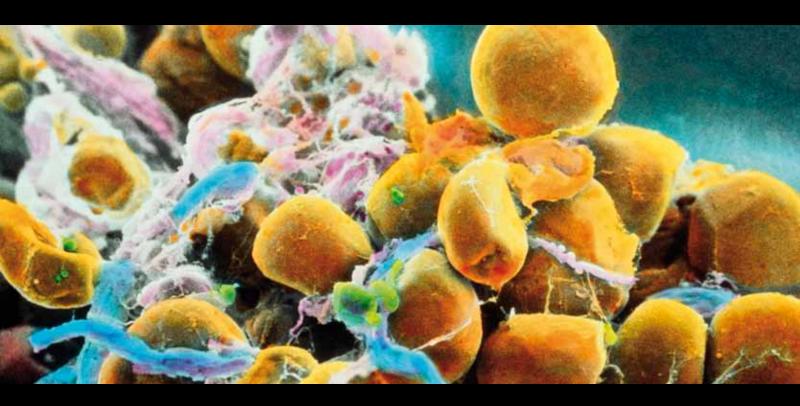
The Influence of Physical Activity on Obesity and Health

Guest Editors: George P. Nassis, Panagiota Klentrou, António Palmeira, and David John Stensel



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Editorial

The Influence of Physical Activity on Obesity and Health

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The eleven articles featured in this issue are grouped into 3 categories based on their topic: (a) complications of obesity, (b) biological effects of physical activity/exercise training on body composition and health related factors, and (c) behavioural aspects related to physical activity and exercise.

To highlight some of the findings and with regards to the first topic, M. J. Duncan and M. Stanley report that the functional movement score is negatively correlated with BMI in 10-11-year-old British children. The authors concluded "Scientists and practitioners ... need to consider interventions which will develop functional movement skills alongside physical activity and weight management strategies in children in order to reduce the risks of orthopaedic abnormality arising from suboptimal movement patterns in later life". The paper from E. Aadland and L. Robertson focuses on the understudied subject of physical activity on severely obese individuals. It is somewhat surprising that this population, that could benefit a lot from being physically active, does not yet have a specific set of guidelines. The data from this study shows that physical activity is mostly associated with weight change in men while it was mostly associated with changes in cardiorespiratory fitness (CRF) in women. The study by D. M. Baur et al. was designed to describe the CRF levels of professionally active career firefighters across different age groups with the objective to determine effect modification by physical activity and BMI levels. Their original results confirm that CRF in career firefighters is significantly reduced with increasing age and clearly show for the first time in this population that the age-related decline in CRF is highly dependent on BMI

and physical activity habits. For older adults with knee osteoathritis (OA), knee pain is associated with difficulty in walking, while obesity is also associated with difficulty in walking and low levels of physical activity and is a primary risk factor for knee OA. D. K. White et al. put these two factors together in order to examine the association of obesity with walking independent of knee pain in a large sample of participants with or at risk for knee OA. Their findings are very interesting suggesting that obesity has an important association with low levels of walking in people with or at high risk of knee OA independent of knee pain.

With regards to topic (b) of this special issue, N. Parekh and colleagues using data from over 15,000 people in the Third National Health and Examination Survey (1986–2006, USA) studied the longitudinal association between leisuretime physical activity and overall cancer mortality. The authors conclude that regular vigorous activity (>6 METS) may reduce the risk of cancer mortality in persons with normal glucose metabolism. Although the authors are not able to elaborate on the mechanism based on this data, the take-home message is that vigorous physical activity may protect from cancer mortality. N. Mirza and colleagues demonstrate that among obese Latino children living in Washington D.C., USA, those who performed 60 minutes of moderate-to-vigorous physical activity per day had lower odds of displaying insulin resistance than those who did not meet this physical activity guideline highlighting the importance of physical activity for obese children from this minority ethnic group. K. Dipla and her colleagues provide an informative review discussing the influence of

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acute bouts of exercise and exercise training on blood pressure control in obese children and adults. Their review focuses in particular on the mechanisms by which exercise can influence autonomic nervous system control of blood pressure. The study by Heydari et al. examined the effect of a 12-week high-intensity, intermittent exercise intervention (3 times/week, 20 min per session) in overweight males, who were assigned to either an intervention or control group. Aerobic power was improved and total, abdominal, and visceral fat were reduced in the intervention group but not in the control group. Despite these beneficial adaptations to training no changes occurred in the levels of insulin, blood lipids, and HOMA-IR. R. E. Lee et al. examined the relationship between sitting time and "cardiometabolic" risk factors in overweight African American women. They observed a positive association between sitting time and blood glucose but an inverse association between sitting time and blood cholesterol suggesting the need for further research to clarify the potential risks of sedentary behaviour in overweight African American women and in other groups.

Although the health benefits of increased physical activity, especially of high-intensity exercise, are widely understood it is interesting to note that overweight and obese individuals often avoid participating in vigorous exercise. Participant attitude is the main focus of the studies which belong to topic (c) of this special issue. To investigate the attitudes of overweight and obese people towards exercise, C. W. Hall et al. studied a group of overweight/obese individuals (age 26-50 y) who were asked to walk at a moderate intensity of 60% of peak aerobic power (VO2 peak), an exercise intensity considered to be cardioprotective, for 30 min. The self-selected intensity corresponded to about 54% of VO₂ peak and this was lower than the predetermined one suggesting that overweight/obese individuals might prefer this metabolic rate to exercise. Interestingly, men walked at a lower percentage of VO₂ peak than women, suggesting that exercise prescription should consider not only the physiological but also the psychological characteristics and responses of overweight and obese individuals. While much of the literature focuses on the social and environmental barriers to physical activity less is known about the individual barriers. Fox et al. and colleagues using data from a survey from a low-income minority community in the USA concluded that individual barriers correlated with lower physical activity levels.

The collective take home message of the studies presented in this issue is that structured physical activity that meets the recommendations and is intensive enough to improve aerobic fitness can improve some health risk factors including body composition and blood pressure. The use of vigorous exercise is a promising health promotion tool although it is associated with practical issues like compliance, especially in untrained and clinical populations. It seems also that regular vigorous activity has the potential to reduce the risk of cancer mortality among persons with normal insulin-glucose metabolism at least for certain populations. Perceptions and other barriers may prevent some people from adopting vigorous exercise and this should be taken into account by policy makers.

Acknowledgments

My colleagues and I had the opportunity to review some very interesting manuscripts submitted to this special issue. Of the 24 manuscripts submitted, 11 were finally accepted for publication. We would like to thank all the authors who submitted their work for consideration to this special issue of the journal.

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Research Article

Barriers to Physical Activity in East Harlem, New York

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Background. East Harlem is an epicenter of the intertwining epidemics of obesity and diabetes in New York. Physical activity is thought to prevent and control a number of chronic illnesses, including diabetes, both independently and through weight control. Using data from a survey collected on adult (age 18+) residents of East Harlem, this study evaluated whether perceptions of safety and community-identified barriers were associated with lower levels of physical activity in a diverse sample. Methods. We surveyed 300 adults in a 2-census tract area of East Harlem and took measurements of height and weight. Physical activity was measured in two ways: respondents were classified as having met the weekly recommended target of 2.5 hours of moderate physical activity (walking) per week (or not) and reporting having engaged in at least one recreational physical activity (or not). Perceived barriers were assessed through five items developed by a community advisory board and perceptions of neighborhood safety were measured through an adapted 7-item scale. Two multivariate logistic regression models with perceived barriers and concerns about neighborhood safety were modeled separately as predictors of engaging in recommended levels of exercise and recreational physical activity, controlling for respondent weight and sociodemographic characteristics. Results. The most commonly reported perceived barriers to physical activity identified by nearly half of the sample were being too tired or having little energy followed by pain with exertion and lack of time. Multivariate regression found that individuals who endorsed a greater number of perceived barriers were less likely to report having met their weekly recommended levels of physical activity and less likely to engage in recreational physical activity controlling for covariates. Concerns about neighborhood safety, though prevalent, were not associated with physical activity levels. Conclusions. Although safety concerns were prevalent in this low-income, minority community, it was individual barriers that correlated with lower physical activity levels.

1. Introduction

Diabetes prevalence for Blacks and Latinos in the US are nearly double that of Whites (11% of Blacks, 10% of Mexican Americans, and 5% of Whites have diabetes) and Blacks and Latinos have higher obesity prevalence than Whites [1, 2]. Physical activity is proven to help prevent and control diabetes, both independently, and through weight control [3].

East Harlem, New York is a predominantly low-income, Black and Latino community at the epicenter of the intertwined epidemics of obesity and diabetes. East Harlem has the highest prevalence of obesity and the highest diabetes mortality rate in New York City and its residents have insufficient resources to cope with the myriad of environmental, social, nutritional, and stress-related forces that fuel

these epidemics [4]. The built environment is believed to play a large role in generating health disparities by creating differential opportunities to engage in recreational physical activity, resulting in "obesogenic environments" [5–7]. Neighborhood safety and aesthetics including maintenance, cleanliness, and open space have been shown to influence the utilization of space for both recreational and routine physical activity [8–10]. Perceptions of neighborhood safety may be particularly salient among those residents in lower-income urban settings, where crime is more prevalent [11]. Indeed, surveys find that racial/ethnic minorities and those of lower socioeconomic status are the most likely to rate their neighborhoods as unsafe [12]. However, research to date on the relationship between perceived neighborhood safety and physical activity levels has generated mixed results,

with many studies finding no relation [13]. Indeed, in low-income neighborhoods with limited personal transportation options, routine physical activity like walking may be a necessity of life in spite of safety concerns and is the least expensive, most accessible, proven effective form of physical activity.

In addition to perceived safety, other perceived barriers might also influence physical activity levels. Research in a variety of communities including older adults [14], high school, [15] and college students [16] have assessed the degree to which perceived barriers act as cognitive constraints on physical activity behavior. A handful of studies have assessed perceived barriers in racial and ethnically diverse samples. For instance, in a study of diverse rural older women, endorsing more personal barriers was negatively associated with physical activity [17] and perceived physical environmental sets of variables were significant predictors of physical activity in a similar study [18, 19].

Few quantitative studies have assessed the relationship between perceived and physical activity levels in predominately African American and Hispanic communities [20], but qualitative research on African American women's attitudes towards physical activity suggests specific barriers that may differ from white populations. For instance, feelings about their physical appearance during physical activities and issues related to personal care after activities have been identified as factors that influence African American's attitudes toward physical activity (i.e., not wanting to mess up ones hair by sweating) [21-24]. A positive body image despite their actual weight may also lower African American women's desire to engage in physical activity for weight loss [25]. One qualitative study of physical activity in Latina women in North Carolina found that gender roles, support from family/husband, child care issues, language, and isolation in the community served as barriers to engaging in physical activity [26]. Few quantitative studies have assessed perceived barriers in predominately Hispanic populations with many first-generation immigrants such as East Harlem [27, 28]. Few, if any, studies have assessed perceived barriers in low-income men.

Using community-based participatory research methods this study evaluated perceived safety and other perceived barriers to physical activity with self-reported data from a 2census tract area of East Harlem, New York. In 2007, a new coalition, "Communities IMPACT (Inspired and Motivated to Prevent And ConTrol) Diabetes" formed. This Centers for Disease Control and Prevention (CDC)-funded Center aimed to use community-based participatory approaches to implement and disseminate interventions that build on local community resources and comprehensively combat the rising tide of diabetes and diabetes-related disparities. IMPACT leaders organized a community-academic taskforce to identify specific elements of the community, built, social, and medical environments that may be altered to foster improved diabetes prevention and control as described elsewhere [29, 30].

As suggested by partners during the grant-writing phase of the partnership, they selected a subneighborhood (two

census tracts) within East Harlem (known as the Sector of Excellence to Eliminate Disparities, or "SEED") for intensive focus. The taskforce reasoned that local concentrated efforts could harness disparate local resources and bring together diverse clinical, community, service, and religious leaders to take action. After discussing many potential interventions to enhance physical activity, healthy eating, and diabetes detection, prevention, and management, they decided they first needed to better understand local barriers to preventing and controlling diabetes. To achieve this goal, the partnership surveyed local community residents to gain insight into the ways characteristics of the neighborhood, particularly safety and perceived barriers, influence physical activity levels.

2. Methods

2.1. Survey Sample. An evaluation subcommittee of the task-force, with community and academic members, developed, piloted, and conducted the survey to assess sociodemographics, medical history, diabetes risk perception, physical activity, diet and weight, community resources, and diabetes knowledge and to gather anthropometric measures of weight and height from willing participants. Sociodemographic data was designed to be comparable to the data for these same 2 census tracts in the 2000 Census. Inclusion criteria consisted of living in the SEED, age 18 and older, and the ability to speak either English or Spanish.

In contrast with population surveys generated from random digit dialing that may undersample hard to reach lower-income residents, recruitment for the community member survey took place on the sidewalk via street intercept. Potential survey sites were selected based on the following criteria: (1) publicly accessible building or space or permission to use space obtained from building supervisor; (2) space for a semiprivate interview. At each selected site, interviewers approached individuals who appeared to be adults over the age of 18 in front of the venue and invited them to participate. We recruited and surveyed 300 willing residents of the SEED who consented to participate in the survey. Nine participants were excluded as they did not have full data to assess residency within the SEED area, thus, data for 291 individuals were included in the study.

The final sample was predominately female (62.7%), Latino (56.0%), and low income (<15,000/year) (63.0%). Nearly 70% of the sample was overweight or obese. The samples' sociodemographic characteristics are summarized in Table 1.

2.2. Measurements

2.2.1. Dependent Variable. Physical activity and diet are notoriously difficult to measure via self-report with no true gold standard. As such, we chose to limit the respondent burden in these areas and target our queries towards items most likely to provide the community coalition with targets for intervention. Physical activity was measured

Table 1: Sociodemographic characteristics of the survey respondents.

Demographics	% (N)
Female	62.8% (182)
Mean age (years), sd	42.6 ± 16.7
18–24	17.6% (51)
25–34	19.0% (55)
35–44	19.0% (55)
45–54	19.0% (55)
55–64	12.8% (37)
65+	12.8% (37)
Race	
Black/African American	30.8% (90)
Latino	55.8% (162)
White	11.7% (34)
Other	1.7% (5)
Acculturation	
Born in US	58.1% (169)
Speaks mainly English at home	63.6% (185)
Speaks mainly Spanish at home	34.7% (101)
Speaks other language at home	1.7% (5)
Education	
Less than 8th grade	12.7% (30)
Less than high school	25.3% (60)
High school/GED	39.2% (93)
At least some college	22.4% (53)
Low income (<\$15,0000/year)	62.9% (132)
Weight	
Overweight (BMI 25-29.9)	29.0% (79)
Obese (BMI 30+)	40.0% (108)
Overweight or Obese (BMI 25+)	69.0% (187)

using 4 self-report items assessing average daily and weekly walking time and recreational activities modified from the International Physical Activity Questionnaire (IPAQ) [31]. To capture both routine physical activity and physical activity specifically for recreation, we generated and assessed two separate dichotomous outcome variables. First, we divided respondents between those who met the CDC's recommendation of engaging in at least 2.5 hours per week of moderate physical activity (see: http://www.cdc.gov/ physicalactivity/everyone/guidelines/adults.html), in this case, those who reported walking for at least 2.5 hours per week or approximately 20 minutes a day and those who did not meet this standard. Second, to capture recreational physical activity, we asked respondents "What do you like to do to be physically active?" For respondents reporting a physical activity other than walking (specifically running/jogging, bicycling, playing a team sport and going to the gym), we coded them as engaging in a leisure time physical activity. Respondents could choose more than one category, but respondents were coded according to whether they participated in a leisure time physical activity at all, not according to how many.

2.2.2. Perceived Safety and Barriers to Physical Activity. Sixitem dichotomous measures of perceived barriers to physical activity were developed with input from the community coalition concerning perceptions of barriers to physical activity believed to be widely held within the community and are summarized in Table 4. Respondents were asked whether or not they agreed with each of the six barriers (Yes/No). The barriers were then summed to create a composite measure of the number of barriers endorsed. In addition, a battery of six items was asked about perceptions of neighborhood safety in relation to neighborhood social capital. This measure was adapted from a measure used in a previous study of perceived neighborhood safety and physical activity [11]. Respondents were asked to rate how safe from crime they considered their neighborhood to be and to rate how safe they feel in a series of locations and situations (e.g., walking alone on a street in your neighborhood at night/day; taking the public bus during the night/day) using a 4-point likert scale. We generated a composite measure of neighborhood safety by dichotomizing the individual questions into generally perceived safe/unsafe categories and then summing the results.

2.2.3. Controls. Because one's excessive weight can affect perceptions of barriers to physical activity including pain with exertion [32], we controlled for participants current body mass index (coded as overweight or obese from our anthropometric measures of height and weight). In addition, current weight-loss efforts were measured using self-report including whether individuals were using physical activity or exercise to lose weight or maintain their current weight. We controlled for a variety of additional demographic factors: sex, age, race/ethnicity, education, and acculturation (if respondents speak Spanish at home and if they were born in the US).

2.3. Analysis. Two multivariate logistic regression models with perceived barriers and concerns about neighborhood safety were modeled separately as predictors of engaging in recommended levels of exercise and recreational physical activity, controlling for respondent weight and sociodemographic characteristics. We first modeled the relationship between the number of perceived barriers to physical activity and reported physical activity levels (time spent walking and participation in recreational physical activity apart from walking), controlling for overweight/obese, age, race/ethnicity, gender, acculturation, and whether or not individuals were trying to lose weight. We then ran the same model with neighborhood safety as the main predictor variable. We stratified the analysis by race/ethnicity and gender to detect group specific differences.

3. Results

With regards to self-reported physical activity, at a median of 7 hours per week or about 1 hour per day, respondents to the survey reported walking a great deal. The overwhelming majority of the sample (80.0%) met the weekly

Table 2: Physical activity (self-report).

Hours per week spent walking	7.0 (median), 12.5 (sd)	
Hours per week spent walking (men) 14.0 (median), 10.8		
Hours per week spent walking (women)	5.25 (median), 14.2 (sd)	
% meeting recommendation of 2.5 hrs of moderate physical activity/wk	80.0% (89.0% men, 75.0% women)	
Physical Activity (what things do you like to do to be physically active?)		
% reporting some physical activity apart from walking	72.0% (80.0% male, 67.0% female)	
% walking	65.2%	
% cycling	20.6%	
% sports	17.9%	
% dance	14.1%	
% going to the gym	13.4%	
% running	11.7%	

recommendation of at least 2.5 hours of moderate intensity exercise, 88.0% of men and 75.0% of women. In regards to recreational physical activity, walking was also the most commonly reported physical activity that respondents liked to do to be physically active (65.2%). Apart from walking, 72.0% of the sample reported engaging in at least one other form of recreational physical activity including cycling (20.6%), playing sports (17.9%), dancing (14.1%), going to the gym (13.4%), and running (11.7%) (Table 2).

While there were many self-reported barriers to physical activity, the most commonly reported was being too tired or having little energy (45%). Pain with exercise (35%), lack of time (30%), dislike of exercise (30%), and difficulty being active (28%) were also commonly identified barriers. Lack of safe spaces for exercise was a relatively less commonly cited barrier, reported by only 20% of the sample, despite nearly half those surveyed (45%) considering the neighborhood to be somewhat or very unsafe (Table 3).

Multivariate regression of perceived barriers found that reporting a greater number of perceived barriers was associated with a lower odds of meeting the weekly recommendation of 2.5 hours of moderate exercise (OR = 0.72, P <0.01). Additionally, individuals who perceived more barriers to physical activity were less likely to report engaging in some form of recreational physical activity other than walking (OR = 0.72, P < 0.01). Perceived safety of the neighborhood, on the other hand, was not associated with significantly lower amounts of time spent walking or engaging in recreational physical activity (Table 4). Men spent more time walking and were more likely to engage in leisure time physical activity compared with women, although safety concerns were not a significant barrier to physical activity for either men or women (analysis not shown). Overall, stratified analysis between men and women and Hispanic and Black participants did not affect the direction or significance of the results (analysis not shown).

4. Discussion

In disadvantaged neighborhoods such as East Harlem, characteristics of the built and social environment, such as perceived safety, are often suggested as the greatest obstacles to increasing physical activity [5–13]. Our results did not support the supposition that concerns about safety serve as a primary barrier to recreational and routine physical activity. Nearly half of the sample perceived their neighborhood as very or somewhat unsafe. Yet, safety concerns were reported as serving as a barrier to physical activity by only 20% of the sample and believing your neighborhood was unsafe was not associated with physical activity levels. The most commonly reported barriers to physical activity identified by nearly half of the sample were motivational—being too tired or having little energy. Lack of time and pain with exertion were also commonly cited barriers and multivariate regression found that endorsing a greater number of individual barriers was associated with reduced time spent walking and engaging in physical activity for sport controlling for covariates.

This finding contrasts with other studies that have found support for the role of perceptions of neighborhood safety in predicting physical activity levels. A study from Boston, for instance, that used a similar measure of perceived neighborhood safety found that female residents of neighborhoods with lower perceived safety at night walked less than similar women in neighborhoods perceived as safer [11]. The effect was only found for women and not for men [11]. Other studies have found mixed results with regards to the role of safety in reducing physical activity levels. Some studies have found a significant inverse association between perceived neighborhood safety and physical activity levels [33–41], but others have found no significant association [42–52].

Another striking finding from this study was the percent of individuals that met the recommendation for moderate-intensity exercise of 2.5 hrs/wk: 80% of the sample met this recommendation through walking alone. Moreover, the median of 7 hours per week of walking was well above the recommended amount and translates into an hour a day of moderate intensity exercise. This contrasts with studies from other parts of the country finding low levels of physical activity in predominately Latino communities. For instance, a study of Latina women in Chicago found much lower rates of physical activity, with only 36% meeting current recommendations for moderate or vigorous physical activity [27]. Although it is possible that respondents exaggerated the actual amount of walking per day, it is also plausible

Table 3: Perceived barriers to physical activity and neighborhood safety.

Perceived Barriers	% Responded yes			
You are too tired or do not have the energy	44.5			
It hurts to exercise	34.5			
You do not have the time	30.4			
You do not like exercising	30.0			
It is difficult to be physically active	28.0			
You do not have a safe place to exercise	20.3			
Mean number of perceived barriers to exercise	2			
Neighborhood safety	% Reporting very or somewhat unsafe			
How safe from crime do you consider your neighborhood to be? 45.3				
Please rate how safe you feel in each of the following locations or situations:				
Walking alone on a street in your neighborhood at night	41.3			
Taking the train/subway at night	46.9			
Taking the public bus at night	35.5			
In a park or playground during the day	17.5			
Taking the train/subway during the day	11.6			
Taking the public bus during the day	3.5			

Table 4: Perceived barriers and safety concerns and engagement in routine and recreational physical activity.

Variables	(1)	(2)	(1)	(2)
variables	Walk 2.5 hrs/wk	Walk 2.5 hrs/wk	Engage in recreational PA	Engage in recreational PA
Perceived PA barriers (count)	0.72*** (0.087)		0.72*** (0.078)	
Perceived neighborhood safety		0.99 (0.096)		1.01 (0.089)
Normal/under weight	0	0	0	0
Overweight	0.58 (0.293)	0.77 (0.402)	1.46 (0.652)	1.83 (0.865)
Obese	1.18 (0.615)	1.28 (0.700)	0.70 (0.307)	0.88 (0.413)
Age 18–35	0	0	0	0
Age 36–55	1.01 (0.016)	1.01 (0.016)	1.02 (0.015)	1.01 (0.015)
Age 56–85	0.72 (0.328)	0.75 (0.358)	0.66 (0.269)	0.54 (0.241)
Male	2.34* (1.056)	2.04 (0.919)	2.27** (0.874)	2.73** (1.110)
Education (high school degree+)	0.81 (0.134)	0.94 (0.160)	1.28 (0.205)	1.51** (0.265)
Race/ethnicity—other	0	0	0	0
Race/ethnicity black	0.71 (0.451)	1.08 (0.642)	0.91 (0.549)	1.06 (0.640)
Race/ethnicity—hispanic	1.43 (0.908)	2.34 (1.443)	0.74 (0.445)	0.80 (0.477)
Speak Spanish at home	0.60 (0.356)	0.64 (0.378)	1.23 (0.633)	1.06 (0.563)
US born	1.27 (0.702)	1.31 (0.707)	1.19 (0.593)	0.97 (0.493)
Trying to lose weight	0.56 (0.234)	0.42* (0.195)	1.86 (0.725)	1.69 (0.707)
Constant	15.22** (16.787)	3.56 (3.698)	1.45 (1.426)	0.49 (0.486)
Observations	248	221	251	224

Standard error in parentheses

that low-income areas of New York are more walkable than low-income areas in other cities. Alternatively, respondents, particularly men, may have active jobs that require a great deal of walking. In addition to walking, 72.0% of the sample reported engaging in some other form of physical activity including team sports, cycling, dancing, running, and going to the gym.

Collectively, these findings contradict typical expectations regarding the need for unique interventions in socially deprived neighborhoods like East Harlem and provides a rich foundation to launch community-based interventions that focus on addressing motivational barriers. In the face of these cognitive barriers, changes to the built environment may go unexploited to physical activity. Given the strong preference for walking, these findings suggest that community-level efforts need to support ways for building activity into daily routines so they are not perceived as adding time to the day and requiring tremendous new sources of energy and motivation. For example, rather than designing a walking path or indoor walking trail in a random location in

^{***}P < 0.01, **P < 0.05, *P < 0.1.

the neighborhood, efforts may be more successful if that path is mapped along a highly used route such as from a central or high traffic bus stop to a housing project or to a supermarket. Furthermore, markers of distance or calories expended could be added to reinforce a sense of accomplishment to community residents as they walk these routes and to encourage more frequent use of them. In addition, efforts should be made to reinforce the notion that engaging in regular, routine exercise can increase energy levels and reduce fatigue.

Although routine exercise was prevalent, so was overweight/obesity. Nearly 70% of the sample was either overweight or obese based on our anthropometric measures. This suggests that routine exercise, even in high dosages, may not be adequate to stave off obesity in the absence of changes in the food environment.

Despite the rich data afforded by this unique community resident assessment, several limitations should be noted. Although substantial efforts were made to replicate a random sample through random selection of pre-selected block faces where recruitment was most feasible, the final sample was ultimately a convenience sample of willing residents that may not be representative of the community as a whole. The use of self-reported physical activity behaviors is inherently subject to significant recall and social desirability biases. The generalizability and comparability of results from this survey are limited by the design, which focused on a single, disadvantaged neighborhood. The findings with regards to the role of perceptions of safety and barriers to physical activity, therefore, pertain to differences among individuals within a single neighborhood rather than between neighborhoods.

5. Conclusions

The objective of this study was to assess levels and types of physical activity in an area with high rates of obesity and diabetes and to analyze determinants of physical activity. East Harlem is an active community, though one with high rates of obesity. Although safety concerns are often believed to deter physical activity in low-income, urban settings we did find this in East Harlem. Rather, more quotidian concerns were associated with lower physical activity levels.

Conflict of Interests

The authors declare no conflict of interests.

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References

- [1] C. C. Cowie, K. F. Rust, D. D. Byrd-Holt et al., "Prevalence of diabetes and impaired fasting glucose in adults in the U.S. population: National Health and Nutrition Examination Survey 1999–2002," *Diabetes Care*, vol. 29, no. 6, pp. 1263–1268, 2006.
- [2] A. V. Diez-Roux, F. J. Nieto, L. Caulfield, H. A. Tyroler, R. L. Watson, and M. Szklo, "Neighbourhood differences in diet: the Atherosclerosis Risk in Communities (ARIC) study," *Journal of Epidemiology and Community Health*, vol. 53, no. 1, pp. 55–63, 1999.
- [3] R. J. Sigal, G. P. Kenny, D. H. Wasserman, C. Castaneda-Sceppa, and R. D. White, "Physical activity/exercise and type 2 diabetes: a consensus statement from the American Diabetes Association," *Diabetes Care*, vol. 29, no. 6, pp. 1433–1438, 2006
- [4] The City of New York, "Community Health Survey," *Epi-query*, 2012, http://www.nyc.gov/html/doh/html/community/community.shtml.
- [5] R. E. Walker and I. Kawachi, "Race, ethnicity and obesity," in The Oxford Handbook of the Social Science of Obesity, J. Cawley, Ed., pp. 257–275, Oxford University Press, 2011.
- [6] K. M. Booth, M. M. Pinkston, and W. S. C. Poston, "Obesity and the built environment," *Journal of the American Dietetic Association*, vol. 105, no. 5, pp. S110–S117, 2005.
- [7] J. F. Sallis, M. A. Adams, and D. Ding, "Physical activity and the built environment," in *The Oxford Handbook of the Social Science of Obesity*, J. Cawley, Ed., pp. 433–451, Oxford University Press, 2011.
- [8] B. E. Molnar, S. L. Gortmaker, F. C. Bull, and S. L. Buka, "Unsafe to play? Neighborhood disorder and lack of safety predict reduced physical activity among urban children and adolescents," *American Journal of Health Promotion*, vol. 18, no. 5, pp. 378–386, 2004.
- [9] M. S. Mujahid, A. V. D. Roux, M. Shen et al., "Relation between neighborhood environments and obesity in the multi-ethnic study of atherosclerosis," *American Journal of Epidemiology*, vol. 167, no. 11, pp. 1349–1357, 2008.
- [10] D. A. Cohen, T. A. Farley, and K. Mason, "Why is poverty unhealthy? Social and physical mediators," *Social Science and Medicine*, vol. 57, no. 9, pp. 1631–1641, 2003.
- [11] G. G. Bennett, L. H. McNeill, K. Y. Wolin, D. T. Duncan, E. Puleo, and K. M. Emmons, "Safe to walk? Neighborhood safety and physical activity among public housing residents," *PLoS Medicine*, vol. 4, no. 10, pp. 1599–1607, 2007.
- [12] D. K. Wilson, K. A. Kirtland, B. E. Ainsworth, and C. L. Addy, "Socioeconomic status and perceptions of access and safety for physical activity," *Annals of Behavioral Medicine*, vol. 28, no. 1, pp. 20–28, 2004.
- [13] N. Humpel, N. Owen, and E. Leslie, "Environmental factors associated with adults' participation in physical activity. A review," *American Journal of Preventive Medicine*, vol. 22, no. 3, pp. 188–199, 2002.

[14] M. L. Booth, N. Owen, A. Bauman, O. Clavisi, and E. Leslie, "Social-cognitive and perceived environment influences associated with physical activity in older Australians," *Preventive Medicine*, vol. 31, no. 1, pp. 15–22, 2000.

