*, M. W. Riledy, Ed., pp. 92–106, Westview Press, Boulder, Colo, USA, 1979.

[74] C. J. Holahan, R. H. Moos, and L. Bonin, "Social support, coping, and psychological adjustment: a resources model," in *Sourcebook of Social Support and Personality*, G. R. Pierce, B. Lakey, I. G. Sarason, and B. R. Sarason, Eds., pp. 169–186, Plenum Press, New York, NY, USA, 1997.

[75] L. W. Poon, G. M. Clayton, P. Martin et al., "The Georgia Centenarian Study," *International Journal of Aging and Human Development*, vol. 34, no. 1, pp. 1–17, 1992.

[76] K. Andersen-Ranberg, M. Schroll, and B. Jeune, "Healthy centenarians do not exist, but autonomous centenarians do: a population-based study of morbidity among danish centenarians," *Journal of the American Geriatrics Society*, vol. 49, no. 7, pp. 900–908, 2001.

[77] V. Gecas, "The social psychology of self-efficacy," *Annual Review of Sociology*, vol. 15, pp. 291–316, 1989.

[78] E. L. Mitty, "Assisted living: aging in place and palliative care," *Geriatric Nursing*, vol. 25, no. 3, pp. 149–163, 2004.

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Review Article

Personality and Longevity: Knowns, Unknowns, and Implications for Public Health and Personalized Medicine

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We review evidence for links between personality traits and longevity. We provide an overview of personality for health scientists, using the primary organizing framework used in the study of personality and longevity. We then review data on various aspects of personality linked to longevity. In general, there is good evidence that higher level of conscientiousness and lower levels of hostility and Type D or "distressed" personality are associated with greater longevity. Limited evidence suggests that extraversion, openness, perceived control, and low levels of emotional suppression may be associated with longer lifespan. Findings regarding neuroticism are mixed, supporting the notion that many component(s) of neuroticism detract from life expectancy, but some components at some levels may be healthy or protective. Overall, evidence suggests various personality traits are significant predictors of longevity and points to several promising directions for further study. We conclude by discussing the implications of these links for epidemiologic research and personalized medicine and lay out a translational research agenda for integrating the psychology of individual differences into public health and medicine.

1. Introduction

The notion that personality and health are linked dates back to Galen [1]. In this paper, we review evidence on associations between personality traits and longevity. The first section provides a conceptual and definitional overview of personality for health scientists not familiar with personality research. Section 2 articulates a basic framework and rationale motivating studies of personality and longevity. Section 3 reviews the data on personality and longevity. Section 4 explains the implications of this literature for understanding and promoting healthy aging and provides a road map for future research spanning psychology and health.

2. Brief Overview of Personality for Aging Researchers and Health Scientists

2.1. Personality Traits. Personality traits reflect distinct sets of interrelated thoughts, feelings, and behaviors [2]. This is a fairly broad definition, and for this reason, personality has been called "the last refuge of the generalist in psychology" [3]. Personality traits are typically operationalized as dimensions, ranging from very high to very low [4, 5]. This is in contrast to a present/absent definition of a trait in, for instance, Mendelian genetics. For instance, extraversion is a personality dimension reflecting sociability, excitement seeking, and a generally consistent positive outlook. A person may possess a relatively high amount of extraversion, a moderate amount, a low amount, or anywhere in between these designations. Operationalizing traits as dimensions provide more nuanced information about people than a crude present/absent designation.

Personality traits are relatively consistent, in the sense that people do not change radically from one day to the next. However, change does occur over the long term, due either to naturalistic forces, such as physiological aging, or to due to intentional intervention [6–8]. Thus, personality represents a core of relatively stable individual differences in which alterations can be intentionally induced or can occur

naturalistically. Personality traits are considered phenotypic dimensions of human variation, reflecting both genetic and environmental influences. Twin studies converge on heritability estimates of major personality traits ranging from 40–50%, with most of the remainder of variation attributable to nonshared environmental influences [9].

Although a few candidate genes are beginning to be identified for personality traits [10, 11], Genome Wide Association Studies (GWAS) have yielded scant findings because personality traits are presumed to be polygenic: they reflect complex interactions between specific genes, rather than the presence of a single gene [12, 13]. This requires more complex analytic models than have been employed to date in molecular genetic studies of personality. Moreover, personality traits are the product of unknown, but probably large degrees of gene-environment interaction [9, 14]. Few studies, even those with GWAS data, include a comprehensive battery of life history and environmental events and exposures. Without this data, it is impossible to identify the environmental conditions under which specific genes may be linked to personality phenotype. As a result, current understanding of personality genetics rests primarily on heritability estimates from twin studies. To the extent that GWAS studies can identify the polygenic and genetic-environmental interfaces underlying phenotypic traits, they will refine understanding of the genetic bases of personality.

A final important piece of personality science includes the measurement of personality traits. Measurement can use self-report inventories containing written questions reflecting specific traits, similar inventories completed by others who know the person, behavioral measures such as the frequency with which one performs various acts, free-response tests where people are asked to provide stories or descriptions of visual stimuli, nonverbal tests in which people report the extent to which pictures describing various personality tendencies describe them, physiological measures such as how reactive a person is to a stressful stimulus, or tests of ability such as a person's capacity to inhibit impulsive responding, resist a temptation, or accurately recognize a facial expression of emotion [15]. In practice, self-report inventories developed through psychometric methods are the most common form of measurement.

Many investigators are taught to distrust self-report as a general measurement strategies because it may be inaccurate [16]. Yet self-reported personality data tends to be accurate and is also more trustworthy than self-reported biomedical data or observer reports of personality. Relatively sophisticated methods have been developed to assess whether a respondent is intentionally dissembling in their response, is trying to be honest but merely has an unrealistically positive self-perception, is responding randomly or haphazardly to questions, merely agreeing with every statement put before them, or engaging in some other behavior that biases measurement [15]. Self-report is more liable to be untrustworthy when respondents are asked to report biomedical data, such as disease exposure or disease diagnoses they may not fully understand. Self-report bias is also more active in "high stakes testing" situations where incentives exist to misrepresent oneself, such as when undergoing a mental health, forensic, injury/disability, or occupational assessment [17, 18]. In contrast, personality instruments ask innocuous questions about the person's daily habits, which people typically know and are able to report accurately, without the bias that occurs when sensitive questions are posed [19, 20].

Self-report is often preferred over observer ratings of personality [21, 22] because observers do not have access to a person's internal motives, emotions, thoughts, and so forth—only to external behavioral manifestations of these. Even then, they are not at the individual's side for extended periods and only observe his or her behavior in certain situations. Only the respondent him or herself is qualified to provide data on his or her own inner tendencies and behavior across all situations over extended periods of time. Other evidence suggests that reporting bias has minimal impact on the prediction of important outcomes [23], and that the tendency to present oneself in a positive light is a personality trait itself [24, 25]. For these reasons, self-report has been the primary mode of personality assessment in studies on personality and longevity. Nevertheless, personality researchers are increasingly considering how to incorporate other measurement methods, such as informant reports, into personality assessment in health research.

3. Approaches to the Study of Personality and Longevity

3.1. Conceptual Framework for Personality and Longevity. Figure 1 summarizes a heuristic model of the ways in which personality is thought to affect longevity [26, 27]. Behavioral causes of mortality, including smoking, diet, exercise, alcohol use, and risk-taking are all linked to basic personality dispositions [28]. Less obvious factors such as health decision-making styles [29] and health risk evaluation [30, 31] have also been linked to personality, as have psychopathology [32, 33] and social relationships [34]. Personality appears to influence health through biological channels as well. Characteristic emotional patterns have been linked to neuro-endocrine interactions involving the Hypothalamic-Pituitary-Adrenal (HPA) and Sympatho-Medullary (SAM) axes [35, 36]. Common genetic bases may also lead to the joint expression of certain personality phenotypes, problematic health behaviors, and resulting chronic diseases [37].

The extent to which any particular mediator affects personality-mortality associations is likely to depend on the personality trait, the form of mortality, and the population.

As denoted in Figure 1, personality traits act on health in social contexts [38]. Personality development is influenced by social-environmental factors such as socioeconomic status (SES) [39, 40]. As well, personality traits can lead to self-selection into different environments [41] and differential responses to socioeconomic disadvantage [40]. As a result, personality-health relations are at least somewhat intertwined with social forces that influence longevity. The role of social inequalities in health is rarely a focus in personality and health research, but in Section 4 we outline three possible models for future investigation.

3.2. The Big 5 Taxonomy and General versus Specific Trait Approaches to Longevity. Because there are thousands of words in natural language used to denote personality traits, personality scientists have developed a taxonomy for organizing and classifying them. This taxonomy consists of 5 general axes representing the primary dimensions of human personality variation [4]. These so called "Big 5" axes are Neuroticism versus Emotional Stability, Extraversion versus Introversion, Openness to Experience versus Closedness, Agreeableness versus Antagonism, and Conscientiousness versus Irresponsibility. The Big 5 dimensions are composites, or clusters of numerous "building block" traits (Figure 2). It is helpful to think of the Big 5 as the primary "molecules" of personality, each molecule composed of the "atoms" of specific traits. The *lexical hypothesis* suggests that the Big 5 reflect basic tendencies of such sociocultural significance that they have become encoded in human languages [4]. Recent work suggests a biological basis for this trait taxonomy as well [42]. Finally, the term "Five-Factor Model" is often used to denote the Big 5 in personality psychology [43]. Minor differences between what each term connotes are of interest to personality scientists, but are not important here.

Because the Big 5 are composites of several specific traits, these measures capture an amalgam of numerous specific facets of personality. The advantage of using a composite measure aggregating each family of traits is that it increases the likelihood that *some* aspect(s) of personality relevant to health have been captured by scores on Big 5 measure. The downside is that unless this composite measure of the entire trait "molecule" can be disaggregated into the specific constituent "atoms", investigators cannot isolate the component(s) of personality most relevant to longevity.

By contrast, another approach to personality and longevity has focused more directly on the specific traits lying within each Big 5 domain. The Big 5 taxonomy did not begin to be used extensively in health research until the early 1990s [44]. Prior to the 1990s, investigators had no choice but to select single traits they believed were associated with mortality. Other investigators preferred this selective, specific approach to personality and longevity even after the Big 5 system had become the major framework in personality research. One reason is that some theoretical traditions within psychology stress the importance of specific traits—for instance, emotional suppression in psychodynamic theory [45] has no obvious analogue in natural language or the Big 5. Another reason some investigators prefer specific traits is that occasionally, a particular trait is not formally measured by the Big 5 system. For instance, sensuality, religiosity, masculinity-femininity, and thriftiness/miserliness versus prodigality have been argued to lay outside of the Big 5 domains [46]. Despite the broad consensus surrounding the Big 5, other investigators continue to examine the constituent components of the domains outside the Big 5 framework. Examples include hostility in interpersonal theory [47], or constructs related to personal control in control theory [48].

The advantage of focusing on specific traits is that they implicate very particular aspects of personality in longevity, providing more precise information for theory and intervention. A disadvantage is that there may be many other specific traits that are relevant to longevity which are omitted from these studies. These traits may be confounders or even interact with the specific trait in question, because all aspects of personality are in simultaneous function in everyday life. The problem is analogous to examining health-related outcomes only as a function of smoking, without considering diet, exercise, alcohol, environmental exposures, health service utilization, medication adherence, or dozens of other behavioral predictors of health outcomes that may themselves be related smoking.

Despite the difference in research motivated by a Big 5, versus specific trait model, it is still possible to organized findings within the Big 5 framework. In the presentation that follows, we group specific traits under the Big 5 taxonomic dimension in which they are primarily classified. For instance, optimism is classified as a trait in the Extraversion domain of traits. However, some of these specific traits may have secondary classification under another Big 5 dimension. For example, (low) optimism/pessimism is sometimes considered to lie secondarily within the Neuroticism domain. One can think of such "cross-classified" traits as sharing some characteristics of more than one Big 5 domain, or laying at the intersection of Big 5 domains. Full listings of Big 5 traits can be found in [49]. A similar idea is the concept of a "compound trait" [50], or a combination of two or more specific traits, often spanning Big 5 dimensions. In later sections, we also discuss the notion of personality prototypes [51] and styles [52], which refer to specific profiles or configurations of the Big 5 dimensions. In conceptualizing factors that enhance or reduce longevity, we also differentiate between all-cause mortality, and cardiovascular disease-(CVD) related and cancer mortality when possible. The CVD versus cancer classification is a common one in epidemiologic studies. Each class contains several diseases unified by a common set of etiologies, and in some cases the personality traits have been implicated more in one set of etiologic processes than another.

4. Evidence Linking the Big 5 Personality Dimensions to Longevity

4.1. Conscientiousness. Conscientiousness involves self-discipline, achievement striving, reliability, and several similar traits related to diligence [53]. This Big 5 dimension has been linked to lower all-cause mortality risk across a number of studies [54–57] (see also [58]). The strength of associations between Conscientiousness and longevity appears comparable to those between longevity and SES and IQ [59]. Conscientiousness appears to be the one Big 5 domain for which no negative results exist. However, some pathologies of Conscientiousness such as perfectionism or compulsive persistence should be noted. In one study, the all-cause mortality risk of Conscientiousness was modeled controlling for perfectionism, [56]. In this case, Conscientiousness was associated with reduced, while perfectionism was associated with increased risk. Immunologic work has suggested that compulsive persistence, defined as the inability to disengage from an impossible, stressful task, induces higher HPA-axis response [60]. It is thus helpful to differentiate "healthy"

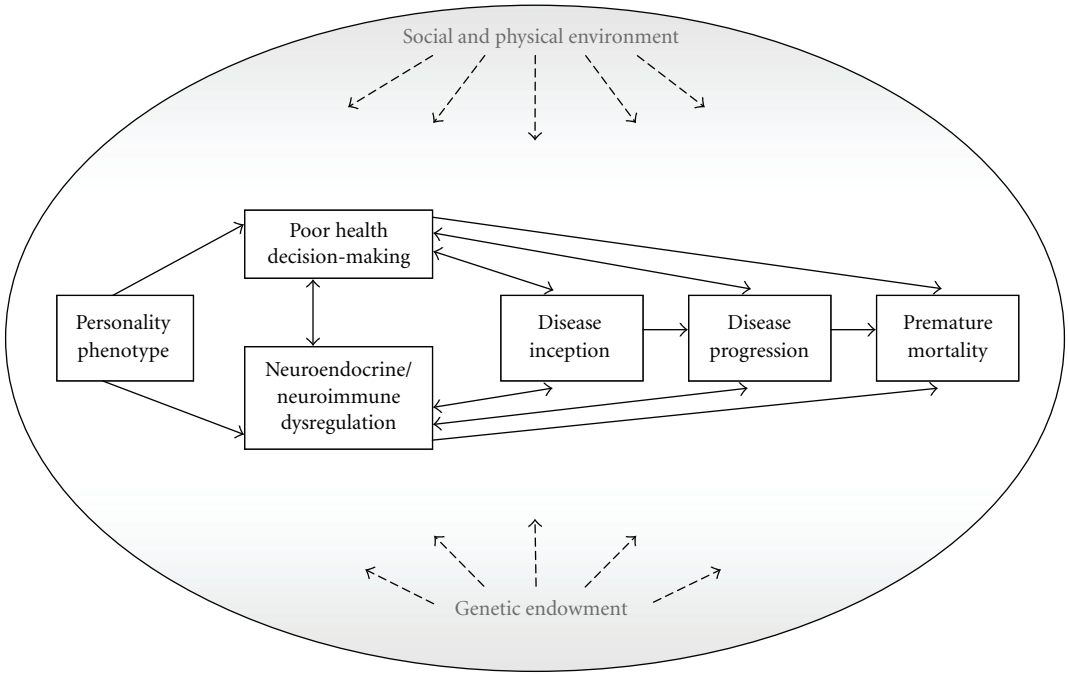

Figure 1: Personality phenotype is thought to influence longevity through physiological and behavioral pathways leading to health decline. This occurs in the context of environmental (physical or social) and individual genetic influences, which may enter into risk chains directly or modify them indirectly.

Conscientiousness from extreme rigidity or compulsion. Conscientiousness involves a strong self-regulatory component, which facilitates adaptive outcomes [61] in part by preventing inflexibility like perfectionism or compulsive behavior.

Because of the self-discipline it entails, Conscientiousness has been linked to a variety of health behaviors including maintaining healthy activity and diet, and abstaining from smoking, alcohol, and substance abuse [28]. However, links between Conscientiousness and longevity are only partly explained by commonly studied health behaviors [62–64]. Less frequently studied health behaviors, such as health decision-making and risky sexual practices, are avenues for further investigation. Biological mechanisms are not well understood, but seem to be implicated in the Conscientiousness-longevity link if only because health behaviors generally account for a surprisingly small portion of the observed association. For instance, higher levels of the inflammatory marker Interleukin (IL)-6 have recently been linked to lower Conscientiousness [65, 66]. IL-6 is associated with chronic stress, a number of chronic diseases, and negative health behaviors, and is a major predictor of mortality in older persons [67, 68]. Studies showing higher IL-6 linked to lower Conscientiousness have controlled, however, for health behaviors and chronic diseases, so a working hypothesis is that Conscientious people are better able to anticipate and prepare for future consequences of potential adversities, more organized, and self-disciplined. These qualities could prevent stressful situations from escalating and could also enhance coping.

Several areas of new research are emerging based on the initial wave of studies linking higher C to longevity. A recent National Institute on Aging workgroup has offered a series of recommendations for advancing research on Conscientiousness, including refining its biological basis and measurement, considering how this trait operates on health in different social contexts, articulating better lifespan models of the effects of Conscientiousness, and intervening in specific areas such as self-regulatory skills. White papers in these areas are due out later in 2011.

4.2. Openness to Experience. Although there is minor disagreement over the range of traits belonging in the Openness to Experience domain, most agree that Openness encompasses cognitive and behavioral flexibility, urbane or cultured tendencies, and attunement to internal and external events and experiences [69]. Although it may reflect a propensity toward an intellectual lifestyle in certain cultures, it is distinct from raw intellectual ability [70] and in fact is observable in nonhuman primates [71]. Higher levels of Openness have been associated with decreased risk for all-cause mortality in a Japanese community sample [54], with lower risk of all-cause and CVD mortality in an American sample of CVD patients [72], and with lower all-cause mortality risk in a cohort of men [73]. The latter two studies were able to decompose Openness into more specific traits and found that cognitive and behavioral flexibility [72] and interest in aesthetics [72, 73] were particularly important predictors of greater longevity. Reduced 5-year all-cause mortality risk has also been documented for curiosity, a facet of Openness, in a mixed-gender older community sample [74]. Finally, one study reported nonsignificant decreases in all-cause mortality risk associated with the general Openness domain [56].

The mechanisms by which Openness may improve longevity are not immediately clear. Beyond basic genetic influences, others factors may include cognitive engagement, flexibility, and the maintenance of cognitive function [75]. These factors probably facilitate the prevention of avoidable health problems, as well as increase adaptive disclosure of health concerns, health decision-making, and capacity to manage problems that are encountered. As with other traits, investigators have begun to examine inflammatory markers as a general biological mechanism accounting for health benefits of Openness. One study found that higher Openness was linked to lower levels of the inflammatory marker Interleukin-6 in older persons [66]. Another possibility is that Openness may be related to markers of synaptic plasticity, such as Insulin-Like Growth Factor (IGF)-1, which has been linked to cognitive function and longevity [76]. In general, the associations between Openness associations with longevity seem to be distinct from the effects of education, which is typically controlled for in analyses. However, few studies to date have investigated the distinct longevity associations for Openness and IQ. This will be an important area for future work.

4.3. Extraversion

4.3.1. The General Extraversion Domain. Extraversion is another broad dimension of personality appearing in some form in most personality trait theories [42]. Extraversion encompasses the tendency toward positive mood, sociability, and activity (in the sense of an active, busy, or engaged lifestyle) [77]. As well, it involves an element of excitement seeking or social disinhibition. This last aspect of Extraversion tended to be very prevalent in an earlier operationalizations of Extraversion [78]. In the Big 5 framework, the desire for excitement is still a part of Extraversion, but pure impulsivity and low self-control are captured by Neuroticism (high) and Conscientiousness (low) [79]. This is relevant to longevity because early Extraversion scales loaded with impulsive content predicted greater all-cause mortality risk over 21 years in a large UK cohort [80] (see also [81]). This effect was accounted for in part by smoking, a health behavior to which more Extraverted people appear susceptible, regardless of whether older or new operationalizations of Extraversion are used [82–85].

Focusing more on elements such as sociability and positive mood, modern operationalizations of Extraversion, tend to find that it is associated with reduced all-cause mortality risk. This was the case over a 9-year followup period in an American [56] community sample, a 5-year follow-up period in a Japanese [54] community samples, and a 6-year follow-up in an older twin sample [87]. The activity facet of Extraversion has also been associated with lower 50-year all-cause mortality risk in a US cohort [88], with suggestive findings over 10 years in another US cohort [62]. Other studies noted no substantial effects for the general Extraversion dimension on 3-year all-cause mortality in an older, ill US community sample [63], in substance and alcohol abusers [89], or in Japanese cancer patients (with a general case-mix of cancer types) [90].

These findings provide some limited evidence that in general community samples, higher levels of Extraversion are associated with greater longevity. However, as excitement-seeking strays into impulsivity and poor self-control, mortality risk may mount. Null findings in specific samples (the ill elderly, alcohol/substance abusers, cancer patients) and links between Extraversion and smoking suggest that the salutary effects of Extraversion, arising from traits such as sociability, activity, and positive mood, may be overpowered by the mortality risks conferred by physical morbidity or destructive addictive behaviors. Another issue is impulsivity tends to decline with age in most cohorts, in part as a normal function of aging [7], but possibly also because more impulsive members of a cohort die earlier. As a result, impulsivity-related mortality risk may be dampened beyond detectable levels in analyses of older samples.

As a technical note, impulsivity is sometimes classified as a Neuroticism trait on some personality instruments, while similar traits such as self-discipline and deliberation appear in the Conscientiousness family [79]. In this vein, one study found that higher impulsivity in the elderly was actually associated with greater probability of 3-year survival [63]. One possibility is that the speed of action entailed by measures of impulsivity actually relates to processing speed, a neurologic factor argued to reflect general integrity of biologic systems [37]. Possibly this could be better distinguished in personality measurement tools from pure sensation seeking or lack of self-discipline, which have clear deleterious effects on health. Research on performance-based measures of impulsivity and self-report scales reveals low correlations [91], suggesting that better measurement may help refine conceptual models differentiating harmful from helpful aspects of impulsivity/sensation-seeking/low levels of deliberation.

4.3.2. Optimism. Optimism is a stable tendency to expect positive future outcomes, while pessimism is the tendency to expect negative outcomes. Sometimes optimism and pessimism are operationalized as two separate constructs [92], sometimes they are considered opposite ends of a single continuum [93], and academic debates exist over the manner in which optimism is related to the Big 5 [94]. For the sake of clarity and utility in health research, optimism-pessimism can safely be considered as a single dimension, defined primarily by high levels of Big 5 Extraversion and secondarily by low levels of Big 5 Neuroticism.

Links between optimism and longevity have been documented in a range of populations. Optimism is associated with lower risk of all-cause mortality over 40 years in both college freshman [95, 96] and over 30 [97] and 40 [96] years in community midlife samples. Short-term ($\leq$1 year) cancer mortality has also been linked to lower levels of optimism in younger patients with a diverse range of cancers [98], and in head and neck cancer patients [99]. Death over a 10-year span related to CVD disease in men [93] and death due to stroke in a general community sample [100] have also been linked to lower optimism. One study found no effect for optimism but elevated cancer mortality risk related to pessimism when these two constructs were

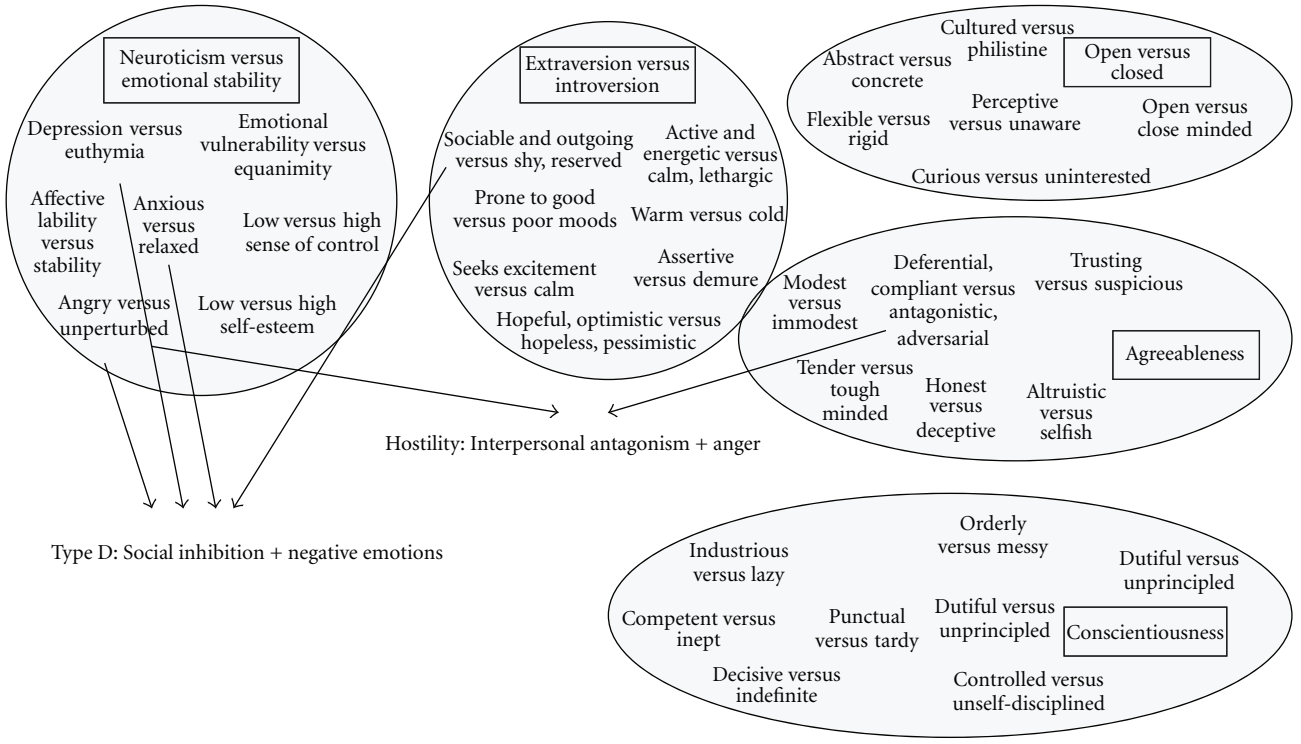

Figure 2: An example of specific traits with major loadings on each of the Big 5. In the diagram, the two compound traits studied with respect to longevity, hostility and Type D, are combinations of traits from different Big 5 dimensions. Numerous other traits and combinations exist; see [49] for a comprehensive mapping of specific traits composing each of the Big 5, and [86] for a comprehensive analysis of compound traits composed of 2 Big 5, with a similar perspective also presented in [52].

conceptualized as separate variables [98]. In our more general framework, this distinction is less relevant and favors greater longevity for people who do not expect negative things of the future, controlling for whether or not they have positive expectations.

More adaptive coping has been posited as one mechanism explaining the association of optimism with lower mortality risk [101]. In addition to, or possibly because of the coping advantage it confers, optimism appears linked to lower level of ambulatory blood pressure [102] and lower levels of inflammatory markers [103]. However, mixed evidence has emerged on the association of optimism with immune function, with one hypothesis being that optimists may experience more frustration when they do not experience immediate success and/or persist longer in stressful situations because they expect positive results [60, 104–106]. Another hint that optimism may not always be adaptive appeared in a study of high IQ children, which found that cheerfulness, defined as optimism and sense of humor, predicts greater mortality risk by midlife. The extent to whether these traits in children measure something similar to adult optimism is not clear, however. While these studies provide some hint that optimism may be maladaptive in some contexts, the preponderance of evidence to date suggests that it tends to promote lower mortality risk in most people in most circumstances.

4.4. The Neuroticism Domain

4.4.1. Findings on Broad Neuroticism. Neuroticism is a dimension of personality present not only in the Big 5 framework, but in nearly every other empirically based personality trait theory [42]. The definition of the Neuroticism domain has subtly shifted over time. Prior to the Big 5 framework, Eysenck primarily defined Neuroticism according to stress-reactivity and emotional lability, with some items that could be interpreted as somatization or somatic sensitivity [107]. The instability component was retained in Big 5 notions of Neuroticism [49], with some emphasizing the chronic experience of specific negative emotions such as anxiety, depression, and anger [79]. Another aspect of Neuroticism that is often neglected is vulnerability, or a sense that one is unable to cope with the challenges of life. This is partially related in the concept of emotional instability, and reflected in items like "I seem to go to pieces easily." It is for this reason that many of the traits related to feeling a sense of control over one's life reflect low Neuroticism. For instance, the correlations between locus of control, self-esteem, and self-efficacy with Neuroticism appear so strong across samples that these traits can essentially be considered aspects of Neuroticism [108].

With respect to health behaviors, elevated has been linked to weight gain and obesity [84, 109] and smoking [110],

general etiologic factors in foreshortened longevity. In fact, Neuroticism has been argued to be of general public health significance because it is associated with so many negative outcomes [111, 112].

Several studies have linked higher Neuroticism to all-cause mortality in general populations from the US [62], Finland [113], and the United Kingdom [80, 81] over as long a follow-up period as 50 years [88, 113], as well as in an older twin sample [87]. As well, higher levels of Neuroticism predicted mortality risk in specific patient populations such as those with end-stage renal disease (ESRD) [57] and congestive heart failure (CHF) [114]. Naturalistic increases over time in Neuroticism have been linked to later all-cause mortality in a sample of older American men [115]. Treatment-induced decreases in Neuroticism also reduced risk of mortality over 9 years in European patients with cardiovascular disease (CVD) [116]. Finally, at least two studies have noted some confounding between socioeconomic status (SES) and Neuroticism, such that some of the socioeconomic inequalities in mortality rate can actually be explained by the fact that persons in more disadvantaged social environments are prone to higher Neuroticism [62, 113].

Other studies have reported negative findings. No associations between Neuroticism and all-cause mortality were observed in US [117] and Japanese [54] community samples. The same was true with respect to cardiovascular disease (CVD) mortality in a US community sample [117] and with respect to cancer mortality in a sample of Japanese cancer patients [90]. Another study reported no association between elevated Neuroticism and increased all-cause mortality risk in a female community sample [118]. However, in a sample of gifted individuals, elevated Neuroticism was linked to *lower* all-cause mortality risk in women but *higher* mortality risk in men [119]. To complicate the picture further, two studies have found that higher Neuroticism was associated with *reduced* all-cause mortality risk in mixed-gender older community samples in the US [63] and Australia [120].

What, then, is one to think of the implications of Neuroticism for longevity? Friedman has proposed a theory of "healthy" versus unhealthy Neuroticism [26]. "Healthy neuroticism" encompasses "worried well" tendencies that may facilitate engagement in preventive health behavior to allay concern about acquiring chronic health problems. Healthy Neuroticism may be represented by individuals higher in anxiety, but not laden by poor self-esteem, vulnerability, and depressive moods. Neuroticism gives rise to medically unfounded symptom complaints and somatic sensitivity [118, 121], which may protect an individual from premature mortality even though it risks burdening health care providers and the health care system. However, Neuroticism has been linked to reduced *quality of life* [122], even controlling for objective measures of disease load [123] raising the question of whether higher levels lead to more quality-adjusted life years. Clarifying the mortality risk of Neuroticism is an important research priority we discuss in the next section.

4.4.2. Low Perceived Control. Locus of control, perceived control, self-efficacy, and sense of coherence are a group of interrelated psychological constructs reflecting the extent to which people feel in control or are able to deal with life stressors and challenges. As suggested by their high loading on the Big 5 Neuroticism dimension [108], these constructs are trait-like, even though they were developed by researchers who were less concerned with stable traits. Although "sense of coherence" suggests existential congruence between one's behavior and one's goals, the construct is really closer to perceptions of control over one's life [124]. In general, this family of traits is theorized to improve adaptation to hardship or life challenges via the use of proactive, effective coping [125].

The range of findings generally suggests that these dispositions are associated with greater longevity. In a large British national cohort, lower mastery was a risk factor for all-cause, CVD, and cancer death over 6 years [126] and for CVD mortality over 11 years, particularly in persons with *low* CVD risk [127, 128]. In this UK cohort, the same pattern of findings emerged for those with low sense of coherence, although this trait was a risk for cancer mortality only in men [128]. Similar findings for all-cause mortality have been reported in a Finnish cohorts [129], and for CVD mortality in a German cohort [130]. Sense of coherence appears to produce more adaptive coping in response to life stressors, which was in turn associated with lower mortality risk [125]. In general, recent research supports the adaptive value of perceived control [131], although not inexorably under all conditions [132].

4.4.3. Negative Affect Blended with Social Inhibition: The Type D Personality. "Type D" stands for the distressed personality, defined by simultaneously high negative affect and social inhibition [133]. It represents high levels of Big 5 Neuroticism, coupled with low levels of the sociability facet of Big 5 Extraversion, with some suggestion of accompanying declivity in Agreeableness and Conscientiousness [134, 135]. Conceptually, Type D has been described as a general propensity toward psychological distress, encompassed by depression, anxiety, and other forms of negative emotion, such as anger [136, 137], and combined with inadequate social resources to offset this distress [138]. This personality configuration was originally identified empirically as a predictor of all-cause mortality over 7.5 years in a sample of CVD patients [139].

Subsequent reports have shown that Type D also increases risk of mortality in CVD patients two years after receiving stents [140], over 2.5 years in patients with chronic heart failure (CHF) [141], over 4 years in peripheral artery disease (PAD) patients who underwent vascular surgery [142], and over 6 years in coronary artery disease (CAD) patients [143]. The majority of mortality in these studies was due to cardiovascular factors. However, negative findings have been reported for all-cause and cardiovascular mortality over 3 years in CVD patients [144], and for 7-year all-cause mortality in patients with chronic obstructive pulmonary disease (COPD) [145]. These latter two studies reported small to modest elevations in mortality risk for Type D that were not statistically significant.

Taken as a whole, these findings indicate that Type D conveys mortality risk in those with or at risk for various forms of CVD. Type D acts through many behavioral risk factors for the inception and progression of CVD, including maintaining a poor diet and smoking [146]. As well, Type D is associated with inflammatory markers [147–150] and oxidative stress [151], which both contribute to the progression of CVD. Others studies suggest that anxiety, associated with sympathetic nervous system disturbance [152], as well as ineffective interaction with health care providers [153], contributes to Type D-related mortality risk. While many studies have documented poorer general health correlates of Type D in community (rather than CVD) samples [154, 155], the risk posed by Type D for mortality due to non-CVD causes, in nonpatient samples, remains to be elaborated. Finally, Type D is a risk factor for depressive episodes, which are thought to dampen prognosis of CVD via inflammation and neglectful health behavior [137, 156].

4.4.4. The Distinction between Neuroticism and Depression. A final note on the relationships between Neuroticism (or other aspects of personality) and depression (or other aspects of psychopathology) is in order. Depression is a particular form of psychopathology considered related to, but distinct from personality. Personality is relatively stable, while depression is defined as an episodic cluster of symptoms reflecting a change in mood and function from a person's normal baseline. Personality refers to the person's "normal baseline". People who are routinely prone to negative emotions and emotionally unstable may be more likely to develop depressed mood, the first cardinal symptom of depression [157]. People who rarely experience positive emotions (an aspect of low Extraversion) may be more liable to experience virtual absence of pleasure or interest, which is the second cardinal symptom of depression. Those who are prone to negative emotion and socially inhibited (the Type D configuration) are similarly prone to develop formal depression syndromes. Other symptoms of depression, such as anergia, may be more liable to emerge given certain personality vulnerabilities, such as low Extraversion. Under this model, the personality predisposition precedes a diagnosis and persists after the depressive symptoms have been treated, in the same way that a compromised immune system precedes the inception of an opportunistic infection and remains after the infection has been treated. Just as the immune system itself can be treated, there is some evidence that Neuroticism can be as well; we consider this in the next section.

4.5. Agreeableness. Agreeableness is a composite of several traits related to maintaining interpersonal harmony: trust, honesty, compliance, interpersonal deference, altruism, and compassion for others [79]. One study to date has demonstrated a protective effect for Agreeableness against all-cause mortality over 3 years in an elderly sample; this was due primarily to specific aspects of the Agreeableness scale reflecting low levels of interpersonal antagonism [63]. We discuss this in detail below when we consider studies focusing specifically on hostility, which involves a combination of low agreeableness and high Neuroticism. Agreeableness did not appear as a robust predictor of longevity in four other studies [54–57]. One study actually found agreeableness was associated with *greater* 10-year all-cause mortality in a US national cohort, when combined with low Conscientiousness [62]. This study used a different measure of agreeableness focusing on compassion and caring. Collectively, evidence that Agreeableness in and of itself promotes or detracts from longevity is not strong. However, one aspect of low agreeableness—interpersonal antagonism—in conjunction with angry emotions, a facet of Neuroticism, yields the personality trait of hostility, which has been a topic of considerable research.

4.5.1. Hostility. Research on hostility and longevity grew out of the theory of Type A personality. This theory was first introduced by a cardiologist in the 1950s and proposed that the combination of impatience, hard-driving, competitiveness, and hostility was associated with cardiovascular disease (CVD) [158]. However, in the early 1980s investigators began to study hostility in particular [159, 160], concluding that it was the primary "toxic" component of the Type A personality [161, 162].

Within US samples, hostility appeared to elevate risk for 25-year all-cause mortality among male medical students [159], for 10-year all-cause, CVD, and cancer mortality among midlife men [160], for worse health trajectories involving earlier mortality in male veterans [163]. Hostility also conferred 3-year CVD and all-cause mortality in midlife Finnish men [164], elevated 25-year all-cause mortality in a mixed-gender midlife sample in Denmark [165], elevated 6-year CVD mortality [166], and 16-year all-cause mortality, [167] in mixed-gender community samples, the latter even with mean ages below 30 at baseline. Others found that hostility was *not* associated with CVD risk or 25-year all-cause mortality in male physicians [168], with 33-year risk for mortality from any cause among a sample of 19-year olds [169], or with 5-year [170] or 1-year [171] mortality in CVD patient samples. Another sample following postmenopausal women for 4 years found that hostility was associated with recurrent nonfatal myocardial infarctions, but not CVD mortality risk [172].

These discrepancies were largely reconciled when investigators decomposed hostility into 6 components and found that only some were predictive of earlier mortality. In particular, aggressive responding, hostile affect, and cynicism predicted nearly 30-year all-cause mortality in lawyers, while hostile attributions, social avoidance, and "other" hostile items did not [173]. Supporting this were studies linking follow-up mortality in mixed-gender older samples to suspiciousness [174], which is similar to cynicism, and to social dominance in men (defined by cutting off and speaking over an interviewer) [175], which is similar to aggressive responding. When revised scales focusing on these components were utilized, they predicted 15-year CVD mortality in CVD patients [176] and all-cause and CVD mortality in other large cohorts [117, 177, 178]. As a result, many have concluded that despite earlier mixed findings,

hostility defined by cynicism, interpersonal antagonism, and angry affect confers risk for foreshortened lifespan, particularly via CVD death [47]. In the context of the Big 5 taxonomy, hostility may be thought of as a multifaceted trait blending the angry affect component of Neuroticism with the adversarial interpersonal component of low Agreeableness [161, 179].

4.6. Suppression and the Grossarth-Maticeck Personality Types. Suppression, sometimes referred to as emotional or anger suppression or antiemotionality, is a tendency to inhibit the expression of anger and negative emotion [45]. This notion arose out of psychodynamic theory. Although psychodynamic theory is primarily concerned with cognition and mental health [180], not physical health, the health correlates of psychodynamic constructs have been examined [181]. Suppression was first identified in a Yugoslavian cohort as predictive of all-cause and CVD death over 10 years, but was particularly predictive of cancer death in a study by Grossarth-Maticeck [45]. Grossarth-Maticeck called this the "cancer prone" personality type and developed several additional personality configurations: rational, CVD prone, antisocial, and healthy. A report linking these personality types with the diseases they were purported to predict incited considerable controversy over the validity of Grossarth-Maticeck's data [182]. A review of this debate is beyond the scope of the present paper. However, objections arose over the question of specificity, or the idea that specific personality tendencies are associated with specific diseases processes. Instead, personality was thought to affect a common set of etiologic factors for multiple diseases (i.e., smoking, obesity, and neuroendocrine dysregulation, which are implicated in several disease), which would make it hard to connect specific traits only to certain outcomes. This is generally supported by current understanding of mechanisms linking traits to disease, in Figure 1. However, Figure 1 might also include an arrow directly from personality to disease reflect as-yet unknown mechanisms. To the extent that such mechanisms were trait and disease specific, the so-called "doctrine of specific etiology" would be more plausible.

Nevertheless, subsequent independent research has suggested that some of the Grossarth-Maticeck personality types may play a role in longevity. A study in Japan found that among patients who already had cancer, moderate levels of suppression actually improved survival, compared to low levels [183]. This hints at the possibility that neither extreme (total suppression or failure to suppress any emotions) is salubrious, consistent with other psychological theories on the role of emotional control in general adaptation [184]. Other independent studies have noted deleterious effects of anger suppression on all-cause mortality over 17 years in a community sample [143, 185] and over 6 years in CVD patients [143], although one study in a German cohort found no robust effect for anger control after adjusting for locus of control [130]. A systematic review on repression in cancer survival yielded somewhat inconclusive results [186, 187]. A large French study found that both the CVD-prone and antisocial personalities predicted both all-cause, cardiovascular, and external-cause (i.e., accidents, homicides) mortality [188]. Ultimately, less is understood about the Grossarth-Maticeck types and how they affect longevity than other personality constructs, due to the earlier controversies surrounding them. Nevertheless, evidence suggests they warrant further consideration as dispositional risks for foreshortened lifespan. Table 1 summarizes the evidence for each personality dimension discussed above.

5. Next Steps in Translational and Applied Research

5.1. Beyond the Broad Big 5: Clarifying the Contribution of Specific Personality Traits to Longevity. As we have seen, the Big 5 is a useful taxonomic framework for classifying personality traits and organizing investigations of personality. Yet the Big 5 was designed as a classification system for many personality traits, not as the ultimate level of analysis in personality research. Few biologists, for instance, would stop at grouping animals into reptiles, mammals, birds, and fish—most are interested in questions at lower levels of aggregation in the Linnaean taxonomy. This is the challenge for emerging research on personality and longevity—having spent an era examining the Big 5 themselves, we now need to drill down further to understand exactly what elements of enduring dispositions are associated with greater longevity. This was, in fact, proposed as a general principle of epidemiologic personality research over a decade ago [189], so we are long overdue in putting this idea into practice. How then is one to actually implement this principle in studies?

In some cases, Big 5 instruments come with built-in subscales for specific traits. For instance, the 240 item NEO-Personality Inventory Revised (NEO-PI R) [79] was designed to capture the Big 5 general dimensions, as well as 30 more specific personality traits subordinate to the Big 5. Far more studies have used abbreviated Big 5 measures, such as the NEO-Five Factor Inventory (NEO-FFI), which was designed originally only to capture the Big 5 broad composites [79]. However, in some cases like the NEO-FFI, subsequent psychometric work identified subscales reflecting specific constituent traits of each of the Big 5 [190, 191] useful in health studies [66, 192].

In other instances, one includes a measure of broad personality dimensions that has not yet been disaggregated into more specific traits. In these cases, there are two primary options. The first approach is to use expert knowledge and theory to select items reflecting a specific trait, and combine these items into a scale. Examples of this approach include a scale reflecting lack of self-discipline from items off the impulsiveness, excitement seeking, and low deliberation scales of the NEO-PI R [110], creating a Neuroticism scale from items measuring negative affect on the MMPI [193, 194], and examining discrete emotions using items on the NEO-PI R [195]. A basic test of whether the effort is successful is whether the resulting scales are internally consistent, and whether they are associated with theoretically expected outcomes; auxiliary datasets may be helpful to verify this. For instance, one project developed scales for a number of traits from an interpersonal theory of personality and found that they correlated as expected with scales in an

Table 1: Summary of studies in personality and longevity.

Personality dimension	Summary of findings	Strength of overall evidence
Conscientiousness	Numerous studies report reduced risk of all-cause mortality across diverse samples	Strong
Openness	Fewer studies, but results suggest reduced risk of all-cause and possibly CVD mortality	Modest
Extraversion	Results somewhat mixed, with findings of reduced, increased, and no mortality risk	Inconsistent
Optimism	Many studies suggesting reduced risk for all-cause, cardiovascular, and in some cases cancer mortality	Strong
Neuroticism	Some studies report increased, while other report decreased or no risk for all-cause and CVD mortality	Inconsistent
Control	Fewer studies, but findings tend to suggest reduced risk of all-cause mortality	Modest
Agreeableness	Fewer studies, with no few substantial effects	Weak
Hostility	Many studies, finding increased risk for all-cause and CVD mortality, driven by interpersonal antagonism and angry emotion	Strong
Type D	Modest number of studies in CVD populations, most finding increased CVD mortality risk	Strong
Suppression	Fewer studies, controversial interpretations of data, some studies suggest increased risk particularly for cancer mortality, others inconclusive	Inconsistent

auxiliary dataset that were directly designed to capture these traits [196]. A variation of this theoretically driven approach has derived measures of similarity to theoretically important personality profiles or trait configurations, such as Type D or the "undercontrolled" personality, using general Big 5 scales, with good success in identifying personality-health links [51, 197]. Another variant is descriptive categorizations of individuals' standing on combinations of the Big 5 called "styles" [52], although different analytic approaches to this problem exist.

If there is no theory strong enough to suggest good a priori subscales, one may take an empirical approach by using factor analysis or some other sort of multivariate structural technique. Details on the statistical disaggregation of broad personality traits are provided in [198]; once a reliable representation of specific traits has been found, those traits can be examined alone and in conjunction to better understand the elements of a Big 5 dimension driving an outcome. An important note is that modeling the simultaneous effects of several highly correlated facets can create analytic and interpretive challenges. For instance, gregariousness with sociability partialed out or positive affect with optimism partialed out are difficult characteristics to conceptualize, even though they can be statistically created. Thus, researchers who decompose the Big 5 tend to examine each facet separately, or create linear combinations of facets [63, 192], in order to isolate personality effects on health. Next, we turn to another intriguing challenge facing researchers: the interface of personality and socioeconomic inequalities in longevity.

5.2. Clarifying the Personality-SES Interface in Longevity. While most of the studies above controlled for education as a proxy measure of SES, very few focused on issues outlined at the end of Section 2 about the role of personality in socioeconomic inequalities in longevity. Personality analyzed outside of social context—or social inequalities analyzed devoid of personal characteristics—provides only one piece of the puzzle of longevity [199]. It is important for personality epidemiology [189], social epidemiology [200], and life course epidemiology [201] to continue building bridges and mounting studies integrating each of their respective foci.

At least three conceptual models may guide this line of research [199, 202]. The *compensatory-cumulative* model suggests that personality traits may independently add to or detract from mortality risk conferred by disadvantaged social environments, and/or the benefits conferred by social advantage. The *indirect selection* hypothesis originally suggested that persons with certain characteristic self select into lower SES, meaning health inequalities are an indirect effect of this selection process [203]. More recent formulations of indirect selection have argued that the relationship between personality and socioeconomic circumstances is mutually reinforcing over time and must be viewed as a bidirectional process in which personality and SES mutually reinforce one another over time [62]. As a result, personality may explain some of the social inequalities in longevity. Finally, the *selective vulnerability* hypothesis suggests that social adversity engenders more health problems in those with vulnerable personality configurations. Each model is presented diagrammatically in Figure 3.

Limited studies on personality, SES, and longevity to date suggest a mild degree of confounding between mortality risk related to Neuroticism and low control, and mortality risk related to low social position (supporting indirect selection). These traits thus far explain less than 20% of the associations between low-SES and mortality [62, 129, 204]. In these studies, both personality and SES still exert independent mortality risks when confounding is controlled. Therefore, despite the presence of indirect selection, the mortality risk of low SES can still be partially offset by adaptive personality traits, and vice versa (a compensatory cumulative model).

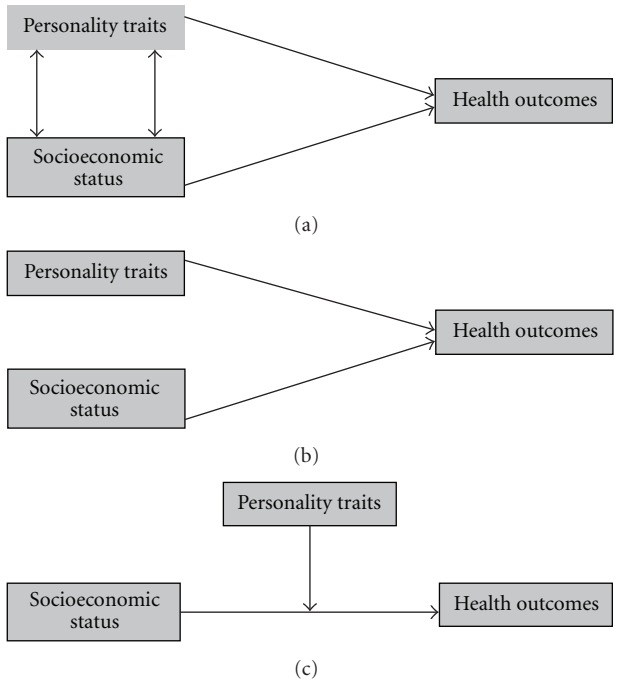

Figure 3: The indirect selection (a), compensatory-cumulative (b), and vulnerability (c) models of the personality-SES interface in health.

Studies are rarely powered to detect interactions between personality and SES however, making it difficult to test the selective vulnerability model adequately. One study did find that mortality risk was reduced by sense of coherence only at higher levels of SES [205], perhaps because such individuals have the resources, in addition to the belief, that they can exert control. Different models may also characterize different personality traits and outcome [32, 206–210]. Another area for further study is the interface between IQ, personality, and SES links to longevity: while mortality risk due to low IQ is partially associated with mortality risk due to low SES, the role of maladaptive personality traits in these associations has been rarely studied [37].

5.3. Comprehensive Measurement of Personality in Health Studies. As noted above, there are limitations to both overly general measures of personality that aggregate across several traits (i.e., only some of the content may be relevant to a given health outcome), but also to overly specific traits (i.e., selective focus on one narrow aspect of personality out of context of other traits). The solution to this problem is to employ a personality measurement tool that taps broad personality dimensions in such a way that they can be broken down into narrow, more constituent facets [189]. In other words, one can use a comprehensive measure of the broad Big 5 dimensions to avoid omitting large spans of personality variation, and then statistically disaggregate the composite Big 5 scales into subscales reflecting more specific traits such as hostility and optimism.

A related issue concerns how personality information is used. In addition to constructing dimensional trait scores, it is also possible to examine configuration, or profiles of multiple traits. Classically, such configurations have been called *personality types,* and more recently joint standing on two Big 5 dimensions at a time has been studied under the moniker of *personality styles* [52]. Categorizing continuous personality dimensions is often done to examine the effects of a certain trait configurations. However, the resulting categories may reflect relatively heterogeneous groups of people, few of whom resemble the prototype personality profile. A solution to this problem has been to generate personality profiles of interest, then measure each individual's similarity to these profiles on a continuous or dimensional scale [51]. A person's distance to a particular pathologic configuration of multiple traits is then collapsed into one summary dimension. Rather than modeling the association of absolute levels of different traits and mortality, one models similarity to a particularly trait configuration as a risk factor. It is also important to examine nonlinearities and trait interactions [211, 212] in predicting mortality, a notion supported by the evolutionary theory that no single trait at high or low levels is likely to be globally adaptive [213].

5.4. Clarifying Mechanisms of Action. Studies to date have often attempted to identify the links between personality and longevity by focusing on health behaviors. While major health behaviors such as poor diet, inactivity, alcohol consumption, and lack of exercise have received general support as mediators of personality-longevity links, three questions remain. First, other health behaviors may play a role, yet are rarely measured. An example is health service utilization, the many determinants of which [214] may include personality [215–217]. Psychological processes at the interface of cognition and behavior, such as health decision-making and risk perception, are also correlated with personality [29]. Definitive studies on this have yet to be conducted, however. Second, the increasing inclusion of biomarkers in personality-longevity studies is beginning to illuminate biological pathways such as systemic inflammation [65, 66, 218, 219] and artery calcification [220, 221], but many other pathways are theoretically plausible. Particularly intriguing is the possibility that common genetic bases may lead to the associations between some personality phenotypes and longevity [37]. Third, the relative strength of each mediator is far from universally established. Mediational confounding by unobserved pathways may occur in studies that include only one a few mediators, making it difficult to gauge how much personality risk is transmitted through different pathways. Finally, the strength of different pathways is likely to differ across populations. The pathways linking personality to longevity are thus only vaguely understood as a partial function of common health behaviors and largely unknown biological mechanisms. Table 2 lists a number of potential behavioral and biological mechanisms.

Methodologically, an interesting framework has recently been articulated for attaching causal (rather than just prospective associative) interpretations to mediation analyses [222]. A key requirement is control for confounders of the mediation pathway—just as a direct main effect may confounded by unobserved factors, so may a mediated

Table 2: Potential mediators of personality-longevity associations.

Behavioral	Biological
Alcohol use	Cortisol, markers of HPA Axis function
Cigarette smoking and tobacco use	Epinephrine/Norepinephrine, markers of SAM Axis Function
Physical activity and exercise	Inflammatory Markers
BMI	Adaptive Immune Markers (Natural Killer Cells, White Blood Cell Count)
Illicit drug use; abuse of prescription medications	Markers of oxidative stress
Specific eating habits	Lipids and sclerotic activity
Accidents	Neurodegeneration associated with HPA axis dysregulation
Sexual risk behavior	Markers of metabolic dysregulation such as insulin resistance
Health risk perception and evaluation	Genetic Markers of Disease and Longevity
Health service use	Telomere shortening and other markers of impaired genetic replication
Health care adherence; communication with health care providers; trust in health care delivery system	Mechanisms of pain and somatic sensitivity
Sleep behavior; dental hygiene	
Complementary and Alternative Medicine Use	
Health beliefs and somatic sensitivity	
Recruitment of social support; informal help-seeking	

effect [222]. Other issues involve the functional form of the mediational pathways (linear versus nonlinear), getting correct standard error estimates for quantities such as the indirect effect, and the complications posed by nonnormal outcome distributions. Equivalent methods for computing indirect effects for linear equations (i.e., the products versus differences of relevant coefficients) are not equivalent with one or more nonlinear (i.e., logistic, survival) models in the mediation system [223]. One solution is to simply use the mediated fraction [224], which is statistically identical to the confounded fraction above but simply assumes that the third variable is a mediator, rather than confounder [225]. Separate examinations of mediating paths in different strata may help identify moderated mediation, reflecting different causal chains in different populations [226].

Understanding pathways between personality and longevity is important if one wishes to conduct targeted interventions in certain at-risk personality populations. To the extent that numerous pathways are operating, however, interventions in at-risk personality populations would need to attempt to modify multiple mediators [227]. Another, possibly more cost-effective, option is to focus on changing the pathogenic personality tendencies in question, which we consider below.

5.5. Integrating Personality into Personalized Medicine. Interest in individual differences in health risk and treatment response has gained great traction over the last decade under the rubric of *personalized medicine* [228]. The goal of personalized medicine is to individualize care according to the unique characteristics of the patient. Typically, these are demographic or biomedical dimensions of individual differences, such as age, lipid levels, and disease conditions. However, the increasing interest in personalized medicine has really come on the back of GWAS studies, under the rationale that patient risk can be prognosticated with increasing precision as more is learned about the roles of genes in disease [228]. Based on these forecasts, an individual may receive closer monitoring for early signs and symptoms of some disease(s), receive a particular preventive treatment that has benefited others like him or her, and/or have existing treatment adapted to fit better with his or her characteristics [229]. However, personality is conspicuously absent in formulations of personalized medicine. Given the links between personality and health, an obvious opportunity exists for translational research integrating personality phenotype into personalized medicine.

Personality phenotype may complement genetic information in several ways. First, since health is a complex function of behavior and biology, incorporating information from both domains into prediction equations should improve forecasting accuracy. As noted above, only some of the associations between personality and longevity are accounted for by commonly measured health behaviors. This means that personality may provide *incremental predictive power* in forecasting health, above and beyond the data contained by an individual's health behavior, demographic, or genetic profile. Psychological tests are able to predict behavioral criteria with an accuracy similar to which medical tests predict medical criteria [230], and since health outcomes are multidetermined by biological and behavioral causes, combining assessments of these factors may be cost-effective. Second, genetic risk profiles typically select specific candidate genes that have been shown to predict health outcomes. There is some concern that this approach has not provided the additional predictive power hoped for, particularly relative to the costs of genetic screening [231]. In contrast, personality information can range from quite specific to very general and is relatively low cost given information it may add to health risk profiling. Third, genetic information without data on environmental experiences may weaken the extent to which genes can be said to have implications for health. Personality phenotype reflects the results of gene-environment interaction and encodes environmental variance at a broad behavioral level. Fourth, just as in psychometric measurement, genetic risk profiling can be error-prone [232]. Integrating genotypic with phenotypic measurement may offset the potential cost of measurement error in either by providing supplemental data related to important outcomes.

An obvious question for translational research is how to incorporate personality trait assessment in standard medical practice, particularly in primary care where most diseases, including the chronic diseases of aging, are managed. Ideas range from including relatively specific screens for traits such as hostility [233], to modern initiatives to incorporate broader personality measures in electronic medical record (EMR) systems. Short Big 5 measures [234, 235] might be used as potential screeners. Individuals scoring above or below empirically identified cut-offs might spark a more thorough, detailed subsequent assessment. This serial testing strategy has proven to be quite effective in medicine and can be thought of as a 2-step algorithm where one minimizes false negatives in the first step, and then weeds out false positives on the second step [236]. Methods exist for identifying cut points for sequential screening scales according to the outcome, scale, and whether one wishes to maximize net sensitivity or specificity from the two-step process [236]. Other methods exist to determine the point at which a continuous risk factor shows the steepest rise for several risks [237].

If indicated, the second, more detailed step of personality assessment in primary care would accomplish three things. First, the additional personality data would increase the accuracy of individual risk forecasting models for outcomes of interest. The idea would be to have a comprehensive measurement of risk for several potentially serious diseases that can be predicted based on combinations of items from the broader personality inventory. Second, a more detailed personality assessment would provide the physician with a wealth of psychosocial data that would otherwise take a long time to gather, preparing the physician for what s/he may expect behaviorally from a particular patient. This would require assessment infrastructures which interpret the personality data for the physicians, such as computer-generated reports for health practitioners, and available consultation with psychologists. In this sense, personality assessment may be thought of as further automation of the primary care practice of background screening or history taking, which typically relies on self-reported information delivered to the physician during an interview. Formal personality assessment would gather more systematic and broader information, in a more time-effective way, enabling physicians to better know and understand their patients as individuals. Third, this type of information is likely to help guide and tailor treatment planning, enhancing the probability of successful care. In general, personality assessment could improve the provision of patient-centered care because the physicians better understand how to approach and interact with different kinds of patients. The mere presence of these assessment tools in primary care waiting rooms would convey to patients that the provision of high quality health care is not solely about ordering diagnostic tests, arriving at the correct diagnosis, and prescribing appropriate treatments. It is also about expressing concern and empathy and understanding the patient's perspective [238, 239], activities that we believe could be facilitated by personality assessment.

Finally, it is important to consider the difference between measuring personality and measuring depressive symptoms in primary care using an instrument like the Patient Health Questionnaire (PHQ)-9. Instruments such as the PHQ-9 are case-finders, identifying prevalent cases of depression, rather than screeners, which identify a pool at-risk for incident depression. Certainly, both activities are important. However, if one wants to engage in any sort of preventive care, people with the risk-factors for outcomes must be identified, not just people already suffering from the undesired outcome itself. Personality assessment thus offers a comprehensive screening strategy for psychosocial health risks, not a case-finding strategy for people with prevalent symptoms for one specific form of psychopathology.

Comprehensive Big 5 measures [240] can be used to develop brief screeners for outcomes of interest using the items on these inventories most predictive of those outcomes. Subsequently, the remainder of the inventory can be administered if a second, more complete assessment is desired. Another approach is to develop highly predictive screeners not embedded in any particular existing instrument, and using these in the first stage, followed by a more comprehensive test. Predictive modeling is an active area of quantitative research and numerous techniques have emerged in the last ten years to facilitate the construction of highly predictive scales [241]. Advances in measurement technology, such as the use of informant report and behavioral measures of personality tendencies, might also be considered. The key is to study these possibilities from a translational viewpoint focused on feasibility of implementation. For instance, health care systems such as Kaiser Permanente and Group Health have begun to incorporate general psychosocial information into electronic medical records.

5.6. Intervening to Change Personality Risk Factors. For some time, it was believed that personality was largely fixed after age 30 [242]. Although there is debate about the degree of naturalistic personality stability [8, 243], many now feel that enough documentation of change over the life-course has appeared to justify the study of personality development and change after age 30 [244]. More recent research has shown naturalistic change over the lifespan indicating a pattern of increasing maturity with age, owing to increasing responsibilities such as marriage and child-rearing and increases in work responsibility [7]. Others have argued that changes are due largely to the biology of aging [8]. Personality also changes in neural-degenerative diseases such as Alzheimer's [245]. In addition, psychotherapy [242] or pharmacotherapy [246] may alter personality tendencies. Regardless, there is emerging evidence that naturalistic declines in Conscientiousness and Extraversionover at a 10-year period are associated with worse self-rated health at follow-up [247], suggesting that personality-related interventions warrant serious consideration.

With respect to traits associated with mortality, psychosocial interventions have successfully reduced hostility [248], Type D traits [116], and deficits in social and emotional functioning in CVD patients [249]; in the latter two studies, patients showed improved 9-year survival rates [116]

and reductions in sclerosis in the fundus of the eye [249]. Similar psychosocial treatment has been mounted in cancer patients [250], with one study noting small improvement in sclerosis [249] and another enhanced survival time and lymphocyte breast count in breast cancer patients [251]. Studies examining different type of psychosocial intervention generally found health improvements for behavior therapy, which focuses on altering patterns of thoughts and behavior, but not for psychoanalytic therapy, which focused on developing insight into unconscious motivations [251, 252]. As these studies were conducted prior to the advent of reporting standards for clinical trials, it is not surprising that they provided very little information bearing on therapist allegiance or fidelity. Their conclusions must be regarded as tentative. Other studies have noted that selective serotonin reuptake inhibitors (SSRIs) tend to reduce Neuroticism and improve Extraversion [246, 253] in depressed samples, while mindfulness-based stress reduction reduced Neuroticism and improved Conscientiousness in a study of practicing physicians [254]. Another paper showed that Openness to Experience improves as a result of cognitive training in older persons [255]. While these results are suggestive, considerably more data is needed on interventions targeting risky personality traits. A recent National Institute of Aging workgroup on this issue pointed specifically to the need for research on what risk-prone personality tendencies are most apt to change, in whom, and with which intervention modalities. This is an area of emerging research likely to receive considerable attention in the coming decade.

5.7. Quantifying the Public Health Impact of Personality. Another agenda for personality and longevity research is to quantify the health impact of personality using traditional public health metrics. This is particularly important since personality traits, unlike BMI, annual household income or assets, years of education, or other continuous risk factors, have no inherently meaningful metric. As an initial step, traits can be continuously scaled so that a relative risk (RR) reflects, for instance, the difference in risk between people of 1 or 2 standard deviations (SDs) difference in the level of the trait, or the difference in risk between a person at the 25th and 75th percentile of the trait distribution [256]. If personality were height, the difference between a person at the 25th and 75th percentile of a trait would be like the difference between a person 5′6″ tall and one 6′0″ tall, or a difference in personality that would be qualitatively noticeable.

This logic can be extended to common public health metrics such as population attributable risk (PAR) [62]. PAR combines information about the relative risk of a trait such as low conscientiousness, with the prevalence of that trait in the population [257]. This means that traits that have mild effects on an outcome may have a large public health impact if they are relatively common in the population. The converse is also true: a very large conventional effect size for a trait may not necessarily mean it is important in population health, if the level of trait necessary for the effect is relatively rare. The PAR itself is a proportion, ranging between 0 and 1, which provides the maximum bound by which population mortality can be reduced if the trait is reduced to some reference level in the population. It can be used to pinpoint prevention and intervention avenues that will yield the most benefit to the population, and to derive other epidemiologic estimates such as Number Needed to Treat (NNT), the number of people who must be treated down to a lower level of the trait to prevent one death in the population [257]. PARs are often computed for categorical risk factors, but methods do exist for estimating them based on continuous risk factors such as personality traits [258]. The one study to date estimating hypothetical PARs showed that reducing Neuroticism to −1 SD could decrease population mortality by 13%, controlling for SES [62]. A similar result was observed for raising Conscientiousness to +1 SD in those also high on Agreeableness. By contrast, eliminating smoking could lead to an 8.8% reduction, obesity (independent of physical inactivity) a 4.7% reduction, and inactivity (independent of obesity) a 13% reduction in population mortality. This illustrates the possibility that intervening to change personality traits may bring somewhat greater benefits because one is treating an underlying cause of multiple mediators (e.g., poor health behaviors) rather than targeting only one or a limited number of the intervening factors [259].

A second way the public health impact of personality may be quantified is by drawing an analogy to aging, which is known to be one of the most potent predictors of longevity. Certain traits are associated with accelerated aging, and the amount of acceleration could be quantified. For instance, one study found that a difference of 1 SD in Conscientiousness and a difference of 8.8 years of age were associated with comparable amounts of physician-assessed medical burden as [192]. Another found that Type D personality was associated with elevations in inflammatory cytokines that was comparable to elevations conferred by 10 years of aging [150]. One caveat is that these studies were cross-sectional, so personality effects were compared to *age differences between people*, which is not the same as years of ageing within people. However, a 5-year longitudinal followup of older adults in the Conscientiousness study was able to make this distinction [260] and found that −1 SD in Conscientiousness and −1 SD in agreeableness were equivalent to roughly 4.5 years of chronic disease progression. The authors also placed this in the metric of a common physician-based measure of morbidity, equating it to moderate deterioration in one organ system, or mild deterioration across 2 organ systems [260].

A final public health metric relevant to longevity is the Years of Potential Life Lost (YPLL) associated with, for instance, the 75th versus 25th percentile of a personality trait. A crude way to compute this is to set a reference age, such as the average age of survival achieved by those at the mean of Conscientiousness. The age at death of those below the mean is subtracted from this, and averaged. More elaborate computations can be made from parametric survival models that have accurately specified the distribution of survival time [257]. A number of similar measures, including quality-adjusted life years (QALYs) and disability-adjusted life years (DALYs) could also be computed. Such measures would help

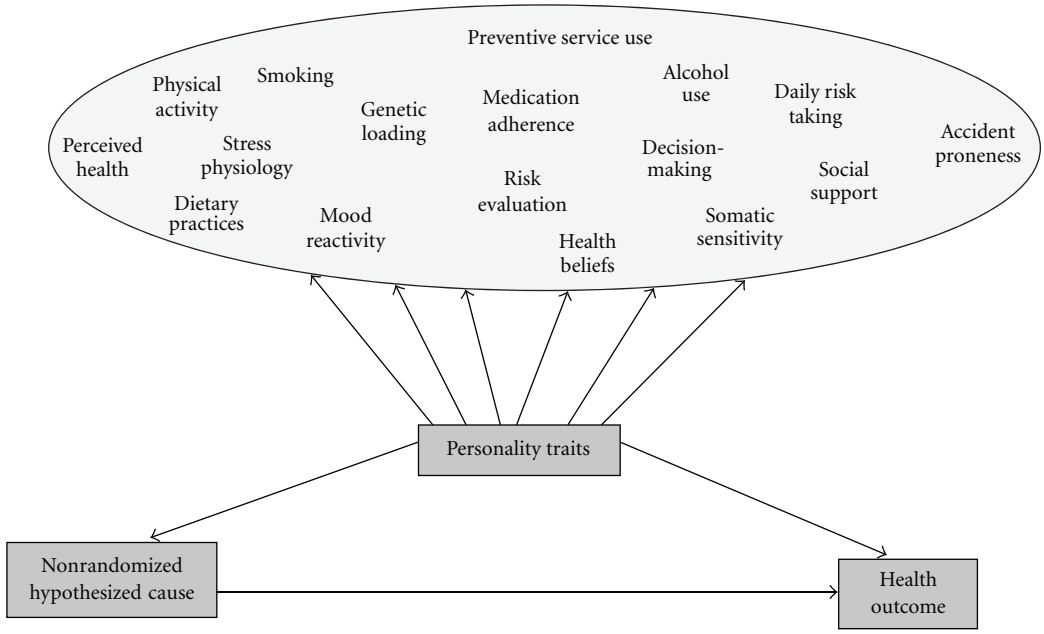

Figure 4: Personality reflects a wide array of unmeasured factors relevant to health that can confound associations between nonrandomized predictors and health outcomes; measuring and adjusting for personality reduces this "healthy subject" bias in a parsimonious fashion.

clarify the individual and population impact of personality traits on longevity.

5.8. Methodologic Uses of Personality to Improve General Research Design. Another implication of personality-longevity associations lays in the design and conduct of general epidemiologic studies. Numerous biases plague observational studies and to some extent randomized clinical trials [261]. Investigators attempt to reduce and control them, with skepticism over observational studies particularly great after they yielded effect estimates for hormone replacement therapy that were actually the opposite of what subsequent randomized trials showed [262]. Many of these biases are psychological or behavioral in nature and related to personality traits. For instance, *volunteer bias*, a form of selection bias, is the tendency for well-adjusted, open, and compliant people to participate in studies [261], a function of personality [263, 264]. *Compliance biases* are another concern. Adherence to medication and study drop-out alike appears to be related to personality characteristics [265–267]. The *healthy subject effect* is a particularly pernicious form of bias in which subjects with generally healthier lifestyles adopt a particular treatment in a nonrandomized study, resulting in unobserved confounding [268]. Personality captures many of the propensities toward healthy lifestyle [269], meaning healthy subject bias may be controlled if personality can be measured. Figure 4 illustrates this concept, which is rooted in recent theories of causal inference [270]. If one can measure a confounder z that captures a wide range of variance in several other unobserved confounders *a-f*, measuring and adjusting for z can remove a large portion of the confounding due to the entire set *a-f*. As a result, use of z may be more cost-effective and analytically tractable in controlling for healthy subject bias. Finally, the *healthy survivor effect* reflects the tendency for findings in samples of older adults to differ from those in younger samples. As personality is related to longevity, it can be used to better understand and adjust for healthy survivor effects. A detailed discussion of epidemiologic biases can be found in [257], and additional methodological work might study personality measurement as a means of controlling many of these biases.