- [15] K. R. Allison, J. J. M. Dwyer, and S. Makin, "Perceived barriers to physical activity among high school students," *Preventive Medicine*, vol. 28, no. 6, pp. 608–615, 1999.
- [16] S. Wilcox, C. Castro, A. C. King, R. Housemann, and R. C. Brownson, "Determinants of leisure time physical activity in rural compared with urban older and ethnically diverse women in the United States," *Journal of Epidemiology and Community Health*, vol. 54, no. 9, pp. 667–672, 2000.
- [17] S. Wilcox, M. Bopp, L. Oberrecht, S. K. Kammermann, and C. T. McElmurray, "Psychosocial and perceived environmental correlates of physical activity in rural and older African American and white women," *Journals of Gerontology*, vol. 58, no. 6, pp. P329–P337, 2003.
- [18] A. Daskapan, E. H. Tuzun, and L. Eker, "Perceived barriers to physical activity in university students," *Journal of Sports Science and Medicine*, vol. 5, no. 4, pp. 615–620, 2006.
- [19] S. G. Trost, N. Owen, A. E. Bauman, J. F. Sallis, and W. Brown, "Correlates of adults' participation in physical activity: review and update," *Medicine and Science in Sports and Exercise*, vol. 34, no. 12, pp. 1996–2001, 2002.
- [20] A. E. Eyler, S. Wilcox, D. Matson-Koffman et al., "Correlates of physical activity among women from diverse racial/ethnic groups," *Journal of Women's Health and Gender-Based Medicine*, vol. 11, no. 3, pp. 239–253, 2002.
- [21] K. A. Henderson and B. E. Ainsworth, "A synthesis of perceptions about physical activity among older African American and American Indian women," *American Journal of Public Health*, vol. 93, no. 2, pp. 313–317, 2003.
- [22] A. C. King, C. Castro, S. Wilcox, A. A. Eyler, J. F. Sallis, and R. C. Brownson, "Personal and environmental factors associated with physical inactivity among different racial—ethnic groups of U.S. middle-aged and older-aged women," *Health Psychology*, vol. 19, no. 4, pp. 354–364, 2000.
- [23] G. R. Dutton, J. Johnson, D. Whitehead, J. S. Bodenlos, and P. J. Brantley, "Barriers to physical activity among predominantly low-income African-American patients with type 2 diabetes," *Diabetes Care*, vol. 28, no. 5, pp. 1209–1210, 2005.
- [24] I. R. Mabry, D. R. Young, L. A. Cooper, T. Meyers, A. Joffe, and A. K. Duggan, "Physical activity attitudes of African American and White adolescent girls," *Ambulatory Pediatrics*, vol. 3, pp. 312–316, 2003.
- [25] K. R. Evenson, O. L. Sarmiento, M. L. Macon, K. W. Tawney, and A. S. Ammerman, "Environmental, policy, and cultural factors related to physical activity among Latina immigrants," *Women and Health*, vol. 36, no. 2, pp. 43–57, 2002.
- [26] J. Wilbur, P. J. Chandler, B. Dancy, and H. Lee, "Correlates of physical activity in urban Midwestern Latinas," *American Journal of Preventive Medicine*, vol. 25, no. 3, pp. 69–76, 2003.
- [27] E. A. Walker, A. Caban, C. B. Schechter et al., "Measuring comparative risk perceptions in an urban minority population: the risk perception survey for diabetes," *Diabetes Educator*, vol. 33, no. 1, pp. 103–110, 2007.
- [28] E. P. Cromley, L. Kleinman, M. Ramos et al., "A community-engaged approach to select geographic areas for interventions to reduce health disparities," *Progress in Community Health Partnerships: Research, Education, and Action*, vol. 5, no. 3, pp. 299–305, 2011.

[29] L. Kleinman, D. Lutz, E. Plumb et al., "A partnered approach for structured observation to assess the environment of a neighborhood with high diabetes rates," *Progress in Community Health Partnerships: Research, Education and Action*, vol. 5, no. 3, pp. 249–259, 2011.

- [30] B. E. Ainsworth, C. A. Macera, D. A. Jones et al., "Comparison of the 2001 BRFSS and the IPAQ physical activity questionnaires," *Medicine and Science in Sports and Exercise*, vol. 38, no. 9, pp. 1584–1592, 2006.
- [31] P. Ekkekakis, E. Lind, and S. Vazou, "Affective responses to increasing levels of exercise intensity in normal-weight, overweight, and obese middle-aged women," *Obesity*, vol. 18, no. 1, pp. 79–85, 2010.
- [32] R. A. Harrison, I. Gemmell, and R. F. Heller, "The population effect of crime and neighbourhood on physical activity: an analysis of 15 461 adults," *Journal of Epidemiology and Community Health*, vol. 61, no. 1, pp. 34–39, 2007.
- [33] F. N. Piro, Ø. Nœss, and B. Claussen, "Physical activity among elderly people in a city population: the influence of neighbourhood level violence and self perceived safety," *Journal of Epidemiology and Community Health*, vol. 60, no. 7, pp. 626–632, 2006.
- [34] C. M. Hoehner, L. K. Brennan Ramirez, M. B. Elliott, S. L. Handy, and R. C. Brownson, "Perceived and objective environmental measures and physical activity among urban adults," *American Journal of Preventive Medicine*, vol. 28, no. 2, pp. 105–116, 2005.
- [35] S. P. Hooker, D. K. Wilson, S. F. Griffin, and B. E. Ainsworth, "Perceptions of environmental supports for physical activity in African American and white adults in a rural county in South Carolina," *Preventing Chronic Disease*, vol. 2, no. 4, article A11, 2005.
- [36] F. Li, K. J. Fisher, A. Bauman et al., "Neighborhood influences on physical activity in middle-aged and older adults: a multilevel perspective," *Journal of Aging and Physical Activity*, vol. 13, no. 1, pp. 87–114, 2005.
- [37] R. R. Suminski, W. S. C. Poston, R. L. Petosa, E. Stevens, and L. M. Katzenmoyer, "Features of the neighborhood environment and walking by U.S. adults," *American Journal of Preventive Medicine*, vol. 28, no. 2, pp. 149–155, 2005.
- [38] N. Humpel, N. Owen, D. Iverson, E. Leslie, and A. Bauman, "Perceived environment attributes, residential location, and walking for particular purposes," *American Journal of Preventive Medicine*, vol. 26, no. 2, pp. 119–125, 2004.
- [39] J. L. Thompson, V. K. Wolfe, N. Wilson, M. N. Pardilla, and G. Perez, "Personal, social, and environmental correlates of physical activity in Native American women," *American Journal of Preventive Medicine*, vol. 25, no. 3, pp. 53–60, 2003.
- [40] J. Wilbur, P. J. Chandler, B. Dancy, and H. Lee, "Correlates of physical activity in urban Midwestern Latinas," *American Journal of Preventive Medicine*, vol. 25, no. 3, pp. 69–76, 2003.
- [41] J. Kuo, C. C. Voorhees, J. A. Haythornthwaite, and D. R. Young, "Associations between family support, family intimacy, and neighborhood violence and physical activity in urban adolescent girls," *American Journal of Public Health*, vol. 97, no. 1, pp. 101–103, 2007.
- [42] R. W. Motl, R. K. Dishman, R. P. Saunders, M. Dowda, and R. R. Pate, "Perceptions of physical and social environment variables and self-efficacy as correlates of self-reported physical activity among adolescent girls," *Journal of Pediatric Psychology*, vol. 32, no. 1, pp. 6–12, 2007.
- [43] H. L. Burdette and R. C. Whitaker, "A national study of neighborhood safety, outdoor play, television viewing, and

- obesity in preschool children," *Pediatrics*, vol. 116, no. 3, pp. 657–662, 2005.
- [44] K. Lim and L. Taylor, "Factors associated with physical activity among older people—a population-based study," *Preventive Medicine*, vol. 40, no. 1, pp. 33–40, 2005.
- [45] J. Mota, M. Almeida, P. Santos, and J. C. Ribeiro, "Perceived Neighborhood Environments and physical activity in adolescents," *Preventive Medicine*, vol. 41, no. 5-6, pp. 834–836, 2005.
- [46] R. W. Motl, R. K. Dishman, D. S. Ward et al., "Perceived physical environment and physical activity across one year among adolescent girls: self-efficacy as a possible mediator?" *Journal of Adolescent Health*, vol. 37, no. 5, pp. 403–408, 2005.
- [47] G. J. Norman, B. A. Schmid, J. F. Sallis, K. J. Calfas, and K. Patrick, "Psychosocial and environmental correlates of adolescent sedentary behaviors," *Pediatrics*, vol. 116, no. 4, pp. 908–916, 2005.
- [48] S. Adkins, N. E. Sherwood, M. Story, and M. Davis, "Physical activity among African-American girls: the role of parents and the home environment," *Obesity research*, vol. 12, pp. S38–S45, 2004.
- [49] B. E. Ainsworth, S. Wilcox, W. W. Thompson, D. L. Richter, and K. A. Henderson, "Personal, social, and physical environmental correlates of physical activity in African-American women in South Carolina," *American Journal of Preventive Medicine*, vol. 25, no. 3, pp. S23–S29, 2003.
- [50] D. R. Young and C. C. Voorhees, "Personal, social, and environmental correlates of physical activity in urban African-American women," *American Journal of Preventive Medicine*, vol. 25, no. 3, pp. S38–S44, 2003.
- [51] B. K. Sanderson, H. R. Foushee, V. Bittner et al., "Personal, social, and physical environmental correlates of physical activity in rural African-American women in Alabama," *American Journal of Preventive Medicine*, vol. 25, no. 3, pp. S30–S37, 2003.
- [52] K. R. Evenson, O. L. Sarmiento, K. W. Tawney, M. L. Macon, and A. S. Ammerman, "Personal, social, and environmental correlates of physical activity in North Carolina Latina immigrants," *American Journal of Preventive Medicine*, vol. 25, no. 3, pp. S77–S85, 2003.

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Research Article

The Effect of High-Intensity Intermittent Exercise on Body Composition of Overweight Young Males

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To determine the effect of a 12-week high intensity intermittent exercise (HIIE) intervention on total body, abdominal, trunk, visceral fat mass, and fat free mass of young overweight males. Participants were randomly assigned to either exercise or control group. The intervention group received HIIE three times per week, 20 min per session, for 12 weeks. Aerobic power improved significantly (P < 0.001) by 15% for the exercising group. Exercisers compared to controls experienced significant weight loss of 1.5 kg (P < 0.005) and a significant reduction in total fat mass of 2 kg (P < 0.001). Abdominal and trunk adiposity was also significantly reduced in the exercising group by 0.1 kg (P < 0.05) and 1.5 kg (P < 0.001). Also the exercise group had a significant (P < 0.01) 17% reduction in visceral fat after 12 weeks of HIIE, whereas waist circumference was significantly decreased by week six (P < 0.001). Fat free mass was significantly increased (P < 0.05) in the exercising group by 0.4 kg for the leg and 0.7 kg for the trunk. No significant change (P > 0.05) occurred in levels of insulin, HOMA-IR, and blood lipids. Twelve weeks of HIIE resulted in significant reductions in total, abdominal, trunk, and visceral fat and significant increases in fat free mass and aerobic power.

1. Introduction

Obesity levels continue to increase in both developed and developing countries [1]. As being overweight is associated with numerous health problems, effective fat loss strategies are required [2]. Although dieting has been the major fat loss method, aerobic exercise programs have been shown to increase cardiorespiratory fitness [3] and preserve fat-free mass [4]. Most aerobic exercise interventions have consisted of moderate-intensity steady-state exercise, for about 30 to 40 min for 3 to 4 days per week, over a four- to six-month period. Disappointingly, these kinds of exercise programs have resulted in minimal fat loss [5, 6].

In contrast, high-intensity intermittent exercise (HIIE) has been shown to result in greater fat loss [7]. For example, Trapp et al. [8] conducted a HIIE program in young women for 15 weeks with three 20 min sessions per week. HIIE consisted of an 8 s sprint followed by 12 s of low intensity cycling, repeated for 20 min. Another group of women carried out an aerobic cycling protocol for 40 min each session. Results

showed that women in the HIIE group lost 2.5 kg of subcutaneous fat, whereas no change occurred with steady state aerobic exercise. Fat loss accruing through 15 weeks of HIIE was attained with 50% less exercise time commitment and a similar energy expenditure to that of steady-state exercise. Importantly, the women in this study also showed a significant 0.6 kg increase in fat-free mass (FFM) after HIIE, whereas FFM of the steady state exercise group was unchanged. The lack of increase in FFM accompanying steady-state exercise is in agreement with prior research in this area [9].

With regard to abdominal fat, 15 weeks of HIIE led to a 0.15 kg reduction of fat in previously untrained young women [8]. As women in this study possessed moderate levels of abdominal fat it is feasible that the greater abdominal, trunk, and visceral fat of men may show greater reductions after exposure to HIIE. For example, Boudou et al. [10] studied older type 2 diabetic males and found that after 8 weeks of HIIE, abdominal adiposity was decreased by 44%. Whether regular HIIE will also reduce the abdominal and visceral fat of young nondiabetic but overweight males is undetermined.

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Therefore, the purpose of this study was to examine the effects of 20 min bouts of HIIE, repeated three times weekly for 12 weeks, on body composition of overweight males. It was hypothesized that HIIE would result in significant reductions in total abdominal, trunk, and visceral fat and a significant increase in fat-free mass and aerobic power.

2. Subjects and Methods

- 2.1. Subjects. Forty-six inactive, overweight men were recruited from a university population and randomly allocated into either exercise (n=25) or control groups (n=21). The exercisers and controls were similar in terms of age (24.7 ± 4.8 and 25.1 ± 3.9 years) and body mass index (BMI: 28.4 ± 0.5 and 29 ± 0.9 kg m $^{-2}$). The study received approval from a University Research Ethics Committee. Forty-six subjects underwent initial testing, however, for various reasons five withdrew from the exercise group and three from the control group. There was no significant difference for any variable between the nonadherents and those males who completed the study.
- 2.2. Procedures. Subjects were advised to avoid strenuous activity and caffeine consumption for 24 hours prior to testing, and attended the laboratory after a 10-hour overnight fast. Tests for all subjects in control and exercise groups were completed at the same time of day. The Physical Activity Readiness Questionnaire [11] was filled out and information on subjects' personal and familial medical history obtained. Fasting blood (300 mL) was drawn at baseline, and at weeks 3, 6, and 12 from an antecubital vein in EDTA vacutainers. An automated enzymatic method (Cholestech LDX, USA) was applied to quantify blood lipid profiles and glucose concentrations from whole blood. The remaining whole blood in EDTA tubes was spun immediately in a chilled centrifuge (Model Megafuge 1.0R, Heraeus, Germany) at 4°C and frozen at −86°C for later analysis. Aerobic power was assessed using a TrueMax 2400 Metabolic Cart (ParvoMedics Inc, USA) and an electronically braked cycle ergometer, Monark 869 (Monark, Sweden). For subjects who could not achieve the criteria for $\dot{V}O_{2max}$, due to the strenuous nature of the exercise session $\dot{V}O_{2peak}$ was used as an indicant of aerobic power.
- 2.3. Resting Metabolic Rate (RMR). Fasted subjects relaxed in a reclined position for 30 minutes. Resting heart rate, resting energy expenditure (REE), $\dot{V}O_2$, and $\dot{V}CO_2$ were assessed using a metabolic cart (TrueMax 2400 Metabolic Cart, ParvoMedics Inc, USA).

 $\dot{V}O_2$ represents the rate of oxygen utilised by subjects during exercise, whereas $\dot{V}CO_2$ represents the rate of carbon dioxide exhaled. Subjects were advised not to sleep and breathe naturally during testing. The first 10 minutes of data

collection were excluded from analysis to allow for subject stabilization.

- 2.4. Diet. Subjects in both exercise and control groups were advised to maintain their normal eating habits during the study. On their first and last visit to the laboratory subjects provided a 3-day diet inventory which was analyzed using diet analysis software (SERVE Nutrition Management Systems, Professional Edition, version 5, Australia).
- 2.5. Body Composition. A Dual Energy X-Ray Absorptiometry (DEXA) scan with a Lunar Prodigy scanner (software version 7.51, GE Corporation, USA) was used to measure body mass and percentage body fat. Fat mass (FM) along with FFM in kg was measured for the whole body. DEXA also provided information on abdominal and trunk fat, as indicators of central adiposity. Computerised tomography (CT) scans (Philips Gemini GXL 16, the Netherlands) were also used to measure abdominal and visceral fat distribution. Axial slices $(3 \times 10 \,\mathrm{mm})$ were performed through the abdomen at L2/L3 and L4/L5. Fat density of 0.9 mg/L was assumed [12], and it was automatically selected at any tissue between 150 to 50 Hounsfield Units (HU). Gemini software (GXL Host system) was used to analyse the CT images. Abdominal, visceral, and subcutaneous fat were determined at the levels of L2/L3 and L4/L5. BMI was calculated by dividing weight by height squared (kg m^{-2}).
- 2.6. High-Intensity Intermittent Exercise Training. Subjects in the exercise group completed supervised exercise (8 s sprint, 12 s recovery) continuously throughout each 20-min session. The HIIE workload was set at 80–90% of each subject's heart rate (HR) peak at a cadence between 120 and 130 r.p.m and recovery was set at the same amount of resistance but at a cadence of 40 r.p.m. Subjects were instructed to keep their exercise intensity at a level necessary to produce a HR between 80-90% of HR peak. As subjects adapted to HIIE training, workload was increased so HR stayed at the appropriate 80-90% HR peak level. HIIE was coordinated with a prerecorded compact disc counting down each sprint in a 3-2-1 manner. Subjects performed a 5-min warm-up and cooldown on the bike prior to and after each exercise session. All training cycling data included continuous recording of HR and r.p.m, whereas rating of perceived exertion [13] (RPE) was assessed at 5-min intervals.
- 2.7. Assays. Insulin was measured using commercially available ELISA immunoassay kits. The degree of enzymatic turnover of the substrate was determined by dual wavelength absorbance measurement at 450 and 620 nm (Dako K6219, Denmark). HOMA-IR, an insulin resistance index [14], was calculated as follows:

TABLE 1: Change in body composition, aerobic power, resting heart rate, RQ, resting energy expenditure, carbohydrate, and fat oxidation for
the high-intensity intermittent exercise and no exercise control group $(N = 38; mean and standard error)$.

	Exercise		Control	
	Pre*	Post	Pre*	Post
Weight (kg)	87.8 ± 2.7	86.3 ± 2.7**	89 ± 2.9	89.4 ± 3.1
BMI $(kg m^{-2})$	28.4 ± 0.5	$27.9 \pm 0.5**$	29 ± 0.9	29.1 ± 0.9
Waist circumference (cm)	93.3 ± 1.4	$89.8 \pm 1.4^{**}$	93.7 ± 1.9	95.1 ± 1.9
Fat mass (kg)	29.8 ± 1.6	$27.8 \pm 1.5^{**}$	31.7 ± 2.2	31.8 ± 2.3
% Fat mass	34.8 ± 1.1	$32.8 \pm 1.1**$	36.3 ± 1.4	36.0 ± 1.5
Fat-free mass (kg)	54.3 ± 1.5	$55.5 \pm 1.4**$	53.8 ± 1.3	54.2 ± 1.3
$\dot{V}O_{2peak}~(l~min^{-1})$	3.0 ± 0.1	$3.4 \pm 0.1^{**}$	2.6 ± 0.1	2.7 ± 0.1
$\dot{V}O_{2peak}~(mLkg^{-1}min^{-1})$	34.2 ± 1.0	$39.4 \pm 0.8**$	29.1 ± 1.3	30.6 ± 1.4
Work output (watts)	246.3 ± 8.1	$289.8 \pm 8.0**$	224.4 ± 7.3	225.9 ± 6.3
HR (bpm)	62.2 ± 2.5	$57.9 \pm 1.8^{**}$	62.7 ± 2.0	63.7 ± 1.7
RQ	0.85 ± 0.01	$0.83 \pm 0.01^{**}$	0.82 ± 0.02	0.86 ± 0.01
REE (Kcal/day)	1793 ± 54	1841 ± 56	1788 ± 58	1794 ± 53
Carbohydrate oxidation (g/day)	232.6 ± 14.3	$201.5 \pm 13.1**$	186.7 ± 22.3	252.1 ± 21.2
Fat oxidation (g/day)	93.8 ± 6.6	$106.1 \pm 6.5^{**}$	110.2 ± 10.0	82.0 ± 10.9

^{*}Pre vales were used as covariates for ANCOVA.

2.8. Statistical Analysis. Data were analysed with the Statistical Package for Social Science for Windows software (SPSS 18, USA). To examine changes after the intervention, an analysis of covariance (ANCOVA) was used to evaluate differences between the two groups for variables that did not violate ANCOVA assumptions. Preintervention values were used as covariates. Where assumptions were violated, an independent *t*-test was conducted on the difference scores. The statistical analysis was considered significant when the probability level was less than 0.05.

3. Results

There was no significant difference between the two groups for body mass, BMI (Table 1), and age prior to the training program.

- 3.1. Exercise Heart Rates, RPE, and Work Load. The average HR during the HIIE training sessions for the exercise group was 160 ± 9 beats min⁻¹ which corresponded to 88% of HR peak and the average RPE was 13.6 ± 0.5 . Maximal work load significantly increased in the exercise group (P < 0.001) by 43.5 watts (Table 1).
- 3.2. Response in Aerobic Power following the Intervention. HIIE resulted in a significant increase in both absolute and relative $\dot{V}O_{2peak}$ (P < 0.005) with absolute $\dot{V}O_{2peak}$ being increased by 13% and relative $\dot{V}O_{2peak}$ by 15% (Table 1).
- 3.3. Total Body Mass and Body Fat Assessed by DEXA. Total body mass significantly decreased (P < 0.005) in the exercise group (Table 1) by 1.5 kg (2%), whereas total

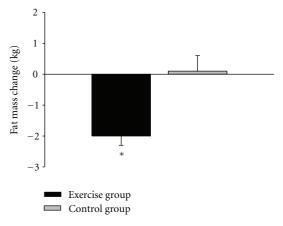


FIGURE 1: Total fat change for the high-intensity intermittent exercise and no exercise control groups (N = 38, mean and standard error). *Significantly different from control group (P < 0.05).

FM significantly decreased (P < 0.005) by 2.0 kg (6.7%; Figure 1). The FM of controls was unchanged after 12 weeks (Table 1). Percent body fat in exercisers at pretest was not correlated to changes in percent body fat after the intervention (r = 0.17, P > 0.05).

3.4. Abdominal and Trunk Fat Assessed by DEXA. There was a significant decrease in abdominal fat by 0.14 kg (6.6%) for the exercise group (P < 0.05) with no change for the control group (Table 2). The exercise group also significantly decreased (P < 0.001) trunk fat by 1.4 kg (8.4%), whereas trunk fat was slightly increased in controls (Table 2).

^{**} *P* < 0.05, change in exercise group significantly greater compared to that of control group. BMI: body mass index; REE: resting energy expenditure; HR: heart rate; RQ: respiratory quotient; REE: resting energy expenditure.

Table 2: Regional changes in body composition for the high-intensity intermittent exercise and no exercise control groups ($N = 38$; mean
and standard error).

Region of fat mass	Ex	tercise	Control	
	Pre*	Post	Pre*	Post
Leg fat (kg)	9.6 ± 0.8	9.0 ± 0.7	9.9 ± 0.7	9.8 ± 0.7
Leg lean (kg)	18.6 ± 0.6	$19.0 \pm 0.6^{**}$	18.5 ± 0.6	18.5 ± 0.5
Arm fat (kg)	2.7 ± 0.2	$2.5 \pm 0.2**$	2.6 ± 0.1	2.7 ± 0.2
Arm lean (kg)	6.7 ± 0.2	6.7 ± 0.2	6.4 ± 0.4	6.6 ± 0.3
Abdominal fat (kg)	2.3 ± 0.1	$2.2 \pm 0.1**$	2.4 ± 0.2	2.4 ± 0.2
Abdominal lean (kg)	3.7 ± 0.1	3.7 ± 0.1	3.5 ± 0.1	3.5 ± 0.1
Trunk fat (kg)	17.0 ± 0.9	$15.5 \pm 0.9**$	17.2 ± 1.2	17.3 ± 1.3
Trunk lean (kg)	24.9 ± 0.7	$25.6 \pm 0.7^{**}$	24.0 ± 0.8	23.9 ± 0.7

^{*}Prevalues were used as covariates for ANCOVA.

^{**}P < 0.05, change significantly greater compared to that of control group.

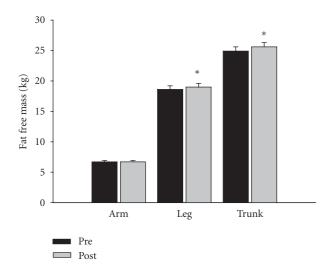


FIGURE 2: Fat-free mass change for the high-intensity intermittent exercise groups (N=38; mean and standard error). *Significant difference between pre- and posttesting (P < 0.05).

3.5. Regional Body Composition Assessed by DEXA. There was no significant difference between groups in absolute FM loss in the leg (P > 0.05), whereas arm FM loss was greater for exercisers (P < 0.01; Table 2).

Total, leg, and trunk FFM (P < 0.05) were significantly increased after 12 weeks of HIIE in the exercise group compared to the control group, whereas arm FFM (P > 0.05) showed no significant change after the intervention (Figure 2).

3.6. Abdominal, Visceral, and Subcutaneous Fat Mass Assessed by CT. Total, abdominal, visceral, and subcutaneous FM at levels of L2/L3 and L4/L5 were significantly reduced (P < 0.05) after 12 weeks of HIIE compared to the control group (Table 3). Abdominal fat decreased by 8.5% at L2/L3 and 6.6% at L4/L5. Visceral fat was significantly decreased (P < 0.05) by 17% at level L2/L3 and 10% at level L4/L5 (Table 3; Figure 3).

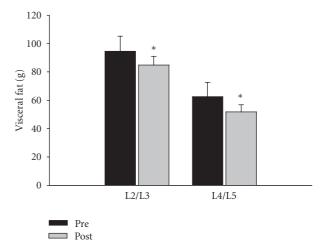


FIGURE 3: Visceral fat change for the high-intensity intermittent exercise and no exercise control groups (N = 38; mean and standard error). *Significantly different from control group (P < 0.05).

3.7. Change in Body Composition after 3, 6, and 9 Weeks. At weeks 3 and 6 there were no change in body mass and BMI, however, after 9 weeks body mass (P < 0.005) and BMI (P < 0.005) were significantly decreased. At the end of the trial, body mass and BMI were significantly decreased (P < 0.001; Table 1), yet, at week 6, WC was significantly lower than that at baseline, from 93.3 to 90.7 cm (P < 0.001). There was a further WC reduction from week 6 (90.7 cm) to week 12, (89.8 cm), which was not significant (P > 0.05). Also the major reduction in visceral fat brought about by HIIE appears to have occurred within the first six weeks as reduction in waist circumference was significantly correlated (P = 0.57, P < 0.05) with reduction in visceral fat (L4/L5) at week six.

3.8. Response in Resting Metabolic Rate following the Intervention. After the intervention exercise subjects had significantly lower resting HR (P < 0.01) and respiratory quotient (RQ; P < 0.01) compared to subjects in the control group. There was no significant change in resting metabolic

Table 3: Changes in computed tomography scan variables for the high-intensity intermittent exercise and no exercise control groups (N = 38; mean and standard error).

	Exercise		Control	
	Pre*	Post	Pre*	Post
L2/L3 total fat mass (g)	564.0 ± 22.3	538.0 ± 21.4**	587.1 ± 26.3	591.0 ± 30.6
L2/L3 abdominal fat (g)	280.0 ± 21.4	256.1 ± 19.6**	304.8 ± 26.5	311.9 ± 30.6
L2/L3 visceral fat (g)	94.6 ± 10.6	$84.8 \pm 9.9^{**}$	102.1 ± 11.5	101.5 ± 11.4
L2/L3 subcutaneous (g)	177.3 ± 16.3	$161.7 \pm 14.7^{**}$	194.2 ± 20.2	200.4 ± 23.4
L4/L5 total fat mass (g)	576.9 ± 24.3	$555.7 \pm 21.7^{**}$	595.7 ± 28.4	602.2 ± 32.7
L4/L5 abdominal fat (g)	327.8 ± 23.0	$306.3 \pm 20.9^{**}$	346.5 ± 27.3	355.5 ± 31.0
L4/L5 visceral fat (g)	62.6 ± 6.2	$51.8 \pm 5.1^{**}$	69.7 ± 9.7	67.3 ± 8.4
L4/L5 subcutaneous (g)	259.7 ± 22.1	$247.8 \pm 20.0^{**}$	271.7 ± 26.1	281.7 ± 27.7

^{*}Prevalues were used as covariates for ANCOVA.

Table 4: Glucose, insulin, HOMA-IR, and lipid change for the high-intensity intermittent exercise and no exercise control groups (N = 38; mean and standard error).

	Exercise		Control	
	Pre	Post	Pre	Post
Glucose (mmoL·l ⁻¹)	4.86 ± 0.14	4.97 ± 0.10	4.91 ± 0.14	4.91 ± 0.14
Insulin ($\mu U \cdot mL^{-1}$)	6.98 ± 0.66	6.72 ± 0.63	8.67 ± 0.90	8.29 ± 0.67
HOMA-IR	1.51 ± 0.15	1.47 ± 0.14	1.90 ± 0.24	1.82 ± 0.17
Total cholesterol (mmo $L \cdot l^{-1}$)	4.18 ± 0.25	3.97 ± 0.24	4.59 ± 0.21	4.36 ± 0.18
Triglycerides (mmoL·l⁻¹)	1.20 ± 0.27	1.18 ± 0.36	1.52 ± 0.21	1.31 ± 0.18
High-density lipoprotein $(mmoL \cdot l^{-1})$	1.31 ± 0.09	1.35 ± 0.09	1.08 ± 0.09	1.03 ± 0.08
Low-density lipoprotein $(mmoL \cdot l^{-1})$	2.51 ± 0.16	2.35 ± 0.18	2.82 ± 0.18	2.81 ± 0.16
Very low-density lipoprotein (mmo $L \cdot l^{-1}$)	0.48 ± 0.07	0.43 ± 0.10	0.66 ± 0.09	0.56 ± 0.09
TC: HDL ratio	3.51 ± 0.32	2.99 ± 0.20	4.62 ± 0.31	4.52 ± 0.27

rate after the intervention (P > 0.05), however, subjects in the exercise group had significantly higher (13%) fat oxidation (P < 0.001) and lower carbohydrate oxidation (P < 0.001) after the intervention compared to the control group (Table 1).

3.9. Response in Blood Variables following the Intervention. Fasting glucose, plasma insulin, glucose, HOMA-IR, and lipid levels were unchanged in the exercise compared to the control group (P > 0.05). For exercisers and controls preintervention levels of insulin, glucose, HOMA-IR, total cholesterol, triglyceride, low-density lipoprotein, high-density lipoprotein (Table 4) were within the normal range for males of this age [15].

3.10. Diet. There was no significant change in macro- or micronutrient content before or after the intervention for the 3-day diet diary of the exercise or control group. Macronutrient levels before and after the 12-week intervention are shown in Table 5.

4. Discussion

The major findings of this study were that HIIE significantly increased $\dot{V}O_{2peak}$ and significantly reduced total, abdominal,

trunk, and visceral fat of young, overweight males. Also trunk and leg fat-free mass was significantly increased after HIIF.

The 15% increase in $\dot{V}O_{2max}$ is similar to results of a previous study that used a 8 s/12 s HIIE protocol [8]. Talanian et al. [16] also found that a HIIE program significantly elevated aerobic power. In this paper the oxidative enzyme β -hydroxyacyl-CoA dehydrogenase was used as a marker of mitochondrial volume and showed that intermittent sprinting enhances mitochondrial capacity. Different forms of HIIE have also been shown to significantly increase aerobic power [17, 18]. Thus, collectively these data show that HIIE results in significant improvements in aerobic fitness. The improvement in cardiorespiratory fitness after HIIE is an attractive feature of this mode of exercise as aerobic fitness has been shown to be an important predictor of positive health [19].

That HIIE resulted in significant subcutaneous fat reduction supports prior research in women using a similar protocol [8]. Results of the present study with males extend these findings showing that HIIE is an effective and efficient way of controlling body composition in both genders. With regard to abdominal fat, it has been found that 15 weeks of HIIE led to significantly reduced abdominal fat (0.15 kg) in untrained young women [8]. The 0.13 kg decrease in abdominal fat and 1.5 kg decrease in trunk fat found in

^{**}P < 0.05, change significantly greater compared to that of control.

TABLE 5: Macronutrient levels before and after the 12-week-intervention ((N = 38; mean and standard error).
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	Exercise		Control	
	Pre	Post	Pre	Post
Kilojoules	8102 ± 428	8142 ± 414	8569 ± 343	8642 ± 343
% carbohydrate	43.6 ± 1.8	42.9 ± 1.5	46.2 ± 1.8	46.8 ± 1.9
% protein	19.8 ± 1.1	19.5 ± 1.1	18.2 ± 1.1	18.3 ± 1.5
% fat	36.4 ± 1.7	37.5 ± 1.4	35.7 ± 1.7	34.9 ± 1.4
% saturated fat	13.2 ± 0.8	13.0 ± 0.7	12.4 ± 0.7	11.3 ± 0.6
% monounsaturated fat	13.6 ± 0.7	14.2 ± 0.7	13.6 ± 0.7	13.8 ± 0.7
% polyunsaturated fat	6.3 ± 0.6	7.0 ± 0.7	6.3 ± 0.6	6.5 ± 0.7
Cholesterol (mg)	397.3 ± 37.5	394.7 ± 41.9	304.8 ± 41.6	381.8 ± 57.0
Fibre (g)	19.5 ± 2.5	19.5 ± 2.4	20.3 ± 2.4	21.4 ± 2.8
Sodium (mg)	2564 ± 320	2709 ± 335	2390 ± 345	2547 ± 335

the current study demonstrates that this effect is also present in young males and supports findings by Boudou et al. [10] who showed that 8 weeks of HIIE significantly reduced abdominal adiposity in older diabetic males.

The significant 17% decrease in visceral fat found in the present study extends the findings of Mourier et al. [20] who showed a significant reduction in visceral fat measured by MRI, following an exercise regimen consisting of steady-state exercise and HIIE for 8 weeks. These findings also add to the results of studies that have shown that aerobic training interventions decrease visceral adipose tissue [21]. The present study, however, appears to be the first to examine the effects of 20 min bouts of HIIE on visceral fat of young males. As visceral compared to overall obesity is more strongly associated with cardiovascular disease risk [22] the ability of HIIE to reduce visceral fat may have positive health implications. For example, it was shown that reduction in visceral fat was associated with improvement in glucose and lipid metabolism, [3] whereas Okauchi et al. [23] found that a reduction in visceral fat lowered the risk of atherosclerotic cardiovascular disease. Interestingly, Ohkawara et al. [21] estimated the optimal dose of aerobic exercise necessary to significantly reduce visceral fat and concluded that 3,780 kcal expended per week was needed. As an exercise session (e.g., cycling on a stationary cycle ergometer) lasting around an hour at a moderate exercise intensity expends about 520-550 kcal then to reach an optimal exercise caloric expenditure of 3,780 kcal per week an individual would have to perform approximately seven one-hour exercise sessions per week. In contrast, subjects in the present study exercised for only one hour per week. Also Donnelly et al. [24] conducted 16 months, 5 hours of aerobic exercise per week program with overweight young males and recorded a 23% decrease in visceral fat. Thus, it appears HIIE can bring about significant decreases in visceral fat with programs that are both significantly shorter in length (e.g., 16 months versus 3 months) and have less exercise commitment per week (5 hours versus 1 hour). Also the major decrease in visceral fat brought about by HIIE may have occurred within the first six weeks as reduction in visceral fat was correlated with

reduction in waist circumference (r = 0.57, P < 0.05) at week six after which waist circumference did not further decrease.

Although the effect of HIIE on FFM has not been extensively examined, one study using DEXA found that trunk muscle mass was significantly increased in young females by 0.6 kg after 15 weeks of HIIE, [8] whereas another study using MRI showed a significant increase in thigh muscle cross sectional area of older males and females after HIIE [10]. The 1.2 kg increase in total FFM found after HIIE in the present study confirms the ability of this type of exercise to increase FFM. However, the length of this 12week intervention was 3 weeks less than that conducted by Trapp et al. [8] that used females as subjects. As exercise HRs and relative exercise intensity of the two trials were similar it appears that males responded with a similar decrease in total fat but a greater increase in FFM after HIIE. FFM increase in the trunk after HIIE was 0.7 kg for males and 0.4 kg for females, whereas in the legs was 0.4 kg for males and 0.1 kg for females. Thus, males compared to females recorded greater increases in FFM in the trunk and legs. This characteristic may be important for fat loss programs as it has been shown that muscle mass is typically decreased after dietary restriction [25] and is typically unchanged after aerobic exercise training [9]. The significant increase in leg FFM may also reflect important metabolic adaptations resulting in enhanced insulin sensitivity [26].

Possible mechanisms underlying the HIIE-induced fat loss effect are undetermined but may include enhanced exercise and postexercise fat oxidation and suppressed postexercise appetite [7]. For example, Burgomaster et al. [26] and Talanian et al. [16] have shown that 6 to 7 sessions of HIIE had significant increases in whole body and skeletal muscle capacity for fatty acid oxidation. The excess postexercise oxygen consumption response to HIIE does not appear to have been examined, however, it is feasible that the significant levels of catecholamines generated during acute HIIE [27] could elevate postexercise fat oxidation. The significant catecholamine response to HIIE is in contrast to moderate, steady-state aerobic exercise that results in small increases in epinephrine and norepinephrine [28]. Also the high

levels of catecholamines produced by HIIE may underlie its ability to reduce visceral fat, as catecholamines have been shown to drive lipolysis and are mainly responsible for fat release from visceral fat stores [29]. Also significantly, more β -adrenergic receptors have been found in visceral compared to subcutaneous fat [30] suggesting that

HIIE may have greater potential than steady-state exercise (e.g., jogging, cycling) to reduce visceral fat. Furthermore, increased fat oxidation after HIIE may occur as a result of the need to remove lactate and H⁺ and to resynthesize glycogen. Uncoupled respiration, protein turnover, and sympathetic nervous system activity may also contribute to increased energy expenditure and fat oxidation after exercise [9]. Finally, HIIE may also have a suppressive effect on appetite as exposing rats to hard exercise has been repeatedly reported to reduce food intake [31].

As this HIIE program required minimal time commitment, it has implications regarding subject compliance with exercise interventions. Thus, physical activity prescriptions, which require the least effort, while still producing adequate reductions in subcutaneous and visceral fat are likely to be optimal [9] and HIIE would seem to fall under this category as subject's total exercise commitment was 60 min per week. In conclusion, 20 min of HIIE, performed three times per week for 12 weeks, resulted in significant reductions in total body, abdominal, trunk, and visceral fat and a significant increase in fat-free mass of overweight young males.

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References

- P. W. Speiser, M. C. J. Rudolf, H. Anhalt, C. Comacho-Hubner, F. Chiarelli, A. Elakim et al., "Childhood obesity," *Journal of Clinical Endocrinology & Metabolism*, vol. 90, pp. 1871–1887, 2005.
- [2] J. M. Jakicic, K. Clark, E. Coleman et al., "Appropriate intervention strategies for weight loss and prevention of weight regain for adults," *Medicine and Science in Sports and Exercise*, vol. 33, no. 12, pp. 2145–2156, 2001.
- [3] R. Ross, D. Dagnone, P. J. H. Jones et al., "Reduction in obesity and related comorbid conditions after diet-induced weight loss or exercise-induced weight loss in men," *Annals of Internal Medicine*, vol. 133, no. 2, pp. 92–103, 2000.
- [4] E. M. Evans, M. J. Saunders, M. A. Spano, A. Arngrimsson, R. D. Lewis, and K. J. Cureton, "Effects of diet and exercise on the density and composition of the fat- free mass in obese women," *Medicine and Science in Sports and Exercise*, vol. 31, no. 12, pp. 1778–1787, 1999.
- [5] T. Wu, X. Gao, M. Chen, and R. M. Van Dam, "Long-term effectiveness of diet-plus-exercise interventions vs. diet-only interventions for weight loss: a meta-analysis," *Obesity Reviews*, vol. 10, no. 3, pp. 313–323, 2009.
- [6] S. H. Boutcher and S. L. Dunn, "Factors that may impede the weight loss response to exercise-based interventions," *Obesity Reviews*, vol. 10, no. 6, pp. 671–680, 2009.

[7] S. H. Boutcher, "High-intensity intermittent exercise and fat loss," *Journal of Obesity*, vol. 2011, Article ID 868305, 2011.

- [8] E. G. Trapp, D. J. Chisholm, J. Freund, and S. H. Boutcher, "The effects of high-intensity intermittent exercise training on fat loss and fasting insulin levels of young women," *Interna*tional Journal of Obesity, vol. 32, no. 4, pp. 684–691, 2008.
- [9] P. Stiegler and A. Cunliffe, "The role of diet and exercise for the maintenance of fat-free mass and resting metabolic rate during weight loss," *Sports Medicine*, vol. 36, no. 3, pp. 239–262, 2006.
- [10] P. Boudou, E. Sobngwi, F. Mauvais-Jarvis, P. Vexiau, and J.-F. Gautier, "Absence of exercise-induced variations in adiponectin levels despite decreased abdominal adiposity and improved insulin sensitivity in type 2 diabetic men," *European Journal of Endocrinology*, vol. 149, no. 5, pp. 421–424, 2003.
- [11] S. Thomas, J. Reading, and R. J. Shephard, "Revision of the physical activity readiness questionnaire (PAR-Q)," *Canadian Journal of Sport Sciences*, vol. 17, no. 4, pp. 338–345, 1992.
- [12] T. Lohman, M. Slaughter, R. Boileau, and L. Lussier, "Bone mineral measurements and their relation to body density in children, youth and adults," *Human Biology*, vol. 56, no. 4, pp. 667–679, 1984.
- [13] G. A. Borg, "Psychophysical bases of perceived exertion," Medicine and Science in Sports and Exercise, vol. 14, no. 5, pp. 377–381, 1982.
- [14] S. E. Stern, K. Williams, E. Ferrannini, R. A. DeFronzo, C. Bogardus, and M. P. Stern, "Identification of individuals with insulin resistance using routine clinical measurements," *Diabetes*, vol. 54, no. 2, pp. 333–339, 2005.
- [15] Expert panel on detection, evaluation, and treatment of high blood cholesterol in adults, "Executive summary of the third report of the national cholesterol education program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (adult treatment panel III)," *The Journal of the American Medical Association*, vol. 285, no. 19, pp. 2486–2497, 2001.
- [16] J. L. Talanian, S. D. R. Galloway, G. J. F. Heigenhauser, A. Bonen, and L. L. Spriet, "Two weeks of high-intensity aerobic interval training increases the capacity for fat oxidation during exercise in women," *Journal of Applied Physiology*, vol. 102, no. 4, pp. 1439–1447, 2007.
- [17] M. Weston, W. Helsen, C. MacMahon, and D. Kirkendall, "The impact of specific high-intensity training sessions on football referees' fitness levels," *The American Journal of Sports Medicine*, vol. 32, no. 1, pp. 54S–61S, 2004.
- [18] E. Gorostiaga, C. Walter, C. Foster, and R. Hickson, "Uniqueness of interval and continuous training at the same maintained exercise intensity," *European Journal of Applied Physiology and Occupational Physiology*, vol. 63, no. 2, pp. 101–107, 1991.
- [19] S. N. Blair, "Physical inactivity: the biggest public health problem of the 21st century," *British Journal of Sports Medicine*, vol. 43, no. 1, pp. 1–2, 2009.
- [20] A. Mourier, J.-F. Gautier, E. D. Kerviler et al., "Mobilization of visceral adipose tissue related to the improvement in insulin sensitivity in response to physical training in NIDDM: effects of branched-chain amino acid supplements," *Diabetes Care*, vol. 20, no. 3, pp. 385–391, 1997.
- [21] K. Ohkawara, S. Tanaka, M. Miyachi, K. Ishikawa-Takata, and I. Tabata, "A dose-response relation between aerobic exercise and visceral fat reduction: systematic review of clinical trials," *International Journal of Obesity*, vol. 31, no. 12, pp. 1786–1797, 2007.
- [22] J. L. Kuk, P. T. Katzmarzyk, M. Z. Nichaman, T. S. Church, S. N. Blair, and R. Ross, "Visceral fat is an independent predictor

of all-cause mortality in men," *Obesity*, vol. 14, no. 2, pp. 336–341, 2006.

- [23] Y. Okauchi, H. Nishizawa, T. Funahashi et al., "Reduction of visceral fat is associated with decrease in the number of metabolic risk factors in Japanese men," *Diabetes Care*, vol. 30, no. 9, pp. 2392–2394, 2007.
- [24] J. E. Donnelly, J. O. Hill, D. J. Jacobsen et al., "Effects of a 16-month randomized controlled exercise trial on body weight and composition in young, overweight men and women: the midwest exercise trial," *Archives of Internal Medicine*, vol. 163, no. 11, pp. 1343–1350, 2003.
- [25] W. Saris, "The role of exercise in the dietary treatment of obesity," *International Journal of Obesity*, vol. 17, no. 1, pp. S17–S21, 1993.
- [26] K. A. Burgomaster, S. C. Hughes, G. J. F. Heigenhauser, S. N. Bradwell, and M. J. Gibala, "Six sessions of sprint interval training increases muscle oxidative potential and cycle endurance capacity in humans," *Journal of Applied Physiology*, vol. 98, no. 6, pp. 1985–1990, 2005.
- [27] E. G. Trapp, D. J. Chisholm, and S. H. Boutcher, "Metabolic response of trained and untrained women during highintensity intermittent cycle exercise," *American Journal of Phy*siology, vol. 293, no. 6, pp. R2370–R2375, 2007.
- [28] H. Zouhal, C. Jacob, P. Delamarche, and A. Gratas-Delamarche, "Catecholamines and the effects of exercise, training and gender," *Sports Medicine*, vol. 38, no. 5, pp. 401–423, 2008.
- [29] B. Issekutz, "Role of beta-adrenergic receptors in mobilization of energy sources in exercising dogs," *Journal of Applied Physiology Respiratory Environmental and Exercise Physiology*, vol. 44, no. 6, pp. 869–876, 1978.
- [30] M. Rebuffé-Scrive, B. Andersson, L. Olbe, and P. Björntorp, "Metabolism of adipose tissue in intraabdominal depots of nonobese men and women," *Metabolism*, vol. 38, no. 5, pp. 453–458, 1989.
- [31] J. Bilski, A. Teległów, J. Zahradnik-Bilska, A. Dembiński, and Z. Warzecha, "Effects of exercise on appetite and food intake regulation," *Medicina Sportiva*, vol. 13, no. 2, pp. 82–94, 2009.

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Clinical Study

Physical Activity Is Associated with Weight Loss and Increased Cardiorespiratory Fitness in Severely Obese Men and Women Undergoing Lifestyle Treatment

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We aimed to examine the relationship between physical activity (PA) and change in body weight and cardiorespiratory fitness (CRF) in severely obese men and women. Thirty-five subjects (10 men, body mass index $43.2 \pm 5.1 \, \text{kg/m}^2$) who participated in a 10-month lifestyle treatment programme were included. The PA duration correlated only with weight change for men (r = -0.69, P = .027 versus r = -0.19, P = .372 for women). Conversely, the PA intensity correlated only with CRF for women (r = 0.61, P = .003 versus r = 0.39, P = .340 for men). PA explained 55.8 and 5.6% of weight change for men and women, respectively, whereas the corresponding explained variances for CRF were 15.6 and 36.7%. We conclude that PA was associated with change in body weight and CRF; however, there was a trend towards a gender specific effect between severely obese men and women.

1. Introduction

As physical activity (PA) is an important contributor to energy expenditure, it is a widely used weight reduction intervention. However, most studies find limited evidence for a decrease in body weight with physical activity alone (1–3 kg) [1–3] or in combination with dietary interventions (3.5– 13 kg) [1–6]. A limitation when reporting such changes on a group level is the large interindividual variation in responses. Because there is a dose-response relationship between PA and weight reduction [7–9], this variation may arise from differences in PA level and other health behaviours. Variation could also be due to gender, as intervention studies suggest that PA may be more effective in reducing weight among men, than among women [10-12]. Because gender may be a moderator of response to PA interventions in obesity treatment, clinical studies in which results are analysed separately for men and women allow for better development of tailored lifestyle intervention programmes [4, 13].

Physical activity is shown to decrease weight in severely obese subjects over both the short term [14–18] and the long term [17, 19–25]. Although some studies suggest that greater

weight reduction may be related to a higher PA level [17, 20, 23], studies on the relationship between PA and weight change are scarce in this population. In addition to reducing weight, lifestyle interventions incorporating PA can increase cardiorespiratory fitness (CRF), which is an important risk factor for cardiovascular disease (CVD) and mortality [26]. As far as we know, the relationship between PA and change in CRF has not been studied in severely obese subjects.

Therefore, the objective of this study was to explore the effect of PA on changes in weight and CRF in severely obese men and women participating in a lifestyle intervention programme. We argue that because distinct gender-related differences might exist in response to PA, analysis should be performed separately for men and women. Such an analysis will facilitate the tailoring of treatment interventions that ultimately improve success rates.

2. Materials and Methods

2.1. Participants. Seventy-one severely obese patients who chose lifestyle treatment over bariatric surgery were enrolled

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at the Red Cross Haugland Rehabilitation Centre (RCHRC) in Norway between August 2006 and May 2009. The inclusion criteria for participation in the programme were age between 18 and 60 years old and body mass index (BMI) > 40 kg/m² without comorbidity, or a BMI > 35 with comorbidity. The exclusion criteria were pregnancy, heart disease, drug or alcohol abuse, previous bariatric surgery and mental disorders and physical impairment that could reduce the ability to comply with the programme. Written informed consent was obtained from each study participant before inclusion in the study. The study meets the standards of the Declaration of Helsinki and was approved by the Regional Committee for Medical Research Ethics.

2.2. Study Protocol. The programme consisted of intermittent inpatient periods of six weeks, four weeks, and three periods of two weeks over a two-year period, separated by three to five months. The present study presents data from the first to the third inpatient period (i.e., 10 months of followup).

An interdisciplinary team of health professionals (dietician, nurse, medical doctor, physiotherapist, and exercise specialist) were responsible for the programme, which consisted of three main components: diet, PA, and cognitive behaviour therapy. Both theoretical and practical sessions were incorporated. With respect to diet, each subject followed a high-fibre, low-fat, reduced-energy meal plan based on the Nordic Nutrition Recommendations [27] that included three main meals and 2-3 snacks each day. Regarding PA, subjects participated in a supervised and structured exercise programme consisting of 20-30 minutes of brisk walking before breakfast and two 45-60 minute exercise sessions (e.g., swimming, aerobics, ball games, hiking, strength training) five days per week. No specific target regarding intensity was applied. Throughout the stay at RCHRC, this programme amounted to between 110 and 150 minutes of PA per day. Together with a team member, each subject developed an individualised plan for PA at home. The main objective of this plan was to increase PA level and strengthen each patient's mastery of everyday life. As such, the plan was based on opportunities at home and subject preferences.

2.3. Measurements. The patient's body weight, waist circumference (WC), height, and CRF were measured at the beginning of the first and third inpatient period. Subjects were weighed to the nearest 0.1 kg in light clothing before breakfast, using a digital scale (BWB-800, Tanita Corp., Tokyo, Japan). Height was measured to the nearest 0.5 cm using a wall-mounted stadiometer. Waist circumference was measured to the nearest 0.5 cm at the level of the iliac crest at the end of a normal expiration using a nonstretchable tape measure. Cardiorespiratory fitness was estimated using the Astrand/Ryhming test according to suggested guidelines [28, 29]. Each subjects heart rate was measured between minute five and six at one submaximal work load on a bicycle ergometer (Ergometrics 900, Jaeger GmbH, Wursburg, Germany) while pedalling at a constant rate of 50 rpm. The measured heart rate was used to estimate maximal oxygen consumption (VO_{2max}), with a correction factor for age. Because we used an indirect measure of VO_{2max} that has not

been validated in severely obese subjects, validity and reliability were examined in a subsample of our subjects (n = 13). The subjects performed a maximal treadmill test using a modified Balke-protocol on day one and performed three Åstrand/Ryhming tests thereafter (on day three, four and five, resp.). Åstrand/Ryhming test number one was used as a learning trial, test number two was used for validation, and test two and three were used for assessing reliability. Oxygen consumption on the treadmill test was measured using the Metamax I and the Metasoft v. 1.11.05 software (Cortex Biophysic, Leipzig, Germany). A one-point gas calibration using ambient air and a volume calibration using a threeliter syringe (SensorMedics Corporation, CA, USA) were performed between each test. The results were comparable to results in normal-weight subjects [30, 31]. There was no systematic bias compared to direct measurement of VO_{2max} (-0.20 mL/kg/min difference, P = .900). For test-retest reliability, we found a Pearson correlation coefficient of r = 0.92(P < .001) and a standard error of measurement (calculated as suggested by Hopkins [32]) of 1.13 mL/kg/min. These results show that the Astrand/Ryhming test has acceptable precision in detecting changes in CRF over time in our sub-

Subjects self-monitored PA using simple training diaries. The mode, duration, and PA intensity were recorded. Intensity was reported as rate of perceived exertion (RPE) on the Borg 6-20 scale [33], which has acceptable validity [34]. Because of the overwhelming amount of data in these diaries, the first week in each month was extracted for analysis. The duration of PA per week as well as the duration for specific modes of PA was calculated. Intensity was calculated as the mean RPE for all training sessions per week. Both measures were averaged over the number of valid months. Because most diaries contained information only about modes of PA, such as strength training, bicycling and walking, activities such as housework and gardening were excluded from the analysis on the assumption that these activities would be performed by most subjects and therefore would be equally distributed. Inclusion criteria for the analysis were valid data on both the duration and PA intensity for at least seven out of the ten months of followup. The energy expenditure (EE) was calculated using reported duration and PA intensity, age, baseline BMI and CRF and the regression equation suggested by Jakicic et al. [35].

2.4. Statistical Analyses. Data are presented as mean \pm standard deviation (SD). Differences between groups were analysed with a one-way ANOVA and the Bonferroni post-hoc test. Where the assumptions of normality or homogeneity of variances were violated, differences were tested using the Kruskall-Wallis test with the Mann-Whitney test and Bonferroni adjustment for multiple comparisons. Differences over time were tested using a one-sample t-test and tested against a value of 0. Correlation between pairs of variables was analysed using Pearson's correlation coefficients as variables were found to approximate normality. Ninety-five percent confidence intervals (CIs) for correlations were obtained using $10\,000$ bootstrap samples. Regression analysis was performed using the partial-least-squares- (PLSs-) method allowing for

Table 1: Baseline characteristics and change values for the group with valid training diaries. (Mean \pm SD n; when different from the total in each group). WC: waist circumference; BMI: body mass index; VO_{2max}: estimated maximal oxygen consumption; EE: energy expenditure.

	Valid training diaries	Men	Women	P between sexes
Baseline				
N	35	10	25	
Age	47.9 ± 8.8	48.8 ± 8.6	47.5 ± 9.0	.694
Height (cm)	168.8 ± 9.5	178.8 ± 8.1	164.8 ± 6.8	.000
Weight (kg)	123.4 ± 20.9	134.9 ± 22.3	118.7 ± 18.8	.036
WC (cm)	$125.8 \pm 13.4 [n = 30]$	$134.6 \pm 17.1 [n = 8]$	$122.6 \pm 10.5 [n = 22]$.056
BMI (kg/m ²)	43.2 ± 5.1	42.0 ± 4.5	43.6 ± 5.3	.400
VO _{2max} (L/min)	2.37 ± 0.57	2.81 ± 0.68	2.18 ± 0.41	.004
VO _{2max} (mL/kg/min)	19.5 ± 5.0	21.5 ± 5.8	18.7 ± 4.6	.171
Change to 10 months				
Weight	-10.73 ± 7.05	-10.67 ± 7.52	-10.76 ± 7.01	.975
WC	$-9.22 \pm 7.18 \ [n = 28]$	$-7.71 \pm 7.29 [n = 7]$	$-9.72 \pm 7.25 [n = 21]$.531
VO _{2max} (L/min)	$0.17 \pm 0.48 \ [n = 30]$	$0.20 \pm 0.62 [n = 8]$	$0.16 \pm 0.44 [n = 22]$.855
VO _{2max} (mL/kg/min)	$3.54 \pm 4.54 \ [n = 30]$	$3.54 \pm 4.54 [n = 8]$	$3.41 \pm 4.52 [n = 22]$.800
Physical activity				
Duration (min/week)	403 ± 171	506 ± 198	361 ± 143	.022
Intensity (RPE)	13.9 ± 1.4	13.8 ± 0.9	13.9 ± 1.6	.855
EE (kcal/week)	3751 ± 1928	5313 ± 2191	3183 ± 1506	.005
Mode of activity				
Walking (%)	57.7 ± 23.2	59.7 ± 28.5	57.0 ± 21.3	.759
Bicycling (%)	11.7 ± 19.2	19.1 ± 26.9	8.7 ± 14.8	.483
Swimming (%)	13.2 ± 9.8	10.9 ± 8.6	14.1 ± 10.3	.398
Strength training (%)	12.2 ± 11.3	8.7 ± 7.1	13.6 ± 12.4	.250
Other (%)	5.3 ± 6.9	1.7 ± 2.8	6.7 ± 7.5	.031

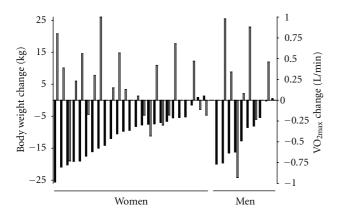
multivariate modeling in small samples. Changes in weight and CRF were used as dependent variables. Four models were established for each dependent variable for men and women separately. Variables to be included were selected on the basis of the bivariate associations with the dependent variables. In model 1, age, baseline BMI, and baseline CRF were used as independent variables. Model 2 included the bivariate relationship between the PA duration and weight change as well as the bivariate relationship between PA intensity and the change in CRF. In model 3, PA duration and intensity were included. Model 4 contained the variables from models 2 and 3. In all PLS analyses, independent variables were standardised to variance because several different units of measurement were used. In order to assess the robustness of the findings, all models were cross-validated excluding every fourth subject. PLS analysis and the cross-validation were performed using Sirius v. 8.0 (Pattern Recognition Systems, Bergen, Norway), while all other analyses were performed using SPSS v. 17.0 (SPSS Inc., Chicago, USA). A two-sided P < .05 indicated significant differences.

3. Results

3.1. Analysis of Attrition. A total of 71 subjects were included in the lifestyle treatment programme. Of these, nine subjects did not consent to take part in this study and six quit during the course of the programme: one to undergo bariatric

surgery, one having reached his weight goal, and four for unknown reasons. This left 56 subjects for analysis, among whom 35 subjects (10 men) had valid training diaries. Subjects without valid training diaries were younger than subjects with valid training diaries (39.3 \pm 11.1 versus 47.9 \pm 8.8 years, P=.005). Dropouts were taller than subjects with valid training diaries (179.8 \pm 8.4 versus 168.8 \pm 9.5 cm, P=.029). All other baseline- and change values were similar between groups. Further results are based on the group with valid training diaries.

3.2. Baseline Characteristics and Outcomes at the Group Level. Table 1 show that the men were taller, heavier and had better CRF (L/min) than the women at baseline. Changes in weight, WC, and CRF were similar between genders. Results regarding changes over time are therefore collapsed. All variables changed significantly over time (P < .001), except for CRF expressed as an absolute value (L/min) (P = .061). The weight and WC changes were $-8.7 \pm 5.8\%$ (95% CI -10.7 to -6.7%) and $-7.2 \pm 5.5\%$ (CI -9.3 to -5.1%), respectively. VO_{2max} increased 9.7 \pm 21.1% (CI 1.8 to 17.5%) and 21.1 \pm 25.8% (CI 11.4 to 30.7%) when expressed as absolute and relative values, respectively. Taken together, subjects spent about 58 minutes on PA per day during the intervention, which corresponds to an EE of about 536 kcal/day. The duration and volume of PA were significantly higher for men than for women, whereas the intensity was similar between



- Body weight change (left *y*-axis)
- VO_{2max} change (right *y*-axis)

FIGURE 1: Individual changes in body weight and cardiorespiratory fitness (n = 22 for women; n = 8 for men). Subjects are sorted within gender with respect to body weight change.

genders. With regard to the mode of PA, the pattern was similar between men and women, except for "other activities," which was reported more often by women.

3.3. Relationships between PA and Change in Weight and CRF. Figure 1 shows individual changes in weight and CRF. The figure clearly shows large individual variation in both outcome variables. Most subjects lost weight and displayed increases in CRF; however, the opposite was also seen. Changes in the two outcomes were not related (r = -0.16, P = .401, n = 30).

Correlation between the PA duration and weight change was r = -0.33 (P = .050); the correlation between the PA intensity and weight change was r = -0.17 (P = .336). The correlation between the PA duration and the change in CRF was r = -0.04 (P = .853, n = 30); the correlation between the PA intensity and the change in CRF was r = 0.51 (P =.004, n = 30). The relationship between the PA duration and weight change was much stronger for men than for women (r = -0.69, CI - 0.88 to -0.41, P = .027 versus r = -0.19,CI -0.60 to 0.26, P = .372). Conversely, the relationship between the PA intensity and change in CRF was stronger for women than for men (r = 0.61, CI 0.10 to 0.85, P = .003(n = 22) versus r = 0.39, CI -0.29 to 0.86, P = .340 (n = 8). Scatterplots of PA duration versus change in weight and PA intensity versus change in CRF are shown in Figures 2(a) and 2(b), respectively. There were no relationship between PA duration and WC (r = -0.10, P = .624, n = 28), nor between PA intensity and WC (r = -0.01, P = .967, n = 28).

3.4. Regression Analysis. Tables 2(a) and 2(b) show the PLS regression analysis with respect to changes in weight and CRF, respectively. A correlation matrix including independent and dependent variables is shown in Table 3. For weight change, model 1 (baseline variables) was quite similar for both genders, explaining about 20% of the variance. Model 2 (PA duration) yielded remarkably different results between

genders, explaining 47.8% of the variance for men compared to 4% for women. The PA variables together (model 3) explained 55.8% of the weight change for men, whereas 5.6% of the weight change was explained for women. Together, baseline characteristics and PA (model 4) explained about two-thirds of the weight change for men, whereas only one-third of the change for women was explained.

Regarding the change in CRF, model 1 explained 40.4 and 34.2% of the variance for men and women, respectively. The corresponding variances explained by model 2 were 14.5 and 37.5%. For women, the PA variables together (model 3) explained one-third of the change in CRF, whereas 15.6% was explained for men. Taken together, the baseline variables and PA variables explained 81.6% of the change in CRF for men and 60.6% for women (model 4). However, the SEEs for men were generally almost twice as high as for women, reflecting considerable uncertainty in the predictions.

4. Discussion

This study found that severely obese subjects undergoing a 10-month lifestyle intervention showed great individual variation in the change in weight and CRF. Physical activity was related differently to change in weight and CRF in men and women. Duration and intensity of PA explained 55.8% of the weight change in men and only 5.6% in women. For CRF the opposite pattern was seen. While PA explained only 15.6% of the change for men, it explained 36.7% of the change for women.

4.1. Weight Change. We found a decrease in weight of -10.7 kg (-8.7%) over 10 months in both genders. This is in line with other studies investigating lifestyle treatment for severely obese subjects [17, 24, 25]. However, other studies have shown more beneficial results after similar programmes [21] and larger weight losses have been achieved with more severe lifestyle interventions using low-energy or very low-energy diets [22, 23]. Weight losses in the present study seem consistent with results seen in overweight and moderately obese subjects undergoing lifestyle interventions [3–5]. Thus, regarding weight loss, lifestyle treatment programmes seem to work equally well in severely obese and less obese persons.

Few previously published studies of severely obese subjects have reported PA levels. Interestingly, Hofsø et al. [24] reported a median physical activity level of approximately the same magnitude as in the present study (65 minutes per day); the weight losses were also nearly identical. Unfortunately, Goodpaster et al. [25], who recently published the first randomised controlled trial in the field of lifestyle intervention for severely obese subjects, did not report total minutes spent in moderate to vigorous physical activity or EE, although PA was measured both objectively and through training diaries. Comparing the present study results with the results of Maffiuletti et al. [17] is also difficult because PA levels were determined using a scoring system that, while allowing for acceptable internal validity, limits its external validity.