6. Conclusions

Evidence has mounted over the past 30 years implicating personality traits in longevity. The association with longevity of some aspects of personality, such as Conscientiousness, is fairly well established. However, mixed or insufficient data exists for a number of other dispositional factors. Future work will benefit from approaches to personality measurement that allow for both the study of specific traits, and for comprehensive coverage of personality. Our focus has been on longevity. Two related issues are quality of life and the *compression of morbidity* that has come with extended lifespans over the 20th Century. This refers to the aspiration (already partially achieved) to confine severe disability, life-threatening disease burden, and other indicators of very poor quality of life to the very end of life, so that most of the extended lifespan can be lived with quality of life. Since considerable research suggests that personality is linked to a number of quality of life measures [122, 271, 272], even controlling for objective disease burden [123, 273], personality assessment may play a role in prognosticating and improving quality of life in old age as well. Better understanding

of the interface between personality and sociostructural influences on longevity is also needed. Personality may also help general health researchers adjust for a number of usually unmeasured behavioral and psychological biases in their studies. Finally, personality traits offer comprehensive information that could be used as personalized medicine begins to gain traction. Translational research is needed to determine how personality information can best be leveraged by personalized medicine.

References

[1] H. S. Friedman and L. R. Martin, *The Longevity Project: Surprising Discoveries for Health and Long Life from the Landmark Eight-Decade Study*, Penguin, New York, NY, USA, 2001.

[2] W. Mischel and Y. Shoda, "Integrating dispositions and processing dynamics within a unified theory of personality: the cognitive-affective personality system," in *Handbook of Personality: Theory and Research*, L. A. Pervin and O. P. John, Eds., pp. 197–218, Guilford Press, New York, NY, USA, 1999.

[3] W. Revelle, Psychology Faculty Profile: William Revelle, 2011, http://www.wcas.northwestern.edu/psych/people/faculty/faculty_individual_pages/Revelle.htm.

[4] L. R. Goldberg, "The structure of phenotypic personality traits," *American Psychologist*, vol. 48, no. 1, pp. 26–34, 1993.

[5] R. R. McCrae and P. T. Costa Jr., "Personality trait structure as a human universal," *American Psychologist*, vol. 52, no. 5, pp. 509–516, 1997.

[6] B. W. Roberts and W. F. DelVecchio, "The rank-order consistency of personality traits from childhood to old age: a quantitative review of longitudinal studies," *Psychological Bulletin*, vol. 126, no. 1, pp. 3–25, 2000.

[7] B. W. Roberts, K. E. Walton, and W. Viechtbauer, "Patterns of mean-level change in personality traits across the life course: a meta-analysis of longitudinal studies," *Psychological Bulletin*, vol. 132, no. 1, pp. 1–25, 2006.

[8] P. T. Costa Jr. and R. R. McCrae, "Age changes in personality and their origins: comment on Roberts, Walton, and Viechtbauer (2006)," *Psychological Bulletin*, vol. 132, no. 1, pp. 26–28, 2006.

[9] S. South and R. F. Krueger, "An interactionist perspective on genetic and environmental contributions to personality," *Personality and Social Psychology Compass*, vol. 2, no. 2, pp. 929–948, 2008.

[10] A. Terracciano, T. Tanaka, A. R. Sutin et al., "BDNF Val66Met is associated with introversion and interacts with 5-HTTLPR to influence neuroticism," *Neuropsychopharmacology*, vol. 35, no. 5, pp. 1083–1089, 2010.

[11] A. Terracciano, T. Tanaka, A. R. Sutin et al., "Genome-wide association scan of trait depression," *Biological Psychiatry*, vol. 68, no. 9, pp. 811–817, 2010.

[12] R. Plomin and A. Caspi, "DNA and personality," *European Journal of Personality*, vol. 12, no. 5, pp. 387–407, 1998.

[13] R. Plomin and A. Caspi, "Behavioral genetics and personality,," in *Handbook of Personality: Theory and Research*, pp. 251–276, Guilford Press, New York, NY, USA, 1999.

[14] B. W. Roberts and J. J. Jackson, "Sociogenomic personality psychology," *Journal of Personality*, vol. 76, no. 6, pp. 1523–1544, 2008.

[15] D. J. Ozer, "Four principles for personality assessment," in *Handbook of Personality: Theory and Research*, A. Lawrence, Ed., pp. 671–688, Guilford Press, New York, NY, USA, 2nd edition, 1999.

[16] D. Dunning, C. Heath, and J. M. Suls, "Flawed self-assessment implications for health, education, and the workplace," *Psychological Science in the Public Interest, Supplement*, vol. 5, no. 3, pp. 69–106, 2004.

[17] R. Bagby, M. B. Marshall, A. S. Bury, J. R. Bacchiochi, and L S. Miller, "Assessing underreporting and overreporting response styles on the MMPI-2," in *MMPI-2: A Practitioner's Guide*, J. N. Butcher, Ed., pp. 39–69, American Psychological Association, Washington, DC, USA, 2006.

[18] S. R. Millis and C. T. Volinsky, "Assessment of response bias in mild head injury: beyond malingering tests," *Journal of Clinical and Experimental Neuropsychology*, vol. 23, no. 6, pp. 809–828, 2001.

[19] J. Barnett, "Sensitive questions and response effects: an evaluation," *Journal of Managerial Psychology*, vol. 13, no. 1-2, pp. 63–76, 1998.

[20] N. C. Schaeffer, "Asking questions about threatening topics: a selective overview," in *The Science of Self Report: Implications for Research and Practice*, A. Stone, J. S. Turkkan, C. A. Bachrach, J. B. Jobe, H. S. Kurtzman, and V. S. Cain, Eds., pp. 105–121, Lawrence Erlbaum Associates, Mahway, NJ, USA, 2000.

[21] D. C. Funder, "On the accuracy of personality judgment: a realistic approach," *Psychological Review*, vol. 102, no. 4, pp. 652–670, 1995.

[22] S. Vazire, "Who knows what about a person? The self-other knowledge asymmetry (SOKA) model," *Journal of Personality and Social Psychology*, vol. 98, no. 2, pp. 281–300, 2010.

[23] R. E. McGrath, M. Mitchell, B. H. Kim, and L. Hough, "Evidence for response bias as a source of error variance in applied assessment," *Psychological Bulletin*, vol. 136, no. 3, pp. 450–470, 2010.

[24] R. R. McCrae and P. T. Costa Jr., "Social desirability scales: more substance than style," *Journal of Consulting and Clinical Psychology*, vol. 51, no. 6, pp. 882–888, 1983.

[25] J. E. Kurtz, S. J. Tarquini, and E. A. Iobst, "Socially desirable responding in personality assessment: still more substance than style," *Personality and Individual Differences*, vol. 45, no. 1, pp. 22–27, 2008.

[26] H. S. Friedman, "Long-term relations of personality and health: dynamisms, mechanisms, tropisms," *Journal of Personality*, vol. 68, no. 6, pp. 1089–1107, 2000.

[27] H. S. Friedman, "The multiple linkages of personality and disease," *Brain, Behavior, and Immunity*, vol. 22, no. 5, pp. 668–675, 2008.

[28] T. Bogg and B. W. Roberts, "Conscientiousness and health-related behaviors: a meta-analysis of the leading behavioral contributors to mortality," *Psychological Bulletin*, vol. 130, no. 6, pp. 887–919, 2004.

[29] K. E. Flynn and M. A. Smith, "Personality and health care decision-making style," *Journals of Gerontology—Series B Psychological Sciences and Social Sciences*, vol. 62, no. 5, pp. P261–P267, 2007.

[30] S. E. Hampson, J. A. Andrews, M. Barckley, E. Lichtenstein, and M. E. Lee, "Conscientiousness, perceived risk, and risk-reduction behaviors: a preliminary study," *Health Psychology*, vol. 19, no. 5, pp. 496–500, 2000.

[31] S. E. Hampson, J. A. Andrews, M. Barckley, E. Lichtenstein, and M. E. Lee, "Personality traits, perceived risk, and risk-reduction behaviors: a further study of smoking and radon," *Health Psychology*, vol. 25, no. 4, pp. 530–536, 2006.

[32] J. Neeleman, J. Ormel, and R. V. Bijl, "The distribution of psychiatric and somatic III health: associations with personality and socioeconomic status," *Psychosomatic Medicine*, vol. 63, no. 2, pp. 239–247, 2001.

[33] J. Neeleman, S. Sytema, and M. Wadsworth, "Propensity to psychiatric and somatic ill-health: evidence from a birth cohort," *Psychological Medicine*, vol. 32, no. 5, pp. 793–803, 2002.

[34] G. R. Pierce, B. Lakey, I. G. Sarason, and B. R. Sarason, *Sourcedbook of Social Support and Personality*, Plenum, New York, NY, USA, 1987.

[35] S. C. Segerstrom, "Personality and the immune system: models, methods, and mechanisms," *Annals of Behavioral Medicine*, vol. 22, no. 3, pp. 180–190, 2000.

[36] S. C. Segerstrom, "Individual differences, immunity, and cancer: lessons from personality psychology," *Brain, Behavior, and Immunity*, vol. 17, no. 1, pp. S92–S97, 2003.

[37] I. J. Deary, A. W. Weiss, and G. D. Batty, "Intelligence and personality as predictors of illness and death: how researchers in differential psychology and chronic disease epidemiology are collaborating to understand and address health inequalities," *Psychological Science in the Public Interests*, vol. 11, no. 2, pp. 53–79, 2010.

[38] B. W. Roberts, "Contextualizing personality psychology," *Journal of Personality*, vol. 75, no. 6, pp. 1071–1082, 2007.

[39] R. D. Conger and M. B. Donnellan, "An interactionist perspective on the socioeconomic context of human development," *Annual Review of Psychology*, vol. 58, pp. 175–199, 2007.

[40] M. B. Donnellan, K. J. Conger, K. K. McAdams, and T. K. Neppl, "Personal characteristics and resilience to economic hardship and its consequences: conceptual issues and empirical illustrations," *Journal of Personality*, vol. 77, no. 6, pp. 1645–1676, 2009.

[41] A. Caspi, B. W. Roberts, and R. L. Shiner, "Personality development: stability and change," *Annual Review of Psychology*, vol. 56, pp. 453–484, 2005.

[42] C. G. DeYoung, "Toward a theory of the big five," *Psychological Inquiry*, vol. 21, no. 1, pp. 26–33, 2010.

[43] R. R. McCrae and P.T. Costa Jr., "A five-factor yheory of personality," in *Handbook of Personality: Theory and Research*, L. A. Pervin and O. P. John, Eds., pp. 139–153, Guilford Press, New York, NY, USA, 1999.

[44] G. N. Marshall, C. B. Wortman, R. R. Vickers, J. W. Kusulas, and L. K. Hervig, "The five-factor model of personality as a framework for personality-health research," *Journal of Personality and Social Psychology*, vol. 67, no. 2, pp. 278–286, 1994.

[45] R. Grossarth-Maticek, J. Bastiaans, and D. T. Kanazir, "Psychosocial factors as strong predictors of mortality from cancer, ischaemic heart disease and stroke: the Yugoslav prospective study," *Journal of Psychosomatic Research*, vol. 29, no. 2, pp. 167–176, 1985.

[46] S. V. Paunonen and D. N. Jackson, "What is beyond the big five? Plenty!," *Journal of Personality*, vol. 68, no. 5, pp. 821–835, 2000.

[47] T. W. Smith, K. Glazer, J. M. Ruiz, and L. C. Gallo, "Hostility, anger, aggressiveness, and coronary heart disease: an interpersonal perspective on personality, emotion, and health," *Journal of Personality*, vol. 72, no. 6, pp. 1217–1270, 2004.

[48] J. Heckhausen and R. Schulz, "A life-span theory of control," *Psychological Review*, vol. 102, no. 2, pp. 284–304, 1995.

[49] L. R. Goldberg, "An alternative "Description of Personality": the big-five factor structure," *Journal of Personality and Social Psychology*, vol. 59, no. 6, pp. 1216–1229, 1990.

[50] D. S. Ones, C. Viswesvaran, and S. Dilchert, "Personality at work: raising awareness and correcting misconceptions," *Human Performance*, vol. 18, no. 4, pp. 389–404, 2005.

[51] B. Chapman and L. G. Goldberg, "Replicability and 40-year predictive power of childhood ARC types," *Journal of Personality and Social Psychology*. In press.

[52] P.T. Costa Jr. and R. L. Piedmont, "Multivariate assessment: NEO-PI R profiles of madeline G," in *Paradigms of Personality Assessment*, J. S. Wiggins, Ed., pp. 262–280, Guilford Press, New York, NY, USA, 2003.

[53] B. W. Roberts, O. S. Chernyshenko, S. Stark, and L. R. Goldberg, "The structure of conscientiousness: an empirical investigation based on seven major personality questionnaires," *Personnel Psychology*, vol. 58, no. 1, pp. 103–139, 2005.

[54] H. Iwasa, Y. Masui, Y. Gondo, H. Inagaki, C. Kawaai, and T. Suzuki, "Personality and all-cause mortality among older adults dwelling in a japanese community: a five-year population-based prospective cohort study," *American Journal of Geriatric Psychiatry*, vol. 16, no. 5, pp. 399–405, 2008.

[55] R. S. Wilson, C. F. Mendes De Leon, J. L. Bienias, D. A. Evans, and D. A. Bennett, "Personality and mortality in old age," *Journals of Gerontology—Series B Psychological Sciences and Social Sciences*, vol. 59, no. 3, pp. P110–P116, 2004.

[56] P. S. Fry and D. L. Debats, "Perfectionism and the five-factor personality traits as predictors of mortality in older adults," *Journal of Health Psychology*, vol. 14, no. 4, pp. 513–524, 2009.

[57] A. J. Christensen, S. L. Ehlers, J. S. Wiebe et al., "Patient personality and mortality: a 4-year prospective examination of chronic renal insufficiency," *Health Psychology*, vol. 21, no. 4, pp. 315–320, 2002.

[58] M. L. Kern and H. S. Friedman, "Do conscientious individuals live longer? A quantitative review," *Health Psychology*, vol. 27, no. 5, pp. 505–512, 2008.

[59] B.W. Roberts, N. R. Kuncel, R. Shiner, A. Caspi, and L. Goldberg, "The power of personality: the comparative validity of personality traits, socioeconomic Status, and Cognitive Ability for Predicting Important Life Outcomes," *Perspectives in Psychological Science*, vol. 4, no. 2, pp. 313–346, 2007.

[60] S. C. Segerstrom, J. O. Castañeda, and T. E. Spencer, "Optimism effects on cellular immunity: testing the affective and persistence models," *Personality and Individual Differences*, vol. 35, no. 7, pp. 1615–1624, 2003.

[61] T. E. Moffitt, L. Arseneault, D. Belsky et al., "A gradient of childhood self-control predicts health, wealth, and public safety," *Proceedings of the National Academy of Sciences of the United States of America*, vol. 108, no. 7, pp. 2693–2698, 2011.

[62] B. P. Chapman, K. Fiscella, I. Kawachi, and P. R. Duberstein, "Personality, socioeconomic status, and all-cause mortality in the United States," *American Journal of Epidemiology*, vol. 171, no. 1, pp. 83–92, 2010.

[63] A. Weiss and P. T. Costa Jr., "Domain and facet personality predictors of all-cause mortality among medicare patients aged 65 to 100," *Psychosomatic Medicine*, vol. 67, no. 5, pp. 724–733, 2005.

[64] L. R. Martin, H. S. Friedman, and J. E. Schwartz, "Personality and mortality risk across the life span: the importance of conscientiousness as a biopsychosocial attribute," *Health Psychology*, vol. 26, no. 4, pp. 428–436, 2007.

[65] A. R. Sutin, A. Terracciano, B. Deiana et al., "High Neuroticism and low Conscientiousness are associated with interleukin-6," *Psychological Medicine*, vol. 40, no. 9, pp. 1485–1493, 2010.

[66] B. P. Chapman, E. van Wijngaarden, C. L. Seplaki, N. Talbot, P. Duberstein, and J. Moynihan, "Openness and conscientiousness predict 34-SSweek patterns of Interleukin-6 in older persons," *Brain, Behavior, and Immunity*, vol. 25, no. 4, pp. 667–673, 2011.

[67] T. B. Harris, L. Ferrucci, R. P. Tracy et al., "Associations of elevated interleukin-6 and C-reactive protein levels with mortality in the elderly," *American Journal of Medicine*, vol. 106, no. 5, pp. 506–512, 1999.

[68] T. L. Gruenewald, T. E. Seeman, C. D. Ryff, A. S. Karlamangla, and B. H. Singer, "Combinations of biomarkers predictive of later life mortality," *Proceedings of the National Academy of Sciences of the United States of America*, vol. 103, no. 38, pp. 14158–14163, 2006.

[69] R. R. McCrae, "Openness to experience: expanding the boundaries of factor V," *European Journal of Personality*, vol. 8, no. 4, pp. 251–272, 1994.

[70] C. G. DeYoung, N. A. Shamosh, A. E. Green, T. S. Braver, and J. R. Gray, "Intellect as distinct from openness: differences revealed by fMRI of working memory," *Journal of Personality and Social Psychology*, vol. 97, no. 5, pp. 883–892, 2009.

[71] A. Weiss, MJ Adams, A. G. M. S. Widdig, and M. S. Gerald, "Rhesus macaques (Macaca mulatta) as living fossils of hominid personality and subjective well-being," *Journal of Comparative Psychology*, vol. 135, pp. 72–83, 2011.

[72] C. R. Jonassaint, S. H. Boyle, R. B. Williams, D. B. Mark, I. C. Siegler, and J. C. Barefoot, "Facets of openness predict mortality in patients with cardiac disease," *Psychosomatic Medicine*, vol. 69, no. 4, pp. 319–322, 2007.

[73] N. A. Turiano, A. Spiro, and D. K. Mcroczek, "Openness to experience and mortality in men: analysis of trait and facets," under review.

[74] G. E. Swan and D. Carmelli, "Curiosity and mortality in aging adults: a 5-year follow-up of the Western Collaborative Group Study," *Psychology and Aging*, vol. 11, no. 3, pp. 449–453, 1996.

[75] P. R. Duberstein, B. P. Chapman, H. A. Tindle et al., "Personality and risk for Alzheimer's disease in adults 72 years of age and older: a 6-year follow-up," *Psychology and Aging*, vol. 26, no. 2, pp. 351–362, 2011.

[76] A. R. Cappola, Q. L. Xue, L. Ferrucci, J. M. Guralnik, S. Volpato, and L. P. Fried, "Insulin-like growth factor I and interleukin-6 contribute synergistically to disability and mortality in older women," *Journal of Clinical Endocrinology and Metabolism*, vol. 88, no. 5, pp. 2019–2025, 2003.

[77] R. E. Lucas, E. Diener, A. Grob, E. M. Suh, and L. Shao, "Cross-cultural evidence for the fundamental features of extraversion," *Journal of Personality and Social Psychology*, vol. 79, no. 3, pp. 452–468, 2000.

[78] W. Revelle, M. S. Humphreys, L. Simon, and K. Gilliand, "The interactive effect of personality, time of day, and caffeine: a test of the arousal model," *Journal of Experimental Psychology: General*, vol. 109, no. 1, pp. 1–31, 1980.

[79] P. T. Costa Jr. and R. R. McCrae, *Revised NEO Personality Inventory and NEO Five Factor Inventory: Professional Manual*, Psychological Assessment, Odessa, Fla, USA, 1992.

[80] G. B. Ploubidis and E. Grundy, "Personality and all cause mortality: evidence for indirect links," *Personality and Individual Differences*, vol. 47, no. 3, pp. 203–208, 2009.

[81] B. A. Shipley, A. Weiss, G. Der, M. D. Taylor, and I. J. Deary, "Neuroticism, extraversion, and mortality in the UK health and lifestyle survey: a 21-year prospective cohort study," *Psychosomatic Medicine*, vol. 69, no. 9, pp. 923–931, 2007.

[82] H. J. Eysenck, R. Grossarth-Maticek, and B. Everitt, "Personality, stress, smoking, and genetic predisposition as synergistic risk factors for cancer and coronary heart disease," *Integrative Physiological and Behavioral Science*, vol. 26, no. 4, pp. 309–322, 1991.

[83] B. Chapman, K. Fiscella, P. Duberstein, and I. Kawachi, "Education and smoking: confounding or effect modification by phenotypic personality traits?" *Annals of Behavioral Medicine*, vol. 38, no. 3, pp. 237–248, 2009.

[84] B. P. Chapman, K. Fiscella, P. Duberstein, M. Coletta, and I. Kawachi, "Can the influence of childhood socioeconomic status on Men's and Women's adult body mass be explained by adult socioeconomic status or personality? Findings from a national sample," *Health Psychology*, vol. 28, no. 4, pp. 419–427, 2009.

[85] R. Grossarth-Maticek and H. J. Eysenck, "Personality, smoking, and alcohol as synergistic risk factors for cancer of the mouth and pharynx," *Psychological Reports*, vol. 67, no. 3, part 1, pp. 1024–1026, 1990.

[86] W. K. B. Hofstee, B. de Raad, and L. R. Goldberg, "Integration of the Big Five and circumplex approaches to trait structure," *Journal of Personality and Social Psychology*, vol. 63, no. 1, pp. 146–163, 1992.

[87] S. Read, G. P. Vogler, N. L. Pedersen, and B. Johansson, "Stability and change in genetic and environmental components of personality in old age," *Personality and Individual Differences*, vol. 40, no. 8, pp. 1637–1647, 2006.

[88] A. Terracciano, C. E. Löckenhoff, A. B. Zonderman, L. Ferrucci, and P. T. Costa Jr., "Personality predictors of longevity: activity, emotional stability, and conscientiousness," *Psychosomatic Medicine*, vol. 70, no. 6, pp. 621–627, 2008.

[89] R. P. Murray, G. E. Barnes, and O. Ekuma, "Does personality mediate the relation between alcohol consumption and cardiovascular disease morbidity and mortality?" *Addictive Behaviors*, vol. 30, no. 3, pp. 475–488, 2005.

[90] N. Nakaya, Y. Tsubono, Y. Nishino et al., "Personality and cancer survival: the Miyagi cohort study," *British Journal of Cancer*, vol. 92, no. 11, pp. 2089–2094, 2005.

[91] G. W. Edmonds, T. Bogg, and B. W. Roberts, "Are personality and behavioral measures of impulse control convergent or distinct predictors of health behaviors?" *Journal of Research in Personality*, vol. 43, no. 5, pp. 806–814, 2009.

[92] L. D. Kubzansky, P. E. Kubzansky, and J. Maselko, "Optimism and pessimism in the context of health: bipolar opposites or separate constructs?" *Personality and Social Psychology Bulletin*, vol. 30, no. 8, pp. 943–956, 2004.

[93] L. D. Kubzansky, D. Sparrow, P. Vokonas, and I. Kawachi, "Is the glass half empty or half full? A prospective study of optimism and coronary heart disease in the normative aging study," *Psychosomatic Medicine*, vol. 63, no. 6, pp. 910–916, 2001.

[94] G. N. Marshall, C. B. Wortman, J. W. Kusulas, L. K. Hervig, and R. R. Vickers, "Distinguishing optimism from pessimism: relations to fundamental dimensions of mood and personality," *Journal of Personality and Social Psychology*, vol. 62, no. 6, pp. 1067–1074, 1992.

[95] B. H. Brummett, M. J. Helms, W. G. Dahlstrom, and I. C. Siegler, "Prediction of all-cause mortality by the Minnesota

multiphasic personality inventory optimism-pessimism scale scores: study of a college sample during a 40-year follow-up period," *Mayo Clinic Proceedings*, vol. 81, no. 12, pp. 1541–1544, 2006.

[96] B. R. Grossardt, J. H. Bower, Y. E. Geda, R. C. Colligan, and W. A. Rocca, "Pessimistic, anxious, and depressive personality traits predict all-cause mortality: the mayo clinic cohort study of personality and aging," *Psychosomatic Medicine*, vol. 71, no. 5, pp. 491–500, 2009.

[97] T. Maruta, R. C. Colligan, M. Malinchoc, and K. P. Offord, "Optimists vs pessimists: survival rate among medical patients over a 30-year period," *Mayo Clinic Proceedings*, vol. 75, no. 2, pp. 140–143, 2000.

[98] R. Schulz, J. Bookwala, M. Scheier, J. E. Knapp, and G. M. Williamson, "Pessimism, age, and cancer mortality," *Psychology and Aging*, vol. 11, no. 2, pp. 304–309, 1996.

[99] P. J. Allison, C. Guichard, K. Fung, and L. Gilain, "Dispositional optimism predicts survival status 1 year after diagnosis in head and neck cancer patients," *Journal of Clinical Oncology*, vol. 21, no. 3, pp. 543–548, 2003.

[100] H. Nabi, M. Koskenvuo, A. Singh-Manoux et al., "Low pessimism protects against stroke: the health and social support (hessup) prospective cohort study," *Stroke*, vol. 41, no. 1, pp. 187–190, 2010.

[101] L. Solberg Nes and S. C. Segerstrom, "Dispositional optimism and coping: a meta-analytic review," *Personality and Social Psychology Review*, vol. 10, no. 3, pp. 235–251, 2006.

[102] K. Räikkönen, K. A. Matthews, J. D. Flory, J. F. Owens, and B. B. Gump, "Effects of optimism, pessimism, and trait anxiety on ambulatory blood pressure and mood during everyday life," *Journal of Personality and Social Psychology*, vol. 76, no. 1, pp. 104–113, 1999.

[103] B. Roy, A. V. Diez-Roux, T. Seeman, N. Ranjit, S. Shea, and M. Cushman, "Association of optimism and pessimism with inflammation and hemostasis in the multi-ethnic study of atherosclerosis (MESA)," *Psychosomatic Medicine*, vol. 72, no. 2, pp. 134–140, 2010.

[104] S. C. Segerstrom, "Optimism, goal conflict, and stressor-related immune change," *Journal of Behavioral Medicine*, vol. 24, no. 5, pp. 441–467, 2001.

[105] S. C. Segerstrom, "Optimism and immunity: do positive thoughts always lead to positive effects?" *Brain, Behavior, and Immunity*, vol. 19, no. 3, pp. 195–200, 2005.

[106] S. C. Segerstrom, "How does optimism suppress immunity? Evaluation of three affective pathways," *Health Psychology*, vol. 25, no. 5, pp. 653–657, 2006.

[107] S. B. G. Eysenck and H. J. Eysenck, "An improved short questionnaire for the measurement of extraversion and neuroticism," *Life Sciences*, vol. 3, no. 10, pp. 1103–1109, 1964.

[108] T. A. Judge, A. Erez, J. E. Bono, and C. J. Thoresen, "Are measures of self-esteem, neuroticism, locus of control, and generalized self-efficacy indicators of a common core construct?" *Journal of Personality and Social Psychology*, vol. 83, no. 3, pp. 693–710, 2002.

[109] B. H. Brummett, M. A. Babyak, R. B. Williams, J. C. Barefoot, P. T. Costa Jr., and I. C. Siegler, "NEO personality domains and gender predict levels and trends in body mass index over 14 years during midlife," *Journal of Research in Personality*, vol. 40, no. 3, pp. 222–236, 2006.

[110] A. Terracciano and P. T. Costa Jr., "Smoking and the five-factor Model of personality," *Addiction*, vol. 99, no. 4, pp. 472–481, 2004.

[111] M. A. Ellenbogen, C. S. Ostiguy, and S. Hodgins, "Intergenerational effects of high neuroticism in parents and their public health significance," *American Psychologist*, vol. 65, no. 2, pp. 135–136, 2010.

[112] B. B. Lahey, "Public health significance of neuroticism," *American Psychologist*, vol. 64, no. 4, pp. 241–256, 2009.

[113] K. A. Stamatakis, J. Lynch, S. A. Everson, T. Raghunathan, J. T. Salonen, and G. A. Kaplan, "Self-esteem and mortality: prospective evidence from a population-based study," *Annals of Epidemiology*, vol. 14, no. 1, pp. 58–65, 2004.

[114] T. A. Murberg, E. Bru, and T. Aarsland, "Personality as predictor of mortality among patients with congestive heart failure: a two-year follow-up study," *Personality and Individual Differences*, vol. 30, no. 5, pp. 749–757, 2001.

[115] D. K. Mroczek and A. Spiro III, "Personality change influences mortality in older men: research report," *Psychological Science*, vol. 18, no. 5, pp. 371–376, 2007.

[116] J. Denollet and D. L. Brutsaert, "Reducing emotional distress improves prognosis in coronary heart disease: 9-Year mortality in a clinical trial of rehabilitation," *Circulation*, vol. 104, no. 17, pp. 2018–2023, 2001.

[117] S. J. Almada, A. B. Zonderman, R. B. Shekelle et al., "Neuroticism and cynicism and risk of death in middle-aged men: the Western Electric study," *Psychosomatic Medicine*, vol. 53, no. 2, pp. 165–175, 1991.

[118] P. T. Costa Jr. and R. R. McCrae, "Neuroticism, somatic complaints, and disease: is the bark worse than the bite?" *Journal of personality*, vol. 55, no. 2, pp. 299–316, 1987.

[119] H. S. Friedman, M. L. Kern, and C. A. Reynolds, "Personality and health, subjective well-being, and longevity," *Journal of Personality*, vol. 78, no. 1, pp. 179–216, 2010.

[120] A. E. Korten, A. F. Jorm, Z. Jiao et al., "Health, cognitive, and psychosocial factors as predictors of mortality in an elderly community sample," *Journal of Epidemiology and Community Health*, vol. 53, no. 2, pp. 83–88, 1999.

[121] R. Noyes, D. B. Watson, E. M. Letuchy et al., "Relationship between hypochondriacal concerns and personality dimensions and traits in a military population," *Journal of Nervous and Mental Disease*, vol. 193, no. 2, pp. 110–118, 2005.

[122] A. Jerant, B. Chapman, P. Duberstein, and P. Franks, "Effects of personality on self-rated health in a 1-year randomized controlled trial of chronic illness self-management," *British Journal of Health Psychology*, vol. 15, no. 2, pp. 321–335, 2010.

[123] B. Chapman, P. Duberstein, and J. M. Lyness, "Personality traits, education, and health-related quality of life among older adult primary care patients," *Journals of Gerontology—Series B Psychological Sciences and Social Sciences*, vol. 62, no. 6, pp. P343–P352, 2007.

[124] A. Antonovsky, *Health, Stress, and Coping*, Jossey-Bass, San Francisco, Calif, USA, 1979.

[125] P. G. Surtees, N. W. J. Wainwright, and K. T. Khaw, "Sense of coherence, social adversity and mortality," *Journal of Psychosomatic Research*, vol. 61, no. 3, pp. 376–381, 2006.

[126] P. G. Surtees, N. W. J. Wainwright, R. Luben, K. T. Khaw, and N. E. Day, "Mastery, sense of coherence, and mortality: evidence of independent associations from the EPIC-Norfolk prospective cohort study," *Health Psychology*, vol. 25, no. 1, pp. 102–110, 2006.

[127] P. G. Surtees, N. W. J. Wainwright, R. Luben, N. J. Wareham, S. A. Bingham, and K. T. Khaw, "Mastery is associated with cardiovascular disease mortality in men and women at apparently low risk," *Health Psychology*, vol. 29, no. 4, pp. 412–420, 2010.

[128] P. Surtees, N. Wainwright, R. Luben, K. T. Khaw, and N. Day, "Sense of coherence and mortality in Men and Women in the EPIC-Norfolk United Kingdom prospective cohort study," *American Journal of Epidemiology*, vol. 158, no. 12, pp. 1202–1209, 2003.

[129] F. V. A. van Oort, F. J. van Lenthe, and J. P. Mackenbach, "Material, psychosocial, and behavioural factors in the explanation of educational inequalities in mortality in the Netherlands," *Journal of Epidemiology and Community Health*, vol. 59, no. 3, pp. 214–220, 2005.

[130] T. Stürmer, P. Hasselbach, and M. Amelang, "Personality, lifestyle, and risk of cardiovascular disease and cancer: follow-up of population based cohort," *British Medical Journal*, vol. 332, no. 7554, pp. 1359–1362, 2006.

[131] F. J. Infurma, D. Gerstorf, N. Ram, J. Schupp, and G. G. Wagner, "Long-term antecedents and outcomes of perceived control," *Psychology and Aging*. In press.

[132] N. C. Hall, J. G. Chipperfield, J. Heckhausen, and R. P. Perry, "Control striving in older adults with serious health problems: a 9-year longitudinal study of survival, health, and well-being," *Psychology and Aging*, vol. 25, no. 2, pp. 432–445, 2010.

[133] J. Denollet and G. L. Van Heck, "Psychological risk factors in heart disease what type D personality is (not) about," *Journal of Psychosomatic Research*, vol. 51, no. 3, pp. 465–468, 2001.

[134] J. Denollet, "Personality and coronary heart disease: the type-D scale-16 (DS16)," *Annals of Behavioral Medicine*, vol. 20, no. 3, pp. 209–215, 1998.

[135] F. De Fruyt and J. Denollet, "Type D personality: a five-factor model perspective," *Psychology and Health*, vol. 17, no. 5, pp. 671–683, 2002.

[136] J. Denollet, P. De Jonge, A. Kuyper et al., "Depression and type D personality represent different forms of distress in the myocardial iNfarction and depression—intervention trial (MIND-IT)," *Psychological Medicine*, vol. 39, no. 5, pp. 749–756, 2009.

[137] J. Denollet and S. S. Pedersen, "Anger, depression, and anxiety in cardiac patients. The complexity of individual differences in psychological risk," *Journal of the American College of Cardiology*, vol. 53, no. 11, pp. 947–949, 2009.

[138] J. Denollet, A. A. Schiffer, and V. Spek, "A general propensity to psychological distress affects cardiovascular outcomes: evidence from research on the type D (distressed) personality profile," *Circulation: Cardiovascular Quality and Outcomes*, vol. 3, no. 5, pp. 546–557, 2010.

[139] J. Denollet, S. U. Sys, N. Stroobant, H. Rombouts, T. C. Gillebert, and D. L. Brutsaert, "Personality as independent predictor of long-term mortality in patients with coronary heart disease," *The Lancet*, vol. 347, no. 8999, pp. 417–421, 1996.

[140] S. Pedersen et al., "Increased risk of mortality or myocardial infarction 2 years post-pci in Type D patients treated with sirolimus-eluting stents," *Psychology & Health*, vol. 20, pp. 206–207, 2005.

[141] A. A. Schiffer, O. R. F. Smith, S. S. Pedersen, J. W. Widdershoven, and J. Denollet, "Type D personality and cardiac mortality in patients with chronic heart failure," *International Journal of Cardiology*, vol. 142, no. 3, pp. 230–235, 2010.

[142] A. E. Aquarius, K. G. Smolderen, J. F. Hamming, J. De Vries, P. W. Vriens, and J. Denollet, "Type D personality and mortality in peripheral arterial disease: a pilot study," *Archives of Surgery*, vol. 144, no. 8, pp. 728–733, 2009.

[143] J. Denollet, Y. Gidron, C. J. Vrints, and V. M. Conraads, "Anger, suppressed anger, and risk of adverse events in patients with coronary artery disease," *American Journal of Cardiology*, vol. 105, no. 11, pp. 1555–1560, 2010.

[144] A. J. Pelle, S. S. Pedersen, A. A. Schiffer, B. Szabó, J. W. Widdershoven, and J. Denollet, "Psychological distress and mortality in systolic heart failure," *Circulation: Heart Failure*, vol. 3, no. 2, pp. 261–267, 2010.

[145] J. N. de Voogd, J. B. Wempe, K. Postema et al., "More evidence that depressive symptoms predict mortality in COPD patients: is type D personality an alternative explanation?" *Annals of Behavioral Medicine*, vol. 38, no. 2, pp. 86–93, 2009.

[146] N. Kupper and J. Denollet, "Type D personality as a prognostic factor in heart disease: assessment and mediating mechanisms," *Journal of Personality Assessment*, vol. 89, no. 3, pp. 265–276, 2007.