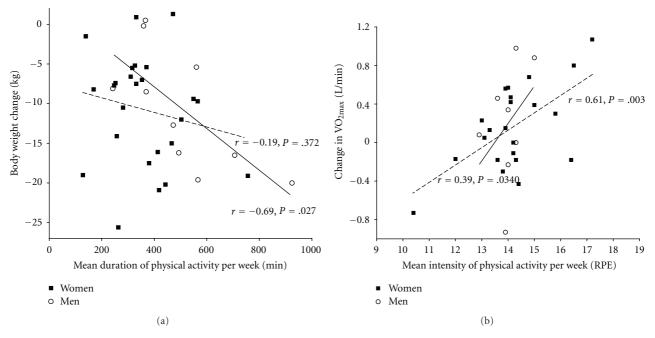


FIGURE 2: (a) Scatterplot showing weight change versus duration of physical activity for men (full regression line) and women (dotted regression line). (b) Scatterplot showing change in cardiorespiratory fitness versus intensity of PA for men (full regression line) and women (dotted regression line).

Although reports on dose-response relationships between PA and weight change in severely obese subjects are scarce, some studies have shown convincing results in less obese subjects [7–9]. The most recent study by Wadden et al. [7] showed a consistent increase in one-year weight loss of 4.4, 7.1, 9.0, and 11.9% for increasing quartiles of mean weekly minutes of physical activity (25.9, 84.8, 148.7, and 287.1 min) in 2570 subjects with a baseline BMI of about 36, randomised to lifestyle intervention.

Overall, a correlation of r=0.41 was found, yielding a 16.1% explained variance in weight change. This relationship is of approximately the same strength as in the present study when the male and female groups are collapsed (r=0.33). However, we found a marked difference between genders. In men, a moderate-to-high correlation was found (r=-0.69), whereas in women, a nonsignificant association with weight change was found (r=-0.19). Although it should be noted that the CIs overlapped between genders, there was a clear difference in the strength of the relationships. Although our results must be interpreted carefully due to the small samples, the results are in line with previous intervention studies suggesting larger weight losses due to physical activity for men than for women [10–12].

4.2. Change in Cardiorespiratory Fitness. An overall mean increase in CRF of 3.5 mL/kg/min (21.1%) was achieved in the present study. This change parallels other study results for both severely obese and less obese subjects undergoing lifestyle interventions incorporating PA [17, 18, 36]. Such an increase in CRF may reduce the risk of all-cause mortality and the incidence of cardiovascular disease in healthy men

and women by 13 and 15%, respectively [26]. As studies consistently show an especially large risk reduction with an increase in CRF beyond some minimal threshold [26, 37], any increase would be beneficial to health for most severely obese subjects, whose CRF is generally low [17, 18, 38, 39]. Moreover, low baseline CRF was a significant predictor for increased CRF in both genders in the present study, showing that subjects with the highest intervention potential benefitted the most. The increase in CRF may also benefit the subjects by allowing them to increase their PA level and thereby lose more weight [4]. This is indicated by the inverse relationship between baseline CRF and PA duration found in the present study (r = 0.40, P = .024, results not shown).

Duration of PA was not correlated with change in CRF in the present study, whereas PA intensity was significantly correlated with increases in CRF in the group as a whole, as well as for women. For men, this relationship was nonsignificant. The importance of PA intensity to increase CRF is well established [40]. The variation in change in CRF explained by PA for women (36.7%) is consistent with nine studies reviewed by Williams [37] in which PA explained 35% of the variation in CRF. The weaker explanation of the variation among men in the present study (15.6%) may be a result of men being a relatively small homogenous group, which resulted in a minimal range in PA intensities.

4.3. Interpretation of the Results. We found that regression models based on both baseline measures and PA variables explained 69.4 and 36.8% of the change in weight for men and women, respectively, with corresponding values for the change in CRF of 81.6 and 60.6%. These values are quite

Table 2: (a) Regression equations, standard errors, and explained variance in change in weight. All models are cross-validated excluding every fourth subject. Independent variables that have a significant contribution (P < .05) to the model are labeled[†]. SEE: standard error of the estimate; SECV: standard error of the cross-validation. (b) Regression equations, standard errors, and explained variance in change in cardiorespiratory fitness. All models are cross-validated excluding every fourth subject. Independent variables that have a significant contribution (P < .05) to the model are labeled[†]. SEE: standard error of the estimate; SECV: standard error of the cross-validation.

		(a)				
	Regression equation (standardized)	Regression equation (actual)	SEE	SECV	R^2	Adj R ²
Men						
Model 1	e	21.60 - 0.14* Age $-0.50*$ Baseline BMI $-1.62*$ Baseline VO _{2max}	7.17	8.68	0.193	0.092
Model 2		2.620 – 0.03* Duration	5.76	5.62	0.478	0.412
Model 3		41.30 – 0.02* Duration – 3.00 * Intensity	5.30	5.45	0.558	0.502
Model 4	BMI^{\dagger} – 1.11 * Baseline VO_{2max} – 3.72 *	68.40 - 0.14* Age $-0.50*$ Baseline BMI $-1.62*$ Baseline VO _{2max} $-0.02*$ Duration $-2.69*$ Intensity	4.41	5.86	0.694	0.656
Women						
Model 1		10.60 - 0.01* Age $-0.60*$ Baseline BMI + 2.40 * Baseline VO _{2max}	6.38	7.85	0.208	0.173
Model 2		-7.43 - 0.01* Duration	6.88	7.04	0.040	0.000
Model 3	0.95 - 1.5/* Direction' - 0.89 * Intensity	0.95 – 0.01* Duration – 0.56 * Intensity	6.96	7.38	0.056	0.015
Model 4	$BMI^{\dagger} + 2.00 * Baseline VO_{2max} - 2.84 *$	33.40 - 0.11* Age $-0.74*$ Baseline BMI + $4.84*$ Baseline VO _{2max} $-0.02*$ Duration $-0.72*$ Intensity	5.83	8.19	0.368	0.310
		(b)				
	Regression equation (standardized)	Regression equation (actual)	SEE	SECV	R^2	Adj R ²
Men						
Model 1	$0.88 + 0.56 * Age^{\dagger} - 0.14 * Baseline BMI - 0.55 * Baseline VO_{2max}^{\dagger}$	0.55 + 0.04* Age $-0.02*$ Baseline BMI $-0.49*$ Baseline VO _{2max}	0.56	0.58	0.404	0.165
Model 2		-5.24 + 0.39 * Intensity	0.56	0.62	0.145	0.002
Model 3	-8.85 + 0.10 * Duration + 0.38 * Intensity	-5.47 + 0.00 * Duration + 0.39 * Intensity	0.61	0.59	0.156	0.015
Model 4	$-19.00 + 0.73*$ Age $^{\dagger} + 0.33*$ Baseline BMI $^{\dagger} - 1.01*$ Baseline VO _{2max} $^{\dagger} + 0.09*$ Duration + 0.71 * Intensity †	-11.7 + 0.05* Age $+ 0.04*$ Baseline BMI $- 0.90*$ Baseline $VO_{2max} + 0.00*$ * Duration $+ 0.72*$ Intensity	0.35	0.62	0.816	0.677
Women						
Model 1	3.46-0.15* Age [†] $+0.14*$ Baseline BMI $-0.63*$ Baseline VO _{2max} [†]	1.51 - 0.01 * Age + 0.01 * Baseline BMI $- 0.66 * Baseline VO_{2max}$	0.36	0.41	0.342	0.310
Model 2		-2.39 + 0.18 * Intensity	0.35	0.34	0.375	0.344
Model 3	-4.86 - 0.12* Duration + 0.58 * Intensity	-2.12 - 0.00* Duration + 0.17 * Intensity	0.36	0.36	0.367	0.336
Model 4	$-3.93-0.15*$ Age $+0.25*$ Baseline BMI † $0.47*$ Baseline VO $_{2max}^{\dagger}+0.12*$ Duration $0.56*$ Intensity †		0.29	0.32	0.606	0.564

high compared to other prospective studies, where explained variances in weight change are in the range of 12 to 38% [10, 41, 42]. However, the residuals are substantial in all models for both genders, indicated by the SEEs of \sim 6 kg for weight and 0.3–0.6 L/min for VO_{2max}. Therefore, care should be taken to ensure the ethical use of such models in clinical settings. Although selection strategies may ensure more effective use of limited resources [43], we believe individuals in need of essential healthcare who are found to

have a low probability of success should not be given less attention and followup based on such predictions.

If the dissimilar responses to PA between genders are accurate, tailoring the content and focus of lifestyle treatment programmes to men and women may improve the rate of treatment success. It can be argued that men and women in our sample used different strategies to lose weight because PA explains 55.8 versus 5.6% of the changes in body weight for men and women, respectively, whereas both genders lose

Table 3: Correlation matrix between independent and dependent variables in the regression analyses for men (n = 8-10) in the lower left corner and women (n = 22-25) in the upper right corner (Pearson r (P value)). BMI: body mass index; VO_{2max}: estimated maximal oxygen consumption.

	Age	Baseline BMI	Baseline VO _{2max}	PA duration	PA intensity	Change in weight	Change in VO _{2max}
Age	1	-0.15 (.467)	$-0.40\;(.064)$	-0.08 (.696)	-0.48 (.014)	-0.01 (.947)	-0.12 (.605)
Baseline BMI	0.11 (.755)	1	0.14 (.549)	-0.20 (.349)	-0.02 (.945)	-0.42 (.039)	0.11 (.632)
Baseline VO _{2max}	0.45 (.224)	0.35 (.350)	1	0.29 (.189)	-0.18 (.416)	0.14 (.526)	-0.49 (.019)
PA duration	0.32 (.372)	0.13 (.725)	0.33 (.384)	1	-0.19 (.362)	-0.19(.372)	-0.12 (.584)
PA intensity	-0.22 (.541)	-0.15 (.671)	-0.21 (.589)	0.26 (.471)	1	-0.11 (.605)	0.61 (.003)
Change in weight	-0.22 (.545)	-0.43 (.218)	-0.24 (.532)	-0.69 (.027)	-0.45 (.195)	1	-0.24 (.274)
Change in VO _{2max}	0.26 (.527)	-0.21 (.613)	-0.41 (.313)	0.10 (.809)	0.39 (.340)	0.03 (.951)	1

the same amount of weight. In other words, factors other than PA may cause the weight reduction in women and might not benefit men equally. Although we have no measure of the diet component of this lifestyle intervention, we can speculate that women relied more on changes in eating to regulate energy balance, than men did. As the literature suggests [44, 45], women may have a closer regulation of appetite than men in short periods of increased energy expenditure, which leads to a partial compensation for the negative energy balance. This would influence weight change in our study and may imply that women should increase their PA level together with having a strict diet. This finding is also in line with results showing an interaction between PA and diet for women such that PA was only beneficial when performed together with changes in diet [10]. Hence, women should be advised to increase their PA level and to make dietary changes. Moreover, PA intensity is important to increase CRF. Thus, we recommend that PA should be an important part of lifestyle treatment programmes for severely obese subjects of both genders.

4.4. Strengths, Limitations, and Suggestions for Further Research. The present study has several limitations. First, as the sample was small, relationships may be made spurious by low representativeness or by increasing the influence of specific cases or outliers in the data. However, all regression models were cross-validated, with minor changes made to the standard errors. Second, as weight regain following weight loss is very common, the 10-month followup in the present study may not have been long enough to determine the clinical importance of our results. However, PA is consistently seen as a predictor of weight maintenance [46, 47], so its effect could become more important over time. Third, as precise measures are important to reveal true relationships, the measures used could undermine our conclusions. In particular, body weight has low sensitivity to changes in fatness after PA interventions. Because PA stimulates muscle hypertrophy, decreases in fat mass may be masked [48, 49]. Furthermore, self-report measures show large deviations compared to objective measures of PA [50]. However, as no gold standard exists, PA measurement remains a topic for debate. There is no evidence, as far as we know, that these measures have less reliability in obese subjects than in other subjects.

While our use of training diaries may be a limitation as discussed above, it may also be a strength. We calculated PA duration and intensity as the mean for at least seven of the ten months of followup, thereby avoiding variation in PA from week to week and during seasons as a source of error. This variation could be a serious threat to studies where PA levels are measured for single points in time, as illustrated by correlations ranging between r=-0.43 and 0.00 between PA duration for each of the ten months and total weight change in the present study. Finally, the lack of diet as a research measure, one of the two main components determining energy balance, constitutes a limitation for the prediction of changes in weight.

Due to the methodological weaknesses described above, our results are preliminary. Further research examining PA and CRF in severely obese subjects undergoing lifestyle treatment should include objective measures to determine PA level and direct measurements of CRF (i.e., by measuring oxygen consumption and/or by performance tests). Furthermore, results should be reported for men and women separately. Finally, comprehensive studies including both objective and subjective measures of health should be conducted, and relationships with PA and CRF should be determined.

5. Conclusion

This study showed beneficial changes in weight and CRF for severely obese subjects undergoing lifestyle treatment. Individual variation in outcomes was large, and PA was related differently to changes in weight and CRF between men and women. Further research is needed to determine the effects of PA in severely obese subjects. Until then, it is reasonable to state that more PA is better.

Conflict of Interests

The authors declared that they have no conflict of interests.

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References

- [1] K. Shaw, H. Gennat, P. O'Rourke, and C. del Mar, "Exercise for overweight or obesity.," *Cochrane database of systematic reviews*, no. 4, Article ID CD003817, 2006.
- [2] M. J. Franz, J. J. VanWormer, A. L. Crain et al., "Weight-loss outcomes: a systematic review and meta-analysis of weightloss clinical trials with a minimum 1-year follow-up," *Journal* of the American Dietetic Association, vol. 107, no. 10, pp. 1755– 1767, 2007.
- [3] W. C. Miller, D. M. Koceja, and E. J. Hamilton, "A metaanalysis of the past 25 years of weight loss research using diet, exercise or diet plus exercise intervention," *International Journal of Obesity*, vol. 21, no. 10, pp. 941–947, 1997.
- [4] V. A. Catenacci and H. R. Wyatt, "The role of physical activity in producing and maintaining weight loss," *Nature Clinical Practice Endocrinology and Metabolism*, vol. 3, no. 7, pp. 518–529, 2007.
- [5] C. Galani and H. Schneider, "Prevention and treatment of obesity with lifestyle interventions: review and meta-analysis," *International Journal of Public Health*, vol. 52, no. 6, pp. 348– 359, 2007.
- [6] T. Wu, X. Gao, M. Chen, and R. M. van Dam, "Long-term effectiveness of diet-plus-exercise interventions vs. diet-only interventions for weight loss: a meta-analysis: obesity management," *Obesity Reviews*, vol. 10, no. 3, pp. 313–323, 2009.
- [7] T. A. Wadden, D. S. West, R. H. Neiberg et al., "One-year weight losses in the look AHEAD study: factors associated with success," *Obesity*, vol. 17, no. 4, pp. 713–722, 2009.
- [8] J. M. Jakicic, B. H. Marcus, K. I. Gallagher, M. Napolitano, and W. Lang, "Effect of exercise duration and intensity on weight loss in overweight, sedentary women: a randomized trial," *Journal of the American Medical Association*, vol. 290, no. 10, pp. 1323–1330, 2003.
- [9] C. A. Slentz, B. D. Duscha, J. L. Johnson et al., "Effects of the amount of exercise on body weight, body composition, and measures of central obesity: STRRIDE—a randomized controlled study," *Archives of Internal Medicine*, vol. 164, no. 1, pp. 31–39, 2004.
- [10] C. L. Dunn, P. J. Hannan, R. W. Jeffery, N. E. Sherwood, N. P. Pronk, and R. Boyle, "The comparative and cumulative effects of a dietary restriction and exercise on weight loss," *International Journal of Obesity*, vol. 30, no. 1, pp. 112–121, 2006.
- [11] J. E. Donnelly, J. O. Hill, D. J. Jacobsen et al., "Effects of a 16-month randomized controlled exercise trial on body weight and composition in young, overweight men and women: the midwest exercise trial," *Archives of Internal Medicine*, vol. 163, no. 11, pp. 1343–1350, 2003.
- [12] K. R. Westerterp, G. A. L. Meijer, E. M. E. Janssen, W. H. M. Saris, and F. T. Hoor, "Long-term effect of physical activity on energy balance and body composition," *British Journal of Nutrition*, vol. 68, no. 1, pp. 21–30, 1992.
- [13] J. C. Lovejoy and A. Sainsbury, "Sex differences in obesity and the regulation of energy homeostasis: etiology and pathophysiology," *Obesity Reviews*, vol. 10, no. 2, pp. 154–167, 2009.
- [14] A. Sartorio, C. L. Lafortuna, G. Conte, G. Faglia, and M. V. Narici, "Changes in motor control and muscle performance after a short-term body mass reduction program in obese subjects," *Journal of Endocrinological Investigation*, vol. 24, no. 6, pp. 393–398, 2001.
- [15] A. Sartorio, C. L. Lafortuna, V. Vangeli, A. Tavani, C. Bosetti, and C. la Vecchia, "Short-term changes of cardiovascular risk factors after a non-pharmacological body weight reduction

- program," European Journal of Clinical Nutrition, vol. 55, no. 10, pp. 865–869, 2001.
- [16] J. R. Shapiro, A. L. Stout, and G. J. Musante, "Structure-size me: weight and health changes in a four week residential program," *Eating Behaviors*, vol. 7, no. 3, pp. 229–234, 2006.
- [17] N. A. Maffiuletti, F. Agosti, P. G. Marinone, G. Silvestri, C. L. Lafortuna, and A. Sartorio, "Changes in body composition, physical performance and cardiovascular risk factors after a 3-week integrated body weight reduction program and after 1-y follow-up in severely obese men and women," *European Journal of Clinical Nutrition*, vol. 59, no. 5, pp. 685–694, 2005.
- [18] J. Ø. Pedersen, E. Zimmermann, B. M. Stallknecht et al., "Lifestyle intervention in the treatment of severe obesity," *Ugeskrift* for Laeger, vol. 168, no. 2, pp. 167–172, 2006.
- [19] H. Bjorvell and S. Rossner, "Short communication: a ten-year follow-up of weight change in severely obese subjects treated in a combined behavioural modification programme," *International Journal of Obesity*, vol. 16, no. 8, pp. 623–625, 1992.
- [20] T. Christiansen, J. M. Bruun, E. L. Madsen, and B. Richelsen, "Weight loss maintenance in severely obese adults after an intensive lifestyle intervention: 2- to 4-year follow-up," *Obesity*, vol. 15, no. 2, pp. 413–420, 2007.
- [21] C. Martins, M. Strømmen, O. A. Stavne, R. Nossum, R. Mårvik, and B. Kulseng, "Bariatric surgery versus lifestyle interventions for morbid obesity—changes in body weight, risk factors and comorbidities at 1 year," *Obesity Surgery*, vol. 21, no. 7, pp. 841–849, 2011.
- [22] J. W. Anderson, S. B. Conley, and A. S. Nicholas, "One hundred-pound weight losses with an intensive behavioral program: changes in risk factors in 118 patients with long-term follow-up," *American Journal of Clinical Nutrition*, vol. 86, no. 2, pp. 301–307, 2007.
- [23] J. W. Anderson, L. Grant, L. Gotthelf, and L. T. P. Stifler, "Weight loss and long-term follow-up of severely obese individuals treated with an intense behavioral program," *International Journal of Obesity*, vol. 31, no. 3, pp. 488–493, 2007.
- [24] D. Hofsø, N. Nordstrand, L. K. Johnson et al., "Obesity-related cardiovascular risk factors after weight loss: a clinical trial comparing gastric bypass surgery and intensive lifestyle intervention," *European Journal of Endocrinology*, vol. 163, no. 5, pp. 735–745, 2010.
- [25] B. H. Goodpaster, J. P. DeLany, A. D. Otto et al., "Effects of diet and physical activity interventions on weight loss and cardiometabolic risk factors in severely obese adults: a randomized trial," *Journal of the American Medical Association*, vol. 304, no. 16, pp. 1795–1802, 2010.
- [26] S. Kodama, K. Saito, S. Tanaka et al., "Cardiorespiratory fitness as a quantitative predictor of all-cause mortality and cardiovascular events in healthy men and women: a meta-analysis," *Journal of the American Medical Association*, vol. 301, no. 19, pp. 2024–2035, 2009.
- [27] Nordic Council of Ministers, Nordic Nutrition Recommendations 2004—Integrating Nutrition and Physical Activity, Nordic Publishing, Copenhagen, Denmark, 2004.
- [28] I. Astrand, "Aerobic work capacity in men and women with special reference to age," *Acta Physiologica Scandinavica*, *Supplementum*, vol. 49, pp. 1–92, 1960.
- [29] P. O. Astrand and I. Ryhming, "A nomogram for calculation of aerobic capacity (physical fitness) from pulse rate during sub-maximal work," *Journal of Applied Physiology*, vol. 7, pp. 218–221, 1954.
- [30] R. E. Cink and T. R. Thomas, "Validity of the Astrand-Ryhming nomogram for predicting maximal oxygen intake," *British Journal of Sports Medicine*, vol. 15, no. 3, pp. 182–185, 1981.

- [31] A. Macsween, "The reliability and validity of the Astrand nomogram and linear extrapolation for deriving (V)over-dot-O-2max from submaximal exercise data," *Journal of Sports Medicine and Physical Fitness*, vol. 41, no. 3, pp. 312–317, 2001.
- [32] W. G. Hopkins, "Measures of reliability in sports medicine and science," *Sports Medicine*, vol. 30, no. 1, pp. 1–15, 2000.
- [33] G. Borg, "Perceived exertion as an indicator of somatic stress," *Scandinavian Journal of Rehabilitation Medicine*, vol. 2, no. 2, pp. 92–98, 1970.
- [34] M. J. Chen, X. Fan, and S. T. Moe, "Criterion-related validity of the Borg ratings of perceived exertion scale in healthy individuals: a meta-analysis," *Journal of Sports Sciences*, vol. 20, no. 11, pp. 873–899, 2002.
- [35] J. M. Jakicic, J. E. Donnelly, N. P. Pronk, A. F. Jawad, and D. J. Jacobsen, "Prescription of exercise intensity for the obese patient: the relationship between heart rate, VO2 and perceived exertion," *International Journal of Obesity*, vol. 19, no. 6, pp. 382–387, 1995.
- [36] G. A. Kelley, K. S. Kelley, and Z. Vu Tran, "Aerobic exercise, lipids and lipoproteins in overweight and obese adults: a meta-analysis of randomized controlled trials," *International Journal of Obesity*, vol. 29, no. 8, pp. 881–893, 2005.
- [37] P. T. Williams, "Physical fitness and activity as separate heart disease risk factors: a meta-analysis," *Medicine and Science in Sports and Exercise*, vol. 33, no. 5, pp. 754–761, 2001.
- [38] M. Hulens, G. Vansant, A. L. Claessens, R. Lysens, and E. Muls, "Predictors of 6-minute walk test results in lean, obese and morbidly obese women," *Scandinavian Journal of Medicine and Science in Sports*, vol. 13, no. 2, pp. 98–105, 2003.
- [39] M. Hulens, G. Vansant, R. Lysens, A. L. Claessens, and E. Muls, "Exercise capacity in lean versus obese women," *Scandinavian Journal of Medicine and Science in Sports*, vol. 11, no. 5, pp. 305–309, 2001.
- [40] H. A. Wenger and G. J. Bell, "The interactions of intensity, frequency and duration of exercise training in altering cardiores-piratory fitness," *Sports Medicine*, vol. 3, no. 5, pp. 346–356, 1986.
- [41] M. A. Lavery and J. W. Loewy, "Identifying predictive variables for long-term weight change after participation in a weight loss program," *Journal of the American Dietetic Association*, vol. 93, no. 9, pp. 1017–1024, 1993.
- [42] R. C. Klesges, L. M. Klesges, C. K. Haddock, and L. H. Eck, "A longitudinal analysis of the impact of dietary intake and physical activity on weight change in adults," *American Journal* of Clinical Nutrition, vol. 55, no. 4, pp. 818–822, 1992.
- [43] K. Elfhag and S. Rössner, "Who succeeds in maintaining weight loss? A conceptual review of factors associated with weight loss maintenance and weight regain," *Obesity Reviews*, vol. 6, no. 1, pp. 67–85, 2005.
- [44] R. J. Stubbs, A. Sepp, D. A. Hughes et al., "The effect of graded levels of exercise on energy intake and balance in free-living men, consuming their normal diet," *European Journal of Clinical Nutrition*, vol. 56, no. 2, pp. 129–140, 2002.
- [45] R. J. Stubbs, A. Sepp, D. A. Hughes et al., "The effect of graded levels of exercise on energy intake and balance in free-living women," *International Journal of Obesity*, vol. 26, no. 6, pp. 866–869, 2002.
- [46] D. F. Tate, R. W. Jeffery, N. E. Sherwood, and R. R. Wing, "Long-term weight losses associated with prescription of higher physical activity goals. Are higher levels of physical activity protective against weight regain?" *American Journal of Clinical Nutrition*, vol. 85, no. 4, pp. 954–959, 2007.

[47] R. R. Wing and S. Phelan, "Long-term weight loss maintenance," *The American Journal of Clinical Nutrition*, vol. 82, supplement 1, pp. 2228–222S, 2005.

- [48] W. J. Kraemer, J. S. Volek, K. L. Clark et al., "Influence of exercise training on physiological and performance changes with weight loss in men," *Medicine and Science in Sports and Exercise*, vol. 31, no. 9, pp. 1320–1329, 1999.
- [49] G. R. Hunter, N. M. Byrne, B. Sirikul et al., "Resistance training conserves fat-free mass and resting energy expenditure following weight loss," *Obesity*, vol. 16, no. 5, pp. 1045–1051, 2008.
- [50] S. A. Prince, K. B. Adamo, M. E. Hamel, J. Hardt, S. Connor Gorber, and M. Tremblay, "A comparison of direct versus self-report measures for assessing physical activity in adults: a systematic review," *International Journal of Behavioral Nutrition and Physical Activity*, vol. 5, article 55, 2008.

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Research Article

Age-Related Decline in Cardiorespiratory Fitness among Career Firefighters: Modification by Physical Activity and Adiposity

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Firefighting is a very hazardous occupation, and strenuous fire duties require high levels of physical fitness. In the general adult population, cardiorespiratory fitness (CRF) declines with aging. We sought to investigate the effect of increasing age on CRF in male career firefighters as well as the modifying effects of physical activity and adiposity. We cross-sectionally examined 804 male career firefighters from two Midwestern states. CRF was determined from symptom-limited maximal treadmill exercise testing in metabolic equivalents (METS) following the Bruce protocol. Physical activity self-reports were extracted from responses to a health and lifestyle questionnaire. We found as expected that CRF declines with advancing age; however, the decline is greatly attenuated among leaner firefighters who report more physical activity. Furthermore, in a linear regression model including age, BMI, and variables describing physical activity behaviors, we could predict CRF ($R^2 = 0.6286$). The total weekly duration of aerobic exercise as well as the duration of weight lifting sessions both had significant impacts on age-related decline. We conclude that firefighters are more likely to maintain the high levels of CRF needed to safely perform their duties if they engage in frequent exercise and maintain healthy weights.

1. Introduction

Cardiorespiratory fitness (CRF) declines with aging in the general population. A decrement in VO₂max (mL/kg/min) of about 1.6% per year in both men and women has been described [1].

Similarly, among untrained individuals, a decline in peak VO_2 of 5–10% per decade of life has been observed [2]. Low CRF is an important risk factor for the development of obesity, hypertension, and other cardiovascular risk factors as well as coronary heart disease, stroke, loss of independence, and premature mortality [1, 3–6], which all increase with advancing age.

Firefighting is known to be a dangerous occupation that is physically demanding. However, inadequate physical activity makes firefighters prone to increased obesity and the metabolic syndrome. Obese firefighters are more susceptible to gain further weight [7] and associated with decline in health status [7, 8]. We have shown previously that low physical activity and CRF are associated with unfavorable CVD risk profiles as well as higher prevalence of the metabolic syndrome (MetSyn) [9–11].

Many US firefighters receive medical- and physical-abilities testing at the beginning of their professional careers and no subsequent formal reassessment over a 20–30-year work span [12]. Nonetheless, older firefighters are expected

to be able to perform the same essential job functions as young recruits. Studies of simulated firefighting tasks suggest that the minimum aerobic capacity as measured by oxygen consumption (VO₂) necessary to safely perform firefighting duties ranges from 33.9 to 45 mL/kg/min (9.7–12.8 METS; 1 MET = 2.5 mL/kg/min) [13, 14]. Therefore, all firefighters should continue to require such relatively high aerobic capacities in order to safely perform their duties regardless of their age. Thus, this study focused on describing CRF across different age categories among professionally active career firefighters. Furthermore, we sought to determine effect modification by physical activity and BMI levels across different ages groups. We hypothesized that leaner and more active firefighters would be more likely to maintain higher aerobic capacities regardless of their age.

2. Methods

2.1. Study Population. Eligible male career firefighters, between 18 and 59 years of age were recruited from 10 fire departments in two Midwestern states. Inclusion criteria included having completed a maximal exercise test during the course of a fire department-sponsored medical examination, no work restrictions at examination, and completing the study questionnaire. Excluded subjects failed to meet one or more of the above criteria or had undergone the index exercise tests for the evaluation of symptoms, retirement pensions, disability, and/or exit examinations. The study was approved by the IRB of Harvard School of Public Health and local IRBs as appropriate, and all participants signed an informed consent.

2.2. Assessment of Cardiovascular Risk Factors. Height was measured in the standing position with a clinic stadiometer. Body weight was measured with bare feet and in light clothes on a calibrated scale. Body mass index (BMI) was calculated as the weight in kilograms divided by the square of height in meters. BMI values were also categorized according to the World Health Organization (WHO) classification.

2.3. Cardiorespiratory Fitness. CRF was determined from symptom-limited maximal treadmill exercise testing with ECG monitoring following the Bruce protocol. The participants were encouraged to continue exercise until volitional exhaustion, even after exceeding 85% of their maximum predicted heart rate defined as 220 minus age. Accordingly, the cohort achieved an average of 97.9% (SD 6.6) of maximal age-predicted heart rate on these tests. During the exercise test, total treadmill time was recorded in minutes and seconds, and then seconds were converted to fractional minutes by dividing by 60 (e.g., 8 minutes 30 seconds = 8.50 minutes). CRF was then estimated according to a widely used formula for VO₂max estimation and the Bruce protocol:

VO₂ max (mL/kg/min) =
$$14.76 - (1.379 \times T)$$

+ $(0.451 \times T^2) - (0.012 \times T^3)$,

For details on the equation see [15], "T" represents "total exercise tolerance test time" (measured in minutes). Finally, CRF estimates in maximal METS were derived by dividing the VO₂max estimates by 3.5 as per the standard conversion factor.

2.4. Assessment of Physical Activity (PA). Self-reports of PA were extracted from responses to a health and lifestyle questionnaire as previously described from our group [11]. Briefly, consented study participants were given standardized written instructions for completing the multiple-choice survey regarding eating, health, exercise, sleep, and work habits as honestly and as best as they could. They were also informed that the completed questionnaires would be confidential and would not become part of their fire department or medical record. The answers to four selected multiplechoice questions about involvement in sports and exercise were analyzed here: (1) "Most weeks, I exercise (include home/work/gym & elsewhere)"; (2) "Most times that I do cardio or aerobic exercise (e.g., jogging, brisk walking, bike, treadmill, etc.), I do an average of ... each session"; (3) "Most times that I lift weights or do strength training, I do ... on average each session"; (4) "Most times that I exercise, I sweat ... on average each session". Additionally, as described previously an additional variable was calculated to evaluate the combined effects of PA frequency and PA aerobic or cardiosession duration as total weekly aerobic exercise. Each of the six original alternatives in the frequency and aerobic session duration dimension questions received a numerical value equivalent to the middle point of the range of each response choice. Thus, a value of 2.5 times per week was assigned to the frequency response of 2-3 times per week, 3.5 for 3-4 times per week, and so on. The same methodology was applied to the duration responses: 22.5 minutes was assigned for 15 to 30 minutes, 37.5 minutes for durations of 30 to 45 minutes, and so on. Subsequently, the resulting frequency and duration values were multiplied to estimate total weekly exercise in minutes per week.