[147] J. Denollet, A. A. Schiffer, M. Kwaijtaal et al., "Usefulness of type D personality and kidney dysfunction as predictors of interpatient variability in inflammatory activation in chronic heart failure," *American Journal of Cardiology*, vol. 103, no. 3, pp. 399–404, 2009.

[148] V. M. Conraads, J. Denollet, L. S. De Clerck, W. J. Stevens, C. Bridts, and C. J. Vrints, "Type D personality is associated with increased levels of tumour necrosis factor (TNF)-α and TNF-α receptors in chronic heart failure," *International Journal of Cardiology*, vol. 113, no. 1, pp. 34–38, 2006.

[149] J. Denollet, V. M. Conraads, D. L. Brutsaert, L. S. De Clerck, W. J. Stevens, and C. J. Vrints, "Cytokines and immune activation in systolic heart failure: the role of type D personality," *Brain, Behavior, and Immunity*, vol. 17, no. 4, pp. 304–309, 2003.

[150] J. Denollet, C. J. Vrints, and V. M. Conraads, "Comparing Type D personality and older age as correlates of tumor necrosis factor-α dysregulation in chronic heart failure," *Brain, Behavior, and Immunity*, vol. 22, no. 5, pp. 736–743, 2008.

[151] N. Kupper, Y. Gidron, J. Winter, and J. Denollet, "Association between type D personality, depression, and oxidative stress in patients with chronic heart failure," *Psychosomatic Medicine*, vol. 71, no. 9, pp. 973–980, 2009.

[152] K. C. van den Broek, I. Nyklíček, P. H. van der Voort, M. Alings, A. Meijer, and J. Denollet, "Risk of ventricular arrhythmia after implantable defibrillator treatment in anxious type D patients," *Journal of the American College of Cardiology*, vol. 54, no. 6, pp. 531–537, 2009.

[153] A. J. Pelle, A. A. Schiffer, O. R. Smith, J. W. Widdershoven, and J. Denollet, "Inadequate consultation behavior modulates the relationship between Type D personality and impaired health status in chronic heart failure," *International Journal of Cardiology*, vol. 142, no. 1, pp. 65–71, 2010.

[154] F. Mols and J. Denollet, "Type D personality among non-cardiovascular patient populations: a systematic review," *General Hospital Psychiatry*, vol. 32, no. 1, pp. 66–72, 2010.

[155] F. Mols and J. Denollet, "Type D personality in the general population: a systematic review of health status, mechanisms of disease, and work-related problems," *Health and Quality of Life Outcomes*, vol. 8, article no. 9, 2010.

[156] A. J. Pelle, J. Denollet, A. D. Zwisler, and S. S. Pedersen, "Overlap and distinctiveness of psychological risk factors in patients with ischemic heart disease and chronic heart failure: are we there yet?" *Journal of Affective Disorders*, vol. 113, no. 1-2, pp. 150–156, 2009.

[157] R. F. Krueger, A. Caspi, T. E. Moffitt, P. A. Silva, and R. McGee, "Personality traits are differentially linked to mental disorders: a multitrait-multidiagnosis study of an adolescent

birth cohort," *Journal of Abnormal Psychology*, vol. 105, no. 3, pp. 299–312, 1996.

[158] M. Friedman, *Type A Behavior: Its Diagnosis and Treatment*, Plenum, New York, NY, USA, 1996.

[159] J. C. Barefoot, W. G. Dahlstrom, and R. B. Williams Jr., "Hostility, CHD incidence, and total mortality: a 25-year follow-up study of 255 physicians," *Psychosomatic Medicine*, vol. 45, no. 1, pp. 59–63, 1983.

[160] R. B. Shekelle, M. Gale, A. M. Ostfeld, and O. Paul, "Hostility, risk of coronary heart disease, and mortality," *Psychosomatic Medicine*, vol. 45, no. 2, pp. 109–114, 1983.

[161] T. M. Dembroski and P. T. Costa Jr., "Coronary prone behavior: components of the type A pattern and hostility," *Journal of Personality*, vol. 55, no. 2, pp. 211–235, 1987.

[162] T. M. Dembroski, J. M. MacDougall, P. T. Costa Jr., and G. A. Grandits, "Components of hostility as predictors of sudden death and myocardial infarction in the Multiple Risk Factor Intervention Trial," *Psychosomatic Medicine*, vol. 51, no. 5, pp. 514–522, 1989.

[163] C. M. Aldwin, A. Spiro, M. R. Levenson, and A. P. Cupertino, "Longitudinal findings from the normative aging study: III. Personality, individual health trajectories, and mortality," *Psychology and Aging*, vol. 16, no. 3, pp. 450–465, 2001.

[164] M. Koskenvuo, J. Kaprio, R. J. Rose et al., "Hostility as a risk factor for mortality and ischemic heart disease in men," *Psychosomatic Medicine*, vol. 50, no. 4, pp. 330–340, 1988.

[165] J. C. Barefoot, S. Larsen, L. von der Lieth, and M. Schroll, "Hostility, incidence of acute myocardial infarction, and mortality in a sample of older Danish men and women," *American Journal of Epidemiology*, vol. 142, no. 5, pp. 477–484, 1995.

[166] P. G. Surtees, N. W. J. Wainwright, R. Luben, N. E. Day, and K. T. Khaw, "Prospective cohort study of hostility and the risk of cardiovascular disease mortality," *International Journal of Cardiology*, vol. 100, no. 1, pp. 155–161, 2005.

[167] C. Iribarren, D. R. Jacobs, C. I. Kiefe et al., "Causes and demographic, medical, lifestyle and psychosocial predictors of premature mortality: the CARDIA study," *Social Science and Medicine*, vol. 60, no. 3, pp. 471–482, 2005.

[168] E. W. McCranie, L. O. Watkins, J. M. Brandsma, and B. D. Sisson, "Hostility, coronary heart disease (CHD) incidence, and total mortality: lack of association in a 25-year follow-up study of 478 physicians," *Journal of Behavioral Medicine*, vol. 9, no. 2, pp. 119–125, 1986.

[169] M. D. Hearn, D. M. Murray, and R. V. Luepker, "Hostility, coronary heart disease, and total mortality: a 33-year follow-up study of university students," *Journal of Behavioral Medicine*, vol. 12, no. 2, pp. 105–121, 1989.

[170] M. W. Ketterer, J. Huffman, M. A. Lumley et al., "Five-year follow-up for adverse outcomes in males with at least minimally positive angiograms: importance of 'denial' in assessing psychosocial risk factors," *Journal of Psychosomatic Research*, vol. 44, no. 2, pp. 241–250, 1998.

[171] M. W. Kaufmann, J. P. Fitzgibbons, E. J. Sussman et al., "Relation between myocardial infarction, depression, hostility, and death," *American Heart Journal*, vol. 138, no. 3, pp. 549–554, 1999.

[172] L. A. Chaput, S. H. Adams, J. A. Simon et al., "Hostility predicts recurrent events among postmenopausal women with coronary heart disease," *American Journal of Epidemiology*, vol. 156, no. 12, pp. 1092–1099, 2002.

[173] J. C. Barefoot, K. A. Dodge, B. L. Peterson, W. G. Dahlstrom, and R. B. Williams, "The Cook-Medley hostility scale: item content and ability to predict survival," *Psychosomatic Medicine*, vol. 51, no. 1, pp. 46–57, 1989.

[174] J. C. Barefoot, I. C. Siegler, J. B. Nowlin, B. L. Peterson, T. L. Haney, and R. B. Williams, "Suspiciousness, health, and mortality: a follow-up study of 500 older adults," *Psychosomatic Medicine*, vol. 49, no. 5, pp. 450–457, 1987.

[175] B. K. Houston, M. A. Babyak, M. A. Chesney, G. Black, and D. R. Ragland, "Social dominance and 22-year all-cause mortality in men," *Psychosomatic Medicine*, vol. 59, no. 1, pp. 5–12, 1997.

[176] S. H. Boyle, R. B. Williams, D. B. Mark et al., "Hostility as a predictor of survival in patients with coronary artery disease," *Psychosomatic Medicine*, vol. 66, no. 5, pp. 629–632, 2004.

[177] S. A. Everson, J. Kauhanen, G. A. Kaplan et al., "Hostility and increased risk of mortality and acute myocardial infarction: the mediating role of behavioral risk factors," *American Journal of Epidemiology*, vol. 146, no. 2, pp. 142–152, 1997.

[178] C. Lemogne, H. Nabi, M. Zins et al., "Hostility may explain the association between depressive mood and mortality: evidence from the french gazel cohort study," *Psychotherapy and Psychosomatics*, vol. 79, no. 3, pp. 164–171, 2010.

[179] L. Musante, J. M. MacDougall, T. M. Dembroski, and P. T. Costa Jr., "Potential for hostility and dimensions of anger," *Health Psychology*, vol. 8, no. 3, pp. 343–354, 1989.

[180] M. H. Erdelyi, *Psychoanalysis: Freud's Cognitive Psychology*, W. H. Freeman, New York, NY, USA, 1985.

[181] P. R. Duberstein and J. M. Masling, *Psychodynamic Perspectives on Sickness and Health*, American Psychological Association, Washington, DC, USA, 2000.

[182] H. J. Eysenck, "Reply to criticisms of the Grossarth-Maticek studies," *Psychological Inquiry*, vol. 2, no. 3, pp. 297–323, 1991.

[183] K. Hirokawa, C. Nagata, N. Takatsuka, and H. Shimizu, "The relationships of a rationality/antiemotionality personality scale to mortalities of cancer and cardiovascular disease in a community population in Japan," *Journal of Psychosomatic Research*, vol. 56, no. 1, pp. 103–111, 2004.

[184] J. H. Block and J. Block, "The role of ego control and ego resiliency in the organization of bheavior," in *Development of Cognition, Affect, and Social Relations: The Minnesota Symposium on Child Psychology*, W. A. Collins, Ed., vol. 13, pp. 39–101, Lawrence Erlbaum Associates, New Jersey, NJ, USA, 1980.

[185] E. Harburg, M. Julius, N. Kaciroti, L. Gleiberman, and M. A. Schork, "Expressive/suppressive anger-coping responses, gender, and types of mortality: a 17-year follow-up (Tecumseh, Michigan, 1971–1988)," *Psychosomatic Medicine*, vol. 65, no. 4, pp. 588–597, 2003.

[186] M. Petticrew, R. Bell, and D. Hunter, "Influence of psychological coping on survival and recurrence in people with cancer: systematic review," *British Medical Journal*, vol. 325, no. 7372, pp. 1066–1069, 2002.

[187] M. Watson, J. Davidson-Homewood, J. Haviland, and J. Bliss, "Psychological coping and cancer. Study results should not have been dismissed," *BMJ*, vol. 326, no. 7389, p. 598; author reply 598, 2003.

[188] H. Nabi, M. Kiviméki, M. Zins et al., "Does personality predict mortality: results from the GAZEL French prospective cohort study," *International Journal of Epidemiology*, vol. 37, no. 2, pp. 386–396, 2008.

[189] R. F. Krueger, A. Caspi, and T. E. Moffitt, "Epidemiological personology: the unifying role of personality in

population-based research on problem behaviors," *Journal of Personality*, vol. 68, no. 6, pp. 967–998, 2000.

[190] G. Saucier, "Replicable item-cluster subcomponents in the NEO five-factor Inventory," *Journal of Personality Assessment*, vol. 70, no. 2, pp. 263–276, 1998.

[191] B. P. Chapman, "Bandwidth and fidelity on the NEO-five factor inventory: replicability and reliability of Saucier's (1998) item cluster subcomponents," *Journal of Personality Assessment*, vol. 88, no. 2, pp. 220–234, 2007.

[192] B. P. Chapman, J. M. Lyness, and P. Duberstein, "Personality and medical illness burden among older adults in primary care," *Psychosomatic Medicine*, vol. 69, no. 3, pp. 277–282, 2007.

[193] A. C. Phillips, G. D. Batty, A. Weiss et al., "Neuroticism, cognitive ability, and the metabolic syndrome: the Vietnam Experience Study," *Journal of Psychosomatic Research*, vol. 69, no. 2, pp. 193–201, 2010.

[194] A. Weiss, C. R. Gale, G. D. Batty, and I. J. Deary, "Emotionally stable, intelligent men live longer: the Vietnam experience study cohort," *Psychosomatic Medicine*, vol. 71, no. 4, pp. 385–394, 2009.

[195] L. Seidlitz, Y. Conwell, P. Duberstein, C. Cox, and D. Denning, "Emotion traits in older suicide attempters and non-attempters," *Journal of Affective Disorders*, vol. 66, no. 2-3, pp. 123–131, 2001.

[196] E. K. Traupman, T. W. Smith, B. N. Uchino, C. A. Berg, K. K. Trobst, and P. T. Costa Jr., "Interpersonal circumplex octant, control, and affiliation scales for the NEO-PI-R," *Personality and Individual Differences*, vol. 47, no. 5, pp. 457–463, 2009.

[197] B. P. Chapman, P. R. Duberstein, and J. M. Lyness, "The distressed personality type: replicability and general health associations," *European Journal of Personality*, vol. 21, no. 7, pp. 911–929, 2007.

[198] L. R. Goldberg, "Doing it all bass-ackwards: the development of hierarchical factor structures from the top down," *Journal of Research in Personality*, vol. 40, no. 4, pp. 347–358, 2006.

[199] J. P. MacKenbach, "New trends in health inequalities research: now it's personal," *The Lancet*, vol. 376, no. 9744, pp. 854–855, 2010.

[200] L. F. Berkman and I. Kawachi, *Social Epidemiology*, Oxford University Press, New York, NY, USA, 2000.

[201] D. Kuh, Y. Ben-Shlomo, J. Lynch, J. Hallqvist, and C. Power, "Life course epidemiology," *Journal of epidemiology and community health*, vol. 57, no. 10, pp. 778–783, 2003.

[202] J. Gallacher, "Commentary: personality and health inequality: inconclusive evidence for an indirect hypothesis," *International Journal of Epidemiology*, vol. 37, no. 3, pp. 602–603, 2008.

[203] D. S. Black, *Inequalities in Health: The Black Report*, Penguin, Harmondsworth, UK, 1988.

[204] H. Nabi, M. Kivimäki, M. G. Marmot et al., "Does personality explain social inequalities in mortality? The French GAZEL cohort study," *International Journal of Epidemiology*, vol. 37, no. 3, pp. 591–602, 2008.

[205] E. Poppius, L. Tenkanen, M. Hakama, R. Kalimo, and T. Pitkänen, "The sense of coherence, occupation and all-cause mortality in the Helsinki Heart Study," *European Journal of Epidemiology*, vol. 18, no. 5, pp. 389–393, 2003.

[206] M. E. Lachman and S. L. Weaver, "The sense of control as a moderator of social class differences in health and well-being," *Journal of Personality and Social Psychology*, vol. 74, no. 3, pp. 763–773, 1998.

[207] M. Kivimäki, M. Elovainio, K. Kokko, L. Pulkkinen, M. Kortteinen, and H. Tuomikoski, "Hostility, unemployment and health status: testing three theoretical models," *Social Science and Medicine*, vol. 56, no. 10, pp. 2139–2152, 2003.

[208] J. W. Lynch, G. A. Kaplan, and J. T. Salonen, "Why do poor people behave poorly? Variation in adult health behaviours and psychosocial characteristics by stages of the socioeconomic lifecourse," *Social Science and Medicine*, vol. 44, no. 6, pp. 809–819, 1997.

[209] M. G. Marmot, R. Fuhrer, S. L. Ettner, N. F. Marks, L. L. Bumpass, and C. D. Ryff, "Contribution of psychosocial factors to socioeconomic differences in health," *Milbank Quarterly*, vol. 76, no. 3, pp. 403–448, 1998.

[210] H. Bosma, H. D. van de Mheen, and J. P. Mackenbach, "Social class in childhood and general health in adulthood: questionnaire study of contribution of psychological attributes," *British Medical Journal*, vol. 318, no. 7175, pp. 18–22, 1999.

[211] T. W. Smith and A. Spiro, "Personality, health, and aging: prolegomenon for the next generation," *Journal of Research in Personality*, vol. 36, no. 4, pp. 363–394, 2002.

[212] C. R. Cloninger, "How does personality influence mortality in the elderly?" *Psychosomatic Medicine*, vol. 67, no. 6, pp. 839–840, 2005.

[213] D. Nettle, "The evolution of personality variation in humans and other animals," *American Psychologist*, vol. 61, no. 6, pp. 622–631, 2006.

[214] R. M. Andersen, "Revisiting the behavioral model and access to medical care: does it matter?" *Journal of Health and Social Behavior*, vol. 36, no. 1, pp. 1–10, 1995.

[215] J. G. Chipperfield and L. Greenslade, "Perceived control as a buffer in the use of health care services," *Journals of Gerontology—Series B Psychological Sciences and Social Sciences*, vol. 54, no. 3, pp. P146–P154, 1999.

[216] R. D. Goodwin, C. W. Hoven, J. S. Lyons, and M. B. Stein, "Mental health service utilization in the United States. The role of personality factors," *Social Psychiatry and Psychiatric Epidemiology*, vol. 37, no. 12, pp. 561–566, 2002.

[217] B. P. Chapman, K. Fiscella, I. Kawachi, and P. R. Duberstein, "Personality traits predict emergency department utilization over three years in older patients," *American Journal of Geriatric Psychiatry*, vol. 17, pp. 526–535, 2009.

[218] A. R. Sutin, P. T. Costa Jr., M. Uda, L. Ferrucci, D. Schlessinger, and A. Terracciano, "Personality and metabolic syndrome," *Age*, vol. 32, no. 4, pp. 513–519, 2010.

[219] B. P. Chapman, A. Khan, M. Harper et al., "Gender, race/ethnicity, personality, and interleukin-6 in urban primary care patients," *Brain, Behavior, and Immunity*, vol. 23, no. 5, pp. 636–642, 2009.

[220] A. R. Sutin, A. Scuteri, E. G. Lakatta et al., "Trait antagonism and the progression of arterial thickening: women with antagonistic traits have similar carotid arterial thickness as men," *Hypertension*, vol. 56, no. 4, pp. 617–622, 2010.

[221] A. R. Sutin, A. Terracciano, B. Deiana et al., "Cholesterol, triglycerides, and the five-factor model of personality," *Biological Psychology*, vol. 84, no. 2, pp. 186–191, 2010.

[222] K. Imai, L. Keele, and D. Tingley, "A general approach to causal mediation analysis," *Psychological Methods*, vol. 15, no. 4, pp. 309–334, 2010.

[223] B. Huang, S. Sivaganesan, P. Succop, and E. Goodman, "Statistical assessment of mediational effects for logistic mediational models," *Statistics in Medicine*, vol. 23, no. 17, pp. 2713–2728, 2004.

[224] J. S. Kaufman, R. F. MacLehose, S. Kaufman, and S. Greenland, "The mediation proportion," *Epidemiology*, vol. 16, no. 5, p. 710, 2005.

[225] D. P. MacKinnon, J. L. Krull, and C. M. Lockwood, "Equivalence of the mediation, confounding and suppression effect," *Prevention Science*, vol. 1, no. 4, pp. 173–181, 2000.

[226] K. J. Preacher, D. D. Rucker, and A. F. Hayes, "Addressing moderated mediation hypotheses: theory, methods, and prescriptions," *Multivariate Behavioral Research*, vol. 42, no. 1, pp. 185–227, 2007.

[227] NIH, NIH Science of Behavior Change Meeting Summary, 2009.

[228] M. A. Hamburg and F. S. Collins, "The path to personalized medicine," *The New England Journal of Medicine*, vol. 363, no. 4, pp. 301–304, 2010.

[229] G. S. Ginsburg and H. F. Willard, "Genomic and personalized medicine: foundations and applications," *Translational Research*, vol. 154, no. 6, pp. 277–287, 2009.

[230] G. J. Meyer, S. E. Finn, L. D. Eyde et al., "Psychological testing and psychological assessment: a review of evidence and issues," *American Psychologist*, vol. 56, no. 2, pp. 128–165, 2001.

[231] T. A. Manolio, "Genomewide association studies and assessment of risk of disease," *The New England Journal of Medicine*, vol. 363, no. 21, pp. 2076–2077, 2010.

[232] P. C. Ng, S. S. Murray, S. Levy, and J. C. Venter, "An agenda for personalized medicine," *Nature*, vol. 461, no. 7265, pp. 724–726, 2009.

[233] L. D. Kubzansky, I. Kawachi, and D. Sparrow, "Socioeconomic status, hostility, and risk factor clustering in the normative aging study: any help from the concept of allostatic load?" *Annals of Behavioral Medicine*, vol. 21, no. 4, pp. 330–338, 1999.

[234] S. D. Gosling, P. J. Rentfrow, and W. B. Swann Jr., "A very brief measure of the Big-Five personality domains," *Journal of Research in Personality*, vol. 37, no. 6, pp. 504–528, 2003.

[235] M. B. Donnellan, F. L. Oswald, B. M. Baird, and R. E. Lucas, "The Mini-IPIP scales: tiny-yet-effective measures of the Big Five factors of personality," *Psychological Assessment*, vol. 18, no. 2, pp. 192–203, 2006.

[236] M. Woodward, *Epidemiology: Study Design and Data Analysis*, Chapman & Hall, New York, NY, USA, 2nd edition, 2005.

[237] L. Ryan, "Quantitative risk assessment for developmental toxicity," *Biometrics*, vol. 48, no. 1, pp. 163–174, 1992.

[238] I. O. Medicine, *Crossing the Quality Chasm: A New Health System for the 21st Century*, The National Academies Press, Washington, DC, USA, 2001.

[239] R. M. Epstein, P. Franks, K. Fiscella et al., "Measuring patient-centered communication in Patient-Physician consultations: theoretical and practical issues," *Social Science and Medicine*, vol. 61, no. 7, pp. 1516–1528, 2005.

[240] B. De Raad and M. Perugini, *Big Five Assessment*, Hogrefe & Huber Publishers, Toronto, Canada, 2002.

[241] T. Hastie, R. Tibshirani, and J. Friedman, *The Elements of Statistical Learning: Data Mining, Inference, and Prediction*, Springer, New York, NY, USA, 2nd edition, 2009.

[242] P. T. Costa Jr. and R. R. McCrae, *Personality in Adulthood: A Five Factor Theory Perspective*, Guilford Press, New York, NY, USA, 2nd edition, 2033.

[243] B. W. Roberts, K. E. Walton, and W. Viechtbauer, "Personality traits change in adulthood: reply to Costa and McCrae (2006)," *Psychological Bulletin*, vol. 132, no. 1, pp. 29–32, 2006.

[244] G. W. Edmonds, "Is character fate, or is there hope to change my personality yet?" *Social and Personality Psychology Compass*, vol. 2, no. 1, pp. 399–413, 2008.

[245] J. M. Duchek, D. A. Balota, M. Storandt, and R. Larsen, "The power of personality in discriminating between healthy aging and early-stage Alzheimer's disease," *Journals of Gerontology—Series B Psychological Sciences and Social Sciences*, vol. 62, no. 6, pp. P353–P361, 2007.

[246] T. Z. Tang, R. J. DeRubeis, S. D. Hollon, J. Amsterdam, R. Shelton, and B. Schalet, "Personality change during depression treatment: a placebo-controlled trial," *Archives of General Psychiatry*, vol. 66, no. 12, pp. 1322–1330, 2009.

[247] N. A. Turiano, L. M. Pitzer, and C. Armour, "Personality trait level and change as predictors of health outcomes: findings from a national study of Americans (MIDUS)," *Journal of Gerontology: Psychological Science*. In press.

[248] Y. Gidron, K. Davidson, and I. Bata, "The short-term effects of a hostility-reduction intervention on male coronary heart disease patients," *Health Psychology*, vol. 18, no. 4, pp. 416–420, 1999.

[249] R. Grossarth-Maticek, H. Eysenck, G. Gallasch, H. Vetter, and R. Frentzel-Beyme, "Changes in degree of sclerosis as a function of prophylactic treatment in cancer-prone and CHD-prone probands," *Behaviour Research and Therapy*, vol. 29, no. 4, pp. 343–351, 1991.

[250] R. Grossarth-Maticek, "Social Psychotherapy and course of the disease. First experiences with cancer patients," *Psychotherapy and Psychosomatics*, vol. 33, no. 3, pp. 129–138, 1980.

[251] R. Grossarth-Maticek and H. J. Eysenck, "Length of survival and lymphocyte percentage in women with mammary cancer as a function of psychotherapy," *Psychological Reports*, vol. 65, no. 1, pp. 315–321, 1989.

[252] R. Grossarth-Maticek and H. J. Eysenck, "Prophylactic effects of psychoanalysis on cancer-prone and coronary heart disease-prone probands, as compared with control groups and behaviour therapy groups," *Journal of Behavior Therapy and Experimental Psychiatry*, vol. 21, no. 2, pp. 91–99, 1990.

[253] P. T. Costa Jr., R. M. Bagby, J. H. Herbst, and R. R. McCrae, "Personality self-reports are concurrently reliable and valid during acute depressive episodes," *Journal of Affective Disorders*, vol. 89, no. 1–3, pp. 45–55, 2005.

[254] M. S. Krasner, R. M. Epstein, H. Beckman et al., "Association of an educational program in mindful communication with burnout, empathy, and attitudes among primary care physicians," *JAMA*, vol. 302, no. 12, pp. 1284–1293, 2009.

[255] J. J. Jackson, P. L. Hill, B. R. Payne, B. W. Roberts, and A. L. Stine-Morrow, "Can an old dog learn (and want to experience) new tricks? Cognitive training increases openness to experience in older adults," under review.

[256] F. E. Harrell Jr., *Regression Modeling Strategies*, Springer, New York, NY, USA, 2001.

[257] K. J. Rothman, S. Greenland, and T. L. Lash, *Modern Epidemiology*, Lippincott Williams & Wilkins, Philadelphia, Pa, USA, 3rd edition, 2008.

[258] S. Greenland and K. Drescher, "Maximum likelihood estimation of the attributable fraction from logistic models," *Biometrics*, vol. 49, no. 3, pp. 865–872, 1993.

[259] N. N. I. O. Health, *NIH Science of Behavior Change Meeting Summary*, National Institutes of Heath, Bethesda, Md, USA, 2009.

[260] B. P. Chapman, B. W. Roberts, J. M. Lyness, and P. R. Duberstein, "Personality trait predictors of physician-assessed illness burden over 4 year in older adults," *American Journal of Geriatric Psychiatry*, In press.
[261] T. L. Lash, M. P. Fox, and K. Fink, *Applying Quantitative Bias Analysis to Epidemiologic Data*, Springer, New York, NY, USA, 2009.
[262] J. P. A. Ioannidis, "Why most published research findings are false," *PLoS Medicine*, vol. 2, no. 8, pp. 0696–0701, 2005.
[263] J. A. Walsh and M. M. Nash, "Personality characteristics of volunteers for medical research," *Criminal Justice and Behavior*, vol. 5, no. 2, pp. 99–116, 1978.
[264] J. E. Lönnqvist, S. Paunonen, M. Verkasalo, S. Leikas, A. Tuulio-Henriksson, and J. Lönnqvist, "Personality characteristics of research volunteers," *European Journal of Personality*, vol. 21, no. 8, pp. 1017–1030, 2007.
[265] A. J. Christensen, "Patient-by-treatment context interaction in chronic disease: a conceptual framework for the study of patient adherence," *Psychosomatic Medicine*, vol. 62, no. 3, pp. 435–443, 2000.
[266] A. Jerant, E. P. Chapman, P. Duberstein, and P. Franks, "Is personality a key predictor of missing study data? An analysis from a randomized controlled trial," *Annals of Family Medicine*, vol. 7, no. 2, pp. 148–156, 2009.
[267] A. Jerant, B. Chapman, P. Duberstein, J. Robbins, and P. Franks, "Personality and medication non-adherence among older adults enrolled in a six-year trial," *British Journal of Health Psychology*, vol. 16, no. 1, pp. 151–169, 2011.
[268] J. P. A. Ioannidis, A. B. Haidich, M. Pappa et al., "Comparison of evidence of treatment effects in randomized and nonrandomized studies," *JAMA*, vol. 286, no. 7, pp. 821–830, 2001.
[269] G. Edmonds, *Personality and the Healthy Lifestyle as Predictors of Physical Health: Can the Healthy Lifestyle be Explained by Personality?* Department of Psychology, University of Illinois at Urbana-Champaign, Urbana, Ill, USA, 2009.
[270] J. Pearl, "An introduction to causal inference," *International Journal of Biostatistics*, vol. 6, no. 2, article 7, 2010.
[271] A. Jerant, B. Chapman, P. Duberstein, and P. Franks, "Effects of personality on self-rated health in a 1-year randomized controlled trial of chronic illness self-management," *British Journal of Health Psychology*, vol. 15, no. 2, pp. 321–335, 2010.
[272] B. P. Chapman, P. Franks, P. R. Duberstein, and A. Jerant, "Differences between individual and societal health state valuations: any link with personality?" *Medical Care*, vol. 47, no. 8, pp. 902–907, 2009.
[273] B. P. Chapman, P. R. Duberstein, S. Sörensen, and J. M. Lyness, "Personality and perceived health in older adults: the five factor model in primary care," *Journals of Gerontology—Series B Psychological Sciences and Social Sciences*, vol. 61, no. 6, pp. P362–P365, 2006.

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Research Article

Successful Aging and Longevity in Older Old Women: The Role of Depression and Cognition

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Based in successful aging theory and terminal cognitive drop research, this paper investigates cerebrovascular burden (CVB), depressive symptoms, and cognitive decline as threats to longevity. A subsample of stroke-free women over the age of 80 was identified in the Health and Retirement Survey (years 2000–2008). Mortality at 2, 6, and 8 year intervals was predicted using CVB (diabetes, heart disease, hypertension), depressive symptoms (Center for Epidemiological Studies Depression Scale), and cognitive decline (decline of 1 standard deviation or more on the 35-point Telephone Interview for Cognitive Status over 2 years). At most waves (2002, 2004, and 2006) mortality was predicted by CVB, depressive symptoms, and cognitive drop measured 2 years prior. CVB and depressive symptoms at the 2000 wave predicted mortality at 6 and 8 years. Older women with the greatest longevity had low CVB, robust cognitive functioning, and few depression symptoms, supporting successful aging theory and terminal cognitive drop.

1. Introduction

Rowe and Kahn [1] proposed criteria for successful aging comprised of avoidance of disease, maintenance of high cognitive and physical function, and sustained engagement in social and productive activities. This model grew from highly prolific MacArthur Foundation Study of Successful Aging, a $10 million, 10-year research effort led by Rowe and Kahn. The objectives of this study, and the theoretical framework that grew from it, are to better understand risk factors for decline and to inform prevention efforts. For instance, work drawn from this initiative concluded that pulmonary health relates to both gross motor and cognitive functioning in late life, suggesting this as an area for primary intervention in preserving late-life independence [2]. Drawing on the MacArthur Study data, Others have investigated modifiable risk factors for dementia, concluding that late-life depression may be a precursor of cognitive decline [3]. Still other work based in this study found that older adults who frequently felt useful to others had lower rates of disability and mortality than less-engaged elders, emphasizing the importance of social engagement and productive activities [4]. While support for this theory of successful aging has been mixed [5, 6], it provides a useful framework for examining longevity. This paper will examine whether the Rowe and Kahn successful aging variables are each independently related to longevity. In the current paper we chose to examine this theory in older old women over 80 years. Women constitute a majority of all older adults over 80 but more importantly, they are more likely than men to experience disability and to live more years with disability than men [7]. Geriatric syndromes such as cardiovascular disease, cognitive decline, and depression compromise functional independence more with increasing age [8]. Age 80 represents a point when medical treatment planning for elders should be reevaluated given the escalating risks associated with these comorbidities.

Behavioral factors as defined in this paper include psychological aspects of functioning that can be measured through self-report or performance-based assessment, such as mood and cognitive functioning (i.e., depression and cognition). Important behavioral factors such as cognition and depression represent major risk factors for disability, often preceding disability [9] and possibly reducing longevity. Serious disability, especially mobility loss, has been linked to reduced survival [10]. Identifying behavioral factors that

hasten disability onset may then lead to improved models of integrated care. Behavioral factors such as depression and cognitive decline may best be understood when integrated with chronic disease, especially those that enhance vascular risk.

Though Rowe and Kahn interpreted "avoidance of disease" broadly, vascular disease is particularly significant to healthy aging as these chronic conditions (e.g., hypertension, atrial fibrillation, and diabetes) gradually compromise adaptive resources. Neural network functioning is broadly compromised by the effect of vascular disease on cerebral tissue, termed cerebrovascular burden (CVB). CVB is also associated with higher stroke risk, cardiac disease, and sensorimotor impairment. Moreover, high CVB hastens the manifestation of clinically significant cognitive impairment, regardless of the specific etiology of cognitive decline (e.g., normative aging, Alzheimer's disease, Parkinson's disease, and vascular dementia) [11]. Elders with high CVB tend to have less prefrontal white matter volume, more white matter hyperintensities, and comparably impaired executive functioning [12]. Considerable evidence also exists that CVB also contributes to the development of late-life depression symptoms [13, 14]. Thus, in addition to being a broad measure of physical health in the Rowe and Kahn model, inclusion of CVB will distinguish variance in mortality risk directly related to this variable, thereby providing a more stringent test of how depression and cognition independently relate to longevity.

Successful aging theory identifies sustained engagement in social and productive activities as central to healthy aging. Clinical depression throughout the lifespan is characterized by reduced enjoyment in activities and decreased social engagement. As such, depression symptoms in late life represent a significant barrier to successful aging based on this interpretation. Generally speaking, more depression symptoms translate to poorer health outcomes. For instance, people experiencing depression are at greater risk of a first heart attack [15], stroke [16, 17], cancer [18], worse health outcomes after controlling for cardiovascular risks [15], and higher mortality [19]. Depression in later life was found to be a significant risk factor for death. Mehta et al. [20] reported that, in a large sample of community-dwelling elders, mortality was significantly predicted by both number of depressive symptoms and performance on a measure of cognitive functioning. Similarly, it was reported that in a sample of older patients with debilitating or chronic medical diagnoses, after controlling for age, comorbidity and illness severity, functional impairment and cognitive functioning, depressed respondents were 34% more likely to die over three years [21]. In a large ($N = 3065$) Dutch sample of individuals between the ages of 55 and 80, depression was identified as a significant risk factor for death over four years. However, the strength of this effect was partially eroded by the addition of other variables such as chronic disease, smoking, and physical inactivity [22].

The third domain of successful aging theory identifies preservation of cognitive and physical functioning as critical to successful aging. Cognitive impairment limits quality of life by reducing the capacity for meaningful work and social interaction [23], and rapid loss of cognitive faculties often suggests medical decline with a heightened mortality risk [22, 24]. Terminal cognitive drop is identified as an accelerated loss of cognitive functioning preceding death [24], by contrast to terminal decline which is a linear decline function preceding death [25]. A review from 2002 concluded that, largely because testing terminal drop theory requires a repeated-measures design, limited data existed supporting this theory [26]. Since this review, several longitudinal studies have been published, including a recent study concluding that elders with sharp declines on the Minimental State Exam [27], a brief cognitive screening measure, experienced more activity of daily living (ADL) disability and higher mortality rates than elders with more stable scores [28]. While estimates of the temporal relationship between terminal drop and death vary, recent research identified evidence of terminal drop at a mean of 42 months before death [29] in a large sample of dementia-free elders. These estimates are roughly similar to the original estimates of about 5 years reported by K. F. Riegel and R. M. Riegel [24]. Similarly, past work drawing on the Health and Retirement Survey (HRS) data identified a relationship between cognitive impairment and mortality over a 2-year interval [30]; however, this study evaluated cognitive decline cross-sectionally and did not include other markers of decline such as CVB or depression. Of note, Langa et al. reported evidence of compression of cognitive morbidity; elders with moderate or severe cognitive impairment in 2002 had greater risk of death over 2 years than those with similar levels of impairment in 1993.