2.5. Statistical Analysis. Baseline characteristics were described using the mean (SD) in the case of quantitative variables and the frequency in case of categorical variables. Group comparisons were calculated using ANOVA. Linear models and logistic regression models were used to adjust for other variables.

The prediction model for CRF was constructed by dividing the whole dataset at random into a "training" (or analyzed) (n=405) subgroup and a "testing" (or validation) (n=399) subgroup. A linear regression model was used to assess the coefficients for the included variables in the training subsets. To assure linear correlations between outcome and covariates, all covariates were also included additionally as squared variables. After backwards elimination of non-significant squared variables only the squared term for the total weekly exercise duration remained in the final model.

The final model derived from the training set was then used in the testing dataset to predict CRF and compare it to actual peak CRF values measured in the clinic. Pearson's

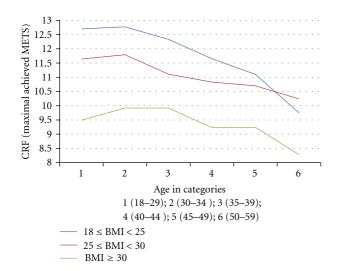


FIGURE 1: Cardiorespiratory fitness across different age categories and stratified by BMI categories.

correlation coefficient and scatter plots were used to compare the predicted values from the model with the actual observed values in the testing dataset.

Analyses were performed using SAS 9.2 (SAS Institute Inc., Cary, NC, USA). All tests presented are two-sided, and a *P* value < 0.05 is considered significant.

3. Results

Table 1 describes the baseline fitness and PA characteristics of our cohort (Table 1). Most of the firefighters are under 40 years of age, with 40% between the age of 30 and 39. Nearly half of the study population is overweight ($25 \le BMI < 30$), and 36% are obese (BMI ≥ 30). The estimated total weekly exercise duration of the cohort is 102.5 minutes (SD 86.9) (Table 1).

Maximal achieved METS—across increasing age and stratified by different BMI categories—are presented in Figure 1. CRF declined over the different age categories in each of the BMI groups, though the lines for the three BMI groups are clearly separated with the normal BMI group demonstrating the highest CRF and the obese group demonstrating the lowest CRF. A similar relationship between CRF, BMI, estimated duration of weekly exercise, and different age categories is illustrated in Figure 2. The trajectory of CRF in firefighters who are obese and exercise little starts and finishes much lower in comparison to leaner colleagues who exercise more.

The predicted relationship between CRF over the career span depending on different BMI categories and weekly exercise duration pattern is illustrated in Figure 3. Even in two hypothetical situations where BMI is the same, firefighters who exercise more have less decrement in fitness levels.

The linear regression model predicting CRF in the training set of our cohort is presented in Table 2. Age, BMI, total weekly aerobic duration (singular and squared), and

Table 1: Baseline characteristics (n = 804).

()	,			
Age mean (SD)	37.4 (8.4)			
BMI mean (SD)	29.3 (4.4)			
CRF (maximal METS) mean (SD)	10.7 (2.0)			
Cardio exercise per session $n(\%)$				
None	44 (5.5)			
<15 minutes	74 (9.3)			
15–30 minutes	340 (42.8)			
30–45 minutes	228 (28.7)			
45–60 minutes	67 (8.4)			
>60 minutes	41 (5.2)			
Minutes of weight training per session $n(\%)$				
None	88 (11.0)			
<15 minutes	106 (13.3)			
15–30 minutes	228 (28.6)			
30–45 minutes	178 (22.3)			
45–60 minutes	106 (13.3)			
>60 minutes	91 (11.4)			
Intensity of training $n(\%)$				
Do not exercise often	28 (3.5)			
Light sweat	146 (18.4)			
Moderate sweat	445 (56.0)			
Heavy sweat	176 (22.1)			
Weekly exercise frequency $n(\%)$				
Never	34 (4.3)			
1 or less	96 (12.1)			
2-3	244 (30.6)			
3-4	246 (30.9)			
5-6	132 (16.6)			
Every day	44 (5.5)			
Estimated total weekly duration mean (SD) (in minutes)				
Tertile one	30.7 (23.2)			
Tertile two	94.6 (18.7)			
Tertile three	195.2 (81.3)			
Estimated total weekly exercise duration mean (SD)	102.5 (86.9)			
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BMI (body mass index), CRF (cardiorespiratory fitness), SD (standard deviation).

duration of weight lifting each week produced statistically significant P values to estimate maximal achieved fitness. Every unit increase in age leads to a decline in METS of -0.05886 times age after adjusting for all the other factors in the model.

The calculated estimates were evaluated in a testing set, and the Pearson's correlation coefficient was computed to be 0.6286. The correlation between the estimated and actual CRF is displayed in a scatter plot (Figure 4). The mean, maximum, and minimum values of the actual observed METS and of the predicted METS based on the model are shown in Table 3.

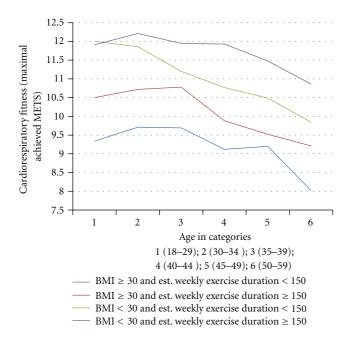


FIGURE 2: Cardiorespiratory fitness across different age categories and stratified by BMI/estimated weekly exercise duration.

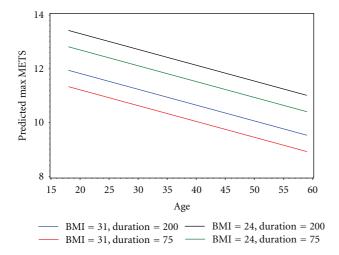


FIGURE 3: Predicted maximal Cardiorespiratory fitness (CRF) over the age span with the model calculated with the training set.

4. Discussion

As expected we found an age-related decline of CRF in our cohort of male career firefighters.

However, the starting, intermediate, and final points for CRF across the career span differ markedly according to the firefighters-measured BMI and self-reported physical activity. While obese firefighters had lower CRF values across the age spectrum compared to normal weight subjects (Figure 1), physical exercise attenuates the decline (Figure 2). Additionally we have demonstrated that CRF can be predicted with a high correlation coefficient including age, BMI, and variables describing physical fitness habits in the model.

Table 2: Linear regression model describing Cardiorespiratory fitness (maximal achieved METS) (in randomly assigned training set n = 405).

Variable	Parameter estimate	Standard error	P value
Age	-0.05886	0.01031	< 0.0001
BMI	-0.21131	0.01994	< 0.0001
Total weekly duration	0.00845	0.00232	0.0003
Total weekly duration squared	-0.00001297	0.000000569	0.0233
Duration of weight lifting	0.18937	0.06393	0.0032
Intensity of exercise	0.21331	0.12362	0.0878

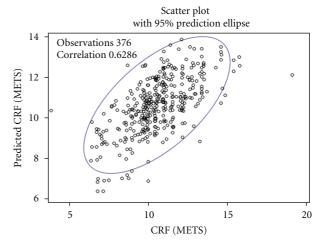


FIGURE 4: Scatter plot of predicted Cardiorespiratory fitness compared to actual values in the testing set.

Table 3: Estimated Cardiorespiratory fitness (maximal achieved METS) and actual maximal METS in testing set (n = 399).

	n	mean (SD)	Minimum	Maximum
Max. METS	386	10.56363 (1.87003)	3.90317	19.01499
Estimated max. METS	384	10.59689 (1.37406)	6.39852	13.84193

Based on our results, albeit cross-sectional, much of the age-related decline in CRF is potentially preventable by maintaining high levels of physical activity and a healthy BMI. The ability to modify aging related declines in CRF is especially important among firefighters who are so dependent on their maximal oxygen consumption in order to safely perform their duties, and as a profession, it has cardiovascular events as the leading cause of on-duty death.

Unpredictable bouts of strenuous activity in otherwise sedentary individuals are a known precursor or acute coronary events [16], as well as periods of emotional stress [17]. Firefighters are much more likely to experience cardiovascular disease (CVD) events during periods of alarm response of fire suppression/active firefighting than during other duties at the firehouse [18].

In the general population the longitudinal decline in peak maximal oxygen consumption is not linear across the age span but accelerates markedly especially in men [2]. This nonlinear decline accelerates especially after 45 years of age [19]. In our cohort, especially in firefighters among higher age groups, the cross-sectional age-related decline in CRF seems to be more prominent.

4.1. Strength of the Present Study. A particular strength of the present study was the large sample size which allowed for adequate power and adjustment for confounders. BMI was calculated from measured weight and height in bare feet and light clothes during medical examinations, which avoided self-reporting biases towards lower weights and taller heights and other misclassification.

4.2. Limitations of the Present Study. Our study does have some modest limitations, including the cross-sectional design, which does not allow us to establish causation. Because of the very small number of female career firefighters, we limited our investigation to male firefighters. We have evaluated physical activity through a questionnaire, which can lead to overestimation of underlying activity levels. However, in our investigation this results in even more conservative findings, that is, would tend to overestimate PA among those with low fitness and lower the effects of PA on CRF. A minor limitation regards the use of a maximal treadmill test without expired gases using peak METS as the outcome estimates true VO₂max, while a measured VO₂max, using expired gases, would have been considered the "gold standard". Furthermore, the prediction model derived from our cohort study will have to be tested in other cohorts.

5. Conclusion

CRF in career male firefighters is significantly reduced with increasing age. The age-related decrements in CRF, however, are highly dependent on firefighters' BMI and physical exercise habits. Firefighters who are fitter and leaner have higher baseline CRF and attenuated CRF decrements. Our investigation supports the importance of measuring CRF, physical activity and parameters of body composition and cardiovascular health at the entry-and at regular time points—throughout a firefighter's career. This would most probably be a valuable tool in preventing the above described excessive decline in CRF with increasing age of the individual firefighter. Furthermore, especially in a profession where preservation of high fitness levels is imperative for firefighters—as well as public safety—fire departments should emphasize the maintenance of frequent physical activity as well as maintaining a healthy weight.

Disclosure

S. N. Kales has served as expert witness in medicolegal cases involving firefighters and is working under contract to revise the Heart Disease Manual of the International Association of Fire Fighters. The other authors report no conflict of interests

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References

- [1] L. Hakola et al., "Cardiorespiratory fitness in aging men and women: the DR's EXTRA study," *Scandinavian Journal of Medicine & Science in Sports*, vol. 21, no. 5, pp. 679–687, 2011.
- [2] J. L. Fleg, C. H. Morrell, A. G. Bos et al., "Accelerated longitudinal decline of aerobic capacity in healthy older adults," *Circulation*, vol. 112, no. 5, pp. 674–682, 2005.
- [3] S. E. Brien, P. T. Katzmarzyk, C. L. Craig, and L. Gauvin, "Physical activity, cardiorespiratory fitness and body mass index as predictors of substantial weight gain and obesity: the Canadian physical activity longitudinal study," *Canadian Journal of Public Health*, vol. 98, no. 2, pp. 121–124, 2007.
- [4] M. R. Carnethon, S. S. Gidding, R. Nehgme, S. Sidney, D. R. Jacobs, and K. Liu, "Cardiorespiratory fitness in young adulthood and the development of cardiovascular disease risk factors," *Journal of the American Medical Association*, vol. 290, no. 23, pp. 3092–3100, 2003.
- [5] M. Hassinen, T. A. Lakka, L. Hakola et al., "Cardiorespiratory fitness and metabolic syndrome in older men and women: the Dose Responses to Exercise Training (DR's EXTRA) study, "Diabetes Care, vol. 33, no. 7, pp. 1655–1657, 2010.
- [6] D. E. Laaksonen, H. M. Lakka, J. T. Salonen, L. K. Niskanen, R. Rauramaa, and T. A. Lakka, "Low levels of leisuretime physical activity and cardiorespiratory fitness predict development of the metabolic syndrome," *Diabetes Care*, vol. 25, no. 9, pp. 1612–1618, 2002.
- [7] E. S. Soteriades, R. Hauser, I. Kawachi, D. Liarokapis, D. C. Christiani, and S. N. Kales, "Obesity and cardiovascular disease risk factors in firefighters: a prospective cohort study," *Obesity Research*, vol. 13, no. 10, pp. 1756–1763, 2005.
- [8] S. N. Kales, G. N. Polyhronopoulos, J. M. Aldrich, E. O. Leitao, and D. C. Christiani, "Correlates of body mass index in hazardous materials firefighters," *Journal of Occupational and Environmental Medicine*, vol. 41, no. 7, pp. 589–595, 1999.
- [9] D. M. Baur et al., "Cardiorespiratory fitness predicts cardiovascular risk profiles in career firefighters," *Journal of Occupa*tional and Environmental Medicine, vol. 53, no. 10, pp. 1155– 1160, 2011.
- [10] D. M. Baur, C. A. Christophi, and S. N. Kales, "Metabolic syndrome is inversely related to cardio-respiratory fitness in male career firefighters," *The Journal of Strength & Conditioning Research*. In press.
- [11] G. Durand, A. J. Tsismenakis, S. A. Jahnke, D. M. Baur, C. A. Christophi, and S. N. Kales, "Firefighters' physical activity: relation to fitness and cardiovascular disease risk," *Medicine*

& Science in Sports & Exercise, vol. 43, no. 9, pp. 1752-1759, 2011

- [12] E. S. Soteriades, D. L. Smith, A. J. Tsismenakis, D. M. Baur, and S. N. Kales, "Cardiovascular disease in US firefighters: a systematic review," *Cardiology in Review*, vol. 19, no. 4, pp. 202–215, 2011.
- [13] K. L. Elsner and F. W. Kolkhorst, "Metabolic demands of simulated firefighting tasks," *Ergonomics*, vol. 51, no. 9, pp. 1418– 1425, 2008.
- [14] I. Holmér and D. Gavhed, "Classification of metabolic and respiratory demands in fire fighting activity with extreme workloads," *Applied Ergonomics*, vol. 38, no. 1, pp. 45–52, 2007.
- [15] C. Foster, A. S. Jackson, and M. L. Pollock, "Generalized equations for predicting functional capacity from treadmill performance," *American Heart Journal*, vol. 107, no. 6, pp. 1229–1234, 1984.
- [16] M. A. Mittleman, M. Maclure, and G. H. Tofler, "Triggering of acute myocardial infarction by heavy physical exertion. Protection against triggering by regular exertion. Determinants of Myocardial Infarction Onset Study Investigators," *The New England Journal of Medicine*, vol. 329, no. 23, pp. 1677–1683, 1993.
- [17] U. Wilbert-Lampen, D. Leistner, S. Greven et al., "Cardiovascular events during World Cup Soccer," *The New England Journal of Medicine*, vol. 358, no. 5, pp. 475–483, 2008.
- [18] S. N. Kales, E. S. Soteriades, C. A. Christophi, and D. C. Christiani, "Emergency duties and deaths from heart disease among firefighters in the United States," *The New England Journal of Medicine*, vol. 356, no. 12, pp. 1207–1215, 2007.
- [19] A. S. Jackson, X. Sui, J. R. Hébert, T. S. Church, and S. N. Blair, "Role of lifestyle and aging on the longitudinal change in cardiorespiratory fitness," *Archives of Internal Medicine*, vol. 169, no. 19, pp. 1781–1787, 2009.

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Review Article

Blood Pressure Control at Rest and during Exercise in Obese Children and Adults

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The hemodynamic responses to exercise have been studied to a great extent over the past decades, and an exaggerated blood pressure response during an acute exercise bout has been considered as an indicator of cardiovascular risk. Obesity is a major factor influencing the blood pressure response to exercise since evidence indicates that the arterial pressure response to exercise is exacerbated in obese compared with lean adults. Signs of augmented responses (such as an exaggerated blood pressure response) to physical exertion appear early in life (from the prepubertal years) in obese individuals. Understanding the mechanisms that drive the altered hemodynamic responses during exercise in obese individuals and prevent the progression to hypertension is vitally important. This paper focuses on the evidence linking obesity with alterations of the autonomic nervous system and discusses the potential mechanisms and consequences of the altered sympathetic nervous system behavior in obese individuals at rest and during exercise. Furthermore, this paper presents the alterations in the reflex regulatory mechanisms ("exercise pressor reflex" and baroreflex) in obese children and adults and addresses the effects of training on obesity-related disturbances.

1. Introduction

The hemodynamic responses to exercise have been extensively studied over the past decades. An exaggerated blood pressure response during an acute dynamic exercise bout (defined as an increase in systolic blood pressure from rest of >10 mmHg per metabolic equivalent or a diastolic blood pressure change of >10 mmHg at any workload) [1] has been considered as an indicator of cardiovascular risk [2–6]. Miyai et al. [7] showed a significant and independent threefold higher risk for future hypertension in middle-aged normotensive men with a disproportionate exercise response. A consistent relationship between resting blood pressure and blood pressure decline during postexercise recovery has also been reported [8-10]. Factors that have been accounted to influence the arterial pressure responses to exercise include resting arterial pressure levels, age, gender, ethnicity, family history of hypertension, and other genetic factors, hyperlipidemia, and obesity and physical fitness levels [11-13]. In

fact, obesity is a major factor influencing the blood pressure response to exercise since evidence indicates that the arterial pressure response to exercise is exacerbated in obese compared with lean adults. Signs of altered responses to physical exertion, such as augmented blood pressure response or a chronotropic incompetence appear early in life (from the prepubertal years) in obese individuals [14, 15]. Understanding the mechanisms that drive the altered hemodynamic responses during exercise in obese individuals and prevent the progression to hypertension is vitally important.

The present paper will focus on the evidence linking obesity with alterations of the autonomic nervous system and discuss the potential mechanisms and consequences of the altered sympathetic nervous system (SNS) behavior in obese individuals at rest and during exercise. First, the mechanisms and reflexes mediating the blood pressure responses to exercise will be introduced. Next, the SNS behavior in obese individuals at rest and the consequences of alterations in SNS behavior to the blood pressure response during exercise will

be presented. Alterations in the reflex regulatory mechanisms (such as the "exercise pressor reflex" and baroreflex) in obese children and adults will also be addressed. Finally, we will conclude with studies examining the effects of training on these obesity-related disturbances and future research recommendations.

2. Mechanisms Mediating Blood Pressure Responses to Exercise

Neural control of the circulation during exercise is a multifactorial phenomenon involving higher brain centers and peripheral reflexes (Figure 1). During exercise, descending signals from a network of higher cerebral regions, from the caudal diencephalon to the rostral mesencephalon, including the ventral tegmental area [16, 17] (termed as "central command"), result in parasympathetic and sympathetic adjustments [18]. In addition, neural signals from peripheral afferents originating from the arterial baroreceptors (termed as "arterial baroreflex") [19] and from the skeletal muscle [20, 21], result in changes in the autonomic outflow. This system mediates the characteristic adjustments to exercise by controlling heart rate, stroke volume, vascular resistance, and thereby, maintaining blood pressure [22, 23].

Central command has been defined as a "feedforward" mechanism capable of activating simultaneously two separate networks, the *motor* and the *cardiovascular* control center. Central command is very important at the onset of exercise for initiating a basal level of autonomic activity and can be dictated, at least partially, by the individual's perception of effort [24, 25]. Central command plays an important role in the resetting of arterial baroreflex during exercise [26]. This effect appears to be mediated via vagal withdrawal coupled to the increase in exercise intensity.

On the other hand, "feedback" mechanisms, consisting of receptors within the exercising muscle, activate afferent nerves, and induce circulatory and respiratory adjustments to provide sufficient blood flow and remove the metabolic byproducts of the working muscle. This neural mechanism arising from the mechanical and chemical receptors within the muscle is termed the "exercise pressor reflex" [18, 21, 27]. The mechanical receptors are composed mainly by group III afferents which discharge early at the onset of contraction (mechanoreflex), whereas the metabolic receptors are composed mainly by group IV afferents that discharge later (with a latency of 5–60 seconds) with the accumulation of metabolites.

Bradykinin, K⁺, arachidonic acid, analogues of ATP, diprotonated phosphate, and prostaglandins are some of the substances that have been reported to activate the nerve endings in the skeletal muscle and blood vessels [20, 21, 28]. Lactic acid has also been considered as a metabolic stimulus to group IV muscle afferents [29, 30]. Injection of lactic acid into the skeletal muscle artery has been shown to evoke reflex increases in arterial pressure that mimic those occurring during static exercise, whereas, injection of sodium lactate at a neutral pH failed to evoke a similar effect [31]. However, the demonstration that lactic acid elicits an exercise pressor reflex has been difficult since its receptor on thin

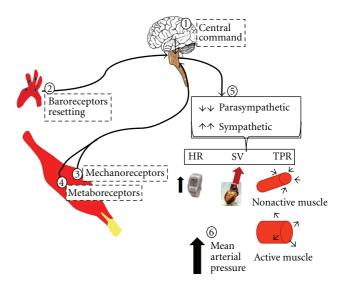


FIGURE 1: During exercise, neural signals originating from higher cerebral regions (1) and peripheral afferents from arterial baroreceptors (2) and the skeletal muscle ("exercise pressor reflex", 3 & 4) result in parasympathetic and sympathetic adjustments (5). Consequently, mean arterial pressure rises (6). HR: Heart Rate; SV: Stroke Volume; TPR: Total Peripheral Resistance.

afferents (group II and IV) has not been clearly identified. Recently, a direct effect of protons (H⁺) on the muscle receptor itself has been suggested [32] and the acid sensing ion channel (ASIC) was shown to play a role in evoking the metabolic component of the exercise pressor reflex [33]. The hemodynamic consequence of mechanometaboreflex activation is a rise in mean arterial blood pressure [21].

Alterations in the reflex regulatory mechanisms of arterial pressure and sympathetic system disturbances have been reported in obese individuals and have been associated with the increased prevalence of hypertension and cardiovascular disorders in obesity. The mechanisms underlying the altered blood pressure responses in obese individuals have not been completely identified. Methodological refinements in the assessment of sympathetic autonomic drive throughout the years have allowed a better understanding of the role of the sympathetic nervous system in the development of obesity-induced hypertension.

3. Methods of Assessing Autonomic Nervous System Behavior

Over the years, different methodological approaches have been used to study the autonomic system behavior in obese individuals. The techniques currently applied for studying sympathetic activity in humans are (i) regional nore-pinephrine spillover, which measures the amount of nore-pinephrine released from sympathetic terminals from the coronary sinus or the renal veins using a radiotracer infusion, such as tritiated norepinephrine, and sampling of blood from the venous drainages of interest, using a central venous catheter, (ii) clinical microneurography, which measures postganglionic sympathetic nerve firing rates, bursts, in

subcutaneous nerves either of skin or skeletal muscle [34], (iii) several types of radio scanning imaging (such as positron emission tomography and single photon emission scanning), and (iv) indirect hemodynamic techniques, using blood pressure and heart rate in the assessment of sympathetic and vagal activity (such as heart rate variability, blood pressure variability, and baroreflex sensitivity measures). The first two methods described, that is, the isotope dilution methodology and sympathetic nerve recording techniques, quantify neurotransmitter release and are considered gold standards for assessing regional sympathetic nervous function in humans [35]. Skeletal muscle sympathetic nerve activity (MSNA) provides valuable information on sympathetic nerve activity at the muscle level and is an online dynamic assessment, which is highly reproducible in humans. However, MSNA does not give access to the sympathetic nerves of internal organs as the rate of spillover of norepinephrine. Ideally, combining measures of cardiac norepinephrine spillover with electrical activity of the cardiac sympathetic nerves would have yielded very conclusive evidence. However, since both techniques are invasive, simultaneous experimentation is very difficult and the information provided is restricted to laboratory environment. Therefore, other noninvasive techniques such as analysis of heart rate variability (based on mathematical models of variations in heart rate) are also available to determine the balance between cardiac sympathetic and vagal activity. Although these noninvasive methods have a number of methodological limitations that should be considered, as described by the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology [36], they can be employed complementary to the other methods or in studies involving healthy participants (including children). Recent advantages in heart rate variability calculation methods, such as geometrical and graphical representation (as the Poincaré plot) [37] are gaining increasing interest and can be used during exercise, since they do not require stationarity of data that was required in the time domain analysis of heart rate variability.

4. Sympathetic Nervous System Behavior in Obese Individuals at Rest: Sympathetic Underactivity versus Sympathetic Overactivity

Until recently, the relationship between obesity and SNS behavior has been controversial, since a number of studies suggested that low SNS was causal in the development of obesity [38, 39], while others, claimed that obesity was associated with high SNS behavior [40]. The reasons for these discrepancies include methods of SNS assessment as well as, the target organ examined, as SNS activity demonstrates regional specificity and thus, SNS outflow to one organ may not be similar to SNS outflow targeting other organs.

In individuals with obesity, whole body norepinephrine spillover rate (an indication of an overall sympathetic activity) has been reported similar to that in lean individuals [41, 42]. However, studies assessing cardiac SNS activity by the

cardiac norepinephrine spillover rate have reported lower spillover rate (by approximately 50%) at the heart level in obese adults compared with nonobese normotensive adults, whereas renal SNS activity has been reported higher (double spillover rate of norepinephrine) in obese compared with their nonobese normotensive counterparts [41, 42]. Reduced norepinephrine spillover has been also suggested in white adipose tissue of obese individuals [43].

Direct recordings of efferent postganglionic muscle sympathetic nerve traffic via microneurography (muscle MSNA) have conclusively documented that obese individuals exhibit a noticeable increase (by as much as a twofold increase) in MSNA compared with nonobese adults during rest [44]. Visceral fat, independently of total body fat was correlated with increased basal MSNA, linking the altered SNS response with body fat distribution [45, 46]. Increased visceral fat and elevated MSNA have also been implicated in the development of obstructive sleep apnea in obese adults [47–51].

Potential mechanisms for the increased SNS activity in obese individuals include hyperinsulinemia [52, 53], hyperleptinemia [34], activation of the renin-angiotensin-aldosterone system [54–56], and mitochondrial dysfunction [57, 58]. Altered neurohumoral signals arising from the hypothalamic pituitary adrenal axis, as well as increased adipokines (adiponectin, ghrelin) [49], and dyslipidemia [59], can also be contributing factors to the observed SNS disturbances [34, 60, 61]. The "neurogenic" hypothesis of obesity has been previously reviewed [42, 59, 62]. Therefore, in this brief review we will next discuss recent findings on the alterations of reflexes controlling the blood pressure response during exercise in obese humans.

5. Sympathetic Nervous System Behavior in Obese Individuals during Exercise

In obese individuals, skeletal muscle sympathetic nerve hyperactivity is evident at rest [63]; however, during physiological stimuli, a reduced SNS responsiveness has been observed [64]. During sympathoexcitation induced by a cold pressor test, forearm vascular resistance (assessed by venous occlusion plethysmography) has been reported significantly higher in obese women compared with lean women; however, blood pressure and heart rate (monitored noninvasively by finger photoplethysmography on a beat by beat basis) similarly increased in obese and lean individuals [65]. Although in the latter study MSNA was higher in the obese than the lean group, the magnitude of the MSNA response was similar between the two groups. Negrão et al. [64] showed that normotensive obese women had enhanced resting MSNA and lower forearm blood flow than lean women [65]. During isometric handgrip exercise at 10% of the maximum voluntary contraction (MVC), when central command and mechanoreceptors are the main contributors to pressure response, the MSNA adaptation to exercise was found to be similar in obese and in lean individuals, although the absolute levels of MSNA were found higher in obese individuals. However, during 30% MVC, when central command, mechanoreceptors, and metaboreceptors are activated, the MSNA exercise adaptation was found to be blunted

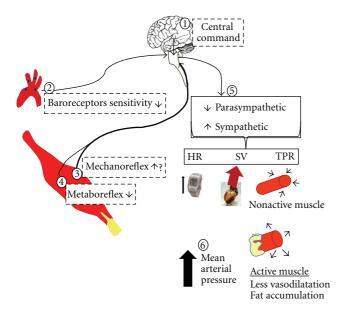


FIGURE 2: Alterations in the control of blood pressure during exercise in obese individuals: the baroreflex is less sensitive to stimulation (i.e., exercise) and the metaboreflex is blunted. Mechanically sensitive afferent neurons may therefore undergo functional changes to compensate for the reduced metaboreflex. These alterations may promote chronic adjustments in peripheral vascular resistance, precipitate fatigue during exercise and delay vasodilatation during exercise recovery. HR: Heart Rate; SV: Stroke Volume; TPR: Total Peripheral Resistance.

in obese women, suggesting that selective activation of metaboreceptors during exercise is impaired in obese females. This view was further explored by isolating the metaboreflex response by postexercise occlusion. During posthandgrip circulatory arrest, when the mechanoreflex and the central command are no longer active, the MSNA responses were blunted in normotensive middle-aged obese compared with lean females [64]. These findings imply that mechanically sensitive afferent neurons may undergo functional changes to compensate for the reduced metaboreflex and result in an increase in mean arterial pressure. During exercise of higher intensity, the mechanoreflex may not be able to compensate for the blunted metaboreflex, muscle perfusion might be impaired, and fatigue may appear earlier. In fact, blunted muscle perfusion to local dynamic exercise has been reported in obese individuals [66]. A proposed model for the altered exercise pressor reflex in obese individuals is presented in Figure 2.

The mechanisms underlying the blunted metaboreflex control in obese individuals are not entirely clear. Possibly, the increased fat content in the skeletal muscle in obese individuals leads to a desensitization of metaboreceptors. The reduced skeletal muscle glucose uptake observed in obesity [67] can also result in an attenuated level of acidosis in the muscle during exercise, and thus, a lower activation of metaboreceptors. A baroreflex involvement can also be implicated to this dysfunction.

Alterations in the arterial baroreflex are linked to SNS hyperactivity and blunted arterial distensibility [68, 69]. Reduced postexercise baroreceptor sensitivity and impaired autonomic regulation have been associated with an attenuated recovery of heart rate and total peripheral resistance following brisk walking in middle-aged obese women [10]. In a recent study, Fardin et al. [70] showed that baroreceptor dysfunction in obese rats (exposed to a high-fat diet) was associated with renal SNS hyperactivity. Whether this finding extend to obese humans remains to be investigated.

Preserved blood flow (assessed by Doppler) during dynamic steady-state forearm (20 contractions/min at 4, 8, and 12 kg) and leg (40 kicks/min at 7 and 14 W) exercise was found in the exercising limb in obese young healthy adults, indicating that steady-state levels of flow can be maintained via compensatory mechanisms [71]. However, in another study [72], a marked impairment in rapid vasodilatation was evident in the immediate postexercise period in obese adults and was greater with increasing workloads (from 20-50% of MVC). Differences in the reported results regarding blood flow during exercise in obese individuals could be attributed to the exercise intensity applied (similar absolute versus similar relative workload) [72], the mode of exercise (dynamic versus isometric), and the hormonal and metabolic profile of the participants (healthy versus hyperinsulinemic). The exact mechanism for the reduced postexercise vasodilatation and the delayed decline in blood pressure in obese humans is not clear. However, impaired potassium channel-mediated vasodilatation has been reported in the skeletal muscle of obese Zucker rats or hamsters [73, 74] and could be partially involved in humans.

Signs of altered reflexes were detectable even when normotensive obese adults (defined in studies as systolic/diastolic blood pressure <140/90 mmHg) were tested [62], suggesting that early detection of these abnormalities can be used as a prognostic tool. Weight loss induced by a hypocaloric diet partially reversed the attenuated metaboreflex response [75] and improved baroreceptor sensitivity in obese normotensive individuals [63]. In addition, weight reduction induced favorable adaptations on the MSNA responses during mental stress in obese women and improved forearm vascular conductance at rest [76]; however, these beneficial adaptations were only obtained when the hypocaloric diet (600 kcal/day dietary reduction) was accompanied by exercise training and not by a similar dietary caloric restriction alone [76]. The diet plus exercise training group in that study performed three 60 min exercise sessions per week of aerobic (40 min per session) and strengthening exercise for four months. Since both groups exhibited a similar weight loss and changes in body composition were not assessed, it is not clear whether increases in lean body mass and a greater decrease in percent body fat resulted in the beneficial effects on MSNA and vascular conductance. Moreover, the effects of weight loss and weight loss maintenance on sympathetic activity might be divergent and organ-specific adaptations might exist. Straznicky et al. [77] showed that after weight maintenance, the beneficial effects of weight loss on norepinephrine spillover rate were preserved, whereas MSNA and baroreflex sensitivity

adaptations were not maintained. Future studies should examine the mechanisms by which exercise training exerts advantageous alterations in the exercise pressor reflex and investigate whether the beneficial effects on sympathovagal control are linked to changes in body composition. The effects of resistance training or high intensity exercise, as well as possible gender differences in the exercise pressor reflex and the muscle metaboreflex should also be investigated.

6. Control of Blood Pressure at Rest and during Exercise in Obese Children

The autonomic nervous system undergoes changes from childhood to adulthood. Body size, muscle characteristics, energy systems involved, and arterial stiffness also change [78–80]. Therefore, applying the information that we have learned from studies in obese adults to studies in obese children, might be not be appropriate. Up-to-date, only a few studies have directly compared the blood pressure responses and the sympathovagal involvement to the control of the exercise pressor reflex in children and in adults. A lower response of blood pressure (using finger photoplethysmography) during isometric handgrip exercise and lower baroreceptor sensitivity has been reported in children than in adults [81]. However, the exercise pressor reflex in that study was examined as a whole, involving all peripheral reflexes; therefore the role of the each of the reflexes (metaboreflex, mechanoreflex) to the control of blood pressure in children is not clear. Only one study [82] attempted to isolate the exercise-induced increase in metabolite concentration (using circulatory occlusion) to the control of blood pressure during handgrip exercise in children and reported similar responses in children and in adults. In the latter study, the blood pressure measurements were not performed on a beat-by-beat basis and the sympathetic/vagal involvement to the blood pressure response was not examined. Furthermore, faster postexercise hemodynamics have been reported in young children compared with adolescents [83] and adults [84, 85], linked with a faster parasympathetic outflow [81] or changes in lean body mass and blood acidosis [83, 86].

In children, increased levels of adiposity have been partially linked to autonomic nervous system dysfunction and an increased prevalence of hypertension in adulthood [87]. Yet, the exact mechanisms by which childhood obesity leads to hypertension remain unclear. Increased resting blood pressure [88], reduced baroreceptor sensitivity [89], and decreased resting forearm blood flow have been reported from an early stage in obese children [90]. During sympathetic stimulation induced by isometric handgrip exercise, the arterial blood pressure response has been reported similar [15] or higher [88] in obese compared with lean aged-matched children. Differences in reported blood pressure responses could be partly or collectively explained by (i) basal blood pressure differences, (ii) variations in body fat distribution [91], (iii) differences in participants' age, since cardiovagal autonomic function undergoes a gradual maturation during childhood [92], and (iv) method of blood pressure measurement (continuous beat-by-beat finger photoplethysmography, or intermittently every 60 or 120 s by a conventional aneroid sphygmomanometer). Genetic factors and family history of hypertension can also be contributing factors, since offsprings of parents with a family history of hypertension exhibit higher basal blood pressure levels and greater mean arterial pressure during mental stress and handgrip exercise than children of normotensive parents [93]. Low fitness levels can also exaggerate the blood pressure response to exercise in children with obesity. In a recent study by Legantis et al. [94], obese and overweight unfit children exhibited an exaggerated systolic blood pressure response (assessed by finger photoplethysmography) during isometric handgrip exercise compared with their fit overweight and obese counterparts.

Even in the absence of any alterations in resting blood pressure, signs of disturbed hemodynamic control during acute isometric handgrip exercise (at 30% MVC) and recovery were evident in obese prepubertal boys [15]. Dipla et al. [15] reported an attenuated increase in exercise heart rate, associated, at least partially with a lower vagal withdrawal in obese compared with lean boys. However, the magnitude of the blood pressure response to exercise (assessed as the change from baseline, using beat-by-beat finger photoplethysmography) was similar in lean and obese boys. In addition, a lower decline in baroreceptor sensitivity during isometric exercise was found in obese versus lean preadolescent children [15].

During dynamic exercise, a blunted muscle perfusion response to lower limbs has been reported in overweight children [66], possibly linked with arterial endothelial dysfunction [56, 95]. Furthermore, early signs of vascular dysfunction in obese children have been demonstrated in the postexercise period, as evident by a reduced capacity for vasodilatation in the recovery from isometric exercise in obese compared with lean boys [15]. These vascular reactivity dysfunctions during exercise (dynamic or isometric) and recovery, are possibly also the result of accumulation of perivascular adipose tissue (surrounding the blood vessels) and intima media thickening [96].

Isolating the metaboreflex activation by postexercise occlusion, Dipla et al. [15] found similar blood pressure responses in normotensive obese and lean prepubertal children. However, the relative contribution of total peripheral resistance and stroke volume to this response was altered in obese preadolescent boys, even in the absence of a fully developed metabolic syndrome.

A limitation of studies currently available in children is that the autonomic nervous system involvement during exercise was assessed indirectly, using hemodynamic parameters and not direct measurement of norepinephrine spillover rate or MSNA. Although heart rate variability provides an indication of the heart's ability to respond to multiple physiological stimuli and can identify phenomena related to autonomic nervous system, it is not considered as precise as the measurement of norepinephrine spillover. However, its noninvasive and nonpharmacologic nature makes it appealing for use in the pediatric population, especially when healthy lean children participate as controls.

7. Effects of Training and Physical Activity on Blood Pressure Control

This section will first discuss findings in studies examining the effects of training to the neural control of blood pressure in obese adults and then continue with findings in obese children. In a large cohort study, Felber-Dietrich et al. [97] showed that participation in regular physical activity programs has beneficial effects on cardiac autonomic function (assessed by 24-hour recordings of heart rate variability) and tends to offset the negative effects of obesity on autonomic nervous system indices. More specifically, obese individuals exercising regularly >2 h/week exhibited heart rate variability values closer to those obtained by normal weight individuals. Cardiac autonomic function in those who gained weight while exercising regularly was improved compared to sedentary subjects who gained weight. Although a major strength of that study is the involvement of a large number of participants, a limitation is that the amount of physical activity was assessed by a questionnaire. In addition, since body composition was not assessed in that study, it is possible that the observed improvements in autonomic function in the active obese individuals, is at least partly the result of an increase in their lean body mass. The important role of physical activity on vascular health has been demonstrated by a study showing that cessation of physical activity in healthy adults during bed rest resulted in a rapid decline of vascular reactivity (within 3-5 days) [98]. Aerobic exercise training in obese individuals induces metabolic alterations, such as increases in bioavailability of nitric oxide and in glucose transporter (GLUT-4) concentration within the skeletal muscle and increases in the number of highly oxidative and insulin-sensitive type I fibers in adults [99, 100]. These changes have been associated with improved endothelial cell function (assessed by brachial artery flow mediated dilatation) [99]. Whether improvements in the autonomic nervous system function in response to training contribute to these alterations remains to be elucidated.

Early detection of vascular dysfunction is important to initiate behavioral interventions to reduce the risk of developing cardiovascular disease. In children, sedentary behavior is a major factor linking adiposity to insulin resistance, inflammation, and endothelial dysfunction. Controlled studies that examine whether exercise training interventions reverse the alterations observed in baroreflex sensitivity and in the exercise pressor reflex in obese children are not currently available. Previous studies have indicated that exercise training improves vascular reactivity. More specifically, Watts et al. [101] showed that obese children and adolescents had a 50% lower vascular reactivity compared with their control peers during rest and reduced free-living physical activity was related to vascular dysfunction (assessed by flow-mediated dilatation) in children [102]. Furthermore, Hopkins et al. [103] showed that the amount of moderate to vigorous activity was closely associated with increased vascular reactivity and health. Prado et al. [104] showed that a hypocaloric diet accompanied by exercise training, accelerated the postexercise heart rate recovery, more than diet alone, suggesting an additive effect of increased cardiorespiratory fitness by exercise training on cardiac autonomic activity. Importantly, the improved arterial endothelial function following an eightweek aerobic exercise training program was evident even in the absence of weight reduction in overweight children [99]. These results highlight the importance of increased fitness levels in vascular health in obese and overweight children [94] and imply that metabolic and hormonal adaptations induced by exercise training plays a major role in lowering risk factors.

In many studies that examined the effect of weight loss on the reflexes involved in the blood pressure control and the involvement of exercise training, it was difficult to isolate the effect of physical activity, independent of changes in body mass consequent to the intervention. In addition, differences in the definition of overweight/obesity among studies, especially in the pediatric population (based on body mass index $\geq 30~\text{kg/m}^2$ versus body mass index > 95th percentile for age and gender) [105], as well as differences in the assessment of childhood or adolescence (based on age versus maturation status using Tanner stage or age at peak height velocity—i.e., the time of maximum growth in stature during adolescence) have created controversies among studies.

8. Overall Summary and Recommendations for Future Research

Given the above findings, a hyperadrenergic state is a hallmark of obesity; however, a differentiation of central nervous system sympathetic outflow, with increased traffic in the renal and skeletal muscle sympathetic nerves and reduced cardiac sympathetic nerve firing is evident in obese individuals. These alterations lead to adjustments in the reflexes that control blood pressure. We can conclude that even when not accompanied by an elevation in arterial blood pressure, alterations in the sympathovagal involvement (at rest and during exercise) may be evident in an obese individual. Adaptations in reflexes including a blunted baroreflex sensitivity, lower metaboreflex, and possibly a greater involvement of mechanoreflex appear even in the preadolescent years. These changes, accompanied by the endothelial dysfunction, result in alterations in hemodynamic control (such as differences in the heart rate responses and vasodilatation) during exercise and recovery. Despite considerable achievements in our understanding in the control of blood pressure in obesity, several issues remain unclear. What cellular and molecular events mediate the alterations in the reflexes that control blood pressure during exercise in obesity? Additional experimentation is required to address the possible mechanisms that reverse these pathological events. Another area of implementation should be the assessment of different exercise training modes (continuous or intermittent, dynamic or isometric) and exercise intensities (high versus low) required to induce beneficial regressions in the reflexes controlling blood pressure in obesity. Importantly, studies involving training interventions should be designed to specifically stratify the independent effect of exercise versus dietary modification. To date, the efficacy of antiadrenergic, antihypertensive drugs to the regression of the alterations observed in the exercise pressor reflex has not been tested.

Future studies should also explore the role of alterations in the macronutrient intake percentages to the blood pressure responses during exercise in obese adults, as well as in the pediatric population.

Conflict of Interests

The authors claim no conflict of interest in the submitted paper.

References

- [1] American College of Sports Medicine, Acsm's Guidelines for Exercise Testing and Prescription, Wolters Clover Lippincott Williams & Willkins, Philadelphia, Pa, USA, 2010.
- [2] L. S. Pescatello, B. A. Franklin, R. Fagard, W. B. Farquhar, G. A. Kelley, and C. A. Ray, "American College of Sports Medicine position stand. Exercise and hypertension.," *Medicine and science in sports and exercise*, vol. 36, no. 3, pp. 533–553, 2004.
- [3] N. Miyai, M. Arita, K. Miyashita, I. Morioka, T. Shiraishi, and I. Nishio, "Blood pressure response to heart rate during exercise test and risk of future hypertension," *Hypertension*, vol. 39, no. 3, pp. 761–766, 2002.
- [4] J. Filipovsky, P. Ducimetiere, and M. E. Safar, "Prognostic significance of exercise blood pressure and heart rate in middle-aged men," *Hypertension*, vol. 20, no. 3, pp. 333–339, 1992.
- [5] R. Mundal, S. E. Kjeldsen, L. Sandvik, G. Erikssen, E. Thaulow, and J. Erikssen, "Exercise blood pressure predicts cardiovascular mortality in middle-aged men," *Hypertension*, vol. 24, no. 1, pp. 56–62, 1994.
- [6] R. Mundal, S. E. Kjeldsen, L. Sandvik, G. Erikssen, E. Thaulow, and J. Erikssen, "Exercise blood pressure predicts mortality from myocardial infarction," *Hypertension*, vol. 27, no. 3 I, pp. 324–329, 1996.
- [7] N. Miyai, M. Arita, I. Morioka, S. Takeda, and K. Miyashita, "Ambulatory blood pressure, sympathetic activity, and left ventricular structure and function in middle-aged normotensive men with exaggerated blood pressure response to exercise," *Medical Science Monitor*, vol. 11, no. 10, pp. CR478– CR484, 2005.
- [8] D. A. Cunningham, P. A. Rechnitzer, J. H. Howard, and A. P. Donner, "Exercise training of men at retirement: a clinical trial," *Journals of Gerontology*, vol. 42, no. 1, pp. 17–23, 1987.
- [9] V. K. Somers, J. Conway, A. Coats, J. Isea, and P. Sleight, "Postexercise hypotension is not sustained in normal and hypertensive humans," *Hypertension*, vol. 18, no. 2, pp. 211–215, 1991.
- [10] A. Figueroa, T. Baynard, B. Fernhall, R. Carhart, and J. A. Kanaley, "Impaired postexercise cardiovascular autonomic modulation in middle-aged women with type 2 diabetes," *European Journal of Cardiovascular Prevention and Rehabilitation*, vol. 14, no. 2, pp. 237–243, 2007.
- [11] S. S. Sawada, I. M. Lee, T. Muto, K. Matuszaki, and S. N. Blair, "Cardiorespiratory fitness and the incidence of type 2 diabetes: prospective study of Japanese men," *Diabetes Care*, vol. 26, no. 10, pp. 2918–2922, 2003.
- [12] S. Sawada, H. Tanaka, M. Funakoshi, M. Shindo, S. Kono, and T. Ishiko, "Five year prospective study on blood pressure and maximal oxygen uptake," *Clinical and Experimental Pharmacology and Physiology*, vol. 20, no. 7-8, pp. 483–487, 1993.

[13] G. E. Alvarez, J. R. Halliwill, T. P. Ballard, S. D. Beske, and K. P. Davy, "Sympathetic neural regulation in endurancetrained humans: fitness vs. fatness," *Journal of Applied Physiology*, vol. 98, no. 2, pp. 498–502, 2005.

- [14] L. Carletti, A. N. Rodrigues, A. J. Perez, and D. V. Vassallo, "Blood pressure response to physical exertion in adolescents: influence of overweight and obesity," *Arquivos Brasileiros de Cardiologia*, vol. 91, no. 1, pp. 25–30, 2008.
- [15] K. Dipla, A. Zafeiridis, I. Koidou, N. Geladas, and I. S. Vrabas, "Altered hemodynamic regulation and reflex control during exercise and recovery in obese boys," *American Journal of Physiology*, vol. 299, no. 6, pp. H2090–H2096, 2010.
- [16] K. Matsukawa, "Central command: control of cardiac sympathetic and vagal efferent nerve activity and the arterial baroreflex during spontaneous motor behaviour in animals," *Experimental Physiology*, vol. 97, pp. 20–28, 2012.
- [17] J. W. Williamson, P. J. Fadel, and J. H. Mitchell, "New insights into central cardiovascular control during exercise in humans: a central command update," *Experimental Physiology*, vol. 91, no. 1, pp. 51–58, 2006.
- [18] J. H. Mitchell, M. P. Kaufman, and G. A. Iwamoto, "The exercise pressor reflex: its cardiovascular effects, afferent mechanisms, and central pathways.," *Annual Review of Physiology*, vol. 45, pp. 229–242, 1983.
- [19] M. J. Joyner, "Baroreceptor function during exercise: resetting the record," *Experimental Physiology*, vol. 91, no. 1, pp. 27–36, 2006.
- [20] M. P. Kaufman, T. G. Waldrop, K. J. Rybicki, G. A. Ordway, and J. H. Mitchell, "Effects of static and rhythmic twitch contractions on the discharge of group iii and iv muscle afferents," *Cardiovascular Research*, vol. 18, pp. 663–668, 1984.
- [21] M. P. Kaufman and S. G. Hayes, "The exercise pressor reflex," *Clinical Autonomic Research*, vol. 12, no. 6, pp. 429–439, 2002.
- [22] D. S. O'Leary, "Autonomic mechanisms of muscle metaboreflex control of heart rate," *Journal of Applied Physiology*, vol. 74, no. 4, pp. 1748–1754, 1993.
- [23] D. S. O'Leary, "Heart rate control during exercise by baroreceptors and skeletal muscle afferents," *Medicine and Science* in Sports and Exercise, vol. 28, no. 2, pp. 210–217, 1996.
- [24] M. Nowak, S. Holm, F. Biering-Sørensen, N. H. Secher, and L. Friberg, "Central command' and insular activation during attempted foot lifting in paraplegic humans," *Human Brain Mapping*, vol. 25, no. 2, pp. 259–265, 2005.
- [25] M. Nowak, K. S. Olsen, I. Law, S. Holm, O. B. Paulson, and N. H. Secher, "Command-related distribution of regional cerebral blood flow during attempted handgrip," *Journal of Applied Physiology*, vol. 86, no. 3, pp. 819–824, 1999.
- [26] P. J. Fadel, "Arterial baroreflex control of the peripheral vasculature in humans: rest and exercise," *Medicine and Science* in Sports and Exercise, vol. 40, no. 12, pp. 2055–2062, 2008.
- [27] M. P. Kaufman, K. J. Rybicki, T. G. Waldrop, and J. H. Mitchell, "Effect on arterial pressure of rhythmically contracting the hindlimb muscles of cats," *Journal of Applied Physiology Respiratory Environmental and Exercise Physiology*, vol. 56, no. 5, pp. 1265–1271, 1984.
- [28] M. P. Kaufman and K. J. Rybicki, "Discharge properties of group III and IV muscle afferents: their responses to mechanical and metabolic stimuli.," *Circulation Research*, vol. 61, no. 4, pp. I60–65, 1987.
- [29] R. G. Victor, L. A. Bertocci, S. L. Pryor, and R. L. Nunnally, "Sympathetic nerve discharge is coupled to muscle pH during exercise in humans," *Journal of Clinical Investigation*, vol. 82, no. 4, pp. 1301–1305, 1988.

[30] L. I. Sinoway, K. J. Wroblewski, S. A. Prophet et al., "Glycogen depletion-induced lactate reductions attenuate reflex responses in exercising humans," *American Journal of Physiology*, vol. 263, no. 5, pp. H1499–H1505, 1992.

- [31] D. M. Rotto, C. L. Stebbins, and M. P. Kaufman, "Reflex cardiovascular and ventilatory responses to increasing H+ activity in cat hindlimb muscle," *Journal of Applied Physiology*, vol. 67, no. 1, pp. 256–263, 1989.
- [32] M. Fischer and S. S. Schäfer, "Effects of changes in pH on the afferent impulse activity of isolated cat muscle spindles," *Brain Research*, vol. 1043, no. 1-2, pp. 163–178, 2005.
- [33] S. G. Hayes, A. E. Kindig, and M. P. Kaufman, "Blockade of acid sensing ion channels attenuates the exercise pressor reflex in cats," *Journal of Physiology*, vol. 581, no. 3, pp. 1271–1282, 2007
- [34] M. Esler, G. Lambert, M. Vaz et al., "Central nervous system monoamine neurotransmitter turnover in primary and obesity-related human hypertension," *Clinical and Experimental Hypertension*, vol. 19, no. 5-6, pp. 577–590, 1997.
- [35] G. Grassi, B. M. Cattaneo, G. Seravalle, M. Colombo, F. Cavagnini, and G. Mancia, "Obesity and the sympathetic nervous system," *Blood Pressure, Supplement*, vol. 5, no. 1, pp. 43–46, 1996.
- [36] "Heart rate variability. Standards of measurement, physiological interpretation, and clinical use. Task force of the european society of cardiology and the north american society of pacing and electrophysiology," European Heart Journal, vol. 17, pp. 354–381, 1996.
- [37] M. P. Tulppo, T. H. Makikallio, T. E. Takala, T. Seppanen, and H. V. Huikuri, "Quantitative beat-to-beat analysis of heart rate dynamics during exercise," *American Journal of Physiol*ogy, vol. 271, pp. H244–H252, 1996.
- [38] K. P. Davy and J. S. Orr, "Sympathetic nervous system behavior in human obesity," *Neuroscience and Biobehavioral Reviews*, vol. 33, no. 2, pp. 116–124, 2009.
- [39] G. A. Bray, "Obesity—a state of reduced sympathetic activity and normal or high adrenal activity (the autonomic and adrenal hypothesis revisited)," *International Journal of Obesity*, vol. 14, no. 3, pp. 77–91, 1990.
- [40] K. P. Davy and J. E. Halle, "Obesity and hypertension: two epidemics or one?" *American Journal of Physiology*, vol. 286, no. 5, pp. R803–R813, 2004.
- [41] M. S. Rumantir, M. Vaz, G. L. Jennings et al., "Neural mechanisms in human obesity-related hypertension," *Journal of Hypertension*, vol. 17, no. 8, pp. 1125–1133, 1999.
- [42] M. Vaz, G. Jennings, A. Turner, H. Cox, G. Lambert, and M. Esler, "Regional sympathetic nervous activity and oxygen consumption in obese normotensive human subjects," *Circulation*, vol. 96, no. 10, pp. 3423–3429, 1997.
- [43] S. W. Coppack, J. F. Horowitz, D. S. Paramore, P. E. Cryer, H. D. Royal, and S. Klein, "Whole body, adipose tissue, and forearm norepinephrine kinetics in lean and obese women," *American Journal of Physiology*, vol. 275, no. 5, pp. E830– E834, 1998.
- [44] G. E. Alvarez, S. D. Beske, T. P. Ballard, and K. P. Davy, "Sympathetic neural activation in visceral obesity," *Circulation*, vol. 106, no. 20, pp. 2533–2536, 2002.
- [45] P. P. Jones, S. Snitker, J. S. Skinner, and E. Ravussin, "Gender differences in muscle sympathetic nerve activity: effect of body fat distribution," *American Journal of Physiology*, vol. 270, no. 2, pp. E363–E366, 1996.
- [46] L. J. Prior, N. Eikelis, J. A. Armitage et al., "Exposure to a high-fat diet alters leptin sensitivity and elevates renal

- sympathetic nerve activity and arterial pressure in rabbits," *Hypertension*, vol. 55, no. 4, pp. 862–868, 2010.
- [47] E. Shinohara, S. Kihara, S. Yamashita et al., "Visceral fat accumulation as an important risk factor for obstructive sleep apnoea syndrome in obese subjects," *Journal of Internal Medicine*, vol. 241, no. 1, pp. 11–18, 1997.
- [48] A. N. Vgontzas, "Does obesity play a major role in the pathogenesis of sleep apnoea and its associated manifestations via inflammation, visceral adiposity, and insulin resistance?" *Archives of Physiology and Biochemistry*, vol. 114, no. 4, pp. 211–223, 2008.
- [49] J. C. M. Lam, A. Xu, S. Tam et al., "Hypoadiponectinemia is related to sympathetic activation and severity of obstructive sleep apnea," *Sleep*, vol. 31, no. 12, pp. 1721–1727, 2008.
- [50] A. Abbas, L. S. Szczepaniak, M. Tuncel et al., "Adiposityindependent sympathetic activity in black men," *Journal of Applied Physiology*, vol. 108, no. 6, pp. 1613–1618, 2010.
- [51] G. E. Alvarez, T. P. Ballard, S. D. Beske, and K. P. Davy, "Subcutaneous obesity is not associated with sympathetic neural activation," *American Journal of Physiology*, vol. 287, no. 1, pp. H414–H418, 2004.
- [52] G. Paolisso, D. Manzella, M. R. Rizzo et al., "Effects of insulin on the cardiac autonomic nervous system in insulin-resistant states," *Clinical Science*, vol. 98, no. 2, pp. 129–136, 2000.
- [53] M. Emdin, A. Gastaldelli, E. Muscelli et al., "Hyperinsulinemia and autonomic nervous system dysfunction in obesity: effects of weight loss," *Circulation*, vol. 103, no. 4, pp. 513–519, 2001.
- [54] S. Engeli and A. M. Sharma, "The renin-angiotensin system and natriuretic peptides in obesity-associated hypertension," *Journal of Molecular Medicine*, vol. 79, no. 1, pp. 21–29, 2001.
- [55] C. M. Boustany, K. Bharadwaj, A. Daugherty, D. R. Brown, D. C. Randall, and L. A. Cassis, "Activation of the systemic and adipose renin-angiotensin system in rats with diet-induced obesity and hypertension," *American Journal of Physiology*, vol. 287, no. 4, pp. R943–R949, 2004.
- [56] K. Rahmouni, M. L. G. Correia, W. G. Haynes, and A. L. Mark, "Obesity-associated hypertension: new insights into mechanisms," *Hypertension*, vol. 45, no. 1, pp. 9–14, 2005.
- [57] E. V. Menshikova, V. B. Ritov, F. G. Toledo, R. E. Ferrell, B. H. Goodpaster, and D. E. Kelley, "Effects of weight loss and physical activity on skeletal muscle mitochondrial function in obesity," *American Journal of Physiology*, vol. 288, pp. E818–E825, 2005.
- [58] F. G. S. Toledo, S. Watkins, and D. E. Kelley, "Changes induced by physical activity and weight loss in the morphology of intermyofibrillar mitochondria in obese men and women," *Journal of Clinical Endocrinology and Metabolism*, vol. 91, no. 8, pp. 3224–3227, 2006.
- [59] G. Grassi, F. Arenare, F. Quarti-Trevano, G. Seravalle, and G. Mancia, "Heart rate, sympathetic cardiovascular influences, and the metabolic syndrome," *Progress in Cardiovascular Diseases*, vol. 52, no. 1, pp. 31–37, 2009.
- [60] M. Esler, M. Rumantir, D. Kaye, and G. Lambert, "The sympathetic neurobiology of essential hypertension: disparate influences of obesity, stress, and noradrenaline transporter dysfunction?" *American Journal of Hypertension*, vol. 14, no. 6, pp. 139S–146S, 2001.
- [61] M. Esler, M. Rumantir, G. Wiesner, D. Kaye, J. Hastings, and G. Lambert, "Sympathetic nervous system and insulin resistance: from obesity to diabetes," *American Journal of Hypertension*, vol. 14, no. 11, pp. 304S–309S, 2001.

[62] G. Grassi, R. Dell'Oro, F. Quarti-Trevano et al., "Neuroadrenergic and reflex abnormalities in patients with metabolic syndrome," *Diabetologia*, vol. 48, no. 7, pp. 1359–1365, 2005.

- [63] G. Grassi, G. Seravalle, M. Colombo et al., "Body weight reduction, sympathetic nerve traffic, and arterial baroreflex in obese normotensive humans," *Circulation*, vol. 97, no. 20, pp. 2037–2042, 1998.
- [64] C. E. Negrão, I. C. Trombetta, L. T. Batalha et al., "Muscle metaboreflex control is diminished in normotensive obese women," *American Journal of Physiology*, vol. 281, no. 2, pp. H469–H475, 2001.
- [65] F. H. S. Kuniyoshi, I. C. Trombetta, L. T. Batalha et al., "Abnormal neurovascular control during sympathoexcitation in obesity," *Obesity Research*, vol. 11, no. 11, pp. 1411–1419, 2003.
- [66] L. Karpoff, A. Vinet, I. Schuster et al., "Abnormal vascular reactivity at rest and exercise in obese boys," *European Journal* of Clinical Investigation, vol. 39, no. 2, pp. 94–102, 2009.
- [67] W. M. Sherman, A. L. Katz, C. L. Cutler, R. T. Withers, and J. L. Ivy, "Glucose transport: locus of muscle insulin resistance in obese Zucker rats," *American Journal of Physiology*, vol. 255, no. 3, pp. E374–E382, 1988.
- [68] M. T. La Rovere, G. Specchia, A. Mortara, and P. J. Schwartz, "Baroreflex sensitivity, clinical correlates, and cardiovascular mortality among patients with a first myocardial infarction: a prospective study," *Circulation*, vol. 78, no. 4 I, pp. 816–824, 1988
- [69] I. Skrapari, N. Tentolouris, D. Perrea, C. Bakoyiannis, A. Papazafiropoulou, and N. Katsilambros, "Baroreflex sensitivity in obesity: relationship with cardiac autonomic nervous system activity," *Obesity*, vol. 15, no. 7, pp. 1685–1693, 2007.
- [70] N. M. Fardin, L. M. Oyama, and R. R. Campos, "Changes in baroreflex control of renal sympathetic nerve activity in highfat-fed rats as a predictor of hypertension," *Obesity*. In press.
- [71] J. K. Limberg, M. D. De Vita, G. M. Blain, and W. G. Schrage, "Muscle blood flow responses to dynamic exercise in young obese humans," *Journal of Applied Physiology*, vol. 108, no. 2, pp. 349–355, 2010.
- [72] G. M. Blain, J. K. Limberg, G. F. Mortensen, and W. G. Schrage, "Rapid onset vasodilatation is blunted in obese humans," *Acta Physiologica*, vol. 205, no. 1, pp. 103–112, 2012.
- [73] B. L. Hodnett, L. Xiang, J. Dearman, C. Carter, and R. L. Hester, "KATP-mediated vasodilation is impaired in obese zucker rats," *Microcirculation*, vol. 15, no. 6, pp. 485–494, 2008
- [74] M. L. Armstrong, A. K. Dua, and C. L. Murrant, "Potassium initiates vasodilatation induced by a single skeletal muscle contraction in hamster cremaster muscle," *Journal of Physiology*, vol. 581, no. 2, pp. 841–852, 2007.
- [75] I. C. Trombetta, L. T. Batalha, M. U. P. B. Rondon et al., "Weight loss improves neurovascular and muscle metaboreflex control in obesity," *American Journal of Physiology*, vol. 285, no. 3, pp. H974–H982, 2003.
- [76] A. C. Tonacio, I. C. Trombetta, M. U. P. B. Rondon et al., "Effects of diet and exercise training on neurovascular control during mental stress in obese women," *Brazilian Journal of Medical and Biological Research*, vol. 39, no. 1, pp. 53–62, 2006.
- [77] N. E. Straznicky, M. T. Grima, N. Eikelis et al., "The effects of weight loss versus weight loss maintenance on sympathetic nervous system activity and metabolic syndrome components," *Journal of Clinical Endocrinology and Metabolism*, vol. 96, no. 3, pp. E503–E508, 2011.

[78] J. P. Finley and S. T. Nugent, "Heart rate variability in infants, children and young adults," *Journal of the Autonomic Nervous System*, vol. 51, no. 2, pp. 103–108, 1995.