Comorbid cognitive impairment and depressive symptoms suggest particularly high risk of death [19, 20]; however, these studies evaluated cognitive functioning cross-sectionally. While impairment on cognition measures *suggests* decline from demographically representative norms, the concept of terminal cognitive drop specifies rapid loss of cognitive functioning over a brief period of time. Relatively few studies relating to cognition and longevity evaluate how decline over brief periods relates to longevity, and even fewer investigate whether depression and cognitive decline are independent predictors of mortality. Because cognition and mood, domains in which impairment may be most evident to family and medical practitioners alike, tend to be interrelated in late life [31, 32], it is important to distinguish the individual relationships of these variables with longevity.

2. Objectives of the Present Study

This investigation seeks to examine whether all three domains of Rowe and Kahn's successful aging paradigm independently predict longevity in a large sample of stroke-free women over the age of 80. In particular, this study emphasizes two behavioral domains that are of particular relevance in clinical settings—depression and cognitive decline. This theoretical orientation, based on a broad reading of the literature, posits that elders with few depression symptoms and preserved cognitive functioning will experience greater longevity. In addition to CVB, other health variables such as self-rated health and Body Mass Index (BMI) will be

included in order to control for general health. The model proposed predicts that both depression and cognitive decline will predict mortality in this sample. Hypothesis 1 looks at survival across the entire study period of 8 years, whereas Hypothesis 2 examines proximate predictors of death across 2-year intervals.

Hypothesis 1. Physical health, depressive symptoms, and cognitive decline at baseline (2000 wave) will all be independent predictors of longevity conceptualized as survival across the entire 8-year study period.

Hypothesis 2. Incidence of death at each wave (2002, 2004, 2006, and 2008) will be predicted by CVB, cognitive decline, and depressive symptoms at the previous wave.

3. Methodology

3.1. Sample. The Health and Retirement Survey (HRS) is a prospective cohort study conducted by the University of Michigan with support from the National Institute on Aging. The first wave of the HRS occurred in 1992 with a 51-to-61 year-old cohort and was merged with the older (70 years and older) Asset and Health Dynamics of the Oldest Old Study (AHEAD) cohort in 1998. Two additional cohorts were added in 1998 to fill in the gaps between these two groups. Briefly, the HRS is a multistage probability cohort sample of US households. Further details on the HRS design and methods have been previously published [33].

The present study utilized the Health and Retirement Survey (HRS) that was prepared by the RAND Center for the Study of Aging (RAND HRS). The selected portion of this publically available, longitudinal dataset includes five waves at two-year intervals from 2000 through 2008 (waves 5 through 9). Inclusion criteria included age over 80 years at the first wave and female sex. This study made use of the 1998 TICS score to identify incidence of cognitive decline from 1998 to 2000. Because stroke is associated with highly variable cognitive performance, participants with history of stroke prior to 1998 were excluded. Respondents who were unable to complete survey materials at the 1998 data collection were also excluded as missing data would have precluded calculation of 1998–2000 cognitive change scores. This data set is demographically representative of the female US population over age 80.

3.2. Measures

3.2.1. Medical Data. Medical data (hypertension, diabetes, history of heart disease, height, and weight) and lifetime history of smoking were collected by self-report. CVB was identified as the number of cerebrovascular risk factors (hypertension, diabetes, and history of heart disease) reported creating a score ranging from 0 to 3.

3.2.2. Depressive Symptoms. A shortened, 8-item form of the original Center for Epidemiological Studies Depression Scale (CESD) was used to evaluate depression [34]. Six of the eight items are negatively worded, and two are positively worded. Participants are asked to respond "yes" or "no" to each item ("was depressed," "everything was an effort," "sleep was restless," "was happy," "felt lonely," "enjoyed life," "felt sad," "could not get going") that occurred within the preceding week. Scores ranged from 0 to 8 with higher scores indicating greater depressive symptoms. Using HRS data, the reliability of the 8-item CESD measure was adequate, with Cronbach's alpha ranging from .81 to .83 between waves [35]. High validity and symptom dimensions similar to those in the longer 20-item CESD have been demonstrated using these 8 items [35, 36]. The CESD is broadly used in the epidemiological study of late-life depression [37]. While the CESD literature describes a clinical cutoff that can be used to distinguish respondents with probable depression[35], this measure was used as a semicontinuous variable representing the full range depressive symptoms in this population sample.

3.2.3. Cognitive Functioning. The HRS data includes a brief standardized 35-point measure of cognitive functioning that was developed for remote screening of cognitive disorders based on the Telephone Interview for Cognitive Status [38]. It includes indices of orientation, concentration, short-term memory, mathematical skills, praxis and language and has a maximum score of 35 points (observed range: 0–35) with higher scores reflecting better functioning. This instrument has a Cronbach's alpha of.69 and past work has identified factors reflecting mental status and memory [39]. The TICS has demonstrated high test-retest reliability and is generally sensitive to cognitive impairment [38, 40–42]. For all of the following models cognitive decline was identified as a decrease in TICS score from one wave to the next wave of more than one standard deviation (6.1 points) based on baseline data.

3.2.4. Self-Rated Health. Self-rated health change was measured with a single question assessing the respondents' perception of change in health since the last data collection 2 years prior. Change in self-rated health was assessed with the question, "Compared to your health when we talked with you in (last wave) would you say that your health is better now, about the same, or worse?" Response options included "much better," "somewhat better," "same," "somewhat worse," "much worse," comprising a 5-point scale. This variable was included as a semicontinuous measure.

4. Statistical Methodology

Binary logistic regression was performed to model the likelihood of death between baseline (2000) and either 2006 or 2008 as a function of demographics, cognitive function, CVB, and depression. While the study spans the years 2000–2008, the logistic regression using 2006 as an end point was included as closer to half of the sample had died at this wave, thereby optimizing the statistical power of the model. Variables were entered blockwise with age, years of education, and body mass index (BMI) entered in the first block. The second block included CVB as reported in the 2000 wave.

Table 1: Sample description at the 2000 wave (baseline).

Variable	Mean	SD
Age	85.63	3.82
Education	11.03	3.42
CVB	1.03	0.82
CESD	2.07	1.98
TICS	18.13	5.86
BMI	24.40	4.86
Self-rated health change	3.36	0.83
Ethnic distribution	Percentage	
White	79.7%	
Black	14.2%	
Hispanic	5.2%	
Other	1.1%	
% high CVB	42.2%	

Note. CVB: number of symptoms comprising cerebrovascular burden (heart disease, diabetes, hypertension scored 0–3). CESD: Center for Epidemiological Studies Depression Scale, TICS: Telephone Interview for Cognitive Status, BMI: Body Mass Index.

The third block included the self-rated health change variable at the 2000 wave. The fourth block included CESD score at the 2000 wave and a variable reflecting incidence of decline greater than 1 standard deviation (>6 points) on the TICS between the 1998 and 2000 waves.

To better track the relationship between TICS score decline, mood, and longevity over the course of the study, four additional logistic regression models were computed. In the first, incidence of death in 2002 was predicted using 2000 CVB, the 2000 CESD score, and the index reflecting a drop in TICS score between 1998 and 2000 exceeding 1 standard deviation. The second model predicted death in 2004 based on CVB in 2002, CESD score in 2002, and incidence of decline in TICS score of more than 1 standard deviation between 2000 and 2002. The third and fourth models predicted death in 2006 and 2008 using similar predictors, respectively.

5. Results

Of the 1368 respondents who met criteria at the 1998 wave, 1186 were living at the 2000 wave. Of these, 417 were living at the 2008 wave representing a 64.8% mortality rate over this 8-year period. The sample at the 2000 wave is described in Table 1. The mean age was 85.6 years (SD = 3.8). On average, respondents had 11 years of formal education (SD = 3.4). The mean BMI at baseline was 24.4 (SD = 4.9). The average number of cerebrovascular risk factors reported was 1 (SD = 0.8). The mean CESD score was 2.1 (SD = 2) suggesting a low rate of depressive symptoms in this population. The mean score on the 35 TICS measure was 18.1 (SD = 5.9).

As can be seen in Table 2, CVB and depressive symptoms were significant predictors of mortality between 2000 and 2006. In addition, age, and BMI were also significant predictors. Cognitive decline and self-rated health showed a trend toward being significant predictors. A slightly different picture emerged with respect to 2008 outcomes. CVB was a significant predictor of mortality, and again BMI and age also predicted mortality. Depressive symptoms showed a trend toward significance, while self-rated health was not a significant predictor. Cognitive decline between 1998 and 2000 was not a significant predictor of mortality at the 2008 wave. Overall, the logistic regressions over 6 and 8 years provided partial support for applying Rowe and Kahn's successful aging model to longevity.

Four additional logistic regressions predicting death in 2002, 2004, 2006, and 2008 were completed to better understand the relationship between CVB, mood, cognitive change, and longevity over brief periods of time. CESD and TICS scores are unavailable for many participants, primarily for reasons of incapacity. As a result of listwise deletion caused by absent data on these predictor variables, these four logistic regression analyses included 69% (2002), 61% (2004), 54% (2006), and 43% (2008) of respondents who died, respectively. As described in Table 3, CVB significantly predicted mortality in the 2002, 2004, and 2008 waves. CESD score was a significant predictor of death in the 2002 and 2004 waves. Decline in TICS score over 2 years of more than 1 standard deviation significantly predicted incidence of death in 2004 and 2008 and showed a trend toward significance in 2002 ($P = .088$).

6. Conclusions

The primary findings of the present research are that, among women over the age of 80, CVB, depressive symptoms, and rapid cognitive decline (terminal drop) predict incidence of mortality over brief periods (2 years). CVB significantly predicted 2-year mortality at 3 of 4 waves, and depressive symptoms and cognitive decline significantly predicted mortality at 2 of 4 waves. Over long periods (6 or 8 years) mortality was significantly predicted by age, BMI, and CVB. Additionally, depressive symptoms significantly predicted mortality over 6 years and showed a trend toward significance in predicting mortality over 8 years. Incidence of rapid cognitive decline was not a significant predictor of death over 6 or 8 years. As demonstrated in the analyses predicting death over 2-year periods, rapid cognitive decline is a robust predictor of proximal death. Together, these findings suggest that, in addition to undermining quality of life and independence as described by Rowe and Kahn, sharp cognitive decline suggests high risk of imminent death. Cognitive drop, even in the older old is a significant indicator of declining health and potentially shortened life. While it is well documented that cognitive abilities decline in those over 80 [43], it is when declines are significantly above the norm when life expectancy is affected among the older old. By contrast, many of those who died toward the end of this study had robust cognitive functioning at this baseline interval. These results are consistent with past work describing the relationship between CVB, mood, cognitive decline, and longevity [19, 20, 22, 24, 28, 29].

The present findings support and extend Rowe and Kahn's [1] successful aging theory by relating to low CVB, relatively few depressive symptoms, and preserved cognitive functioning, representing the three domains of successful

Table 2: Results of logistic regression predicting incidence of mortality between 2000 and 2006.

	Predicting death between 2000 and 2006				Predicting death between 2000 and 2008			
Variable	*B*	SE	Wald	Exp(*B*) 95% CI	*B*	SE	Wald	Exp(*B*) 95% CI
Age	0.15	0.02	55.65$^{\Psi}$	1.12–1.21	0.15	0.02	46.86$^{\Psi}$	1.11–1.22
Education	−0.03	0.02	1.61	.93–1.02	−0.03	0.02	1.54	.93–1.02
BMI	−0.07	0.02	17.27$^{\Psi}$	.91–.97	−0.07	0.02	18.76$^{\Psi}$	.90–.96
CVB	0.46	0.09	26.74$^{\Psi}$	1.33–1.88	0.56	0.09	36.19$^{\Psi}$	1.46–2.10
Self-rated health	0.17	0.09	3.21§	.98–1.42	0.15	0.10	2.37	.96–1.41
2000 CESD score	0.08	0.04	4.26*	1.00–1.16	0.07	0.04	3.46§	.99–1.16
1998–2000 TICS Drop	0.35	0.21	2.81§	.94–2.12	0.05	0.22	0.06	.69–1.62
Constant	−12.00	1.82	43.44		−11.31	1.97	32.87	

Note. BMI: Body Mass Index. CVB: cerebrovascular burden, self-rated health change: change in self-rated health from previous wave, CESD: Center for Epidemiological Studies Depression Scale, TICS: Telephone Interview for Cognitive Status. TICS Drop reflects incidence of decline on TICS score greater than 6 points between 1998 and 2000.
$^{\S}P$ between .05 and .10, $^{*}P < .05$, $^{\Psi}P < .001$, df = 1 for all comparisons.

Table 3: Results of logistic regression analyses predicting death at 2002, 2004, 2006, and 2008 waves based on depressive symptoms and incidence of cognitive decline.

	Predicting death in 2002					Predicting death in 2004			
	B	SE	Wald	Exp(*B*) 95% CI		*B*	SE	Wald	Exp(*B*) 95% CI
2000 CVB	.54	.11	26.83$^{\Psi}$	1.40–2.11	2002 CVB	.43	.12	11.97$^{\Psi}$	1.21–1.97
2000 CESD	.08	.04	3.90*	1.00–1.18	2002 CESD	.17	.05	13.71$^{\Psi}$	1.09–1.31
1998–2000 TICS >1SD	.40	.23	2.92§	.94–2.33	2000–2002 TICS >1SD	.76	.27	8.11^{+}	1.27–3.61
Constant	−2.49	.18	183.52$^{\Psi}$		Constant	−2.68	.22	146.83$^{\Psi}$	
	Predicting death in 2006					Predicting death in 2008			
	B	SE	Wald	Exp(*B*) 95% CI		*B*	SE	Wald	Exp(*B*) 95% CI
2004 CVB	.22	.14	2.61	.95–1.64	2006 CVB	.44	.17	6.93^{+}	1.12–2.16
2004 CESD	.03	.06	0.29	.92–1.15	2006 CESD	.07	.07	1.09	0.94–1.22
2002–2004 TICS > 1SD	.39	.34	1.36	.77–2.85	2004–2006 TICS > 1SD	1.12	.43	6.86^{+}	1.33–7.06
Constant	−1.79	.22	64.51		Constant	−2.15	.30	50.13$^{\Psi}$	

Note. CVB: cerebrovascular burden. CESD: Center for Epidemiological Studies Depression Scale. TICS: Telephone Interview for Cognitive Status. TICS Drop reflects incidence of decline on TICS score greater than 6 points between waves as indicated.
$^{\S}P = .088$, $^{*}P < .05$. $^{+}P < .01$, $^{\Psi}P < .001$, df = 1 for all comparisons.

aging theory, to greater longevity in this sample of older women. In the present study, Rowe and Kahn's formulation of successful aging is expanded from quality of life to length of life. Heightened depression and rapid loss of cognition were significantly related to timing of death, and conversely robust functioning was related to longevity. These findings underscore the significance of behavioral factors to the discussion of longevity. Behavioral factors such as mood and gross cognitive functioning are basic characteristics of the individual patient, and these findings highlight the importance of subjective reports or clinician perceptions of decline in these areas, especially when working with older patients. The finding that CVB predicts longevity broadly supports the large medical and epidemiological literature citing conditions such as heart disease, hypertension, and diabetes as risk factors for death (discussed in [11]). Our finding that depressive symptoms predict longevity corroborate significant past work on this subject [19–22].

These findings are also consistent with the concept of terminal cognitive drop [24]. While some studies support the terminal cognitive decline effect over longer periods of time [24, 28, 29], these findings support other research [19, 20] that terminal cognitive drop theory can be applied over brief periods of time. Additionally, most research supporting terminal cognitive decline relates mortality to impaired performance on cognitive measures, suggesting decline from an idiographic baseline and consequently discounting the time period over which decline occurs. By contrast, the present study contributes to the existing literature by testing this theory among older old women using longitudinal data.

The primary limitation of the present study is that cognitive and mood data are not available for many respondents approaching death. This data is absent largely for reasons of incapacity. Consequently, it is likely that the relationship between depressive symptoms, cognitive decline, and longevity is underrepresented by the present research. Another limitation is the use of categorical self-reported CVB data. However, use of such data is common in population-based samples and reasonable concordance values between self-reports of disease and medical chart reviews have been reported [44, 45]. Future research should build on the present finding by identifying relationships between

markers of cerebrovascular health, depression, and cognitive decline.

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References

[1] J. W. Rowe and R. L. Kahn, "Successful aging," *The Gerontologist*, vol. 37, no. 4, pp. 433–440, 1997.

[2] N. R. Cook, M. S. Albert, L. F. Berkman, D. Blazer, J. O. Taylor, and C. H. Hennekens, "Interrelationships of peak expiratory flow rate with physical and cognitive function in the elderly: MacArthur Foundation Studies of Aging," *Journals of Gerontology—Series A*, vol. 50, no. 6, pp. M317–M323, 1995.

[3] J. Chodosh, D. M. Kado, T. E. Seeman, and A. S. Karlamangla, "Depressive symptoms as a predictor of cognitive decline: MacArthur Studies of Successful Aging," *The American Journal of Geriatric Psychiatry*, vol. 15, no. 5, pp. 406–415, 2007.

[4] T. L. Gruenewald, A. S. Karlamangla, G. A. Greendale, B. H. Singer, and T. E. Seeman, "Feelings of usefulness to others, disability and mortality in older adults: the MacArthur Study of Successful Aging," *Journals of Gerontology—Series B*, vol. 62, no. 1, pp. P28–P37, 2007.

[5] W. J. Strawbridge, M. I. Wallhagen, and R. D. Cohen, "Successful aging and well-being: self-rated compared with Rowe and Kahn," *The Gerontologist*, vol. 42, no. 6, pp. 727–733, 2002.

[6] M. B. Holstein and M. Minkler, "Self, society and the 'New Gerontology'," *The Gerontologist*, vol. 43, no. 6, pp. 787–796, 2003.

[7] S. Arber and H. Cooper, "Gender differences in health in later life: the new paradox?" *Social Science and Medicine*, vol. 48, no. 1, pp. 61–76, 1999.

[8] A. L. Rosso, C. B. Eaton, R. Wallace et al., "Combined impact of geriatric syndromes and cardiometabolic diseases on measures of functional impairment," *Journals of Gerontology—Series A*, vol. 66, no. 3, pp. 349–354, 2011.

[9] M. L. Bruce, T. E. Seeman, S. S. Merrill, and D. G. Blazer, "The impact of depressive symptomatology on physical disability: MacArthur Studies of Successful Aging," *The American Journal of Public Health*, vol. 84, no. 11, pp. 1796–1799, 1994.

[10] M. Hirvensalo, T. Rantanen, and E. Heikkinen, "Mobility difficulties and physical activity as predictors of mortality and loss of independence in the community-living older population," *Journal of the American Geriatrics Society*, vol. 48, no. 5, pp. 493–498, 2000.

[11] L. Flicker, "Cardiovascular risk factors, cerebrovascular disease burden and healthy brain aging," *Clinics in Geriatric Medicine*, vol. 26, no. 1, pp. 17–27, 2010.

[12] N. Raz, K. M. Rodrigue, and J. D. Acker, "Hypertension and the brain: vulnerability of the prefrontal regions and executive functions," *Behavioral Neuroscience*, vol. 117, no. 6, pp. 1169–1180, 2003.

[13] J. R. Sneed, D. Rindskopf, D. C. Steffens, K. R. Krishnan, and S. P. Roose, "The vascular depression subtype: evidence of internal validity," *Biological Psychiatry*, vol. 64, no. 6, pp. 491–497, 2008.

[14] C. E. Coffey, G. S. Figiel, W. T. Djang, and R. D. Weiner, "Subcortical hyperintensity on magnetic resonance imaging: a comparison of normal and depressed elderly subjects," *The American Journal of Psychiatry*, vol. 147, no. 2, pp. 187–189, 1990.

[15] A. H. Glassman and P. A. Shapiro, "Depression and the course of coronary artery disease," *The American Journal of Psychiatry*, vol. 155, no. 1, pp. 4–11, 1998.

[16] M. J. Bos, T. Lindén, P. J. Koudstaal et al., "Depressive symptoms and risk of stroke: the Rotterdam Study," *Journal of Neurology, Neurosurgery and Psychiatry*, vol. 79, no. 9, pp. 997–1001, 2008.

[17] S. L. Larson, P. L. Owens, D. Ford, and W. Eaton, "Depressive disorder, dysthymia and risk of stroke: thirteen-year follow-up from the Baltimore Epidemiologic Catchment Area Study," *Stroke*, vol. 32, no. 9, pp. 1979–1983, 2001.

[18] B. W. Penninx, J. M. Guralnik, M. Pahor et al., "Chronically depressed mood and cancer risk in older persons," *Journal of the National Cancer Institute*, vol. 90, no. 24, pp. 1888–1893, 1998.

[19] C. L. Arfken, P. A. Lichtenberg, and M. E. Tancer, "Cognitive impairment and depression predict mortality in medically ill older adults," *Journals of Gerontology—Series A*, vol. 54, no. 3, pp. M152–M156, 1999.

[20] K. M. Mehta, K. Yaffe, K. M. Langa, L. Sands, M. A. Whooley, and K. E. Covinsky, "Additive effects of cognitive function and depressive symptoms on mortality in elderly community-living adults," *Journals of Gerontology—Series A*, vol. 58, no. 5, pp. 461–467, 2003.

[21] K. E. Covinsky, E. Kahana, M. H. Chin, R. M. Palmer, R. H. Fortinsky, and C. S. Landefeld, "Depressive symptoms and 3-year mortality in older hospitalized medical patients," *Annals of Internal Medicine*, vol. 130, no. 7, pp. 563–569, 1999.

[22] B. W. Penninx, S. W. Geerlings, D. J. Deeg, J. T. van Eijk, W. van Tilburg, and A. T. Beekman, "Minor and major depression and the risk of death in older persons," *Archives of General Psychiatry*, vol. 56, no. 10, pp. 889–895, 1999.

[23] P. Missotten, G. Squelard, M. Ylieff et al., "Quality of life in older Belgian people: comparison between people with dementia, mild cognitive impairment and controls," *International Journal of Geriatric Psychiatry*, vol. 23, no. 11, pp. 1103–1109, 2008.

[24] K. F. Riegel and R. M. Riegel, "Development, drop and death," *Developmental Psychology*, vol. 6, no. 2, pp. 306–319, 1972.

[25] E. Palmore and W. Cleveland, "Aging, terminal decline and terminal drop," *Journals of Gerontology*, vol. 31, no. 1, pp. 76–81, 1976.

[26] H. B. Bosworth and I. C. Siegler, "Terminal change in cognitive function: an updated review of longitudinal studies," *Experimental Aging Research*, vol. 28, no. 3, pp. 299–315, 2002.

[27] M. F. Folstein, S. E. Folstein, and P. R. McHugh, "'Mini-mental state': a practical method for grading the cognitive state of patients for the clinician," *Journal of Psychiatric Research*, vol. 12, no. 3, pp. 189–198, 1975.

[28] K. Yaffe, K. Lindquist, E. Vittinghoff et al., "The effect of maintaining cognition on risk of sisability and death," *Journal of the American Geriatrics Society*, vol. 58, no. 5, pp. 889–894, 2010.

[29] R. S. Wilson, T. L. Beck, J. L. Bienias, and D. A. Bennett, "Terminal cognitive decline: accelerated loss of cognition in the last years of life," *Psychosomatic Medicine*, vol. 69, no. 2, pp. 131–137, 2007.

[30] K. M. Langa, E. B. Larson, J. H. Karlawish et al., "Trends in the prevalence and mortality of cognitive impairment in the

United States: is there evidence of a compression of cognitive morbidity?" *Alzheimer's and Dementia*, vol. 4, no. 2, pp. 134–144, 2008.

[31] B. T. Mast, B. Yochim, S. E. MacNeill, and P. A. Lichtenberg, "Risk factors for geriatric depression: the importance of executive functioning within the vascular depression hypothesis," *Journals of Gerontology—Series A*, vol. 59, no. 12, pp. 1290–1294, 2004.

[32] A. Bielak, D. Gerstorf, K. M. Kiely, K. J. Anstey, and M. Luszcz, "Depressive symptoms predict decline in perceptual speed in older adulthood," *Psychology and Aging*. In press.

[33] S. G. Heeringa and J. Conner, "Technical description of the Health and Retirement Study sample design," HRS/AHEAD Documentation Report DR-002, University of Michigan, Ann Arbor, Mich, USA, 1995.

[34] L. Radloff, "The CES-D Scale: a self-report depression scale for research in the general population," *Applied Psychological Measurement*, vol. 1, no. 3, pp. 385–401, 1977.

[35] D. E. Steffick, "Documentation of affective functioning measures in the Health and Retirement Study," HRS Documentation Report DR-005, Survey Research Center at the Institute for Social Research, Ann Arbor, Mich, USA, 2000.

[36] R. Wallace, A. R. Herzog, M. B. Ofstedal et al., "Documentation of affective functioning measures in the Health and Retirement Study," Tech. Rep., Survey Research Center, University of Michigan, Ann Arbor, Mich, USA, 2000.

[37] A. T. F. Beekman, D. J. H. Deeg, J. Van Limbeek, A. W. Braam, M. Z. De Vries, and W. Van Tilburg, "Criterion validity of the Center for Epidemiologic Studies Depression scale (CES-D): results from a community-based sample of older subjects in the Netherlands," *Psychological Medicine*, vol. 27, no. 1, pp. 231–235, 1997.

[38] J. Brandt, M. Spencer, and M. Folstein, "The telephone interview for cognitive status," *Neuropsychiatry, Neuropsychology and Behavioral Neurology*, vol. 1, no. 2, pp. 111–117, 1988.

[39] A. R. Herzog and R. B. Wallace, "Measures of cognitive functioning in the AHEAD study," *Journals of Gerontology—Series B*, vol. 52, special issue, pp. 37–48, 1997.

[40] K. A. Welsh, J. C. S. Breitner, and K. M. Magruder-Habib, "Detection of dementia in the elderly using telephone screening of cognitive status," *Neuropsychiatry, Neuropsychology and Behavioral Neurology*, vol. 6, no. 2, pp. 103–110, 1993.

[41] D. W. Desmond, T. K. Tatemichi, and L. Hanzawa, "The telephone interview for cognitive status (TICS): reliability and validity in a stroke sample," *International Journal of Geriatric Psychiatry*, vol. 9, no. 10, pp. 803–807, 1994.

[42] T. Järvenpää, J. O. Rinne, I. Räihä et al., "Characteristics of two telephone screens for cognitive impairment," *Dementia and Geriatric Cognitive Disorders*, vol. 13, no. 3, pp. 149–155, 2002.

[43] C. L. Dahle, B. S. Jacobs, and N. Raz, "Aging, vascular risk and cognition: blood glucose, pulse pressure and cognitive performance in healthy adults," *Psychology and Aging*, vol. 24, no. 1, pp. 154–162, 2009.

[44] T. L. Bush, S. R. Miller, A. L. Golden, and W. E. Hale, "Self-report and medical record report agreement of selected medical conditions in the elderly," *The American Journal of Public Health*, vol. 79, no. 11, pp. 1554–1556, 1989.

[45] B. M. Psaty, L. H. Kuller, D. Bild et al., "Methods of assessing prevalent cardiovascular disease in the cardiovascular health study," *Annals of Epidemiology*, vol. 5, no. 4, pp. 255–335, 1995.

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Research Article

Dental Health Behaviors, Dentition, and Mortality in the Elderly: The Leisure World Cohort Study

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In the last decade the effect of oral health on the general health and mortality of elderly people has attracted attention. We explored the association of dental health behaviors and dentition on all-cause mortality in 5611 older adults followed from 1992 to 2009 (median = 9 years) and calculated risk estimates using Cox regression analysis in men and women separately. Toothbrushing at night before bed, using dental floss everyday, and visiting the dentist were significant risk factors for longevity. Never brushing at night increased risk 20–35% compared with brushing everyday. Never flossing increased risk 30% compared with flossing everyday. Not seeing a dentist within the last 12 months increased risk 30–50% compared with seeing a dentist two or more times. Mortality also increased with increasing number of missing teeth. Edentulous individuals (even with dentures) had a 30% higher risk of death compared with those with 20+ teeth. Oral health behaviors help maintain natural, healthy and functional teeth but also appear to promote survival in older adults.

1. Introduction

Only in the last decade has the possible effect of oral health on the general health and mortality of elderly people attracted much attention. An association between number of teeth and mortality has been reported in several studies [1–11]. As people age, many lose teeth. Tooth loss reduces masticatory capacity, which can influence food selection, nutritional status, and general health. Evidence is also increasing that oral infections play a role in the pathogenesis of some systemic diseases and may be especially debilitating in the old and frail [12, 13].

The focus of the present study was to examine the possible role not only of dentition status, especially the number of natural teeth and use of dentures, but also of dental health practices as predictors of mortality in elderly men and women. In addition, we wanted to determine if any observed effects would remain after adjusting for other health and lifestyle factors related to mortality. We hypothesized that more teeth and better dental health behaviors would be related to increased longevity. We report here the results in a large cohort (over 5000) of elderly (median age 81 years) men and women followed for 17 years.

2. Materials and Methods

The Leisure World Cohort Study was established in the early 1980s when 13,978 (8877 female and 5101 male) residents of a California retirement community (Leisure World Laguna Hills) completed a postal health survey. Residents were recruited in four waves: those who owned homes in Leisure World on June 1, 1981; new residents who had moved into the community and were living there on June 1, 1982; on June 1, 1983; on October 1, 1985. The population and the cohort are mostly Caucasian, well educated, upper-middle class, and elderly.

The baseline survey asked demographic information (birth date, sex, marital status, number of children, height, and weight), brief medical history (high blood pressure,

angina, heart attack, stroke, diabetes, rheumatoid arthritis, fractures after age 40, cancer, gallbladder surgery, glaucoma, and cataract surgery), medication use (hypertensive medication, digitalis, and nonprescription pain medication), personal habits (cigarette smoking, exercise, alcohol consumption, and vitamin supplement use), and beverage intake (milk, regular coffee, decaffeinated coffee, black or green tea, and soft drinks).

2.1. Dental Health Status and Behaviors. We mailed a follow-up survey to the 8403 cohort members still alive on November 1, 1992. Emphasis was placed on questions regarding dental health, focusing on the number of natural teeth, dentures worn, number of visits to a dentist, and oral health habits. Participants were asked to indicate how often they did the following: "brush my teeth in the morning, brush my teeth at night before bed, brush my teeth during the day, clean my dentures, use dental floss, use mouth wash, and use a tooth pick" with the response categories of everyday, sometimes, never. The survey was returned by 6173 cohort members (73%). We were unable to contact 13 cohort members and another 384 (5%) died within one year of the mailing. Of those returning the survey, 487 did not indicate the number of teeth or provide information on any dental health practice and were eliminated from the analyses.

Jackson and Murray suggested 16 teeth as the lowest acceptable number of natural teeth in persons older than 60 years for adequate masticatory function [14]. We used this categorization with the additional division of 16+ teeth into 16–25 and 26+ teeth. This was further refined to 10 teeth in the upper jaw and six in the lower jaw [15]. We used this latter criteria to classify our subjects into two groups: those with or without adequate natural dentition. Other studies have defined adequate dental status as at least 20 teeth [2], and we used this as an alternative definition, further classifying those with fewer than 20 teeth as wearing dentures or not.

2.2. Lifestyle Factors. Information on the lifestyle factors came from the original health survey completed in the early 1980s. We previously reported the effects of several lifestyle practices on all-cause mortality in this cohort [16–20]. Other studies have shown that body mass index and obesity [21, 22], smoking [23–25], alcohol consumption [25, 26], and coffee intake [26] are related to the number of teeth.

Based on their reported smoking history we classified participants as never, past, or current smokers [16].

Consumption of alcoholic beverages was asked separately for wine (4 oz), beer (12 oz), and hard liquor (1 oz), each equivalent to about 1/2 oz of alcohol. Response choices for average weekday consumption were never drink, less than 1, 1, 2, 3, and 4 or more drinks. Total alcohol intake per day was calculated by summing the number of drinks consumed of each type [17]. Individuals were then categorized into four groups: 0, $\leq$1, 2-3, and 4+ drinks/day.

We estimated daily caffeine intake by summing the frequency of consumption of each beverage and chocolate multiplied by its average caffeine content (mg/standard unit) as 115, 3, 50, 50, and 6 for regular coffee, decaffeinated coffee, tea, cola soft drinks, and chocolate, respectively [18]. Caffeine intake was categorized as <50, 50–99, 100–199, 200–399, 400+ mg/day.

The amount of time spent on physical activities was ascertained by asking the amount of time spent in active and other (less physically demanding) activities. Activities were categorized as 0, $\leq$1/2, 3/4–1, 2+ hours/day for active activities and <2, 2-3, 4-5, 6+ hours/day for other activities [19].

Body mass index (weight (kg)/height $(m)^2$) was calculated based on self-reported height and weight at baseline and categorized according to federal guidelines: underweight (<18.5), normal weight (18.5–24.9), overweight (25–29.9), and obese (30+) [20, 27].

2.3. Determination of Outcome. Followup of the cohort is maintained by periodic resurvey and determination of vital status by search of governmental and commercial death indexes and ascertainment of death certificates. Participants were followed to death or December 31, 2009, whichever came first. To date, 40 cohort members have been lost to followup; search of death indices did not reveal that these individuals were deceased.

2.4. Statistical Analysis. Hazard ratios (HRs) and 95% confidence intervals (CIs) were obtained using Cox proportional hazard regression analysis [28]. For the Cox models, chronological age was used as the fundamental time scale with study entry being age in 1992 when the dental health survey was completed and the event of interest being age at death. Because women differ from men on many of the dental and potential confounding variables, women live longer on average than men, and for comparison with other studies which stratified by sex, we performed separate analyses for men and women. HRs were calculated for dental status and each dental health behavior adjusted for age (continuous) and then additionally adjusted for lifestyle variables plus seven separate histories (no, yes) of hypertension, angina, heart attack, stroke, diabetes, rheumatoid arthritis, and cancer, previously found to be significant risk factors for death in this cohort [16–20]. For some dental health behaviors separate analyses were done for subjects with teeth and for subjects with dentures. Statistical analyses were performed using SAS version 9.2 (SAS Institute Inc., Cary, NC). No adjustment in the P values was made for multiple comparisons.

Previous reports present details of the methods and validity of exposure and outcome data [16–20, 29–31]. The Institutional Review Boards of the University of Southern California and the University of California, Los Angeles approved the study.

3. Results

After excluding 65 subjects with missing covariates, we analyzed data on 5611 subjects (3847 women and 1764 men). At study entry, the participants ranged in age from 52 to 105 years (median, 81 years). By December 31, 2009, the subjects had contributed 52,441 person-years of followup (median 9

Table 1: Characteristics of the cohort by sex.

	Men		Women	
Number	1764		3847	
	Mean	SD	Mean	SD
Age at time of dental survey (years)	81	6.5	80	7.1
Age at last followup (years)	89	5.9	90	6.2
Followup years	8.2	5.2	9.9	5.3
At time of original survey (1981–1985):				
Active activities (hrs/day)	1.2	1.3	1.1	1.2
Other activities (hrs/day)	3.9	2.8	4.8	2.7
Alcohol (drinks/day)	1.7	1.5	1.3	1.2
Caffeine (mg/day)	190	176	182	169
Body mass index (kg/m^2)	25	2.7	24	3.5
Number of natural teeth	19.0	10.5	20.5	9.9
	No.	%	No.	%
Medical history				
High blood pressure	572	32	1367	36
Angina	192	11	275	7.1
Heart attack	212	12	154	4.0
Stroke	58	3.3	63	1.6
Cancer	99	5.6	444	12
Diabetes	101	5.7	134	3.5
Rheumatoid arthritis	58	3.3	216	5.6
Smoke				
Never	651	37	2065	54
Past	980	56	1320	34
Current	133	7	462	12
Dentures	924	52	1703	44
Deceased by December 31, 2009	1588	90	3165	82

years), and 4753 had died. Age at death ranged from 64 to 108 years (median, 90 years).

Table 1 presents selected characteristics for the participants by sex. Differences between males and females were highly statistically significant ($P < .01$) for all variables except age at study entry ($P = .32$) and caffeine ($P = .11$). The median number of teeth in cohort members was 24 (range 0–32, mean 20) and nearly half had dentures. Men tended to have fewer teeth than women (median 23 versus 24) and had a greater frequency of denture use. Only 11% of the cohort was edentulous.

Table 2 shows the age-adjusted and mutivariable-adjusted HRs of all-cause mortality for number of teeth and the various definitions of adequate dentition for women and men, separately. Adjustment for potential confounders had limited effect, generally attenuating the observed HRs. Number of natural teeth was related to longevity in both men and women. Edentulous individuals had a 30% higher risk of death than subjects with 26–32 teeth; those with 1–15 teeth had a 20% higher risk. Compared with those with adequate natural masticatory function (10+ upper teeth and 6+ lower teeth), those with inadequate function had over a 15% increased risk of death. Ninety percent of subjects with inadequate natural masticatory function had dentures. Among women with inadequate natural masticatory function, those without dentures had a somewhat higher (not significant) risk of death than those with dentures. No difference was seen in men.

Table 3 shows the HRs for the dental health behaviors in individuals with teeth. Daily tooth brushing at night before bed and flossing significantly decreased risk of death while using a tooth pick or mouth wash did not. Both individuals who never brushed at night and those who never flossed had a 20–30% increased mortality risk compared to those who brushed at night or flossed everyday, respectively. Those who reported not brushing their teeth daily had a 41–91% increased risk of death compared with those who brushed three times daily—in the morning, during the day and at night. Brushing at night was the most significant tooth brushing variable. Risk was similar for those brushing at night everyday whether or not they brushed daily in the morning or during the day. Individuals who everyday brushed in the morning or during the day but not everyday at night had a 13–26% increased risk.

Table 2: Hazard ratios for mortality by dental status, Leisure World Cohort Study, 1992–2009.

	Men					Women				
	No.	Model 1*		Model 2*		No.	Model 1*		Model 2*	
		HR	95% CI	HR	95% CI		HR	95% CI	HR	95% CI
Number of natural teeth										
26–32	659	1.00		1.00		1709	1.00		1.00	
16–25	541	1.04	0.92–1.18	1.03	0.91–1.17	1131	1.01	0.93–1.10	0.97	0.89–1.06
1–15	343	1.22	1.07–1.41	1.21	1.05–1.40	627	1.23	1.11–1.36	1.17	1.06–1.30
0	221	1.33	1.13–1.56	1.18	1.00–1.39	380	1.30	1.15–1.47	1.21	1.07–1.37
Number of natural teeth										
20–32	1053	1.00		1.00		2551	1.00		1.00	
1–19	490	1.11	0.99–1.25	1.09	0.97–1.23	916	1.19	1.10–1.30	1.15	1.06–1.25
(i) Dentures—yes	446	1.12	1.00–1.30	1.10	0.98–1.24	809	1.19	1.09–1.30	1.15	1.05–1.25
(ii) Dentures—no	44	1.03	0.75–1.40	1.01	0.74–1.39	107	1.21	0.99–1.49	1.16	0.95–1.43
0	221	1.29	1.11–1.51	1.15	0.98–1.34	380	1.31	1.16–1.47	1.24	1.10–1.39
Adequate masticatory function: 10+ upper teeth and 6+ lower teeth										
Yes	1017	1.00		1.00		2498	1.00		1.00	
No	747	1.16	1.05–1.29	1.12	1.01–1.24	1349	1.22	1.13–1.31	1.17	1.08–1.26
(i) Dentures—yes	680	1.16	1.05–1.29	1.11	1.00–1.24	1205	1.21	1.12–1.30	1.16	1.07–1.25
(ii) Dentures—no	67	1.18	0.91–1.52	1.19	0.92–1.55	144	1.33	1.12–1.59	1.25	1.05–1.50
Dentures										
None	766	1.00		1.00		1786	1.00		1.00	
Partial or partial plus one jaw full	711	1.08	0.97–1.21	1.06	0.95–1.18	1359	1.08	1.00–1.17	1.05	0.97–1.14
Full upper and lower	213	1.29	1.10–1.51	1.14	0.96–1.34	344	1.22	1.08–1.38	1.14	1.01–1.30
Unknown	74	1.25	0.97–1.60	1.27	0.99–1.64	358	0.94	0.83–1.07	0.92	0.81–1.04

* Model 1: adjusted for age at entry.
Model 2: adjusted for age at entry, smoking, alcohol, caffeine, active activities, other activities, body mass index, high blood pressure, angina, heart attack, stroke, diabetes, rheumatoid arthritis, and cancer.

To sort out whether brushing teeth at night or flossing was more important we looked at both together. Among subjects who brushed their teeth at night everyday, never flossing conferred a significantly increased risk of death of about 25% compared with those who flossed everyday. Among subjects who used dental floss everyday, those never brushing their teeth at night had similarly higher risk than subjects who brushed everyday, but the risk was not statistically significant. However, the numbers never brushing were small, 33 men and 63 women, and the estimates of risk were similar to that of the all subjects who never brushed at night. Never brushing teeth at night and never flossing remained significant risk factors after adjusting for adequate dentition. The risk of not brushing was 1.29 (95% CI 1.10–1.51) in men and 1.18 (95% CI 1.01–1.36) in women. The risk of not flossing was 1.25 (95% CI 1.08–1.43) in men and 1.29 (95% CI 1.16–1.42) in women.

Table 4 shows the HRs for the dental health behaviors in individuals with dentures. Never cleaning dentures was a significant risk factor in men (HR = 1.25) but not in women. Use of mouthwash had no effect on mortality.

Compared with having visited a dentist twice in the last 12 months, not seeing a dentist was associated with a higher (25–50%) risk of death in both men and women and in both those with teeth and those with dentures. The risk was slightly higher in men than women and in those with teeth than in those with dentures.

4. Discussion

Our study extends the available literature on the survival benefits of adequate dentition, use of dentures among those without adequate natural masticatory function, brushing teeth at night before bed, and flossing in the elderly. Each of these was associated with reduced death in our elderly men and women. Although brushing teeth at night before bed and flossing were correlated ($r = 0.23$), each independently predicted risk.

Table 3: Hazard ratios for mortality by dental health behaviors among subjects with teeth, Leisure World Cohort Study, 1992–2009.

	Men					Women				
	No.	Model 1*		Model 2*		No.	Model 1*		Model 2*	
		HR	95% CI	HR	95% CI		HR	95% CI	HR	95% CI
Brush my teeth in the morning										
Everyday	1196	1.00		1.00		3014	1.00		1.00	
Sometimes	176	1.03	0.87–1.21	1.08	0.91–1.29	250	1.14	0.99–1.31	1.13	0.98–1.30
Never	171	0.94	0.79–1.12	0.95	0.80–1.12	203	0.99	0.85–1.15	151.01	0.86–1.17
Brush my teeth at night before bed										
Everyday	1112	1.00		1.00		2895	1.00		1.00	
Sometimes	222	1.17	1.01–1.37	1.13	0.97–1.32	350	1.17	1.03–1.32	1.13	1.00–1.28
Never	209	1.36	1.16–1.58	1.34	1.14–1.57	222	1.19	1.02–1.38	1.19	1.02–1.38
Brush my teeth during the day										
Everyday	265	1.00		1.00		965	1.00		1.00	
Sometimes	521	0.99	0.85–1.16	0.95	0.81–1.12	1498	1.01	0.92–1.10	0.98	0.90–1.07
Never	757	1.09	0.94–1.26	1.06	0.91–1.23	1004	1.14	1.03–1.25	1.10	1.00–1.21
Use dental floss										
Everyday	462	1.00		1.00		1572	1.00		1.00	
Sometimes	603	1.18	1.03–1.34	1.14	1.00–1.30	1226	1.05	0.97–1.14	1.03	0.95–1.12
Never	478	1.31	1.15–1.50	1.27	1.11–1.46	669	1.31	1.19–1.45	1.28	1.16–1.42
Use mouth wash										
Everyday	400	1.00		1.00		1134	1.00		1.00	
Sometimes	543	0.99	0.87–1.14	1.01	0.88–1.16	1249	1.00	0.91–1.09	0.98	0.90–1.07
Never	600	0.92	0.80–1.05	0.93	0.81–1.07	1084	0.98	0.89–1.07	1.00	0.91–1.10
Use a tooth pick										
Everyday	363	1.00		1.00		710	1.00		1.00	
Sometimes	661	0.93	0.81–1.07	0.95	0.83–1.10	1306	0.99	0.89–1.10	0.98	0.88–1.09
Never	519	1.00	0.87–1.15	1.01	0.88–1.16	1451	1.05	0.95–1.16	1.04	0.94–1.15
Brush my teeth										
Everyday morning, day and night	217	1.00		1.00		870	1.00		1.00	
Everyday morning and night, not everyday day	646	1.06	0.90–1.24	1.01	0.86–1.20	1679	1.04	0.95–1.14	1.01	0.93–1.11
Everyday night, not everyday morning/day	249	0.96	0.80–1.17	0.96	0.79–1.17	346	1.00	0.87–1.14	0.98	0.86–1.13
Everyday morning/day, not everyday night	350	1.26	1.06–1.51	1.19	0.99–1.43	496	1.13	1.00–1.28	1.09	0.97–1.24
Not everyday	81	1.41	1.08–1.83	1.37	1.05–1.80	76	1.91	1.49–2.44	1.77	1.38–2.28
Brush my teeth at night before bed among subjects who use dental floss everyday										
Everyday	403	1.00		1.00		1432	1.00		1.00	
Sometimes	26	1.20	0.79–1.82	1.28	0.82–1.99	77	0.94	0.72–1.23	0.87	0.66–1.15
Never	33	1.36	0.92–1.99	1.33	0.87–2.01	63	1.23	0.93–1.62	1.21	0.91–1.60
Use dental floss among subjects who brush teeth at night before bed everyday										
Everyday	403	1.00		1.00		1432	1.00		1.00	
Sometimes	422	1.17	1.01–1.36	1.14	0.98–1.33	988	1.04	0.95–1.14	1.02	0.93–1.12
Never	287	1.26	1.08–1.48	1.25	1.06–1.48	475	1.24	1.11–1.39	1.20	1.07–1.34

Table 3: Continued.

	Men					Women				
	No.	Model 1*		Model 2*		No.	Model 1*		Model 2*	
		HR	95% CI	HR	95% CI		HR	95% CI	HR	95% CI
Number of dental visits within the last 12 months										
2+	1173	1.00		1.00		2671	1.00		1.00	
1	235	1.05	0.91–1.22	1.06	0.91–1.23	505	1.04	0.94–1.16	1.07	0.96–1.18
0	135	1.48	1.23–1.79	1.46	1.21–1.77	291	1.33	1.17–1.51	1.31	1.14–1.50

* Model 1: adjusted for age at entry.
Model 2: adjusted for age at entry, smoking, alcohol, caffeine, active activities, other activities, body mass index, high blood pressure, angina, heart attack, stroke, diabetes, rheumatoid arthritis, and cancer.

Table 4: Hazard ratios for mortality by dental health behaviors among subjects with dentures, Leisure World Cohort Study, 1992–2009.

	Men					Women				
	No.	Model 1*		Model 2*		No.	Model 1*		Model 2*	
		HR	95% CI	HR	95% CI		HR	95% CI	HR	95% CI
Clean my dentures										
Everyday	743	1.00		1.00		1443	1.00		1.00	
Sometimes	90	1.11	0.88–1.39	1.11	0.87–1.40	87	1.13	0.91–1.42	1.09	0.87–1.38
Never	165	1.23	1.03–1.46	1.24	1.03–1.48	531	0.85	0.76–0.94	0.87	0.78–0.97
Use mouth wash										
Everyday	297	1.00		1.00		751	1.00		1.00	
Sometimes	308	0.91	0.77–1.08	0.93	0.78–1.10	668	0.95	0.85–1.07	0.95	0.85–1.06
Never	393	0.99	0.84–1.15	1.02	0.87–1.20	642	0.96	0.86–1.08	0.99	0.88–1.11
Number of dental visits within the last 12 months										
2+	612	1.00		1.00		1310	1.00		1.00	
1	137	1.04	0.86–1.26	1.09	0.89–1.33	299	1.09	0.95–1.25	1.10	0.96–1.26
0	249	1.33	1.14–1.55	1.23	1.05–1.45	452	1.26	1.12–1.42	1.20	1.07–1.35

* Model 1: adjusted for age at entry.
Model 2: adjusted for age at entry, smoking, alcohol, caffeine, active activities, other activities, body mass index, high blood pressure, angina, heart attack, stroke, diabetes, rheumatoid arthritis, and cancer.

In the present study, the associations between mortality and the number of teeth and the dental health behaviors were to a large extent independent of established risk factors for mortality. Although lifestyle factors such as smoking, alcohol, and caffeine consumption play important roles in relation to both survival and dental health, the HRs adjusted for these and other confounders were similar to those adjusted for age only. Conscientiousness and other personality traits undoubtedly play a role in health-related behaviors and longevity [32, 33]. However, that only toothbrushing at night and flossing and not toothbrushing in the morning and using mouth wash were related to reduced risk of death suggests that these behaviors are directly related to longevity.

We acknowledge several limitations in our study. All variables used in the analyses are self-reported; we performed no dental examinations. However, previous studies in our population and others support the reliability of self-reported number of teeth [10], health practices and drug usage [29], medical history of major chronic disease [29, 30], and height and weight [29]. Another limitation is that changes over time in all potential risk factors may affect outcome. Additionally, the subjects in our study were mostly white, highly educated, and of middle-class social-economic status (SES), and therefore not representative of the general population. Although this may limit the generalizability of our results, it offers the advantage of reduced potential confounding by race, education, SES, and presumed access to health care. Tooth loss is typically the result of trauma, caries, or periodontal disease and is correlated with lower SES. Confounding by SES is unlikely to explain our results. Nonetheless, although we adjusted for other risk and potential confounding factors, unrecognized and uncontrolled confounders cannot be ruled out in this or any observational study.

This cohort has the advantages of population-based prospective design, large sample size, inclusion of men and women, and data on several potential confounders, including lifestyle practices and chronic disease history previously found to be related to mortality. The long and almost complete followup of the cohort resulted in a large number of outcome events.

Previous studies have identified dental factors that promote health and increase longevity (Table 5) [1–11]. Generally, the smaller the number of teeth the higher the risk of death. The robustness of the finding is illustrated by

Table 5: Studies which evaluated the relationship between dentition status and all-cause mortality in the elderly.

First author year [ref]	Population	Sample size	Followup (years)	Age (years)	Stratification and adjustment[a]	Dental exam % edentulous mean/median no. of teeth	Hazard ratio (95% confidence interval)
Appollonio 1997 [1]	Brescia, Italy	1137	6.5	70–75	Sex, nutrition, smoking, functional status, and health service utilization, education, SES	Yes	Females only 0.67 (0.39–1.15) for adequate dentition 0.83 (0.54–1.27) for inadequate with dentures versus inadequate without dentures
Shimazaki 2001 [2]	Kitakyushu City, Japan	1762	6	59–107	Age, sex, physical mental-health status, cardiovascular disease, musculoskeletal disease, *and* institution, other chronic diseases	Yes 52%	1.8 (1.1–2.8) for 0 teeth without dentures 1.3 (0.8–2.4) for 0 teeth with dentures 1.5 (0.9–2.4) for 1–19 without dentures 1.3 (0.8–2.0) for 1–19 teeth with dentures versus 20+ teeth
Hämäläinen 2003 [3]	Jyväskylä, Finland	226	10	80	Sex, number of chronic diseases, self-rated health	Yes 59% Mean = 11.8 in dentate	2.67 (1.15–6.22) for 1–19 teeth 2.56 (1.12–5.85) for 0 teeth versus 20+ teeth (univariate analysis) 1.03 (1.00–1.05) per missing tooth (adjusted)
Cabrera 2005 [4]	Göteborg, Sweden	1417	24	38, 46, 50, 54, 60	Age, SES Women only	Yes Median = 20	1.27 (1.09–1.47) for >10 missing teeth
Nakanishi 2005 [5]	Settsu Japan	1405	9	65+	Sex, age, overall disability, use of dental health checks, use of general health checks, daily health promotion practices, participation in social activities, life worth living, finding relationships with people difficult	No	1.63 (1.30–2.03) for self-assessed masticatory disability versus no disability
Abnet 2005 [6]	Linxian, China	29584	15	44–59	Sex, age, and smoking	Yes in those reporting missing teeth median = 26	1.07 (1.01–1.14) in females 1.09 (0.98–1.21) in male never-smokers 1.24 (1.16–1.32) in male ever-smokers for loss of > age-specific median of number of teeth

Table 5: Continued.

First author year [ref]	Population	Sample size	Followup (years)	Age (years)	Stratification and adjustment[a]	Dental exam % edentulous mean/median no. of teeth	Hazard ratio (95% confidence interval)
Hämäläinen 2005 [7]	Jyväskylä, Finland	94	5	85	Number of chronic diseases, urgent need of dental treatment, and sex, self-rated health, education, FEV, ESR, CPITN	Yes 39% M, 60% F	0.93 (0.87–0.99) for number of teeth
Yoshida 2005 [8]	Kure City, Japan	1030	8	Mean = 74	Sex, age	Yes	0.78 (0.60–0,99) for adequate occlusion 1.08 (0.85–1.36) for insufficient occlusion versus no occlusion 1.52 (1.24–1.83) for denture non-users versus denture uses among those with no occlusion with their own teeth
Morita 2006 [9]	Tokoname, Japan	118	6.5	Mean = 82	Matched on sex, age, health status, living environment; adjusted for smoking, alcohol	21% M, 30% F	2.71 (1.05–7.05) for < 20 versus ≥ 20 teeth in males; not significant in females
Österberg 2007 [10]	Glostrup, Denmark Jyväskylä, Finland Göteborg, Sweden	1044	7	75	Sex, location, smoking, BMI, self-assessed health, physical activity, circulation disease, ADL	No 45%, 58%, 23% in the 3 locations	0.80 (0.72–0.89) among females 0.92 (0.84–1.01) among males for number of teeth and adjusted only for location 0.87 (0.78–0.97) for women fully adjusted; not significant in men
Österberg 2008 [11]	Göteborg, Sweden	1803	7	70	Sex, ischemic heart disease, number of drugs, plasma glucose, blood hemoglobin, serum triglycerides, BMI, feeling not healthy and others	Yes 16% to 38% in 4 birth cohorts	Males / Females 7-year mortality 0.65 (0.46–0.91) / 0.71 (0.42–1.20) for 1–9 0.51 (0.36–0.74) / 0.65 (0.41–1.02) for 10–19 0.38 (0.24–0.60) / 0.36 (0.19–0.66) for 20–32 versus 0 teeth
			18		Sex, ischemic heart disease, number of drugs, hypertension, plasma glucose, blood hemoglobin, smoking, social activity, and others		18-year mortality 1.02 (0.82–1.27) / 0.81 (0.61–1.07) for 1–9 0.74 (0.58–0.94) / 0.94 (0.74–1.20) for 10–19 0.64 (0.48–0.84) / 0.70 (0.51–0.96) for 20–32 versus 0 teeth
Paganini-Hill (current study)	Leisure World, California, USA	5611	17	52–105	Sex, age, smoking, alcohol, caffeine, exercise, BMI, 7 chronic diseases	No 11% median = 24	See previous tables.

[a] Some included only significant variables in the multivariate models. Those *not* included are preceded by and.

SES: socioeconomic status, FEV: forced respiratory volume, ESR: erythrocyte sedimentation rate, CPITN: community periodontal index of treatment needs, BMI: body mass index, ADL: activities of daily living.

the various populations studied (Italy [1], Scandinavia [3, 4, 7, 10, 11], Japan [2, 5, 8, 9], China [6], and USA (present study)), the different classification schemes for number of teeth, the proportion of subjects edentulous (11 to 60%), the mean/median number of teeth (11 to 26), the length of followup (5 to 24 years), the ages of the subjects (38 to 105), and the adjustments made for a wide variety of health and lifestyle founders that could be potential confounders. Some studies have found significant effects only in one sex group [1, 9, 10]. However, as in our study, loss of teeth is generally seen as a mortality risk factor in both men and women. The number of teeth appears to be a significant predictor of mortality independent of age, sex, education, SES, physical and mental health, body mass index, physical activity, smoking, alcohol consumption, caffeine intake, and country of origin.

Edentulous men and women in our study had a 30% increased 17-year mortality risk compared with those with 20+ teeth. In Sweden, edentulous subjects had 2.8 (95% CI 2.0–4.0) times greater 7-year mortality risk [11], similar to the Finnish study for 10-year mortality [3]. The 18-mortality risk in a Swedish study was lower at about 1.5 (95% CI = 1.1–2.8) times, similar to our finding of 1.3. This suggests that change over time in the risk factor may affect outcome.

Although the wearing of dentures may have a beneficial effect on mastication in those who have lost teeth, our study and others [1, 2] suggest that prostheses do not sufficiently compensate in terms of survival. Our study showed that edentulous subjects (virtually all who had prostheses) had an increase in mortality of 1.3 compared with those with 20 or more teeth. The corresponding ratio in Shimazaki et al. [2] was 1.3 (adjusted for physical-mental health status) and 1.2 compared with adequate natural dentition in Appollonio et al. [1]. Although in one Japanese study [8], the risk of mortality among denture non-users was 1.52-times (95% CI = 1.24–1.83) higher than the risk for denture wearers in subjects who had no occlusal contact (with or without remaining teeth), the group as a whole had 1.3-times 8-year mortality rate of subjects who retained adequate occlusal contacts.

Most of the missing teeth of an elderly subject would have been infected before tooth loss or extractions, and, hence, the number of missing teeth would be a surrogate for previous dental infection. Poor dental status (periodontitis, dental bacteraemia, and oral mucosal infections) links with heart disease, stroke, atherosclerosis, pneumonia, other respiratory disease, and mortality (for a review see [12]). Oral infections may trigger systemic inflammatory reactions and death. Poorer masticatory ability and nutrient intake may also affect general health and survival.

Our study also found that several dental health behaviors were related to mortality. Not visiting a dentist within the last 12 months was significantly related to increased (30–50% higher) mortality in both men and women and in both those with teeth and those with dentures. In a study in Settsu, Japan [5], not using dental health checks was also associated with mortality (HR = 1.4, 95% CI 0.98–1.92).

Oral hygiene measures used in conjunction with regular professional care can prevent caries and most periodontal disease [34]. Toothbrushing and flossing are the most commonly used measures. Toothbrushing with a fluoride toothpaste helps prevent caries and dental floss is the most effective means of removing interdental plaque and reducing interdental gingival inflammations. Our study highlights the advantages on longevity of brushing at night and of flossing everyday.

Although not establishing a cause and effect relationship between mortality and dental status and behaviors, these strong and statistically persistent associations of dental health and mortality provide another basis for encouraging oral health and hygiene and use of dentures. One marker of successful ageing is the maintenance of a natural, healthy, and functional dentition. Retaining healthy teeth not only helps in mastication, nutrition, aesthetics, and self-esteem, but also appears to promote survival.

5. Conclusion

Results in this large elderly cohort with long followup showing a decreased risk of mortality with several dental health behaviors, number of teeth, and dentures suggest that maintenance of good oral hygiene is an important health promoter in aging populations.

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References

[1] I. Appollonio, C. Carabellese, A. Frattola, and M. Trabucchi, "Influence of dental status on dietary intake and survival in community-dwelling elderly subjects," *Age and Ageing*, vol. 26, no. 6, pp. 445–455, 1997.

[2] Y. Shimazaki, I. Soh, T. Saito et al., "Influence of dentition status on physical disability, mental impairment, and mortality in institutionalized elderly people," *Journal of Dental Research*, vol. 80, no. 1, pp. 340–345, 2001.

[3] P. Hämäläinen, J. H. Meurman, M. Keskinen, and E. Heikkinen, "Relationship between dental health and 10-year mortality in a cohort of community-dwelling elderly people," *European Journal of Oral Sciences*, vol. 111, no. 4, pp. 291–296, 2003.

[4] C. Cabrera, M. Hakeberg, M. Ahlqwist et al., "Can the relation between tooth loss and chronic disease be explained by socioeconomic status? A 24-year follow-up from the Population Study of Women in Gothenburg, Sweden," *European Journal of Epidemiology*, vol. 20, no. 3, pp. 229–236, 2005.

[5] N. Nakanishi, H. Fukuda, T. Takatorige, and K. Tatara, "Relationship between self-assessed masticatory disability and 9-year mortality in a cohort of community-residing elderly people," *Journal of the American Geriatrics Society*, vol. 53, no. 1, pp. 54–58, 2005.

[6] C. C. Abnet, Y.-L. Qiao, S. M. Dawsey, Z.-W. Dong, P. R. Taylor, and S. D. Mark, "Tooth loss is associated with increased risk of total death and death from upper gastrointestinal

cancer, heart disease, and stroke in a Chinese population-based cohort," *International Journal of Epidemiology*, vol. 34, no. 2, pp. 467–474, 2005.

[7] P. Hämäläinen, J. H. Meurman, M. Kauppinen, and M. Keskinen, "Oral infections as predictors of mortality," *Gerodontology*, vol. 22, no. 3, pp. 151–157, 2005.

[8] M. Yoshida, H. Morikawa, M. Yoshikawa, K. Tsuga, and Y. Akagawa, "Eight-year mortality associated with dental occlusion and denture use in community-dwelling elderly persons," *Gerodontology*, vol. 22, no. 4, pp. 234–237, 2005.

[9] I. Morita, H. Nakagaki, K. Kato et al., "Relationship between survival rates and numbers of natural teeth in an elderly Japanese population," *Gerodontology*, vol. 23, no. 4, pp. 214–218, 2006.

[10] T. Österberg, G. E. Carlsson, V. Sundh, and B. Steen, "Number of teeth—a predictor of mortality in the elderly? A population study in three Nordic localities," *Acta Odontologica Scandinavica*, vol. 65, no. 6, pp. 335–340, 2007.

[11] T. Österberg, G. E. Carlsson, V. Sundh, and D. Mellström, "Number of teeth—a predictor of mortality in 70-year-old subjects," *Community Dentistry and Oral Epidemiology*, vol. 36, no. 3, pp. 258–268, 2008.

[12] J. H. Meurman and P. Hämäläinen, "Oral health and morbidity—implications of oral infections on the elderly," *Gerodontology*, vol. 23, no. 1, pp. 3–16, 2006.

[13] S. Ajwani, K. J. Mattila, R. S. Tilvis, and A. Ainamo, "Periodontal disease and mortality in an aged population," *Special Care in Dentistry*, vol. 23, no. 4, pp. 125–130, 2003.

[14] D. Jackson and J. Murray, "The loss of teeth in dentate populations," *The Dental Practitioner and Dental Record*, vol. 22, no. 5, pp. 186–189, 1972.

[15] J. D. Manson, "The elderly dental cripple," *Proceedings of the Royal Society of Medicine*, vol. 66, no. 6, pp. 597–598, 1973.

[16] A. Paganini-Hill and G. Hsu, "Smoking and mortality among residents of a California retirement community," *American Journal of Public Health*, vol. 84, no. 6, pp. 992–995, 1994.

[17] A. Paganini-Hill, C. H. Kawas, and M. M. Corrada, "Type of alcohol consumed, changes in intake over time and mortality: The Leisure World Cohort Study," *Age and Ageing*, vol. 36, no. 2, pp. 203–209, 2007.

[18] A. Paganini-Hill, C. H. Kawas, and M. M. Corrada, "Non-alcoholic beverage and caffeine consumption and mortality: The Leisure World Cohort Study," *Preventive Medicine*, vol. 44, no. 4, pp. 305–310, 2007.

[19] A. Paganini-Hill, C. H. Kawas, and M. M. Corrada, "Activities and mortality in the elderly: The Leisure World Cohort Study," *Journals of Gerontology Series A*, vol. 66A, no. 5, pp. 559–567, 2011.

[20] M. M. Corrada, C. H. Kawas, F. Mozaffar, and A. Paganini-Hill, "Association of body mass index and weight change with all-cause mortality in the elderly," *American Journal of Epidemiology*, vol. 163, no. 10, pp. 938–949, 2006.

[21] A. Sheiham, J. G. Steele, W. Marcenes, S. Finch, and A. W. G. Walls, "The relationship between oral health status and Body Mass Index among older people: a national survey of older people in Great Britain," *British Dental Journal*, vol. 192, no. 12, pp. 703–706, 2002.

[22] N. Shah, H. Parkash, and K. R. Sunderam, "Edentulousness, denture wear and denture needs of Indian elderly—a community-based study," *Journal of Oral Rehabilitation*, vol. 31, no. 5, pp. 467–476, 2004.

[23] T. Yanagisawa, M. Ueno, K. Shinada, S. Ohara, F. A. C. Wright, and Y. Kawaguchi, "Relationship of smoking and smoking cessation with oral health status in Japanese men," *Journal of Periodontal Research*, vol. 45, no. 2, pp. 277–283, 2010.

[24] T. Dietrich, N. N. Maserejian, K. J. Joshipura, E. A. Krall, and R. I. Garcia, "Tobacco use and incidence of tooth loss among US male health professionals," *Journal of Dental Research*, vol. 86, no. 4, pp. 373–377, 2007.

[25] L. B. Copeland, E. A. Krall, L. J. Brown, R. I. Garcia, and C. F. Streckfus, "Predictors of tooth loss in two US adult populations," *Journal of Public Health Dentistry*, vol. 64, no. 1, pp. 31–37, 2004.

[26] P. Norlén, I. Johansson, and D. Birkhed, "Impact of medical and life-style factors on number of teeth in 68-year-old men in southern Sweden," *Acta Odontologica Scandinavica*, vol. 54, no. 1, pp. 66–74, 1996.

[27] National Heart, Lung, and Blood Institute, "Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: the evidence report," 1998, National Heart, Lung, and Blood Institute,Bethesda, Md, USA, http://www.nhlbi.nih.gov/guidelines/obesity/ob_gdlns.pdf.

[28] D. R. Cox, "Regression models and life tables (with discussion)," *Journals of the Royal Statistical Society B*, vol. 34, pp. 187–220, 1972.

[29] A. Paganini-Hill and R. K. Ross, "Reliability of recall of drug usage and other health-related information," *American Journal of Epidemiology*, vol. 116, no. 1, pp. 114–122, 1982.

[30] A. Paganini-Hill and A. Chao, "Accuracy of recall of hip fracture, heart attack, and cancer: a comparison of postal survey data and medical records," *American Journal of Epidemiology*, vol. 138, no. 2, pp. 101–106, 1993.

[31] A. Paganini-Hill, R. K. Ross, and B. E. Henderson, "Prevalence of chronic disease and health practices in a retirement community," *Journal of Chronic Diseases*, vol. 39, no. 9, pp. 699–707, 1986.

[32] T. Bogg and B. W. Roberts, "Conscientiousness and health-related behaviors: a meta-analysis of the leading behavioural contributtors to mortality," *Pyscologial Bulletin*, vol. 130, pp. 887–919, 2004.

[33] M. L. Kern and H. S. Friedman, "Do conscientious individuals live longer? A quantitative review," *Health Psychology*, vol. 27, no. 5, pp. 505–512, 2008.

[34] A. Choo, D. M. Delac, and L. B. Messer, "Oral hygiene measures and promotion: review and considerations," *Australian Dental Journal*, vol. 46, no. 3, pp. 166–173, 2001.

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Research Article

The Relationship between Physical Health and Psychological Well-Being among Oldest-Old Adults

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The purpose of this study was to evaluate the relationship between physical health and psychological well-being among oldest-old adults. Structural equation modeling was performed to examine health influences on psychological well-being among 306 octogenarians and centenarians from the Georgia Centenarian Study. Latent variables were created to reflect subjective health, as measured by self-ratings of health and objective health, as measured by physical health impairment (i.e., health problems, past and present diseases, hospitalization) and biomarkers (i.e., hemoglobin and albumin). Psychological well-being was measured by positive and negative affect. There were significant direct effects of subjective health on affect and significant indirect effects of objective health through subjective health on positive affect and negative affect. Subjective health took the role of a mediator between objective health and psychological well-being. These results highlight the status and perceptions of health as a critical indicator for well-being in extreme old age.

1. Introduction

With the unprecedented increase in the number of oldest-old adults, several studies have paid attention to centenarians and their lives exploring factors related to their longevity, such as health, genetic influences, general lifestyle, physical activity, nutrition, and social relationships [1]. Even though many researchers indicate that centenarians have several chronic diseases [2], and health is a significant indicator for psychological well-being among oldest-old adults, only a few studies have focused on health and its impact on psychological well-being in extreme old age. Therefore, there is a need to investigate the association between health and psychological well-being among oldest-old adults.

Usually, *physical health* is the most commonly used index to assess the well-being of individuals. As people grow older, they might perceive that their physical health (e.g., the prevalence rates of chronic conditions) is not as good as it has been in the past. The importance of health among oldest-old adults, especially the prevalence rates of chronic conditions, was shown in a study of Danish centenarians [2]. They found that there were few healthy centenarians and that most Danish centenarians had several common diseases and chronic conditions such as cardiovascular disease (72%), osteoarthritis (54%), hypertension (52%), dementia (51%), and ischemic heart disease (28%). Andersen-Ranberg et al. [2] concluded that it is a challenge to be free from potentially common diseases until the age of 100. This assertion was supported by another centenarian study. After assessing the health history of 424 centenarians, Evert et al. found that even though 19% of centenarians were classified as "escapers" who had reached their 100th birthday without the diagnosis of common age-related diseases, 81% of centenarians were not free from common age-related diseases [3]. Therefore, most oldest-old adults reported chronic health conditions.

Subjective health is "related not only to length of life but also to states of health in the years remaining" [4, p.S315] and

serves as one of the most important determinants for psychological well-being in later life. Hoeymans and colleagues noted that subjective health is a valuable and personalized health indicator, specifying one's perception and evaluation of one's own health, based on an interpretation of the objective physical and mental health status, and expectations and comparisons [5–10]. Because individuals' attitudes, motivations, and beliefs influence perceptions of illness and disability, individual differences in subjective health might play an important role for psychological well-being in later life [11]. For instance, as psychological characteristics involve an individual's ability and willingness to adapt to physical change [12], the subjective experience is influenced by various kinds of diseases or illness histories [11, 13].

Psychological well-being has been examined as an indicator of successful adaptation during old and very old age [14]. Bradburn considered the subjective assessment of well-being as the balance between positive and negative affect [15]. These two dimensions of well-being may be the origin of psychological well-being [15, 16]. The two types of affect may have different adaptive functions. Negative affect refers to a consequence of maladaptive behavior, whereas positive affect may be considered reinforcement for adaptive or appropriate behavior [15, 16]. Larson summarized previous studies of psychological well-being performed over a 30-year period (1940s–1970s) and noted that the construct is strongly associated with physical health status, functional status, and socio-demographic factors, including occupation, income, educational level, and the degree of social interaction [17, 18]. Hamashima examined previous studies of psychological well-being (specifically, quality of life) in Japan and concluded that it was influenced by physical health and other factors such as age, marital status, occupation, and economic status [18–20].