- [79] S. M. Pikkujamsa, T. H. Makikallio, L. B. Sourander et al., "Cardiac interbeat interval dynamics from childhood to senescence: comparison of conventional and new measures based on fractals and chaos theory," *Circulation*, vol. 100, pp. 393–399, 1999.
- [80] Z. Lenard, P. Studinger, B. Mersich, L. Kocsis, and M. Kollai, "Maturation of cardiovagal autonomic function from child-hood to young adult age," *Circulation*, vol. 110, no. 16, pp. 2307–2312, 2004.
- [81] S. Goulopoulou, B. Fernhall, and J. A. Kanaley, "Developmental changes in hemodynamic responses and cardiovagal modulation during isometric handgrip exercise," *International Journal of Pediatrics*, vol. 2010, Article ID 153780, 11 pages, 2010.
- [82] K. R. Turley, "The chemoreflex in young boys and girls," *International Journal of Sports Medicine*, vol. 26, no. 2, pp. 96–101, 2005.
- [83] M. Buchheit, H. Al-Haddad, A. Mendez-Villanueva, M. J. Quod, and P. C. Bourdon, "Effect of maturation on hemodynamic and autonomic control recovery following maximal running exercise in highly trained young soccer players," *Frontiers in Physiology*, vol. 2, p. 69, 2011.
- [84] A. Zafeiridis, A. Dalamitros, K. Dipla, V. Manou, N. Galanis, and S. Kellis, "Recovery during high-intensity intermittent anaerobic exercise in boys, teens, and men," *Medicine and Science in Sports and Exercise*, vol. 37, no. 3, pp. 505–512, 2005.
- [85] K. Dipla, T. Tsirini, A. Zafeiridis et al., "Fatigue resistance during high-intensity intermittent exercise from childhood to adulthood in males and females," *European Journal of Applied Physiology*, vol. 106, no. 5, pp. 645–653, 2009.
- [86] M. Buchheit, P. Duche, P. B. Laursen, and S. Ratel, "Postexercise heart rate recovery in children: relationship with power output, blood ph, and lactate," *Applied Physiology, Nutrition, and Metabolism*, vol. 35, pp. 142–150, 2010.
- [87] K. Jamerson and S. Julius, "Predictors of blood pressure and hypertension. General principles," *American Journal of Hypertension*, vol. 4, no. 11, pp. 598S–602S, 1991.
- [88] M. M. Ribeiro, A. G. Silva, N. S. Santos et al., "Diet and exercise training restore blood pressure and vasodilatory responses during physiological maneuvers in obese children," *Circulation*, vol. 111, no. 15, pp. 1915–1923, 2005.
- [89] Z. Lazarova, I. Tonhajzerova, Z. Trunkvalterova et al., "Baroreflex sensitivity is reduced in obese normotensive children and adolescents," *Canadian Journal of Physiology and Phar*macology, vol. 87, pp. 565–571, 2009.
- [90] A. P. Rocchini, C. Moorehead, V. Katch, J. Key, and K. M. Finta, "Forearm resistance vessel abnormalities and insulin resistance in obese adolescents," *Hypertension*, vol. 19, no. 6, pp. 615–620, 1992.
- [91] Z. Pausova, M. Abrahamowicz, A. Mahboubi et al., "Functional variation in the androgen-receptor gene is associated with visceral adiposity and blood pressure in male adolescents," *Hypertension*, vol. 55, no. 3, pp. 706–714, 2010.
- [92] Z. Lénérd, P. Studinger, B. Mersich, G. Pavlik, and M. Kollai, "Cardiovagal autonomic function in sedentary and trained offspring of hypertensive parents," *Journal of Physiology*, vol. 565, no. 3, pp. 1031–1038, 2005.
- [93] J. W. Cavalcante, L. P. Cavalcante, W. S. Pacheco, M. G. de Menezes, and C. G. Gama Filho, "blood pressure responses in children of normotensive and of hypertensive parents treated

with pressor stimulus," Arquivos Brasileiros de Cardiologia, vol. 69, pp. 323–326, 1997.

- [94] C. D. Legantis, G. P. Nassis, K. Dipla, I. S. Vrabas, L. S. Sidossis, and N. D. Geladas, "Role of cardiorespiratory fitness and obesity on hemodynamic responses in children," *The Journal of Sports Medicine and Physical Fitness*. In press.
- [95] S. Verlohren, G. Dubrovska, S. Y. Tsang et al., "Visceral periadventitial adipose tissue regulates arterial tone of mesenteric arteries," *Hypertension*, vol. 44, no. 3, pp. 271–276, 2004.
- [96] F. Dangardt, W. Osika, R. Volkmann, L. M. Gan, and P. Friberg, "Obese children show increased intimal wall thickness and decreased pulse wave velocity," *Clinical Physiology and Functional Imaging*, vol. 28, no. 5, pp. 287–293, 2008.
- [97] D. Felber-Dietrich, U. Ackermann Liebrich, C. Schindler et al., "Effect of physical activity on heart rate variability in normal weight, overweight and obese subjects: results from the sapaldia study," *European Journal of Applied Physiology*, vol. 104, pp. 557–565, 2008.
- [98] N. M. Hamburg, C. J. McMackin, A. L. Huang et al., "Physical inactivity rapidly induces insulin resistance and microvascular dysfunction in healthy volunteers," *Arteriosclerosis*, *Thrombosis*, and Vascular Biology, vol. 27, no. 12, pp. 2650– 2656, 2007.
- [99] A. S. Kelly, R. J. Wetzsteon, D. R. Kaiser, J. Steinberger, A. J. Bank, and D. R. Dengel, "Inflammation, insulin, and endothelial function in overweight children and adolescents: the role of exercise," *Journal of Pediatrics*, vol. 145, no. 6, pp. 731–736, 2004.
- [100] G. P. Nassis, K. Papantakou, K. Skenderi et al., "Aerobic exercise training improves insulin sensitivity without changes in body weight, body fat, adiponectin, and inflammatory markers in overweight and obese girls," *Metabolism*, vol. 54, no. 11, pp. 1472–1479, 2005.
- [101] K. Watts, P. Beye, A. Siafarikas et al., "Exercise training normalizes vascular dysfunction and improves central adiposity in obese adolescents," *Journal of the American College of Cardiology*, vol. 43, no. 10, pp. 1823–1827, 2004.
- [102] R. A. Abbott, M. A. Harkness, and P. S. W. Davies, "Correlation of habitual physical activity levels with flow-mediated dilation of the brachial artery in 5-10 year old children," *Atherosclerosis*, vol. 160, no. 1, pp. 233–239, 2002.
- [103] N. D. Hopkins, G. Stratton, T. M. Tinken et al., "Relationships between measures of fitness, physical activity, body composition and vascular function in children," *Atherosclerosis*, vol. 204, no. 1, pp. 244–249, 2009.
- [104] D. M. Prado, A. G. Silva, I. C. Trombetta et al., "Exercise training associated with diet improves heart rate recovery and cardiac autonomic nervous system activity in obese children," *International Journal of Sports Medicine*, vol. 31, no. 12, pp. 860–865, 2010.
- [105] J. S. Harrell, A. Jessup, and N. Greene, "Changing our future: obesity and the metabolic syndrome in children and adolescents," *Journal of Cardiovascular Nursing*, vol. 21, no. 4, pp. 322–330, 2006.

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Research Article

Longitudinal Associations of Leisure-Time Physical Activity and Cancer Mortality in the Third National Health and Nutrition Examination Survey (1986–2006)

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Longitudinal associations between leisure-time physical activity (LTPA) and overall cancer mortality were evaluated within the Third National Health and Nutrition Examination Survey (NHANES III; 1988–2006; n =15,535). Mortality status was ascertained using the National Death Index. Self-reported LTPA was divided into inactive, regular low-to-moderate and vigorous activity. A frequency-weighted metabolic equivalents (METS/week) variable was also computed. Hazard ratios (HRs) and 95% confidence intervals (CI) were calculated for overall cancer mortality in the whole sample, by body mass index categories and insulin resistance (IR) status. Nonsignificant protective associations were observed for regular low-to-moderate and vigorous activity, and for the highest quartile of METS/week (HRs range: 0.66–0.95). Individuals without IR engaging in regular vigorous activity had a 48% decreased risk of cancer mortality (HR: 0.52; 95% CI: 0.28–0.98) in multivariate analyses. Conversely, nonsignificant positive associations were observed in people with IR. In conclusion, regular vigorous activity may reduce risk of cancer mortality among persons with normal insulin-glucose metabolism in this national sample.

1. Introduction

Cancer is a major public health concern in the United States with approximately 25% of total deaths attributed to cancer [1]. The projected number of cancer deaths in 2010 alone was 1500 deaths per day, corresponding to a total of over 560,000 deaths. Therefore, investigations into modifiable risk factors that may reduce the rates of cancer mortality continue to be of significant importance. Physical activity has been hypothesized to protect against cancer through obesity reduction, improved insulin sensitivity and sex hormone profiles, and lowered inflammation [2]. The World Cancer Research Fund/American Institute for Cancer Research Expert Panel Report concluded that physical activity likely reduces the risk of some cancers and suggested an active lifestyle for protection against cancer progression

and mortality [3]. However, concrete recommendations for physical activity to prevent cancer mortality are lacking, in part, due to inconsistencies in the existing evidence, and due to varying methodology and differing definitions of physical activity used [4]. Furthermore, at this time associations with physical activity are better established for cancer incidence as compared to mortality [4, 5].

A greater understanding of the protective effects of physical activity may enhance existing cancer control strategies and potentially reduce cancer mortality. The present study strengthens the evidence in the current literature by examining longitudinal associations between leisure-time physical activity (LTPA) in the Third National Health and Nutrition Examination Survey (NHANES III: 1988–1994) linked to mortality data through 2006. Excess adiposity and aberrations in the insulin-glucose axis are hypothesized to promote

carcinogenesis [6, 7] and may offset the protective potential of physical activity. It is noteworthy that research in the area of cancer mortality has typically focused on a single risk factor. Less attention has been devoted to the combined effects of body weight, insulin resistance (IR), physical activity, and cancer mortality [8–11], and the interrelationships of these risk factors warrant investigation. Therefore the current study is designed to expand the physical activity-cancer hypothesis by assessing whether body mass index (BMI) and IR modify the relationships of physical activity and cancer mortality. Existing data available from this national sample on body weight, laboratory values, self-reported physical activity data, and cancer mortality (N = 863) provides a unique opportunity to conduct these analyses. The results of this study provide data to guide clinical trials that may ultimately contribute to individualized physical activity recommendations for cancer control.

2. Materials and Methods

2.1. Study Population. The NHANES III (1988–1994) population, a national sample of civilian noninstitutionalized individuals, was selected through a complex, multistage probability design [12]. Persons who were 17+ or older were eligible for the mortality follow-up from the date of participation (1988-1994) through December 31, 2006. This represents the last NHANES III mortality update. The current analyses included adults 20-89 years. Per the Adult Treatment Panel (ATP) definition, individuals were considered to be adults if they were at least 20 years old [13]. NHANES participants 89+ years were assigned an arbitrary age of 90 years for confidentiality purposes and were excluded from the analyses. Pregnant women were excluded because their baseline physical activity and BMI may not be an accurate reflection of their usual activity or body weight. Additionally, persons with missing values for the pertinent variables were also excluded, resulting in a final sample of 15,535 individuals.

2.2. Data Collection. The NHANES III survey consisted of a structured household interview and a standardized physical examination in a mobile examination center at entry into the study. Participants self-reported their age, race/ethnicity, level of education, leisure-time physical activity, dietary intakes, alcohol use, current prescription medication, and presence of doctor-diagnosed cancer in a personal interview. Race/ethnicity were categorized into (1) non-Hispanic whites, (2) non-Hispanic Blacks, (3) Mexican-Americans and (4) "other." Trained personnel measured height, weight and waist circumference during the in-person examination [12]. The measured height and weight were used to compute BMI. Smoking status was assessed during the in-person interview, in which participants reported the use of cigarettes, pipes, and cigars. A fasting blood sample was obtained during the physical examination that was used to measure fasting plasma glucose concentrations. Details of the NHANES III protocols have been previously published [14]. Figure 1 describes the data collection strategy in NHANES III.

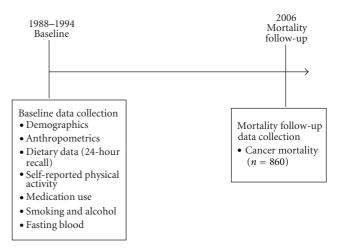


FIGURE 1: NHANES III data collection timeline.

2.3. Exposure: Leisure-Time Physical Activity. A questionnaire consisting leisure-time physical activity questions including type of activity and frequency of the activity in the past month was queried in the Household Adult Ques tionnaire administered once at baseline. There was no additional follow-up information on physical activity and other variables after time of entry into the study until the end of mortality follow-up (Figure 1). At baseline, participants were asked for example, if "In the past month, did you run or jog?" and "In the past month, how often did you jog or run?" Similarly, questions were asked on walking, bicycling, swimming, aerobics and/or aerobic dancing, other dancing, calisthenics, yard work, lifting weights, and engagement in up to four other activities that were not mentioned in the survey. Frequency of engagement in activities over the past 30 days was reported. These responses were standardized to weekly frequency by using the conversion factors of 4.3 weeks/month and 30.4 days/month per NHANES III [15]. A validated intensity rating in metabolic equivalents (MET) was provided by NHANES III for each activity as defined in the Compendium of Physical Activities [16]. For example, jogging/running was assigned an intensity rating of "8 METS." For this study, LTPA was classified as "low intensity," "moderate" and "vigorous" using the standard cut-offs established by Pate et al. [17]. Activities requiring <3 METS were classified as "low-intensity," activities requiring 3-6 METS were classified as "moderate" and >6 METS were classified as "vigorous" [17]. Participants who did not report engagement in any of the activities, were classified as "inactive." Because individuals engaging in "low intensity" activities represented <0.05% (n = 7) of the population, they were combined into the "moderate" activity group.

Next, participants were categorized as performing "regular low-to-moderate" exercises if they reported performing the activity at least 5 times per week and "regular vigorous" if they reported performing vigorous activities at least 3 times per week. These categories were mutually exclusive (so that if an individual performed vigorous and low-to-moderate activities, he/she was included in the vigorous activity group and not in both groups). Additionally, a frequency-weighted

LTPA variable was calculated by multiplying the frequency of each activity by its corresponding intensity value and summed. This variable was divided into quartiles in the regression models. The NHANES III survey did not collect information for duration of each bout of exercise, which limits more precise estimation of physical activity. The physical activity definitions used in this study are consistent with previously published studies using the NHANES III population [18–21].

2.4. Cancer Mortality Ascertainment. Mortality information was obtained from the updated NHANES III Linked Mortality Files that provide mortality follow-up data from the date of participation in the survey (1988–1994) through December 31, 2006 (Figure 1). Mortality was ascertained based upon either death certificates or from a probabilistic match between NHANES III and the National Center for Health Statistics, National Death Index (NDI) records. Cancer mortality was identified using the International Classification of Diseases, Tenth Revision (ICD-10; NCHS 2006; ICD-10 codes C00–C97). Persons were considered alive at the end of the follow-up period if they were not matched to a death record. Details of the mortality files are described elsewhere [22].

2.5. Statistical Methods. Descriptive statistics were generated to assess demographic, lifestyle and dietary attributes related with cancer mortality, and to identify potential confounders. Next, unadjusted univariate hazard ratio (HR) and 95% confidence intervals (95% CI) of overall cancer mortality and level of LTPA were computed using Cox proportional hazard models, using age as the time scale. Persons who died from causes other than cancer were censored at the age of death and persons who were considered alive were censored at the end of the study follow-up period.

We tested demographic, lifestyle, physiological, and dietary factors as potential confounders of the associations of LTPA and cancer mortality, by entering these additional variables singly into the Cox proportional hazards models. If the addition of the variable singly in the model changed the HR for cancer mortality by 10% or more, the variable was added to the final regression model. Potential variables tested were age (continuous and years), race or ethnicity (categorical: white, black, Mexican, and other), cardiovascular disease (categorical: yes/no), BMI (categorical: normal weight, overweight and obese), smoking (categorical: never, current, past), alcohol consumption (continuous: number of drinks/ day), education (categorical: <12 and ≥12 years), aspirin use (yes/no) total dietary proteins (continuous: grams/day), total dietary fats (continuous: grams/day) total dietary carbohydrates (continuous: grams/day), and total calories (continuous: kilocalories/day). We tested for potential effect modification (considered significant for the purpose of these analyses at alpha value of 0.10 or less) by race, age, gender, BMI, and IR status for the relationships of physical activity and cancer mortality. A significance level of 0.05 was used for all the other tests. Sensitivity analyses were conducted by reanalyzing the associations after excluding persons with a baseline report of doctor-diagnosed cancer (n = 588). For

exploratory purposes, we repeated analyses in adults 40+ years at baseline (n=9348). The statistical analyses were performed using SAS v.9.1 and SUDAAN v.10.0.1 Sample weights provided by NHANES III were applied to all the analyses.

3. Results

3.1. Participant Characteristics. Baseline characteristics of NHANES III sample are expressed as either weighted frequencies for categorical variables or weighted means with their corresponding standard errors (SEs), and minimum and maximum values for continuous variables (Table 1). The population consisted of 76.8% non-Hispanic Whites, 10.4% non-Hispanic blacks, 5.2% Mexican-Americans, and 7.8% "other" ethnicities. The mean population age was 45 years. Approximately 85% of the participants reported engaging in any physical activity, with half (53.3%) of the population being moderately and 16.9% being vigorously active on a regular basis. About 15% of the sample was "inactive." Furthermore, 33% of the participants reported being more active, 21% reported being less active, and 44% estimated about the same level of activity as compared to their peers of the same sex and similar age. Over half the population was obese or overweight (BMI > 25), with a mean waist circumference of 92 cm. Twenty-two percent of the population had the insulin resistance syndrome, defined per the ATP III criteria as having at least three of the following five criteria: abdominal obesity (waist circumference >102 cm in men or >88 cm in women), insulin resistance (blood glucose >110 mg/dL), low high density lipoprotein (HDL) (<40 in men or <50 in women), high serum triglycerides concentration (>150 mg/dL), and hypertension (systolic blood pressure >130 mm Hg or diastolic blood pressure >85 mm Hg.) and 10.67% were insulin resistant (defined as blood glucose levels >110 mg/dL) [13]. In this dataset 5.5% (n = 863) of the population had died from cancer at the end of the follow-up period till 2006. Approximately 15% of the patients who had died of cancer had reported doctordiagnosed cancer during their in-person baseline interview.

3.2. Associations between Leisure-Time Physical Activity and Overall Cancer Mortality. Overall, nonsignificant inverse associations were observed for overall cancer mortality among persons engaging in any activity, regular low-tomod- erate or regular vigorous activity in the whole population (Table 2). Individuals who engaged in any activity were 8% and 5% less likely to die of cancer as compared to individuals who were "inactive," after adjusting for age (HR: 0.92; 95% CI: 0.71–1.19) and additional variables (HR: 0.95; 95% CI: 0.72-1.26), respectively. Associations were in a similar direction for regular low-to-moderate activity (HR: 0.92; 95% CI: 0.69–1.21), regular vigorous activity (HR: 0.66; 95% CI: 0.39-1.13), and for the highest versus lowest quartile of frequency-weighted METS per week (HR: 0.89; 95% CI: 0.68-1.16) after adjusting for age, race, sex, and smoking status, albeit not statistically significant. Additional adjustment for BMI did not change the associations.

Table 1: Characteristics of the NHANES III study participants at baseline $(N = 15, 535)^1$.

	Percent (%)	Mean (SE)	Minimum	Maximum
Demographic information				
Non-Hispanic Whites	76.8			
Non-Hispanic Blacks	10.4			
Mexican Americans	5.2			
Other races/ethnicities	7.8			
Sex (men)	48.7			
Age (years)		45.0 (0.14)	20.0	89.0
Anthropometric measurements				
Body mass index (wt (kgs)/ht²(meter)		26.5 (0.04)	14.4	62.1
Waist circumference (cm)		92.0 (0.12)	57.5	174.1
Physical activity				
Inactive	14.8			
Any activity	85.2			
Regular low to moderate intensity activity	53.3			
Regular vigorous activity	16.9			
METS/week ²		3.67 (0.03)	0	53.5
Activity level compared to peers of same age				
More active	33			
Less active	21			
About the same	44			
Blood analyses				
Fasting glucose (mg/dL)		99.0 (0.24)	35.4	642.6
Insulin resistance ³	10.67			
Insulin resistance syndrome ⁴	22.2			
Dietary intake				
Percent kilocalories from carbohydrate		49.71 (0.09)	1.3	96.5
Percent kilocalories from total fat		33.52 (0.08)	1.2	76.20
Percent kilocalories from protein		15.42 (0.04)	2.0	62.8
Total calories ⁵		2069.41 (8.37)	401.0	9,769.0
Cancer	$\% (n)^6$			
Overall cancer mortality	5.5 (863)			
Self-report of doctor-diagnosed cancer at baseline	3.82 (588)			
Individuals with self-reported doctor-diagnosed cancer at baseline who died of cancer	15 (128)			

¹ Data is presented as weighted frequencies (percentages) for categorical variables and as weighted means (SE), minimum, and maximum values for continuous

Analyses were repeated after excluding self-reported history of cancer at baseline (n=588) to assess whether the observed nonsignificant associations persisted. The HRs among individuals with no history of cancer at baseline ranged from 0.82 to 1.09 for all levels of regular LTPA; however,

the HRs were not statistically significant after adjusting for age and additional covariates in the whole population and the conclusions remained unchanged (Table 2). Restricting analyses to adults 40+ years at baseline yielded similar results (data not shown).

²METS were only reported for documented leisure time physical activities in the self-report questionnaire.

³Insulin resistance was defined as blood glucose >110 mg/dL and includes individuals who consumed oral hypoglycemic agents or insulin users.

⁴Insulin resistance syndrome was defined as the presence of at least 3 of 5 criteria per the ATP III criteria.

⁵Only individuals who reported energy intake between 400–10,000 kcal were included in these analyses.

⁶n represent actual unweighted frequencies in the analyses dataset.

Table 2: Hazard ratio (HR) (95% CI) for cancer mortality and measures of leisure-time physical activity in NHANES III (1988–2006) participants 20–89 years (n = 15,535).

	Whole population	After excluding persons with existing cancer $(n = 588)$
Cancer deaths/number at risk	860/15,535	733/14,951
Any activity		
Age-Adjusted	0.92 (0.71–1.19)	1.04 (0.78–1.37)
Adjusted for additional variables ¹	0.95 (0.72–1.26)	1.10 (0.82–1.48)
Additional adjustment for BMI ²	0.97 (0.73–1.29)	1.12 (0.83–1.50)
Regular low-moderate activity		
Age-Adjusted	0.89 (0.69–1.17)	1.01 (0.76–1.34)
Adjusted for additional variables ¹	0.92 (0.69–1.21)	1.07 (0.80–1.43)
Additional adjustment for BMI ²	0.93 (0.70–1.24)	1.09 (0.81–1.48)
Regular vigorous activity		
Age-adjusted	0.62 (0.37–1.06)	0.73 (0.42–1.26)
Adjusted for additional variables ¹	0.66 (0.39–1.13)	0.82 (0.48–1.39)
Additional adjustment for BMI ²	0.69 (0.40–1.19)	0.85 (0.50–1.44)
Frequency-weighted METS/week ³		
Age-adjusted		
Quartile 1	1.00	1.00
Quartile 2	0.84 (0.67–1.05)	0.92 (0.71–1.18)
Quartile 3	0.72 (0.54–0.96)	0.80 (0.59–1.08)
Quartile 4	0.86 (0.68–1.10)	0.97 (0.78–1.26)
P value ⁴	0.120	0.353
Adjusted for additional variables ¹		
Quartile 1	1.00	1.00
Quartile 2	0.85 (0.67–1.08)	0.95 (0.73–1.23)
Quartile 3	0.74 (0.56–0.98)	0.85 (0.64–1.14)
Quartile 4	0.89 (0.68–1.16)	1.04 (0.79–1.36)
P value⁴	0.139	0.422
Additional adjustment for BMI ²		
Quartile 1	1.00	1.00
Quartile 2	0.85 (0.67–1.09)	0.95 (0.73–1.24)
Quartile 3	0.75 (0.57–1.00)	0.87 (0.65–1.15)
Quartile 4	0.91 (0.69–1.30)	1.06 (0.79–1.41)
P value ⁴	0.132	0.426

Adjusted for age, race, sex, and smoking.

Previous research has reported that BMI and IR are associated with cancer mortality [6, 23, 24]. Significant effect modification (a priori considered significant if P < 0.1) by IR in the relationships of physical activity and overall cancer mortality were noted (P = 0.07). Therefore, the analyses were stratified by IR status (yes/no) as shown in Table 3. Among individuals who were not insulin resistant, significant 54% (HR: 0.46; 95% CI: 0.24–0.87) and 49% (HR: 0.51; 95% CI: 0.27–0.97) decreased risk were observed between engagement in regular vigorous activity and overall cancer mortality, after adjusting for age and additional variables,

respectively. Among individuals who were insulin resistant, an 82% increased risk of cancer mortality was observed (HR: 1.82; 95% CI: 0.71–4.65), albeit this finding was not statistically significant and therefore cannot be considered definitive. These associations persisted when the relationships were reevaluated in a sample after excluding persons with a cancer diagnosis at baseline, in sensitivity analyses (data not shown).

Although there was no significant effect modification by BMI (P = 0.355), for exploratory purposes only, the analyses were stratified by BMI < 25 (normal weight) and >25 (obese or overweight). Among persons with a lower

²Adjusted for age, race, sex, smoking, and BMI.

³Quartile cutoffs (METS/week) quartile 1 < 1.16; quartile 2: 1.16–10.47; quartile 3: 10.48–32; quartile 4: >48.33.

⁴P value for difference between quartiles.

Table 3: Hazard ratio (HR) (95% CI) for cancer mortality and leisure-time physical activity in NHANES III (1988–1994) participants 20–89 years, by categories of body mass index and insulin resistance status

	By High a	By High and low BMI ¹		tance status ²
Cancer deaths/number at risk	521/9279	321/5991	228/2300	628/13,166
Cancer deaths/number at risk	High BMI	Low BMI	IR	No IR
Any activity				
Age adjusted	1.02 (0.73-1.42)	0.82 (0.55-1.22)	0.89 (0.56-1.41)	0.94 (0.72-1.23)
Adjusted for additional variables ³	1.03 (0.73–1.46)	0.90 (0.61-1.35)	0.88 (0.54–1.44)	0.99 (0.75–1.32)
Additional adjustment for BMI ⁴	_	_	0.89 (0.54–1.48)	1.00 (0.76–1.32)
Regular low-moderate activity				
Age adjusted	1.00 (0.70-1.41)	0.80 (0.53-1.20)	0.84 (0.53-1.34)	0.93 (0.69-1.24)
Adjusted for additional variables ³	1.00 (0.70-1.42)	0.88 (0.58-1.35)	0.84 (0.51-1.38)	0.97 (0.72-1.31)
Additional adjustment for BMI ⁴	_	_	0.86 (0.51–1.43)	0.98 (0.73–1.32)
Regular Vigorous Activity				
Age-Adjusted	0.68 (0.33-1.39)	0.59 (0.28-1.27)	1.82 (0.71-4.65)	0.46 (0.24-0.87)
Adjusted for additional variables ³	0.74 (0.36–1.52)	0.68 (0.32-1.45)	1.79 (0.67–4.87)	0.51(0.27-0.97)
Additional adjustment for BMI ⁴	_	_	1.83 (0.66-5.04)	0.52 (0.28-0.98)
Frequency-weighted METS/week ⁵				
Age-adjusted				
Quartile 1	1.00	1.00	1.00	1.00
Quartile 2	1.00 (0.08-1.46)	0.92 (0.64-1.34)	0.85 (0.43–1.67)	0.82 (0.63-1.05)
Quartile 3	0.96 (0.66-1.38)	0.61 (0.36-1.02)	0.67 (0.42–1.07)	0.76 (0.53–1.09)
Quartile 4	1.01 (0.69–1.48)	0.78 (0.51-1.18)	1.11 (0.65–1.09)	0.79 (0.6–1.03)
P value ⁶	0.987	0.226	0.145	0.294
Adjusted for additional variables ³				
Quartile 1	1.00	1.00	1.00	1.00
Quartile 2	0.99 (0.65-1.49)	0.99 (0.68-1.44)	0.82 (0.41–1.66)	0.83 (0.65–1.07)
Quartile 3	0.97 (0.69-1.37)	0.68 (0.41–1.13)	0.69 (0.42–1.13)	0.80 (0.57-1.12)
Quartile 4	1.02 (0.69–1.52)	0.91 (0.58-1.45)	1.08 (0.62–1.89)	0.83 (0.62–1.12)
P value ⁶	0.991	0.315	0.262	0.465
Additional adjustment for BMI ⁴	_	_		
Quartile 1			1.00	1.00
Quartile 2			0.82 (0.41–1.66)	0.83 (0.65–1.07)
Quartile 3			0.69 (0.42-1.13)	0.80 (0.57–1.12)
Quartile 4			1.08 (0.62–1.89)	0.83 (0.62–1.12)
P value ⁶			0.262	0.465

 $^{^{1}}$ BMI < 25 and \geq 25.

⁶P value for difference between quartiles.

BMI, the observed inverse associations were more protective for any activity and overall cancer mortality, albeit non-significant after adjusting for age (HR: 0.82; 95% CI: 0.55-1.22) and additional variables (HR: 0.90; 95% CI: 0.61-1.35) as compared to individuals with a BMI $\,>\,25$ ((age-adjusted HR: 1.02; 95% CI: 0.73-1.42) and (adjusted for additional variables HR: 1.03; 95% CI: 0.73-1.46)). The HR were

similarly <1 but remained non-significant among persons with a normal BMI engaging in regular low-to-moderate or regular vigorous LTPA. The direction of the associations persisted when reevaluated after excluding persons with no cancer diagnosis at baseline (data not shown).

Next, the frequency-weighted METS/week variable was divided into quartiles in the Cox proportional regression

²IR was considered to be present if blood glucose concentrations were >110 mg/dL.

³Adjusted for age, race, sex, and smoking.

⁴Adjusted for age, race, sex, smoking, and BMI.

 $^{^5}$ Quartile cutoffs (METS/week) quartile 1: <2.33; quartile 2: 1.24–13.60; quartile 3: 13.61–35.35 quartile 4: >35.36 for "low BMI" group quartile 1: <1.05; quartile 2: 1.06–9.65; quartile 3: 9.66–30.70; quartile 4: >30.72 for "High BMI" group; quartile 1: <1.63; quartile 2: 1.64–11.40; quartile 3: 11.41 to 34.65; quartile 4: >34.65 for "No IR"; quartile 1: 0; quartile 2: 0.1–6.28; quartile 3: 6.29–26.05; quartile 4: >26.05 for IR "Yes".

models. Quartile 1 was the referent group and represented participants with the lowest level of METs expended, and quartile 4 represented the individuals with the highest level of METs expended. Although not significant, the HRs for quartiles 2, 3, and 4 were <1 as compared to quartile 1. These associations persisted among persons without insulin resistance and among participants with a BMI<25. The non-significant decreased risk for cancer mortality persisted after excluding persons with a cancer diagnosis at baseline (data not shown). Furthermore, HRs were similar when analyses were repeated among individuals who were 40 years or older at baseline.

4. Discussion

The current study was undertaken to elucidate the relationships between LTPA and cancer mortality in a nationally representative dataset and its linkage to mortality status (1988–2006). The comprehensive data available in the NHANES III sample on obesity, insulin resistance, and physical activity history provided a unique opportunity to evaluate the *combined* impact of these factors on longitudinally ascertained cancer mortality, an approach that has typically not been used in the previous studies.

Although the results of the current study are not statistically significant in the whole population, they are suggestive of protection against overall cancer mortality, specifically for vigorous activity (HR < 1). This study is similar to previous studies that have evaluated physical activity in relation to overall cancer mortality of site-specific cancer mortality. For example, a recent prospective cohort study among elder Japanese adults noted that high levels of physical activity (5 or more days per week) were associated with a 23% decreased risk of overall cancer mortality (P for trend = 0.02) [25]. Previous studies have shown that physical activity protects against mortality from some common cancers including lung [3], prostate [10, 26], breast [27-29], and colon cancer [11]. We observed a slight attenuation of the non-significant associations in the sensitivity analyses after excluding persons with self-reported cancer at baseline. This could perhaps be due to lifestyle changes among the cancer cases after diagnosis, or due to reverse causality. However, unavailability of follow-up measures of physical activity in the NHANES III survey limits the complete understanding of this phenomenon. Furthermore, the current study suggests that the protection afforded by LTPA may be realized in the absence of aberrations in the insulin-glucose axis. We note that the protective associations of LTPA and cancer mortality were more pronounced among non-insulin-resistant individuals who engaged in vigorous LTPA.

Several biological mechanisms may explain the potential protective role of physical activity against cancer mortality. With respect to this study, IR-related mechanisms seem particularly relevant. Insulin, an anabolic hormone, is hypothesized to promote cell proliferation, inhibit apoptosis, and support cancer progression [30–32]. Regular LTPA has been associated with improved insulin sensitivity [33–36]. Studies have demonstrated that the benefits of physical activity are apparent even in the absence of changes in body

weight [2, 35]. IR is associated with increased levels of sex hormones and increased levels of inflammation [35], that may affect cancer risk [35, 37]. LTPA may also improve hormonal profiles [38, 39], reduce systemic inflammation [2], and ultimately delay cancer mortality.