Based on previous studies, the effect of physical health needs to be considered when accounting for well-being in later life. The importance of physical health for psychological well-being has been reported in a number of studies. Revicki and Mitchell, for example, found that physical health problems were the most important source of life strain among older adults [21]. Physical health can have a major impact on subjective well-being. For instance, Bishop et al. found that poor health was a significant factor associated with lower morale [22]. In addition, there are several studies that have focused on the influence of specific diseases on psychological well-being. For example, positive affect was related to fewer stroke symptoms [23], and low cardiovascular risk was associated not only with better survival but also with better psychological well-being in older adults [24]. These studies all demonstrated that perceived health is associated with objective health [25]. Several studies uncovered the strong relationships between perceived health and long-standing chronic illness, especially among older adults [25–29] and with other health indicators such as number of medications, sick days, or hospitalizations [25, 30–32].

As shown in previous studies, there is a close association between objective and subjective assessments of personal health, and this association influences psychological well-being [11]. In other words, individuals' psychological well-being is affected by medical history, current physical symptoms and body sensations, health beliefs and behaviors, and mental and emotional well-being [11, 33]. Additionally, the major factor of subjective health is objective physical health, that is, chronic conditions and disabilities. Many studies have noted the relations between chronic conditions, disabilities, and subjective health [5, 25, 34–38]. Interestingly, Kempen and colleagues observed that health perceptions were most affected by heart conditions, followed by asthma/chronic bronchitis, joint complaints, back problems, and diabetes [35].

Even though a number of studies have suggested a strong association between physical health (objective and subjective) and psychological well-being, many studies only include individuals between the ages of 60 to 80 years, and there is little information about this association for very old age [39]. Therefore, additional research needs to focus on both physical health markers and subjective health predicting psychological well-being in very late life because this time is often characterized by a functional decline or breakdown of the physical and psychological system [39]. The purpose of this study was to assess the association of different aspects (objective and subjective) of physical health and their direct and indirect effect on psychological well-being (i.e., positive and negative affect) in very old adults.

2. Method

2.1. Participants. The sampling frame of the Georgia Centenarian Study (GCS, Phase III) [40], which provides data for this study, had two components. The first one was to identify the proportion of all residents of skilled nursing facilities (SNFs) and personal care homes (PCHs) in a 44-county area in northern Georgia. Based on census proportions, the project identified residents of SNFs and PCHs. The second recruiting strategy was to use the date-of-birth information in voter registration files to identify community-dwelling residents. Based on these two components and five different characteristics (geographic, age, gender, race, and type of residence) a sample of centenarians and octogenarians was drawn for this study [40].

Obtaining information from oldest-old adults is not always easy or feasible. Especially in old age, individuals' abilities to respond are affected by their physical health, cognitive status, or functional abilities [41]. The different levels of those factors among older adults often lead to the use of proxy ratings of health, functional status, or mental health instead of self-ratings [41–44]. LaRue and colleagues suggested that there was a significant relationship between self and physicians' reports; so self-reports could offer a valid measurement for health assessment in old age [43]. Bassett and colleagues reported that there was a significant correspondence between respondents' and proxy reports on cognitive and mental health [42]. These authors also suggested that self-responses on cognitive and psychological status measures can be substituted with proxy responses when the original informant is unavailable [42]. In addition, several studies also found that proxy information is reliable or less biased when respondents are cognitively

Table 1: Summary of demographic characteristics.

Demographic characteristics	*n*	%
Age		
Octogenarian ($M = 84.58$)	72	23.5
Centenarian ($M = 100.23$)	234	76.5
Gender		
Female	243	79.4
Male	63	20.6
Type of residence		
Private home/apartment	165	54.1
Personal care	48	15.7
Nursing home	92	30.2
Ethnicity		
White/Caucasian	240	78.4
Black/African American	66	21.6
Education		
Less than high school complete	99	34.1
High school diploma	61	21.0
GED/some college	67	23.1
College/graduate degree	63	21.7
Subjective health		
Poor	10	3.3
Fair	69	22.7
Good	148	48.7
Excellent	77	25.3

impaired or depressed [44–46]. Rodgers and Herzog, for example, indicated that there has been a general consensus among researchers that proxy respondents should be used in research focusing on oldest-old adults to avoid biasing the data compared to healthy elderly [47]. Therefore, based on these arguments, using proxies data might be helpful not only to substitute for insufficient information of self-reports but also to have different viewpoints of psychological well-being among oldest-old adults. Therefore, the information in this paper is based on proxy information.

Proxy informants were selected in the following fashion: first close family such as spouses or children was considered as proxies. If more than one child was alive, the oldest-old adults nominated a proxy, or in the case of cognitive impairment, a contacted child made the decision about who could provide the most accurate information. Other relatives served as proxies if no children were alive or available or if so nominated by the participant. If no other relatives were alive or available, friends, neighbors, nurses, clergy, or other knowledgeable person also served as proxies. Most of the proxy informants (59.4%) were adult children. Additional proxies included nieces and nephews (10.0%), granddaughters (7.7%), and miscellaneous informants, such as spouses, siblings, or friends (22.9%).

This study included 306 community-dwelling and institutionalized oldest-old adults aged from 80 to 100 (mean age was 96.55 years). In this study, 79.4% of the participants were women and 75% of participants rated their health as good or excellent. A summary of demographic characteristics is presented in Table 1.

2.2. Measures

2.2.1. Physical Health Impairments. Physical health impairments were measured using items with several indicators: past and current diseases, health conditions, and hospitalization. Past and current diseases were assessed with a comprehensive list of diseases such as congestive heart failure, myocardial infarction, and high blood pressure. Health conditions were assessed with a variety of health problems such as chest discomfort, numbness, arthritis, and dizziness. Lastly, hospitalization was accessed with any recent or lifetime hospitalization. Higher scores reflect more health problems, more diseases, and more hospitalizations.

2.2.2. Biomarkers. Biomarkers included hemoglobin and albumin, which were assessed with a blood draw. Higher scores indicate higher levels of hemoglobin and albumin.

2.2.3. Subjectively Perceived Health. The subjective perception of health was comprised of two questions [48] with an original internal consistency coefficient $\alpha = 0.74$. Proxies were asked: "How would you rate his/her overall health at the present time—excellent, good, fair, or poor?" and was scaled so that 0 = poor to 3 = excellent. The other question was "How much do his/her health troubles stand in the way of his/her doing the things he/she wants to do?" and was scaled so that 0 = a great deal to 2 = not at all. Internal consistency for the proxy ratings of our participants was $\alpha = 0.56$. Physical health was scored so that higher scores indicated higher levels of physical health.

2.2.4. Psychological Well-Being. Psychological well-being was assessed with the Bradburn Affect Balance Scale [15]. The scale consists of two dimensions: positive affect and negative affect. Five positive affect items ($\alpha = 0.80$) and five negative affect items ($\alpha = 0.80$) from proxy reports were used in this study. Proxies were asked to rate centenarians with the following statements for positive affect. During the past two weeks, (1) Did he/she ever feel pleased about having accomplished something? (2) Did he/she ever feel proud because someone complimented him/her on something he/she had done? (3) Did he/she ever feel particularly excited or interested in something? (4) Did he/she ever feel that things were going his/her way? (5) Did he/she ever feel on top of the world? For negative affect, the following statements were asked. (1) Did he/she ever feel depressed and very unhappy? (2) Did he/she ever feel vaguely uneasy? (3) Did he/she ever feel bored? (4) Did he/she ever feel so restless that he/she could not sit long in a chair? (5) Did he/she ever feel very lonely or remote from other people? Ratings were used with a four-point Likert scale: 1 = not at all, 2 = once, 3 = several times, and 4 = often. Higher scores for positive affect indicated better well-being, while higher scores for negative affect indicated lower well-being.

Table 2: Factor loadings in confirmatory factor of health and psychological well-being.

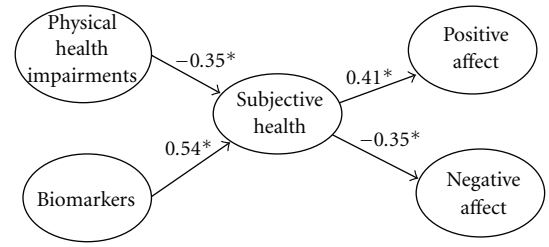
	Physical health impairments	Biomarkers	Subjective health	Positive affect	Negative affect
Past disease	0.89				
Current disease	0.55				
Health problem	0.39	—	—	—	—
Hospitalization	0.35				
Hemoglobin		0.78			
Albumin	—	0.43	—	—	—
Self-rated overall health			0.72		
Self-rated health problem	—	—	0.63	—	—
Pleased				0.73	
Proud	—	—	—	0.70	—
Excited/interested				0.76	
Depressed					0.77
Vaguely uneasy	—	—	—	—	0.76
Bored					0.70

Note. All factor loadings are standardized parameter estimates.

2.3. Plan of Analysis

2.3.1. Confirmatory Factor Analysis (CFA). Confirmatory factor analyses using LISREL 8.71 [49] established the fit of subjective health, objective health, and psychological well-being to the corresponding constructs in this study. Maximum-likelihood estimation was used. The results are summarized in Table 2. In terms of the psychological well-being measure, positive affect and negative affect were initially tested in relation to a model composed of five indicators for each construct. However, the lowest loadings of each construct were dropped after conducting an item analysis, and the model specified three indicators for each affect construct. All the loadings of each factor were significant (Table 2).

2.3.2. Structural Equation Modeling (SEM). Structural equation modeling was used to test the relationship between subjective health, objective health, and psychological well-being with LISREL 8.71.



x^2 (df = 72) = 55.48, P = .93, CFI = 1.00,
TLI (NNFI) = 1.03, RMSEA = 0.00

Figure 1: The latent variable relationship between physical health impairment and psychological well-being. *Note.* Path coefficients are standardized parameter estimates and direct loadings are displayed in solid lines. $^*P < .05$.

3. Results

Three different models were tested to examine the relationship between physical health and psychological well-being (Table 3). Model 1 is the measurement model for objective health, subjective health, and psychological well-being and no relationship among physical health impairments, biomarkers, subjective health, positive affect, and negative affect was hypothesized, χ^2 (df = 78) = 111.19, $P < .05$, CFI = 0.94, TLI (NNFI) = 0.93, and RMSEA = 0.06. Model 2 investigated the relationship between objective health and psychological well-being through subjective health. Model 2 yielded a better fit in comparison to Model 1, χ^2 (df = 72) = 55.48, $P = .93$, χ^2 diff (6) = 55.71, $P < .001$, CFI = 1.00, TLI (NNFI) = 1.03, and RMSEA = 0.00 (Table 3). Model 3 tested the full model of direct effects of objective health (physical health impairments, biomarkers) and subjective health on psychological well-being. All possible relationships between health and psychological well-being were hypothesized to be correlated. In comparison to Model 1, Model 3 yielded a better model fit, χ^2 (df = 68) = 53.75, $P = .90$, χ^2 diff (10) = 57.74, $P < .001$, CFI = 1.00, TLI (NNFI) = 1.03, and RMSEA = 0.00 (Table 3). However, Model 3 was not significantly better in comparison to Model 2, χ^2 diff (4) = 1.73, and $P = .79$. Therefore, Model 2, the more parsimonious model, was selected as the best fitting model (Table 3).

Based on Model 2, the latent variable relationships between health and psychological well-being were inspected (Figure 1). This model examined the mediating effect of subjective health between objective health (i.e., physical health impairments and biomarkers) and psychological well-being (i.e., positive affect and negative affect). Paths between physical health impairments, biomarkers, subjective health, positive affect, and negative affect were investigated (Figure 1).

Table 3: Fit indices for nested sequence of cross-sectional models.

Model	χ^2	df	χ^2 diff	CFI	TLI	RMSEA
(1) Measurement model	111.19	78		0.94	0.93	0.06
(2) Health and psychological well-being relation model	55.48			1.00	1.03	0.00
Difference between model 2 and model 1		72	55.71***			
(3) Fully recursive model	53.75	68		1.00	1.03	0.00
Difference between model 3 and model 2			1.73			
(4) Null model	562.57					

***$P < .001$.

There were several significant direct effects in Model 2. In terms of physical health, physical health impairments were found to have a significant negative direct effect on subjective health ($\beta = -0.35$, $P < .05$). Biomarkers had a significant positive effect on subjective health ($\beta = 0.54$, $P < .05$). Second, subjective health had significant direct effects on positive and negative affect. In other words, subjective health was found to be significantly associated with positive affect, $\beta = 0.41$, $P < .05$, and to have a significant negative association with negative affect, $\beta = -0.35$, $P < .05$. Third, there was an indirect effect of objective health on psychological well-being. Specifically, physical health impairments had significant indirect effects on positive affect, $\beta = -0.14$, $P < .05$, and negative affect, $\beta = 0.12$, $P < .05$. Biomarkers also had significant indirect effects on positive, $\beta = 0.22$, $P < .05$, and negative affect, $\beta = -0.19$, $P < .05$. In other words, there was a mediating effect of subjective health between physical health and positive affect and negative affect.

4. Discussion

The purpose of this study was to highlight the way in which physical health influences psychological well-being among oldest-old adults. Two potentially important findings emerged through structural equation modeling. First, the analysis suggests that subjective health was strongly associated with psychological well-being (e.g., affect) among oldest-old adults. Second, the results further revealed that physical health impairments and biomarkers had independent direct effects on subjective health and they had an indirect association with psychological well-being among oldest-old adults.

There are a couple of reasons why these results emerging from this study are noteworthy. To begin with, the conclusions are based on data that were gathered from an oldest-old population. In general, physical health is recognized as one of the most important indicators of quality of life in later life. Even though the importance of studying very old populations has been noted repeatedly, few studies (e.g., [49]) have explored the relationship of health with psychological well-being for very old persons. In addition, specification of physical health by different assessments such as physical health impairment, biomarkers, and subjective health helps underscore the importance of including different aspects (objective and subjective) of physical health and the different role they play for psychological well-being in very late life. Finally, the findings from this study were based on proxy information. This is important because many researchers indicate that it is less reliable to use proxy information due to proxy bias. However, consistent with earlier studies [44–47], the results of this study contribute to the argument that information from proxies could provide sufficient information and unique perspectives of psychological well-being among oldest-old adults.

The findings of this study were supported by previous studies. First, objective aspects of physical health (e.g., physical impairment, biomarkers) had an independent direct effect on subjective health. Earlier studies showed that chronic diseases were significantly associated with subjective health or perceived health status [44–47]. For example, Jylhä and colleagues found that different factors were associated with self-rated health for different age groups [27]. The results showed that the number of chronic diseases such as high blood pressure was the strongest predictor of self-rated health among older adults aged 70 to 79 [27]. This underlines the importance of physical health impairment for perception of health among old and oldest-old population. Furthermore, the results of this study are consistent with the findings of recent studies that biomarker assessments are used in combination with behavioral and social aspects related to individuals' health and well-being [50–52]. Jylhä et al. showed a significant association between biomarkers and self-rated health. Interestingly, lower levels of hemoglobin were significantly associated with fair or poor self-rated health [53]. Another result of this study confirmed previous findings that reported a significant association between psychological well-being and subjective health among oldest-old adults [6–11]. Perhaps the most noteworthy finding of this study was the significant indirect effect of physical health on the psychological well-being among oldest-old adults. This is consistent with other studies reporting that those higher in positive affect reported fewer severe disease symptoms, and those higher in negative affect reported more severe ones (e.g., [54]). This finding is supported by the work of Temane and Wissing [55] who showed that the subjective perception of health mediated the relationship between individual context such as physical health and psychological well-being [55]. Therefore, the results of our study lead us to conclude that perceived health takes the role of an important mediator between physical health and psychological well-being.

Even though this study made significant contributions to the literature by linking two perspectives of health and

psychological well-being, there are also several limitations of the present study. The sample of this study was from only one geographic area of the United States. Other oldest-old adults in different regions might present different patterns in the relationship between physical health and psychological well-being. Second, although physical health was assessed with the number of present and past diseases, those indicators were examined with a cross-sectional design. Therefore, causal inferences on the relationship between health and well-being cannot be made. Finally, even though most of the indicators were examined by proxy ratings and quite a few papers have demonstrated that proxy informants are reliable and substitutable for self-rated reports to use, we need to consider that disagreement on psychological aspects might result in differences of proxy—and centenarians' self-ratings.

In spite of these limitations, the results of this study support the notion that health, subjective and objective, is an essential factor for psychological well-being in later life. Fewer problems with physical health (i.e., number of diseases, health problems, and hospitalization) and more favorable readings of hemoglobin and albumin influence perceptions of health, and this has a positive effect on positive affect and a negative effect on negative affect among very old persons. Even though physical health problems are common among octogenarians and centenarians, the results confirm that both physical and psychological well-beings are critical factors at the very end of the human life span.

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References

[1] A. Ozaki, M. Uchiyama, H. Tagaya, T. Ohida, and R. Ogihara, "The Japanese centenarian study: autonomy was associated with health practices as well as physical status," *Journal of the American Geriatrics Society*, vol. 55, no. 1, pp. 95–101, 2007.

[2] K. Andersen-Ranberg, M. Schroll, and B. Jeune, "Healthy centenarians do not exist, but autonomous centenarians do: a population-based study of morbidity among danish centenarians," *Journal of the American Geriatrics Society*, vol. 49, no. 7, pp. 900–908, 2001.

[3] J. Evert, E. Lawler, H. Bogan, and T. Perls, "Morbidity profiles of centenarians: survivors, delayers, and escapers," *Journals of Gerontology A*, vol. 58, no. 3, pp. 232–237, 2003.

[4] E. L. Idler and S. V. Kasl, "Self-ratings of health: do they also predict change in functional ability?" *Journals of Gerontology B*, vol. 50, no. 6, pp. S344–S353, 1995.

[5] N. Hoeymans, E. J. M. Feskens, D. Kromhoup, and G. A. M. Van Den Bos, "Ageing and the relationship between functional status and self-rated health in elderly men," *Social Science and Medicine*, vol. 45, no. 10, pp. 1527–1536, 1997.

[6] W. C. Cockerham, K. Sharp, and J. A. Wilcox, "Aging and perceived health status," *Journals of Gerontology*, vol. 38, no. 3, pp. 349–355, 1983.

[7] S. E. Fienberg, E. F. Loftus, and J. M. Tanur, "Cognitive aspects of health survey methodology: an overview," *Milbank Memorial Fund Quarterly, Health and Society*, vol. 63, no. 3, pp. 547–564, 1985.

[8] E. L. Idler, "Age differences in self-assessments of health: age changes, cohort differences, or survivorship?" *Journals of Gerontology*, vol. 48, no. 6, pp. S289–S300, 1993.

[9] N. M. Krause and G. M. Jay, "What do global self-rated health items measure?" *Medical Care*, vol. 32, no. 9, pp. 930–942, 1994.

[10] J. Suls, C. A. Marco, and S. Yobin, "The role of temporal comparison, social comparison, and direct appraisal in the elderly's self-evaluation of health," *Journal of Applied Social Psychology*, vol. 21, pp. 1125–1144, 1991.

[11] Z. Wu and C. M. Schimmele, "Psychological disposition and self-reported health among the 'oldest-old' in China," *Ageing and Society*, vol. 26, no. 1, pp. 135–151, 2006.

[12] W. H. Kuo and Y. M. Tsai, "Social networking, hardiness and immigrant's mental health," *Journal of Health and Social Behavior*, vol. 27, no. 2, pp. 133–149, 1986.

[13] J. Smith, D. Gerstorf, and Q. Li, "Psychological resources for healthy longevity," 2004, http://www.duke.edu/web/cpses/Smith_MPIDR.ppt.

[14] J. Smith, W. Fleeson, B. Geiselmann, R. A. Settersten, and U. Kunzmann, "Sources of well-being in very old age," in *The Berlin Aging Study: Aging from 70 to 100*, P. B. Baltes and K. U. Mayer, Eds., pp. 450–471, Cambridge University Press, New York, NY, USA, 2002.

[15] N. Bradburn, *The Structure of Psychological Well-Being*, Aldine, Chicago, Ill, USA, 1969.

[16] D. Jopp and C. Rott, "Adaptation in very old age: exploring the role of resources, beliefs, and attitudes for centenarians' happiness," *Psychology and Aging*, vol. 21, no. 2, pp. 266–280, 2006.

[17] R. Larson, "Thirty years of research on the subjective well-being of older Americans," *Journals of Gerontology*, vol. 33, no. 1, pp. 109–125, 1978.

[18] H. Iwasa, K. Kawaai, Y. Gondo, H. Inagaki, and T. Suzuki, "Subjective well-being as a predictor of all-cause mortality among middle-aged and elderly people living in an urban Japanese community: a seven-year prospective cohort study," *Geriatrics & Gerontology International*, vol. 6, pp. 216–222, 2006.

[19] C. Hamashima, "The quality of life in aged people," *Japanese Journal of Hygiene*, vol. 49, pp. 533–542, 1994 (Japanese).

[20] H. Iwasa, K. Kawaai, Y. Gondo, H. Inagaki, and T. Suzuki, "Subjective well-being as a predictor of all-cause mortality among middle-aged and elderly people living in an urban Japanese community: a seven-year prospective cohort study," *Geriatrics & Gerontology International*, vol. 6, pp. 216–222, 2006.

[21] D. A. Revicki and J. P. Mitchell, "Strain, social support, and mental health in rural elderly individuals," *Journals of Gerontology*, vol. 45, no. 6, pp. S267–S274, 1990.

[22] D. S. Bishop, N. B. Epstein, and G. I. Keitner, "Stroke: morale, family functioning, health status, and functional capacity," *Archives of Physical Medicine and Rehabilitation*, vol. 67, no. 2, pp. 84–87, 1986.

[23] G. V. Ostir, K. S. Markides, M. K. Peek, and J. S. Goodwin, "The association between emotional well-being and the incidence of stroke in older adults," *Psychosomatic Medicine*, vol. 63, no. 2, pp. 210–215, 2001.

[24] T. E. Strandberg, A. Y. Strandberg, K. H. Pitkälä, V. V. Salomaa, R. S. Tilvis, and T. A. Miettinen, "Cardiovascular risk in midlife and psychological well-being among older men," *Archives of Internal Medicine*, vol. 166, no. 20, pp. 2266–2271, 2006.

[25] J. Rodin and G. McAvay, "Determinants of change in perceived health in a longitudinal study of older adults," *Journals of Gerontology*, vol. 47, no. 6, pp. P373–P384, 1993.

[26] M. S. Goldstein, J. M. Siegel, and R. Boyer, "Predicting changes in perceived health status," *American Journal of Public Health*, vol. 74, no. 6, pp. 611–614, 1984.

[27] M. Jylhä, E. Leskinen, E. Alanen, A. L. Leskinen, and E. Heikkinen, "Self-rated health and associated factors among men of different ages," *Journals of Gerontology*, vol. 41, no. 6, pp. 710–717, 1986.

[28] J. Liang, "Self-reported physical health among aged adults," *Journals of Gerontology*, vol. 41, no. 2, pp. 248–260, 1986.

[29] A. B. Zonderman, "Effects of age, hypertension history, and neuroticism on health perceptions," *Experimental Gerontology*, vol. 21, no. 4-5, pp. 449–458, 1986.

[30] G. G. Fillenbaum, "Social context and self-assessments of health among the elderly," *Journal of Health and Social Behavior*, vol. 20, no. 1, pp. 45–51, 1979.

[31] B. S. Linn and M. W. Linn, "Objective and self-assessed health in the old and very old," *Social Science and Medicine*, vol. 14, no. 4, pp. 311–315, 1980.

[32] T. T. H. Wan, "Predicting self assessed health status: a multivariate approach," *Health Services Research*, vol. 11, no. 4, pp. 464–477, 1976.

[33] G. Kaplan and O. Baron-Epel, "What lies behind the subjective evaluation of health status?" *Social Science and Medicine*, vol. 56, no. 8, pp. 1669–1676, 2003.

[34] R. J. Johnson and F. D. Wolinsky, "The structure of health status among older adults: disease, disability, functional limitation, and perceived health," *Journal of Health and Social Behavior*, vol. 34, no. 2, pp. 105–121, 1993.

[35] G. I. J. M. Kempen, J. Ormel, E. I. Brilman, and J. Relyveld, "Adaptive responses among Dutch elderly: the impact of eight chronic medical conditions on health-related quality of life," *American Journal of Public Health*, vol. 87, no. 1, pp. 38–44, 1997.

[36] S. H. Kim, G. Wolde-Tsadik, and D. B. Reuben, "Predictors of perceived health in hospitalized older persons: a cross-sectional and longitudinal study," *Journal of the American Geriatrics Society*, vol. 45, no. 4, pp. 420–426, 1997.

[37] T. Moum, "Self-assessed health among Norwegian adults," *Social Science and Medicine*, vol. 35, no. 7, pp. 935–947, 1992.

[38] C. D. Mulrow, M. B. Gerety, J. E. Cornell, V. A. Lawrence, and D. N. Kanten, "The relationship between disease and function and perceived health in very frail elders," *Journal of the American Geriatrics Society*, vol. 42, no. 4, pp. 374–380, 1994.

[39] J. Smith, "Well-being and health from age 70 to 100: findings from the Berlin aging study," *European Review*, vol. 9, no. 4, pp. 461–477, 2001.

[40] L. W. Poon, S. M. Jazwinski, R. C. Green et al., "Methodological considerations in studying centenarians: lessons learned from the Georgia Centenarian Studies," in *Annual Review of Gerontology and Geriatrics: Biopsychosocial Approaches to Longevity*, L. W. Poon and T. T. Perls, Eds., vol. 27, pp. 231–264, Springer, New York, NY, USA, 2007.

[41] P. Schönemann-Gieck, C. Rott, M. Martin, V. D'Heureuse, M. Kliegel, and G. Becker, "Similarities and differences between self-rated and proxy-rated health in extreme old ageÜbereinstimmungen und unterschiede in der selbst- und fremdeingeschätzten gesundheit bei extrem hochaltrigen," *Zeitschrift fur Gerontologie und Geriatrie*, vol. 36, no. 6, pp. 429–436, 2003.

[42] S. S. Bassett, J. Magaziner, and J. R. Hebel, "Reliability of proxy response on mental health indices for aged, community-dwelling women," *Psychology and Aging*, vol. 5, no. 1, pp. 127–132, 1990.

[43] A. LaRue, L. Bank, L. Jarvik, and M. Hetland, "Health in old age: how do physicians' ratings and self-ratings compare?" *Journals of Gerontology*, vol. 34, no. 5, pp. 687–691, 1979.

[44] M. Weinberger, G. P. Samsa, K. Schmader, S. M. Greenberg, D. B. Carr, and D. S. Wildman, "Comparing proxy and patients' perceptions of patients' functional status: results from an outpatient geriatric clinic," *Journal of the American Geriatrics Society*, vol. 40, no. 6, pp. 585–588, 1992.

[45] R. L. Kane, R. A. Kane, B. Bershadsky et al., "Proxy sources for information on nursing home residents' quality of life," *Journals of Gerontology B*, vol. 60, no. 6, pp. S318–S325, 2005.

[46] H. Tamim, J. McCusker, and N. Dendukuri, "Proxy reporting of quality of life using the EQ-5D," *Medical Care*, vol. 40, no. 12, pp. 1186–1195, 2002.

[47] W. L. Rodgers and A. R. Herzog, "Collecting data about the oldest: problems and procedures," in *The Oldest Old*, R. M. Suzman, D. P. Willis, and K. G. Manton, Eds., pp. 135–156, Oxford University, New York, NY, USA, 1992.

[48] G. G. Fillenbaum, *Multidimensional Functional Assessment of Older Adults: The Duke Older Americans Resources and Services Procedures*, Lawrence Erlbaum Associates, Hillsdale, Mich, USA, 1988.

[49] K. Jöreskog and D. Sorbom, "Lisrel 8: user's reference guide," SSI, Chicago, Ill, USA, 1996.

[50] T. L. Gruenewald and M. E. Kemeney, "Psychonueroimmunological processes in aging and health," in *Handbook of Health Psychology and Aging*, C. M. Aldwin, C. L. Park, and A. Spiro, Eds., pp. 97–118, Guilford Press, New York, NY, USA, 2007.

[51] J. R. Piazza, D. M. Almeida, N. O. Dmitrieva, and L. C. Klein, "Frontiers in the use of biomarkers of health in research on stress and aging," *Journals of Gerontology B*, vol. 65, no. 5, pp. 513–525, 2010.

[52] B. Singer and C. D. Ryff, *New Horizons in Health: An Integrative Approach*, National Academy Press, Washington, DC, USA, 2001.

[53] M. Jylhä, S. Volpato, and J. M. Guralnik, "Self-rated health showed a graded association with frequently used biomarkers in a large population sample," *Journal of Clinical Epidemiology*, vol. 59, no. 5, pp. 465–471, 2006.

[54] S. Cohen and S. D. Pressman, "Positive affect and health," *Current Directions in Psychological Science*, vol. 15, no. 3, pp. 122–125, 2006.

[55] Q. M. Temane and M. P. Wissing, "The role of subjective perception of health in the dynamics of context and psychological well-being," *Psychotherapy and Psychosomatics*, vol. 75, no. 2, pp. 85–95, 2006.

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Research Article

Body Mass Index Is Associated with Dietary Patterns and Health Conditions in Georgia Centenarians

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Associations between body mass index (BMI) and dietary patterns and health conditions were explored in a population-based multiethnic sample of centenarians from northern Georgia. BMI ≤20 and ≥25 was prevalent in 30.9% and 25.3% of study participants, respectively. In a series of logistic regression analyses controlled for gender and place of residence, the probability of having BMI ≥25 was increased by being black versus white and having a low citrus fruit, noncitrus fruit, orange/yellow vegetable or total fruit and vegetable intake. The probability of having BMI ≤20 was not associated with dietary intake. When controlled for race, gender, residence, and total fruit and vegetable intake, BMI ≥25 was an independent risk factor for diabetes or having a systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg, whereas BMI ≤20 was a risk factor for anemia. Given the many potential adverse consequences of under- and overweight, efforts are needed to maintain a healthy weight, even in the oldest old.

1. Introduction

Body mass index is a simple index of weight for height that is frequently used in the assessment of nutritional status. A low BMI, or underweight status, is often associated with an increased risk of mortality in seriously ill or hospitalized older adults [1, 2]. Conversely, a high BMI, indicative of overweight or obesity, is associated with an exacerbation in age-related physical and cognitive decline [3, 4] and with an increased prevalence or risk of many chronic health conditions common in older adults such as diabetes, hypertension, and cardiovascular disease [3–5]. Such associations are typically determined across the entire spectrum of older adults (aged 60+), with no further demarcation within this age classification. Our finding of a much higher prevalence of several nutritional deficiencies in centenarians as compared with octogenarians [6, 7], suggests that there is considerable heterogeneity in nutrient status in the "older adult" age group. Likewise, there may also be considerable heterogeneity within the older adult age group with regard to chronic health conditions. Thus, it is not known whether the associations between underweight or overweight/obesity and chronic health conditions as observed in previous studies of older adults extend to the very old.

Dietary intake patterns featuring a high intake of nutrient-dense foods such as cereals, fruits, vegetables, and low-fat meat and dairy products have been associated with a number of favorable health outcomes in adults including a decreased prevalence of obesity [8, 9], lower rates of weight gain over time [10], and better quality of life and improved survival [11]. In contrast, low-nutrient dense dietary patterns with high intakes of sweets, desserts, and high-fat dairy products have been associated with higher rates of obesity and poor nutritional status in older adults [9]. Whether these observations extend to the very old is unknown. Studies comparing energy intakes and dietary intake patterns of centenarians to younger older adult cohorts have generally observed lower energy and/or fat intake in the centenarians, while dietary preferences of centenarians are considerably more varied and dependent of the region of study likely reflecting cultural patterns and cohort differences rather than longevity-related differences

per se (reviewed [12]). Nonetheless, there can be considerable variation of body weight status within a given group of centenarians [13], though to our knowledge the extent to which this may be associated with potential differences in dietary intake patterns or selected health conditions has not been explored. Thus, the objectives of this study were to explore associations (1) between BMI and dietary habits and (2) between underweight or overweight/obesity and health status in a population-based multiethnic sample of centenarians (98 years and above) from northern Georgia in the USA. It was hypothesized that overweight/obesity, but not underweight, would be associated with poorer dietary habits and that both overweight/obesity and underweight, as well as dietary habits, would be associated with specific health conditions in this population.

2. Methods

2.1. Study Population. This study was a secondary analysis of data collected by the Georgia Centenarian Study, a population-based multidisciplinary study conducted in 44 counties in northern Georgia (USA) from 2002 to 2005. The original study included 244 centenarians (defined as age 98 and older). The sampling procedures and data collection methods have been described elsewhere [6, 14]. Briefly, recruitment of participants from skilled nursing facilities was based on estimates of the institutionalized population of the area according to the 2000 US Census tabulations. The community dwelling participants resided in private residences and personal care homes and were recruited from voter registration lists. Participants were recruited to match census figures for gender and race/ethnicity (white or black; all were non-Hispanic) and were interviewed by trained personnel in their place of residence. All questionnaires and procedures were approved by the University of Georgia Institutional Review Board on Human Subjects.

2.2. Demographic, Nutrition, and Health Information. Information regarding age, gender, race/ethnicity, living arrangements, health conditions (cardiovascular disease, diabetes, hypertension, etc.), and behaviors (including tobacco and nutritional supplement use) were obtained from each participant (or his/her caregiver) by self-report. Questions regarding food intake, appetite, and weight change were adapted from the Mini-Nutritional Assessment [15, 16] and the response categories for food intake represented current frequency of consumption of food groups, including dairy products (milk, yogurt, and cheese); meat, fish, or poultry; orange/yellow vegetables; green vegetables; citrus and noncitrus fruit and juice. The total food score, ranging from 0 to 5, was based on comparisons with the Dietary Guidelines for Americans [17] 1,600-calorie meal pattern for sedentary older adults, as previously described [16].

Body weight and height were measured by interviewers, obtained from charts or via self-report. In addition, knee height was measured on the right leg, unless contraindicated, to the nearest 0.1 centimeter and used to predict stature as per the formulas of Chumlea et al. [18]. Body mass index (BMI) was calculated as weight (kg)/height (meters)^2. BMI calculated from observed/recorded height and weight was highly correlated with BMI calculated from predicted stature [18] and observed/recorded weight ($r = 0.877$; $P < .0001$). BMI calculated from observed/recorded height and weight was used to form three BMI classifications based on the National Institutes of Health criteria [19] for overweight/obesity and defined as underweight $\leq 20\,kg/m^2$, normal weight >20 and $<25\,kg/m^2$, and overweight/obese $\geq 25\,kg/m^2$. Triceps skinfold (TSF) was measured on the right arm, unless contraindicated, by caliper to the nearest 0.1 millimeter. Mid-arm circumference (MAC) was measured on the right arm, unless contraindicated, to the nearest 0.1 centimeter. Systolic and diastolic blood pressure measurements were obtained with a brachial cuff.

2.3. Biochemical Indices. Nonfasting blood samples were collected as previously described [6, 14]. Hemoglobin was assessed by a clinical diagnostic laboratory (LabCorp, Inc., Burlington, NC) and anemia was defined as hemoglobin $<12\,g/dL$ for females or $<13\,g/dL$ for males [20].

2.4. Exclusions from Data Analysis. Participants missing data for primary variables of interest were excluded from the present analyses. From the original sample of centenarians, 11 individuals were excluded due to missing data for BMI ($n = 7$; includes one double-amputee), food intake patterns ($n = 1$), average grip strength ($n = 1$), and/or systolic/diastolic blood pressure ($n = 3$). Overall characteristics of the 233 centenarians included in the study are given in Table 1. Compared to the included centenarians ($n = 233$), the excluded centenarians tended to be older (102.6 ± 3.6 versus 100.5 ± 1.9 yrs; $P = .052$), but did not differ in gender (90.9% [excluded] versus 84.6% female), race/ethnicity (72.7% versus 79.0% white), or place of residence (36.4% versus 43.4% skilled nursing facility).

2.5. Statistical Analyses. Means, standard deviations, medians, range of values, and/or frequencies were calculated. Differences between participants with different BMI classifications were assessed with the Wilcoxon rank sum test for continuous variables and Chi square analysis for categorical variables. Probabilities reported are unadjusted for multiple tests. The level of significance was set at $P < .05$.

Because the food groups provide calories, a series of logistic regression analyses were performed with BMI ≤ 20 or BMI ≥ 25 as the dependent variable and gender, race/ethnicity, living arrangements, and reported intake of specific food groups as the independent variables. In addition, because BMI can play a causative role as a risk factor for chronic disease, a second series of logistic regression analyses was performed with diabetes, anemia, or other chronic health conditions as the dependent variable and race, gender, residence, and BMI ≤ 20 or BMI >25 as the independent variables (Model 1). A final series of logistic regression analyses was performed with diabetes, anemia, or other chronic health conditions as the dependent variable and race, gender, residence, total fruit, and vegetable intake and BMI ≤ 20 or BMI >25 as the independent variables (Model 2).

Table 1: Characteristics of study participants.

	Mean ± SD, or % (minimum – maximum)
Age (y)	100.5 ± 1.9 (98–108)
Gender	
Women (%)	84.6
Men (%)	15.4
Race	
White (%)	79.0
African American (%)	21.0
Living arrangements	
Skilled nursing facility (%)	43.4
Community (%)	56.6
Total food score[1]	3.1 ± 1.6 (0–5)
BMI (kg/m^2)	22.5 ± 4.2 (14.0– 35.2)
Mid-arm circumference	23.8 ± 4.3 (7.5–37.1)
Triceps skin fold	11.1 ± 5.7 (2.8–34.0)
Blood pressure (mm Hg)	
Systolic	127.7 ± 15.1 (90–190)
Diastolic	73.8 ± 9.4 (38–100)
Hemoglobin (g/dL)	12.0 ± 1.5 (7.5–16.7)

[1] Total food score ranged from 0 to 5 and one point was given for meeting each recommended serving from five food groups as follows: two or more servings of meat, poultry, or fish daily, two or more servings of dairy foods daily, four or more servings of green vegetables weekly, three or more servings of orange or yellow vegetables weekly, and three or more servings of fruit daily [16].

P-values are unadjusted for multiple comparisons, because all comparisons were preplanned. Analyses were conducted using SAS 9.2 (SAS, Cary, NC).

3. Results

Approximately one-third (31.3%) of the centenarians included in the final analytical sample had a BMI of ≤20 (underweight), 43.8% were classified as being in the normal weight range, and 24.9% met the NIH classification for overweight/obesity (BMI ≥25). Triceps skin fold (TSF) and mid-arm circumference (MAC) of centenarians in the lowest BMI classification averaged below the 5th percentile for females 80+ years in USA based on 2003–2006 NHANES data [21]. Both parameters increased with BMI classification in a stepwise manner (data not shown) and were highly correlated with BMI (Pearson Correlation Coefficient between BMI and TSF = 0.482; and between BMI and MAC = 0.624; $P < .0001$ for both).

Chi-square analysis indicated that those with a BMI ≤20 were more likely to be women, live in a skilled nursing facility, eat a modified food diet, have experienced a weight change in the past three months, and have anemia as compared to centenarians classified as normal weight or overweight/obese (Table 2). Conversely, those with a BMI ≥25 were more likely to be black, diabetic, have a higher systolic blood pressure, and/or have a diastolic blood pressure ≥90 mm as compared to centenarians classified as underweight or normal weight. There were no differences according to BMI classification with regard to history of CVD, cancer, stroke, depression, or past or current tobacco use.

Bivariate analysis of diet intake patterns suggested that centenarians with BMI ≤20 had the highest total food scores and were more likely to report eating two or more servings of meat, fish, and poultry per day and three or more total servings of fruit per day as compared with centenarians in the other BMI classifications. In contrast, those with a BMI ≥25 were more likely to report eating less than one serving of citrus or noncitrus fruit per day, less than four servings of orange/yellow vegetables per week, or three total servings of fruit and vegetables per day (Table 2). A series of logistic regression analyses indicated that when controlled for gender, race, and place of residence the odds of having a BMI ≥25 were about two to three times higher in centenarians with lower intakes of citrus and noncitrus fruit (less than one serving per day), orange and yellow vegetables (less than four servings per week), or total fruits and vegetables (less than three servings per day), but were not related to intake of the meat group or dairy group (Table 3). Similar analyses with BMI ≤20 as the dependent variable, failed to show any significant association with dietary intake categories (Table 3).

Finally, associations between BMI classifications and chronic health conditions were determined in a series of logistic regression analyses that included either BMI ≤20 or BMI ≥25 as an independent variable (Table 4). When controlled for gender, race, and place of residence, the odds of having anemia, based on blood hemoglobin values, were over twofold higher in centenarians with BMI ≤20 versus those with BMI >20 whereas the odds of self-reported CVD tended to be reduced in those in the underweight classification (BMI ≤20; $P = .053$). The latter finding became significant in regression models further controlled for total fruit and vegetable intake (Model 2; $P = .048$). In analyses controlled for gender, race, and place of residence, the odds of having self-reported diabetes or systolic blood pressure ≥140 mmHg were approximately three- and twofold higher, respectively, in centenarians with BMI ≥25 versus those with BMI <25. Being overweight/obese (BMI ≥25) also tended to increase the odds of having diastolic blood pressure >90 mmHg (approximately three-fold; $P = .055$) or cardiovascular disease (approximately twofold; $P = .074$). Further controlling for total fruit and vegetable intake (Model 2) strengthened the associations between BMI ≥25 and diabetes and systolic blood pressure >140 mmHg, and resulted in a significant association between BMI ≥25 and diastolic blood pressure ≥90 mmHg. There were no associations between BMI ≤20 or BMI ≥25 and stroke, depression, or cancer in any of the regression models.

4. Discussion

To our knowledge, this is the first study to explore associations between dietary patterns and body weight status in the oldest old segment of the population. Prevalence of overweight/obesity in this population-based study of

Table 2: Demographics, dietary patterns, and health conditions of centenarians of varying BMI classification: The Georgia Centenarian Study.

	BMI ≤20[1] Median, range, mean ± SD, or %	BMI >20 and < 25[1] Median, range, mean ± SD, or %	BMI ≥ 25[1] Median, range, mean ± SD, or %	*P*
Age	100.5, 98.1–106.0 100.8 ± 1.8	100.2, 98.1–105.2 100.5 ± 1.8	99.6, 98.1–108.5 100.2 ± 2.1	.051
Gender[2]				
Women	34.5 (68)	41.1 (81)	24.4 (48)	.042
Men	13.9 (5)	58.3 (21)	27.8 (10)	
Race[2]				
White	33.2 (61)	46.2 (85)	20.6 (38)	.015
African American	24.5 (12)	34.7 (17)	40.8 (20)	
Living arrangements[2]				
Skilled nursing facility	43.6 (44)	35.6 (36)	20.8 (21)	.002
Community	22.0 (29)	50.0 (66)	28.0 (37)	
B-vitamin supplements[3]				
No	61.6 (45)	63.7 (65)	74.1 (43)	.281
Yes	38.4 (28)	36.3 (37)	25.9 (15)	
Total food score[4]	4.0, 0–5 3.5 ± 1.6[a]	3.0, 0–5 3.0 ± 1.5[ab]	2.0, 0–5 2.8 ± 1.6[b]	.046
<3	37.0 (27)	49.0 (50)	55.2 (32)	.097
≥3	63.0 (46)	51.0 (52)	44.8 (26)	
Meat, fish, poultry intake (servings/day)				
<2	41.1 (30)	57.8 (59)	60.3 (35)	.041
≥2	58.9 (43)	42.2 (43)	39.6 (23)	
Milk and dairy product intake (servings/day)				
<2	45.2 (33)	55.9 (57)	59.6 (34)	.210
≥2	54.8 (40)	44.1 (45)	40.4 (23)	
Green vegetable intake (servings/week)				
<4	6.8 (5)	4.9 (5)	8.6 (5)	.644
≥4	93.2 (68)	95.1 (97)	91.4 (53)	
Orange and yellow vegetable intake (servings/week)				
<3	9.6 (7)	11.8 (12)	20.7 (12)	.148
≥3	90.4 (66)	88.2 (90)	79.3 (46)	
Total fruit (servings/day)				
<3	50.7 (37)	66.7 (68)	70.7 (41)	.034
≥3	49.3 (36)	33.3 (34)	29.3 (17)	
Citrus fruit intake (servings/dly)				
<1	27.4 (20)	34.3 (35)	50.0 (29)	.025
≥1	72.6 (53)	65.7 (67)	50.0 (29)	
Noncitrus fruit intake (servings/day)				
<1	24.7 (18)	34.3 (35)	46.6 (27)	.032
≥1	75.3 (55)	65.7 (67)	53.4 (31)	

Table 2: Continued.

	BMI ≤20[1] Median, range, mean ± SD, or %	BMI >20 and < 25[1] Median, range, mean ± SD, or %	BMI ≥ 25[1] Median, range, mean ± SD, or %	*P*
Orange and yellow vegetable intake (servings/week)				
<4	23.3 (17)	24.5 (25)	41.4 (24)	.038
≥4	76.7 (56)	75.5 (77)	58.6 (34)	
Total fruit and vegetables (servings/day)				
<3	17.8 (13)	25.5 (26)	37.9 (22)	.033
≥3	82.2 (60)	74.5 (76)	62.1 (36)	
Needs help at mealtime				
Yes	42.5 (31)	29.4 (30)	24.1 (14)	.060
No	57.5 (42)	70.6 (72)	75.9 (44)	
Eats a typical diet of regular foods				
Yes	50.0 (36)	77.4 (79)	74.1 (43)	.0003
No-foods modified	50.0 (36)	22.6 (23)	25.9 (15)	
Body weight change in past 3 months				
Loss	28.2 (20)	20.6 (20)	12.3 (7)	.040
Gain	15.5 (11)	7.2 (7)	19.3 (11)	
No	56.3 (40)	72.2 (70)	68.4 (39)	
Appetite loss in past 3 months				
Yes (moderate/severe)	11.3 (8)	13.9 (14)	5.3 (3)	.249
No loss	88.7 (63)	86.1 (87)	94.7 (54)	
Systolic BP, mmHg	122, 90–160 124.4 ± 14.5[a]	125, 100–165 126.2 ± 12.7[a]	130, 110–190 134.4 ± 17.6[b]	.0013
<140	86.3 (63)	81.4 (83)	67.2 (39)	.022
≥140	13.7 (10)	18.6 (19)	32.8 (19)	
Diastolic BP, mmHg	72, 56–100 73.0 ± 8.5	73, 46–100 73.2 ± 8.8	75, 38–100 76.0 ± 11.1	.261
<90	95.9 (70)	96.1 (98)	86.2 (51)	.031
≥90	4.1 (3)	3.9 (4)	13.8 (8)	
Diabetes[5]				
Yes	8.2 (6)	3.9 (4)	15.5 (9)	.036
No	91.8 (67)	96.1 (98)	84.5 (49)	
Anemia[6]				
Yes	63.2 (43)	44.4 (44)	47.4 (27)	.048
No	36.8 (25)	55.6 (55)	52.6 (30)	
CVD[5]				
Yes	58.9 (43)	61.8 (63)	69.0 (40)	.482
No	41.1 (30)	38.2 (39)	31.0 (18)	
Cancers[5]				
Yes	31.5 (23)	31.4 (32)	24.1 (14)	.574
No	68.5 (50)	68.6 (70)	75.9 (44)	
Stroke[5]				
Yes	23.3 (17)	21.6 (22)	22.4 (13)	.964

Table 2: Continued.

	BMI ≤20[1] Median, range, mean ± SD, or %	BMI >20 and < 25[1] Median, range, mean ± SD, or %	BMI ≥ 25[1] Median, range, mean ± SD, or %	*P*
No	76.7 (56)	78.4 (80)	77.6 (45)	
Depression[5]				
Yes	20.6 (15)	11.8 (12)	10.3 (6)	.163
No	79.4 (58)	88.2 (90)	89.7 (52)	
Current or past tobacco use[5]				
Yes	25.0 (18)	31.4 (32)	28.1 (16)	.654
No	75.0 (54)	68.6 (70)	71.9 (41)	
Current tobacco use[5]				
Yes	1.4 (1)	3.9 (4)	3.5 (2)	.612
No	98.6 (71)	96.1 (98)	96.5 (55)	

[a,b]Means with different superscripts are significantly different, $P < .05$.
[1]$n = 73$ for BMI ≤20; $n = 102$ for BMI > 20 and < 25; $n = 58$ for BMI ≥ 25.
[2] Percentages add up to 100% across a row. Number of participants represented by each percentage is included in brackets ().
[3]B vitamin supplements included multivitamin/mineral, B vitamins, or single oral supplements of vitamin B12.
[4]Total food score ranged from 0 to 5 and one point was given for meeting each recommended serving from five food groups as follows: two or more servings of meat, poultry, or fish daily, two or more servings of dairy foods daily, four or more servings of green vegetables weekly, three or more servings of orange or yellow vegetables weekly, and three or more servings of fruit daily (16).
[5]Information obtained from participant or a proxy by self-report.
[6]Based on laboratory values and defined as hemoglobin <12 g/dL for females or <13 g/dL for males (20).

Table 3: Associations of dietary intake patterns with underweight or overweight/obesity in Georgia centenarians.

	BMI ≤20[a]		BMI ≥ 25[b]	
Independent Variable	Odds ratio (95% CI)	*P*	Odds ratio (95% CI)	*P*
Meat, <2 servings/day	0.83 (0.40–1.70)	.608	1.17 (0.54–2.53)	.685
Dairy, <2 serving/day	0.93 (0.48–1.78)	.819	1.18 (0.57–2.42)	.660
Fruit & vegetables, <3 servings/day	0.75 (0.35–1.59)	.450	2.12 (1.04–4.32)	.039
Orange/yellow vegetables, <4 servings/week	1.17 (0.56–2.44)	.684	2.27 (1.11–4.65)	.025
Citrus fruit, <1 serving/day	0.77 (0.40–1.50)	.445	2.40 (1.20–4.76)	.013
Noncitrus fruit, <1 serving/day	0.73 (0.36–1.45)	.362	2.18 (1.09–4.38)	.028

[a]Determined by a series of logistic regression analyses with BMI ≤20 versus BMI ≥20 as the dependent variable, controlled for gender, race, and residence.
[b]Determined by a series of logistic regression analyses with BMI ≥25 versus BMI <25 as the dependent variable, controlled for gender, race, and residence.

centenarians was approximately 25%, which was considerably below the prevalence for these conditions in the overall population of older adults, aged 60 and above, in the USA at the time of data collection (~69%; [22]). In analyses controlled for gender, race, and place of residence, several parameters indicative of a low frequency of fruit and vegetable intake were associated with overweight/obesity (BMI ≥25), whereas there were no associations between frequency of intake of meat, dairy, and fruits and vegetables and being underweight (BMI ≤20). Other findings include strong associations of underweight with anemia and of overweight/obesity with diabetes and high blood pressure, extending knowledge of such associations to the very old.

The present research was part of a large, multidisciplinary study across a range of cognitive, mental, physical, and health-associated domains exploring the role of various factors pertinent to the survival and functioning of centenarians. To decrease testing burden for participants, dietary intake data focused on frequency of intake of specific food groups selected based on the Dietary Guidelines for Americans and age-related associations with nutritional deficiencies and chronic diseases [16, 17]. Notably, we observed that based on frequency of intake and regardless of BMI classification, a large percentage of centenarians were not meeting the dietary guidelines for many food groups, with the exception of green and orange/yellow vegetables. This later finding is consistent with an apparent preference for sweet potatoes and green vegetables reported for an earlier convenience sampling of Georgia centenarians [23, 24]. Such preferences likely are reflective of the traditional diet of the Southeastern USA, rather than longevity-related differences in dietary patterns, and may not be replicable in other regions and cultures.

Our initial analysis indicated a greater intake of meat and total fruits in the underweight centenarians, suggesting that they were eating better than those in the normal weight and

Table 4: Associations of underweight or overweight/obesity with health conditions or indicators in Georgia centenarians.

	BMI ≤20[a]		BMI ≥ 25[b]	
Dependent variable	Odds ratio (95% CI)	*P*	Odds ratio (95% CI)	*P*
Diabetes				
Model 1	0.91 (0.30–2.69)	.858	2.86 (1.02–7.99)	.045
Model 2	0.89 (0.30–2.64)	.837	3.11 (1.11–8.75)	.031
Anemia				
Model 1	2.47 (1.32–4.62)	.004	0.72 (0.38–1.36)	.309
Model 2	2.47 (1.32–4.62)	.005	0.72 (0.38–1.37)	.318
Systolic BP ≥140 mmHg				
Model 1	0.72 (0.32–1.61)	.425	2.09 (1.02–4.27)	.043
Model 2	0.70 (0.31–1.57)	.385	2.26 (1.09–4.69)	.029
Diastolic BP ≥90 mmHg				
Model 1	0.81 (0.25–3.18)	.760	2.96 (0.98–9.01)	.055
Model 2	0.71 (0.18–2.79)	.621	3.81 (1.21–12.05)	.022
CVD				
Model 1	0.54 (0.29–1.01)	.053	1.85 (0.94–3.64)	.074
Model 2	0.53 (0.28–1.00)	.048	1.95 (0.98–3.88)	.058
History of cancer				
Model 1	1.09 (0.58–2.07)	.791	0.82 (0.40–1.66)	.575
Model 2	1.08 (0.57–2.05)	.818	0.87 (0.42–1.77)	.694
Stroke				
Model 1	0.91 (0.46–1.82)	.791	1.18 (0.57–2.47)	.655
Model 2	0.91 (0.46–1.83)	.794	1.18 (0.56–2.47)	.667
Depression				
Model 1	1.12 (0.49–2.57)	.782	0.89 (0.32–2.50)	.829
Model 2	1.22 (0.53–2.80)	.640	0.91 (0.32–2.58)	.854

[a]Determined by a series of logistic regression analyses with BMI ≤20 versus BMI >20 as an independent variable, controlled for gender, race, and residence (Model 1) or for gender, race, residence, and total fruit and vegetable intake (<3 servings/day = 1; >3 servings/day = 0) (Model 2).
[b]Determined by a series of logistic regression analyses with BMI ≥25 versus BMI <25 as an independent variable, controlled for gender, race, and residence (Model 1) or for gender, race, residence, and total fruit and vegetable intake (<3 servings/day = 1; >3 servings/day = 0) (Model 2).

overweight/obese classifications. The underweight centenarians also had the highest total food scores, suggesting that they were meeting more of the recommended servings for specific food groups [17]. However, almost twice as many underweight centenarians lived in skilled nursing facilities as compared to the community, and there was no association between low BMI and dietary groups after controlling for race, residence, and gender. In a specific comparison between centenarians residing in skilled nursing facilities and in the community, Johnson et al. [16] reported that those in skilled nursing facilities were more likely to eat three or more meals a day and to have a higher frequency of intake of most food groups. They suggested that such differences may be due to (1) the requirement that skilled nursing facilities serve meals that meet dietary guidelines and other federal nutrition policies [25, 26], (2) the inability of the methodology used to distinguish between food that was served and food that was eaten, and (3) barriers faced by community dwelling centenarians or their caregivers in purchasing, preparing, or consuming appropriate food to meet their nutritional needs. Thus, associations in centenarians between low BMI and dietary status may be quite complex and influenced considerably by place of residence.

In the present study, there was an increase in the relative risk for being overweight/obese in centenarians reporting the lowest frequency of intake of some nutrient dense foods including orange/yellow vegetables and citrus and noncitrus fruits. These observations are consistent with previous studies finding associations between lower reported or inferred consumption of fruits and/or vegetables and increased prevalence of overweight/obesity in children [27] and young to middle-age adults [5, 28–30]. Interestingly, other studies in adults have indicated that increased consumption of fruits and vegetables may be an effective strategy for decreasing energy consumption and for increasing and maintaining weight loss [29, 31, 32]. In addition to potential beneficial effects on body weight, there is considerable evidence in other age groups that high consumption of fruits and vegetables may offer protective effects against and/or to reduce the relative risk of cardiovascular disease, hypertension, diabetes, and certain cancers [33–40]. We observed that centenarians in the highest weight category (BMI ≥25) reported eating lower amounts of certain types of fruits and vegetables than their nonoverweight/nonobese counterparts and also appeared to be at greater risk for diabetes, high blood pressure, and cardiovascular disease.

This suggests that a high intake of fruits and vegetables may be beneficial for maintaining optimal weight, and perhaps decreasing risk of chronic disease, even at very advanced age. However, additional, ideally longitudinal, research collecting more detailed food intake data is needed to support this contention. In addition, as social isolation, missing teeth, digestion difficulties, poor self-reported health, cost and preparation issues have been identified as barriers to fruit and vegetable intake in older adults [41–43], research is needed to determine if and to what degree these or other potential barriers may be influencing the intake of fruit, vegetables, and other low energy, high nutrient dense foods in the very old.

Clinically defined anemia was present in greater than 60% of the centenarians in the lowest BMI grouping. After controlling for demographic covariates, a strong association remained between low BMI and anemia but not with other health conditions and indicators. Accordingly, low BMI had been identified as an independent correlate or risk factor for anemia in some previous studies in older adults and clinical populations [44–46], but not in others [47]. Although older adults with a low BMI are considered to be at nutritional risk [47, 48], it cannot be assumed that the anemia observed in underweight older adults is primarily of dietary or nutritional etiology. Indeed, our previous studies indicate a similar prevalence of anemia in vitamin B12-deficient and vitamin B12-adequate in Georgia centenarians [7] and a high prevalence of inflammatory anemia, either alone or in combination with nutritional deficiencies, in this population [49]. Nonetheless, as anemia is associated with increased mortality in acute and chronic disease states, particularly in those underweight [50, 51], it is important to monitor and treat this condition, as appropriate, in the very old.

In summary, this secondary analysis provides evidence of an inverse association between fruit and vegetable intake and body weight status in a population-based study of centenarians. In addition, both underweight and overweight emerged as potential risk factors for various chronic diseases, emphasizing the importance of monitoring weight and of maintaining a healthy weight, even at very advanced ages. A major strength of the study is the inclusion of a population-based sampling of centenarians with greater diversity in race, place of residence, and functional status than would be typically obtained with a convenience sample. Limitations of the study include the relatively small sample size, lack of information regarding physical activity, and reliance on data of frequency of intake for only a few food groupings rather than the use of a more extensive food frequency questionnaire including individual foods and serving sizes as per our earlier convenience study of centenarians [23, 24]. Absence of intake data on key dietary components including grains/cereals and sweets/desserts necessitated the use a series of nonindependent binary logistic regression models instead of a more complex, single multinomial logistic regression to explore potential associations between BMI and dietary intake patterns. Thus, specifically designed studies including more detailed information of dietary intake, physical activity data, and additional chronic disease indicators are needed to verify associations, or lack thereof, between dietary intake patterns, weight status, and chronic health conditions in the very old. Furthermore, as dietary habits and other characteristics of this sample from Georgia likely differ from those of centenarians from other countries and cultures, our findings need replication in other population groups.

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References

[1] K. C. Neidert and B. Borner, *Nutrition Care of the Older Adult: A Handbook for Dietetics Professionals Working throughout the Continuum of Care*, American Dietetic Association, 2nd edition.

[2] K. Kitamura, K. Nakamura, T. Nishiwaki, K. Ueno, and M. Hasegawa, "Low body mass index and low serum albumin are predictive factors for short-term mortality in elderly Japanese requiring home care," *Tohoku Journal of Experimental Medicine*, vol. 221, no. 1, pp. 29–34, 2010.

[3] D. T. Villareal, C. M. Apovian, R. F. Kushner, and S. Klein, "Obesity in older adults: technical review and position statement of the American Society for Nutrition and NAASO, The Obesity Society," *American Journal of Clinical Nutrition*, vol. 82, no. 5, pp. 923–934, 2005.

[4] D. K. Houston, B. J. Nicklas, and C. A. Zizza, "Weighty concerns: the growing prevalence of obesity among older adults," *Journal of the American Dietetic Association*, vol. 109, no. 11, pp. 1886–1895, 2009.

[5] M. Tjepkema, "Adult obesity," *Health Reports/Statistics Canada*, vol. 17, no. 3, pp. 9–25, 2006.

[6] M. A. Johnson, A. Davey, S. Park, D. B. Hausman, and L. W. Poon, "Age, race and season predict vitamin D status in African American and White centenarians and octogenarians," *Journal of Nutrition, Health and Aging*, vol. 12, pp. 690–695, 2008.

[7] M. A. Johnson, D. B. Hausman, A. Davey, L. W. Poon, R. H. Allen, and S. P. Stabler, "Vitamin B12 deficiency in African American and white octogenarians and centenarians in Georgia," *Journal of Nutrition, Health and Aging*, vol. 14, no. 5, pp. 339–345, 2010.

[8] A. M. Paradis, G. Godin, L. Pérusse, and M. C. Vohl, "Associations between dietary patterns and obesity phenotypes," *International Journal of Obesity*, vol. 33, no. 12, pp. 1419–1426, 2009.

[9] J. H. Ledikwe, H. Smiciklas-Wright, D. C. Mitchell, C. K. Miller, and G. L. Jensen, "Dietary patterns of rural older adults are associated with weight and nutritional status," *Journal of the American Geriatrics Society*, vol. 52, no. 4, pp. 589–595, 2004.

[10] P. K. Newby, D. Muller, J. Hallfrisch, R. Andres, and K. L. Tucker, "Food patterns measured by factor analysis and anthropometric changes in adults," *The American Journal of Clinical Nutrition*, vol. 80, no. 2, pp. 504–513, 2004.

[11] A. L. Anderson, T. B. Harris, F. A. Tylavsky et al., "Dietary patterns and survival in older adults," *Journal of the American Dietetic Association*, vol. 111, pp. 84–91, 2011.

[12] D. B. Hausman, J. G. Fischer, and M. A. Johnson, "Nutrition in centenarians," *Maturitas*, vol. 68, pp. 203–209, 2011.

[13] A. Davey, M. F. Elias, I. C. Siegler et al., "Cognitive function, physical performance, health, and disease: norms from the Georgia centenarian study," *Experimental Aging Research*, vol. 36, pp. 394–425, 2010.

[14] L. W. Poon, S. M. Jazwinski, R. C. Green et al., "Methodological considerations in studying centenarians: lessons learned from the Georgia centenarian studies," *Annual Review of Gerontology and Geriatrics*, vol. 27, pp. 213–264, 2007.

[15] Y. Guigoz, B. Vellas, and P. J. Garry, "Assessing the nutritional status of the elderly: the Mini Nutritional Assessment as part of the geriatric evaluation," *Nutrition Reviews*, vol. 54, no. 1, pp. S59–S65, 1996.

[16] M. A. Johnson, A. Davey, D. B. Hausman et al., "Dietary differences between centenarians residing in communities and in skilled nursing facilities: the Georgia Centenarian Study," *Age*, vol. 28, no. 4, pp. 333–341, 2006.

[17] US Department of Health and Human Services and US Department of Agriculture, "Dietary Guidelines for Americans 2005," 2005, http://www.health.gov/dietaryguidelines/dga2005/document/pdf/DGA2005.pdf.

[18] W. C. Chumlea, S. S. Guo, K. Wholihan, D. Cockram, R. J. Kuczmarski, and C. L. Johnson, "Stature prediction equations for elderly non-Hispanic white, non-Hispanic black, and Mexico-American persons developed from NHANES III data," *Journal of the American Dietetic Association*, vol. 98, no. 2, pp. 137–142, 1998.

[19] National Institutes of Health: National Heart Lung and Blood Institute, North American Association for the Study of Obesity, "Practical Guide to the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults," 2000, http://www.nhlbi.nih.gov/guidelines/obesity/prctgd_c.pdf.

[20] B. Blanc, C. A. Finch, L. Hallberg et al., "Nutritional anaemias. Report of a WHO Scientific Group," *WHO Technical Report Series*, no. 40, pp. 1–40, 1968.

[21] M. A. McDowell, C. D. Fryar, C. L. Ogden, and K. M. Flegal, "Anthropometric reference data for children and adults: United States, 2003–2006," National Health Statistics Reports, Number 10, October 2008, http://www.cdc.gov/nchs/data/nhsr/nhsr010.pdf.

[22] C. L. Ogden, M. D. Carroll, L. R. Curtin, M. A. McDowell, C. J. Tabak, and K. M. Flegal, "Prevalence of overweight and obesity in the United States, 1999–2004," *Journal of the American Medical Association*, vol. 295, no. 13, pp. 1549–1555, 2006.

[23] M. A. Johnson, M. A. Brown, L. W. Poon, P. Martin, and G. M. Clayton, "Nutritional patterns of centenarians," *International Journal of Aging and Human Development*, vol. 34, no. 1, pp. 57–76, 1992.

[24] D. K. Houston, M. A. Johnson, L. W. Poon, and G. M. Clayton, "Individual foods and food group patterns of the oldest old," *Journal of Nutrition for the Elderly*, vol. 13, no. 4, pp. 5–23, 1994.

[25] V. H. Castellanos, "Food and nutrition in nursing homes," *Generations*, vol. 28, no. 3, pp. 65–71, 2004.

[26] N. S. Wellman and B. Kamp, "Federal food and nutrition assistance programs for older people," *Generations*, vol. 28, no. 3, pp. 78–85, 2004.

[27] J. Aranceta, C. Pérez-Rodrigo, L. Serra-Majem et al., "Prevention of overweight and obesity: a Spanish approach," *Public Health Nutrition*, vol. 10, no. 10A, pp. 1187–1193, 2007.

[28] L. Wang, J. M. Gaziano, E. P. Norkus, J. E. Buring, and H. D. Sesso, "Associations of plasma carotenoids with risk factors and biomarkers related to cardiovascular disease in middle-aged and older women," *American Journal of Clinical Nutrition*, vol. 88, no. 3, pp. 747–754, 2008.

[29] K. E. E. Schroder, "Effects of fruit consumption on body mass index and weight loss in a sample of overweight and obese dieters enrolled in a weight-loss intervention trial," *Nutrition*, vol. 26, no. 7-8, pp. 727–734, 2010.

[30] T. Andreyeva, M. W. Long, K. E. Henderson, and G. M. Grode, "Trying to lose weight: diet strategies among Americans with overweight or obesity in 1996 and 2003," *Journal of the American Dietetic Association*, vol. 110, no. 4, pp. 535–542, 2010.

[31] J. Kruger, H. M. Blanck, and C. Gillespie, "Dietary practices, dining out behavior, and physical activity correlates of weight loss maintenance," *Preventing Chronic Disease*, vol. 5, no. 1, p. A11, 2008.

[32] M. C. de Oliveira, R. Sichieri, and R. Venturim Mozzer, "A low-energy-dense diet adding fruit reduces weight and energy intake in women," *Appetite*, vol. 51, no. 2, pp. 291–295, 2008.

[33] S. Liu, J. E. Manson, I. M. Lee et al., "Fruit and vegetable intake and risk of cardiovascular disease: the Women's Health Study," *American Journal of Clinical Nutrition*, vol. 72, no. 4, pp. 922–928, 2000.

[34] K. J. Joshipura, F. B. Hu, J. E. Manson et al., "The effect of fruit and vegetable intake on risk for coronary heart disease," *Annals of Internal Medicine*, vol. 134, no. 12, pp. 1106–1114, 2001.

[35] L. A. Bazzano, J. He, L. G. Ogden et al., "Fruit and vegetable intake and risk of cardiovascular disease in US adults: the first National Health and Nutrition Examination Survey Epidemiologic Follow-up Study," *American Journal of Clinical Nutrition*, vol. 76, no. 1, pp. 93–99, 2002.

[36] E. Riboli and T. Norat, "Epidemiologic evidence of the protective effect of fruit and vegetables on cancer risk," *American Journal of Clinical Nutrition*, vol. 73, supplement 3, pp. 559S–569S, 2003.

[37] L. P. Svetkey, T. P. Erlinger, W. M. Vollmer et al., "Effect of lifestyle modifications on blood pressure by race, sex, hypertension status, and age," *Journal of Human Hypertension*, vol. 19, no. 1, pp. 21–31, 2005.

[38] F. J. van Duijnhoven, H. B. Bueno-de-Mesquita, P. Ferrari et al., "Fruit, vegetables, and colorectal cancer risk: the European Prospective Investigation into Cancer and Nutrition," *American Journal of Clinical Nutrition*, vol. 89, pp. 1441–1452, 2009.

[39] K. Esposito, C. M. Kastorini, D. B. Panagiotakos, and D. Giugliano, "Prevention of type 2 diabetes by dietary patterns: a systemic review of prospective studies and meta-analysis," *Metabolic Syndrome and Related Disorders*, vol. 8, pp. 471–476, 2010.

[40] P. Carter, L. J. Gray, J. Troughton, K. Khunti, and M. J. Davies, "Fruit and vegetable intake and incidence of type 2 diabetes: systemic review and meta-analysis," *British Medical Journal*, vol. 341, p. c4229, 2010.

[41] G. Tsakos, K. Herrick, A. Sheiham, and R. G. Watt, "Edentulism and fruit and vegetable intake in low-income adults," *Journal of Dental Research*, vol. 89, no. 5, pp. 462–467, 2010.

[42] S. J. Hendrix, J. G. Fischer, S. Reddy et al., "Fruit and vegetable intake and knowledge increased following a community-based intervention in older adults in Georgia senior centers," *Journal of Nutrition for the Elderly*, vol. 27, no. 1-2, pp. 155–178, 2008.

[43] N. R. Sahyoun, X. L. Zhang, and M. K. Serdula, "Barriers to the consumption of fruits and vegetables among older adults," *Journal of Nutrition for the Elderly*, vol. 24, no. 4, pp. 5–21, 2006.

[44] C. W. Choi, J. Lee, K. H. Park et al., "Prevalence and characteristics of anemia in the elderly: cross-sectional study of three urban Korean population samples," *American Journal of Hematology*, vol. 77, no. 1, pp. 26–30, 2004.

[45] H. Ohwada, T. Nakayama, N. Nara, Y. Tomono, and K. Yamanaka, "An epidemiological study on anemia among institutionalized people with intellectual and/or motor disability with special reference to its frequency, severity and predictors," *BMC Public Health*, vol. 6, article 85, 2006.

[46] B. DiIorio, M. Cirillo, V. Bellizzi, D. Stellato, and N. G. DeSanto, "Prevalence and correlates of anemia and uncontrolled anemia in chronic hemodialysis patients-the Campania Dialysis Registry," *International Journal of Artificial Organs*, vol. 30, pp. 325–333, 2007.

[47] A. Ramel, P. V. Jonsson, S. Bjornsson, and I. Thorsdottir, "Anemia, nutritional status, and inflammation in hospitalized elderly," *Nutrition*, vol. 24, no. 11-12, pp. 1116–1122, 2008.

[48] L. Watson, W. Leslie, and C. Hankey, "Under-nutrition in old age: diagnosis and management," *Reviews in Clinical Gerontology*, vol. 16, no. 1, pp. 23–34, 2006.

[49] A. Haslam, *Anemia in Georgia centenarians and octogenarians*, M.S. thesis, University of Georgia, Athens, Ga, USA, 2010.

[50] R. D. Semba, M. O. Ricks, L. Ferrucci et al., "Types of anemia and mortality among older disabled women living in the community: the Women's Health and Aging Study I," *Aging Clinical and Experimental Research*, vol. 19, no. 4, pp. 259–264, 2007.

[51] D. Aronson, M. Nassar, T. Goldberg, M. Kapeliovich, H. Hammerman, and Z. S. Azzam, "The impact of body mass index on clinical outcomes after acute myocardial infarction," *International Journal of Cardiology*, vol. 145, pp. 476–480, 2010.