Despite the potential biological mechanisms of physical activity in cancer biology, associations were not significant for all levels of LTPA in the whole population in this study. Some inherent limitations of the NHANES III survey design and assessment methods for physical activity could have contributed to measurement error and the quantitatively weak findings. It has been hypothesized that the time course of risk factors along the continuum of the cancer process is important [40] and previous studies suggest that physical activity after the diagnosis of cancer influences cancer mortality [29, 41]. LTPA information was collected once during the in-person interview at baseline. Changes in LTPA patterns over time and engagement in LTPA after cancer diagnosis during the follow-up period were not captured, potentially biasing the results towards the null. Next, physical activity was self-reported and was therefore vulnerable to recall bias due to under- or overreporting their level of LTPA [42]. Further, the NHANES III dataset was not well powered to investigate site-specific cancer mortality. Lastly, the results of this study might be an underestimation of the associations, because the exposure data were collected in 1988-1994 and the prevalence of IR as well as the proportion of physically inactive persons have increased in the past two decades [43– 45], emphasizing the importance of physical activity in the current context.

5. Conclusion

In conclusion, this study uniquely utilizes a large, nationally representative sample of US adults and assesses the interrelationship between LTPA, obesity, and IR, two hypothesized risk factors of cancer mortality in the previous literature. The results suggest that the protective effects offered by physical activity against cancer mortality may be realized through the maintenance of normal metabolic function and may thereby serve a potential cancer control tool. If confirmed in additional epidemiologic studies, these findings may have important public health implications in the context of the high rates of insulin resistance and cancer mortality and a large proportion of persons with sedentary lifestyles in the USA. Further research is also required to determine the critical periods of exposure to physical activity during the life course with exposure measures before and after cancer diagnosis in relation to cancer outcomes in prospective studies.

Abbreviations

ATP III: Adult Treatment Panel III

BMI: Body mass index
CI: Confidence interval
HDL: High-density lipoprotein

HR: Hazard ratio

ICD-10: International Classification of Diseases,

Tenth Revision Insulin resistance

LTPA: Leisure-time physical activity

METS: Metabolic equivalents NDI: National Death Index

NHANES III: Third National Health and Nutrition

Examination Survey

SE: Standard error.

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References

IR:

- [1] A. Jemal, R. Siegel, J. Xu, and E. Ward, "Cancer statistics, 2010," *CA Cancer Journal for Clinicians*, vol. 60, no. 5, pp. 277–300, 2010.
- [2] A. McTiernan, "Mechanisms linking physical activity with cancer," *Nature Reviews Cancer*, vol. 8, no. 3, pp. 205–211, 2008.
- [3] World Cancer Research Fund/American Institute for Cancer Research, Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective, AICR, Washington, DC, USA, 2007.
- [4] M. Barbaric, E. Brooks, L. Moore, and O. Cheifetz, "Effects of physical activity on cancer survival: a systematic review," *Physiotherapy Canada*, vol. 62, no. 1, pp. 25–34, 2010.
- [5] A. E. Speed-Andrews and K. S. Courneya, "Effects of exercise on quality of life and prognosis in cancer survivors," *Current Sports Medicine Reports*, vol. 8, no. 4, pp. 176–181, 2009.
- [6] N. Parekh, Y. Lin, R. B. Hayes, J. B. Albu, and G. L. Lu-Yao, "Longitudinal associations of blood markers of insulin and glucose metabolism and cancer mortality in the third National Health and Nutrition Examination Survey," *Cancer Causes and Control*, vol. 21, no. 4, pp. 631–642, 2010.
- [7] E. Giovannucci, "Insulin and colon cancer," *Cancer Causes and Control*, vol. 6, no. 2, pp. 164–179, 1995.
- [8] M. J. Borugian, S. B. Sheps, C. Kim-Sing et al., "Insulin, macronutrient intake, and physical activity: are potential indicators of insulin resistance associated with mortality from breast cancer?" *Cancer Epidemiology Biomarkers and Prevention*, vol. 13, no. 7, pp. 1163–1172, 2004.
- [9] E. L. Richman, S. A. Kenfield, M. J. Stampfer, A. Paciorek, P. R. Carroll, and J. M. Chan, "Physical activity after diagnosis and risk of prostate cancer progression: data from the cancer of the prostate strategic urologic research endeavor," *Cancer Research*, vol. 71, no. 11, pp. 3889–3895, 2011.
- [10] S. A. Kenfield, M. J. Stampfer, E. Giovannucci, and J. M. Chan, "Physical activity and survival after prostate cancer diagnosis in the health professionals follow-up study," *Journal of Clinical Oncology*, vol. 29, no. 6, pp. 726–732, 2011.
- [11] K. Y. Wolin, A. V. Patel, P. T. Campbell et al., "Change in physical activity and colon cancer incidence and mortality," *Cancer Epidemiology Biomarkers and Prevention*, vol. 19, no. 12, pp. 3000–3004, 2010.

[12] National Center of Health Statistics, Plan and Operation of the Third National Health and Nutrition Examination Survey, 1988–1994, National Center of Health Statistics, Hyattsville, Md, USA, 1995.

- [13] "Executive summary of the third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (adult treatment panel III)," *Journal of the American Medical Association*, vol. 285, no. 19, pp. 2486–2497, 2001.
- [14] E. W. Gunter, B. G. Lewis, and S. M. Koncikowski, Laboratory Procedures Used for the Third National Health and Nutrition Examination Survey (NHANES III), 1988–1994, U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, Hyattsville, Md, USA, 1996.
- [15] National Health and Nutrition Examination Survey: NHANES III Household Adult Data File Documentation, http://www.cdc.gov/nchs/nhanes/nh3data.htm.
- [16] B. E. Ainsworth, W. L. Haskell, A. S. Leon et al., "Compendium of physical activities: classification of energy costs of human physical activities," *Medicine and Science in Sports and Exercise*, vol. 25, no. 1, pp. 71–80, 1993.
- [17] R. R. Pate, M. Pratt, S. N. Blair et al., "Physical activity and public health: a recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine," *Journal of the American Medical Association*, vol. 273, no. 5, pp. 402–407, 1995.
- [18] G. A. King, E. C. Fitzhugh, D. R. Bassett Jr. et al., "Relationship of leisure-time physical activity and occupational activity to the prevalence of obesity," *International Journal of Obesity*, vol. 25, no. 5, pp. 606–612, 2001.
- [19] S. Liangpunsakul, D. W. Crabb, and R. Qi, "Relationship among alcohol intake, body fat, and physical activity: a population-based study," *Annals of Epidemiology*, vol. 20, no. 9, pp. 670–675, 2010.
- [20] K. H. Schmitz, D. Xie, V. Teal, R. Ballard-Barbash, and D. Berrigan, "Association of IGF axis hormones with waist-to-hip ratio varies by physical activity," *In Vivo*, vol. 25, no. 2, pp. 245–250, 2011.
- [21] D. R. Bassett Jr., E. C. Fitzhugh, C. J. Crespo, G. A. King, and J. E. McLaughlin, "Physical activity and ethnic differences in hypertension prevalence in the United States," *Preventive Medicine*, vol. 34, no. 2, pp. 179–186, 2002.
- [22] NHANES III Linked Mortality File, http://www.cdc.gov/nchs/data_access/data_linkage/mortality/nhanes3_linkage.htm.
- [23] J. R. Jaggers, X. Sui, S. P. Hooker et al., "Metabolic syndrome and risk of cancer mortality in men," *European Journal of Cancer*, vol. 45, no. 10, pp. 1831–1838, 2009.
- [24] J. S. Lee, S. I. Cho, and H. S. Park, "Metabolic syndrome and cancer-related mortality among Korean men and women," *Annals of Oncology*, vol. 21, no. 3, Article ID mdp344, pp. 640– 645, 2009.
- [25] K. Ueshima, K. Ishikawa-Takata, T. Yorifuji et al., "Physical activity and mortality risk in the japanese elderly. a cohort study," *American Journal of Preventive Medicine*, vol. 38, no. 4, pp. 410–418, 2010.
- [26] N. Orsini, R. Bellocco, M. Bottai et al., "A prospective study of lifetime physical activity and prostate cancer incidence and mortality," *British Journal of Cancer*, vol. 101, no. 11, pp. 1932– 1938, 2009.
- [27] A. Emaus, M. B. Veierød, S. Tretli et al., "Metabolic profile, physical activity, and mortality in breast cancer patients," *Breast Cancer Research and Treatment*, vol. 121, no. 3, pp. 651–660, 2010.

[28] M. D. Holmes, W. Y. Chen, D. Feskanich, C. H. Kroenke, and G. A. Colditz, "Physical activity and survival after breast cancer diagnosis," *Journal of the American Medical Association*, vol. 293, no. 20, pp. 2479–2486, 2005.

- [29] R. E. Patterson, L. A. Cadmus, J. A. Emond, and J. P. Pierce, "Physical activity, diet, adiposity and female breast cancer prognosis: a review of the epidemiologic literature," *Maturitas*, vol. 66, no. 1, pp. 5–15, 2010.
- [30] M. G. Vander Heiden, D. R. Plas, J. C. Rathmell, C. J. Fox, M. H. Harris, and C. B. Thompson, "Growth factors can influence cell growth and survival through effects on glucose metabolism," *Molecular and Cellular Biology*, vol. 21, no. 17, pp. 5899–5912, 2001.
- [31] V. Ganapathy, M. Thangaraju, and P. D. Prasad, "Nutrient transporters in cancer: relevance to Warburg hypothesis and beyond," *Pharmacology and Therapeutics*, vol. 121, no. 1, pp. 29–40, 2009.
- [32] M. Okumura, M. Yamamoto, H. Sakuma et al., "Leptin and high glucose stimulate cell proliferation in MCF-7 human breast cancer cells: reciprocal involvement of PKC-α and PPAR expression," *Biochimica et Biophysica Acta*, vol. 1592, no. 2, pp. 107–116, 2002.
- [33] R. K. Dishman, R. A. Washburn, and G. W. Heath, *Physical Activity and Diabetes*, Human Kinetics, Champaign, Ill, USA, 2004.
- [34] J. L. Ivy, "Role of exercise training in the prevention and treatment of insulin resistance and non-insulin-dependent diabetes mellitus," *Sports Medicine*, vol. 24, no. 5, pp. 321–336, 1997.
- [35] H. K. Neilson, C. M. Friedenreich, N. T. Brockton, and R. C. Millikan, "Physical activity and postmenopausal breast cancer: proposed biologic mechanisms and areas for future research," *Cancer Epidemiology Biomarkers and Prevention*, vol. 18, no. 1, pp. 11–27, 2009.
- [36] R. J. Sigal, G. P. Kenny, D. H. Wasserman, and C. Castaneda-Sceppa, "Physical activity/exercise and type 2 diabetes," *Diabetes Care*, vol. 27, no. 10, pp. 2518–2539, 2004.
- [37] B. N. Díaz-Chico, F. G. Rodríguez, A. González et al., "Androgens and androgen receptors in breast cancer," *Journal of Steroid Biochemistry and Molecular Biology*, vol. 105, no. 1–5, pp. 1–15, 2007.
- [38] E. R. Bertone-Johnson, S. S. Tworoger, and S. E. Hankinson, "Recreational physical activity and steroid hormone levels in postmenopausal women," *American Journal of Epidemiology*, vol. 170, no. 9, pp. 1095–1104, 2009.
- [39] A. McTiernan, S. S. Tworoger, K. B. Rajan et al., "Effect of exercise on serum androgens in postmenopausal women: a 12-month randomized clinical trial," *Cancer Epidemiology Biomarkers and Prevention*, vol. 13, no. 7, pp. 1099–1105, 2004.
- [40] E. K. Wei, K. Y. Wolin, and G. A. Colditz, "Time course of risk factors in cancer etiology and progression," *Journal of Clinical Oncology*, vol. 28, no. 26, pp. 4052–4057, 2010.
- [41] A. Vrieling and E. Kampman, "The role of body mass index, physical activity, and diet in colorectal cancer recurrence and survival: a review of the literature," *The American Journal of Clinical Nutrition*, vol. 92, no. 3, pp. 471–490, 2010.
- [42] S. A. Prince, K. B. Adamo, M. E. Hamel, J. Hardt, S. C. Gorber, and M. Tremblay, "A comparison of direct versus self-report measures for assessing physical activity in adults: a systematic review," *International Journal of Behavioral Nutrition and Physical Activity*, vol. 5, article no. 55, 2008.
- [43] E. W. Gregg, Y. J. Cheng, K. M. V. Narayan, T. J. Thompson, and D. F. Williamson, "The relative contributions of different levels of overweight and obesity to the increased prevalence of

- diabetes in the United States: 1976–2004," Preventive Medicine, vol. 45, no. 5, pp. 348–352, 2007.
- [44] D. E. King, A. G. Mainous III, M. Carnemolla, and C. J. Everett, "Adherence to Healthy Lifestyle Habits in US Adults, 1988– 2006," *American Journal of Medicine*, vol. 122, no. 6, pp. 528– 534, 2009.
- [45] Q. Zhang, Y. Wang, and E. S. Huang, "Changes in racial/ethnic disparities in the prevalence of type 2 diabetes by obesity level among US adults," *Ethnicity and Health*, vol. 14, no. 5, pp. 439–457, 2009.

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Research Article

Do Overweight and Obese Individuals Select a "Moderate Intensity" Workload When Asked to Do So?

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The purpose of this study was (1) to determine if overweight/obese individuals (age 26–50 y) would self-select moderate exercise intensity when asked to do so and (2) to determine how this self-selected workload compared to exercising at a workload (60% peak aerobic capacity) that is known to provide cardioprotective health benefits. Oxygen consumption (VO₂) and energy expenditure were measured in 33 men/women (BMI \geq 27 kg/m²) who completed two 30 min walking bouts: (1) self-selected walking pace on an indoor track and (2) prescribed exercise pace (60% VO₂ peak) on a treadmill. The data revealed that (1) the prescribed intensity was 6% higher than the self-selected pace and elicited a higher energy expenditure (P < 0.05) than the self-selected pace (+83 kJ); (2) overweight subjects walked at a slightly lower percentage of VO₂ peak than the obese subjects (P < 0.05); (3) men walked at a lower percentage of VO₂ peak than the women (P < 0.05). In conclusion when asked to walk at a moderate intensity, overweight/ obese individuals tended to select a lower workload in the "moderate intensity" range which could be maintained for 30 min; however, a higher intensity which would be more cardioprotective could not be maintained for 30 min by most individuals.

1. Introduction

Studies have shown that 30 minutes of moderate intensity exercise can confer health benefits if performed on most days of the week (5 days/wk) [1, 2]. These benefits include weight maintenance, weight loss [3], cardiovascular risk management [4, 5], and the avoidance of metabolic dysfunction such as type 2 diabetes [6]. Further, more than 30 min of physical activity a day are necessary for formerly obese individuals to maintain weight loss [7]. Success in obtaining improvements in these health outcomes often occurs because the exercise regimen is carefully controlled in a supervised setting where specific directions are provided [7–9].

As the population has become more obese, numerous guidelines have been established to help the lay public understand the amount of activity that should be incorporated into their lifestyle. For example, the 2008 Physical Activity Guidelines for Americans states that people should participate in

"150 minutes of moderate-intensity aerobic activity every week" (p. vi), which would be approximately 30 minutes of fast walking five days a week [2, 10]. Sedentary individuals, however, may misinterpret the term moderate intensity activity, and their efforts to increase their physical activity levels may result in insufficient workloads for cardioprotection, glucose control, and weight loss and maintenance. Previous research concerned with self-selected exercise pace has used descriptors such as "walk briskly" or "preferred pace" [11–14] but have not examined the pace selected when subjects are told to walk at a "moderate intensity". Yet, "moderate intensity" is the terminology of the physical activity guidelines.

Thus, the purpose of this study was to determine the workload sedentary overweight and obese individuals would self-select when asked to walk at a "moderate intensity". Since exercise at intensities greater than 60% peak oxygen consumption (VO₂ peak) is associated with more health benefits

[15], we compared the self-selected pace to a prescribed pace (60% VO₂ peak). We selected this workload because it was at the low end of the vigorous exercise range, is known to confer health benefits beyond just weight loss, and we anticipated that this intensity would be tolerable by our sedentary participants [16]. We examined the potential differences in heart rate (HR), ratings of perceived exertion (RPE), and energy expenditure at the prescribed versus self-selected "moderate intensity" exercise. Further, we determined if there was a relationship between the self-selected exercise intensity and adiposity or aerobic fitness. We hypothesized that most individuals would self-select a workload at the low end of the moderate intensity exercise pace, when required to walk for 30 min. We further hypothesized that this workload would result in a lower energy expenditure and lower RPE than when the exercise was at an exercise intensity know to confer health benefits.

2. Methods

- 2.1. Subjects. Thirty-three overweight and obese individuals (age 30–50 y) were recruited. Inclusion criteria for the study were a body mass index (BMI) greater than 25 kg/m^2 , non-diabetic status, not on β -blockers, no known cardiovascular disease, no metabolic disorders, no orthopedic limitations and currently not participating in a regular exercise program or coached/guided physical training. All subjects were initially screened by a detailed medical history questionnaire before consideration for the study. All participants were required to complete an informed consent document, approved by the University Institutional Review Board. This study meets the ethical standards of the journal [17].
- 2.2. Experimental Design. All subjects completed 3 visits which included an exercise stress test, a 30 min submaximal walk at a self-selected moderate intensity pace and a 30 min submaximal walk at a 60% of VO₂ peak (prescribed pace). This workload was selected because it is at the low end of the vigorous exercise intensity workload and is known to provide cardioprotective benefits [15]. The submaximal visits were not presented in a randomized fashion because of the potential for learning the ideal walking intensity during the prescribed exercise intensity condition. These visits were a minimum of 72 h apart and no longer than 2 weeks apart.
- 2.3. Exercise Stress Test. Each subject walked on a Quinton Treadmill (Bothell, Washington), and a modified walking protocol was utilized that started at 2.5 mph and 0% grade [18]. Briefly, an increasing workload was administered by initially increasing speed in increments of 0.5 mph per stage until 3.5 mph was achieved. Thereafter, increases in grade of 2.5% per stage were administered until volitional fatigue [19]. At the end of each stage, a rating of perceived exertion (RPE) on a scale of 6–20 was collected from the subject [20]. The American College of Sports Medicine (ACSM) guidelines were followed to establish if a physician needed to be present [21]. During exercise, expired gases were collected and analyzed for volume, O₂ and CO₂ content using a

Cosmed Quark b4 Metabolic Analyzer (Rome, Italy) that was calibrated prior to each test. Oxygen consumed (VO₂) and CO₂ produced (VCO₂) were calculated. Heart rate (HR) was collected continuously with a Polar Heart Rate Monitor (Polar Electro, Lake Success, NY) throughout the study. Criteria for a successful test were matched in accordance with the ACSM [22].

- 2.4. Body Composition. Body composition was assessed using air-displacement plethysmography (Life Measurement, Inc. Concorde, CA) and the test was administered according to the manufacture's guidelines. Height and weight were measured without shoes on prior to testing using a Healthometer (Sunbeam Products Inc, Boca Raton, FL). Body mass index was calculated as weight (kg)/height (m²). Subjects were asked to wear the same clothing on each visit.
- 2.5. Self-Selected Submaximal Exercise Test. During the second visit, subjects were instructed to walk at a "moderate intensity" around an indoor track for thirty minutes. Subjects self-selected the pace they wanted to utilize for the 30 minutes of exercise and received encouragement to keep walking but were given no further directions than to walk at a "moderate intensity". We calculated the distance travelled on the track to determine walking speed. Subjects were not given feedback on their walking speed. Time per lap was monitored with a stopwatch (Timex Ironman Marathon Stopwatch, Timex, N. Little Rock, AR). Speed varied somewhat in a given lap, but there were no significant differences in the lap times documented during the walk for each lap (data not shown). In addition, no significant change in VO₂ was observed between laps. During this test, the subject donned a harness that carried the K4 Cosmed portable metabolic analyzer (Rome, Italy) which was calibrated before each test. HR was measured continuously using Polar Heart Rate technology. Ratings of perceived exertion were collected every five minutes of exercise. At the end of thirty minutes, total distance covered was measured in order to calculate average speed.
- 2.6. Prescribed Exercise at 60% VO_2 Peak. The subjects walked on a treadmill for 30 minutes at \sim 60% VO_2 peak. The exercising pace was selected based on the performance on the VO_2 peak test from the first visit. Wearing the same K4 apparatus as the track visit, the subject walked on a Quinton Treadmill while expired gases were collected. Heart rate was measured continuously, and again RPE was ascertained at the end of every five minutes.
- 2.7. Data Analysis. The breath by breath VO₂ data were averaged into 15 second intervals. The VO₂ peak was considered the highest value achieved at the end of the test while meeting all other ACSM guidelines for a successful stress test [22]. The calculated VO₂ from the track and treadmill evaluations was averaged into one-minute intervals and from these data energy expenditure per minute was calculated [23–26], as well as per kg fat free mass (FFM). Total expenditure for the duration of exercise of both visits was then calculated as the

TABLE 1: Descriptive characteristics of the subject separated by gender.

	Females $(n = 24)$	Males $(n = 9)$
	(16 obese/8 overweight)	(5 obese/4 overweight)
Age (yr)	41.7 ± 1.5	43.3 ± 2.2
rige (yr)	(26–50)	(30–50)
Height (cm)	164.5 ± 1.2	$180.7 \pm 1.7^*$
rieight (em)	(153–175)	(173–191)
Weight (kg)	89.2 ± 2.9	$101.6 \pm 5.6^*$
weight (kg)	(69–123)	80–137)
Body mass	33.1 ± 1.1	31.1 ± 1.3
index (kg/m²)	(27–48)	(27–40)
% body fat	35.6 ± 2.0	$26.3 \pm 2.3*$
70 body lat	(27–48)	(20–48)
VO ₂ peak	27.0 ± 1.2	$37.3 \pm 2.4^*$
(mL/kg/min)	(18-42)	(27–45)

^{*}P < 0.05 between genders. Mean \pm standard error (range of values).

sum of the one-minute averages. Both total kJ and steady state values are presented. Steady state values were used for the calculation of average VO₂ and HR responses to exercise in order to accurately represent level of workload accomplished. Also from the VO₂ data the MET level was calculated with 3.5 mL/kg/min equaling 1 MET [22].

2.8. Statistical Analysis. The data were tested for normality using the Shapiro-Wilk test of normality. A paired t-test was performed to compare variables from the track and treadmill visit. When comparing for gender differences, a 2 (male versus female) X treatment (self-selected versus prescribed) ANOVA with repeated measures or 2 (obese versus overweight) X treatment (self-selected versus prescribed) ANOVA with repeated measures was employed. Using the statistical package for the social sciences (SPSS) for Windows (Chicago, IL ver. 17), all variables were expressed as mean \pm standard error. A Pearson correlation was used to determine the relationship between the differences in energy expenditure and other measured variables, as well as between walking speed and descriptive subject variables. Those descriptive variables that were positively associated with walking speed were put into a linear regression analysis using the enter method to find the best predictor of walking speed. This method allows the entry of variables into the analysis and allows the program to select the order of entering the variable into the model. Significance was accepted at a preset alpha = 0.05.

3. Results

Thirty-three subjects volunteered for this study, 24 females and 9 males. The BMI ranged from $27-40 \text{ kg/m}^2$, and of the 33 subjects 21 subjects were obese and 12 were overweight (Table 1). The men and women were similar in age (\sim 42.5 y) but the men were significantly (P < 0.05) heavier, taller, and

had a greater percent body fat. The men were slightly more fit (P < 0.05) than the women.

Total energy expenditure for the prescribed walking was significantly greater than the energy expenditure for selfselected walking pace (955.7 \pm 39.2 kJ versus 872.7 \pm 39.9 kJ, P < 0.01, resp.), resulting in 83 KJ more being expended at the prescribed pace than in the self-selected pace. The men had a greater energy expenditure than the women (self-selected: males 1057.7 \pm 51.0 kJ and females 803.4 kJ \pm 26.4 kJprescribed: males 1218.6 \pm 35.1 kJ and females 857.1 \pm 22.3 kJ, P < 0.01), and there were no differences in total energy expenditure between the obese and overweight subjects. Adjusting the data for fat free mass did not alter the findings, and the prescribed walking pace (10.4 \pm 0.3 kJ/kg FFM/min) elicited a higher rate of energy expenditure than the self-selected pace (9.4 \pm 0.3 kJ/kg FFM/min, P < 0.05; Figure 1). However, men had a higher energy expenditure (kJ/kg FFM/min) than the women (P < 0.01), and the overweight subjects had a slightly higher energy expenditure than the obese subjects (P < 0.05).

Self-selected exercise resulted in a workload of 52% of VO₂ peak versus 58% of VO₂ peak for the prescribed exercise (P < 0.05). The mean prescribed %VO₂ is lower than we had targeted as many of the subjects could not complete 30 min of exercise at ~60% VO₂ peak. The overweight subjects walked at a lower percentage of VO₂ peak than the obese subjects (P < 0.05), regardless if the exercise pace were self-selected or prescribed. The %HR max for the 30 min of prescribed and self-selected walking were not significantly different (selfselected: 75.1 \pm 11.2 b/min; prescribed: 75.5 \pm 7.9, Table 2). Mean RPE values for the prescribed walking was slightly greater than for the self-selected walking pace (self-selected: mean 11.3 \pm 0.2, range 8–13 versus prescribed: mean 12.5 \pm 0.3, range 11–15, Table 2); no sex differences were observed. Converting the work intensity to METs revealed no differences until 15 minutes of exercise where the MET levels significantly increased and the self-selected intensity was less than the prescribed intensity, and remained lower through the remaining 30 minutes (self-selected 4.2 \pm 0.2, prescribed 4.9 ± 0.2 METs, P < 0.01).

An association was found between the difference in energy expended between the prescribed and self-selected walking and VO₂ peak (r=0.53, P<0.01, Figure 2(a)), and adjusting for sex did not alter this finding. The best predictor of the self-selected walking speed in this cohort of subjects was fat mass which explained 32% of the variability (β coefficient = -0.564, $R^2=0.32$, P<0.001, Figure 2(b)). Fitness, age, height, and sex were not significant predictors of walking speed in this cohort of subjects.

4. Discussion

Although the current physical activity recommendation for health is to accumulate at least 30 min of moderate intensity physical activity daily, the interpretation of moderate can vary considerably between individuals. A misconception of the term "moderate" may potentially result in the selected physical activity workload not being in an appropriate

TABLE 2: Ratings of perceived	d exertion and % max heart ra	ate for the self-selected and	prescribed walking pace	for both the males and females.
TABLE 2. Ratings of perceived	a caci tioni and 70 maa ncart ra	ite for the sem-selected and		

Variable		Female	Males	Total
RPE	Self-selected	11.7 ± 0.2	10.1 ± 0.3	11.3 ± 0.2
	Prescribed pace	$12.6 \pm 0.4**$	$12.2 \pm 0.4**$	12.5 ± 0.3
% max HR	Self-selected	78.0 ± 1.9	67.9 ± 1.9	75.1 ± 2.0
	Prescribed pace	77.4 ± 1.4	70.5 ± 1.4	75.5 ± 1.4

Mean \pm SE. *P < 0.05 between testing days, **P < 0.001 between testing days.

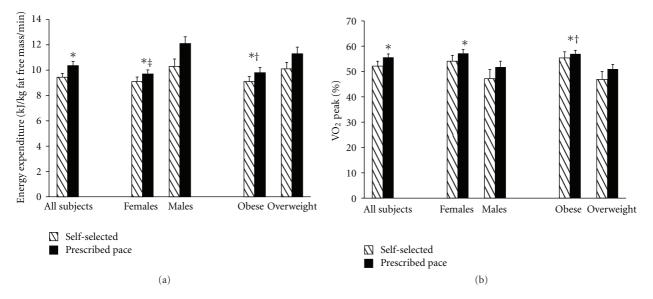


FIGURE 1: The rate of energy expenditure (a) and percent peak oxygen consumption (VO₂ peak) (b) for the self-selected walking pace and the prescribed walking pace for 33 overweight subjects during 30 minutes of exercise. *P < 0.05 between the self-selected and prescribed pace; †P < 0.05 between obese and overweight subjects; †P < 0.01 between men and women.

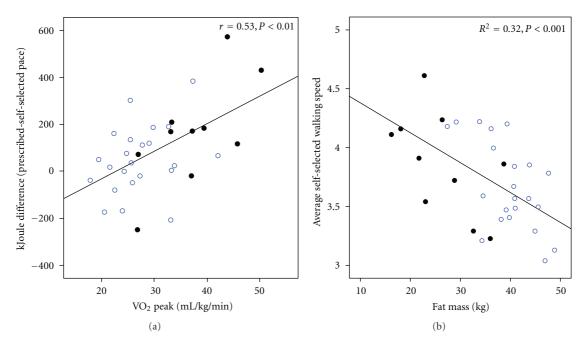


FIGURE 2: (a) The association between VO₂ peak and the difference in energy expended between the prescribed and self-selected walking pace and (b) the association between self-selected walking speed and fat mass. ○ Women, ● Men.

intensity range to be cardioprotective. The present study revealed the following: (1) the self-selected pace (52% VO₂ peak) was in the range that is defined as moderate (40–59% VO₂ peak), (2) the HR response was similar between experimental conditions yet, (3) RPE was greater in the prescribed bout than when the exercise was self-selected, and (4) a lower energy expenditure occurred with the self-selected workload than with the prescribed workload. These findings indicate that when participants are allowed to self-select exercise intensity, the workload would meet the ACSM standards and potentially would be effective in increasing cardiorespiratory fitness; however, the workload may not be adequate to provide other health benefits [27].

Our subjects were able to select a workload in the range of moderate intensity when asked; however, when the subjects were asked to increase the workload slightly to the high end of the moderate intensity range (~58% of VO₂ peak), 68% of the subjects could not complete our prescribed workload, and the workload had to be reduced after 15–20 min of exercise. In addition, this reduction in workload may have resulted in slightly smaller differences in energy expenditure between the self-selected and prescribed pace. Further the obese individuals worked at a higher %VO₂ peak than the overweight subjects, in agreement with findings by Mattsson et al. [14]. Possibly, obese individuals receive more information on the appropriate exercise pace or possibly they have a smaller VO₂ reserve than an overweight individual thus working at a higher percentage of their aerobic capacity.

The inability to complete 30 min at the higher intensity parallels the findings of Ekkekakis and Lind [28] who noted that imposing a speed that is just 10% higher than what overweight women self-selected led to a decline in reported pleasure and decreased affective responses. This may explain why studies in controlled settings obtain more dramatic effects on health variables [29, 30] than when subjects participate in unsupervised exercise. Moreover, other studies have shown that subjects told to exercise at a "brisk pace" and not at their "preferred pace" the exercise intensity increased to levels well above the minimum of the recommended range [11, 12]. Hence, this provides further evidence that the descriptor used in the physical activity recommendation must be carefully considered.

Although there was no difference in the exercise HR between the self-selected and prescribed trial, RPE increased with the prescribed workload, indicating cardiophysiological tolerance but increased perception of the workload [31]. This finding highlights that there may be a mismatch between perceived-exertion and physiological responses of Borg's model of effort continua in sedentary obese individuals. More specifically, a higher intensity of exercise is less pleasurable and enjoyable, thus perceived as greater exertion. Lind et al. [13] have shown that declines in affective valence and consistent ratings of perceived exertion are found when subjects are at an exercise intensity that exceeds the transition across the lactate threshold. We did not measure the lactate threshold in the present study, but a workload at 60% VO2 peak most likely would have been at or above their lactate threshold, based on other studies in sedentary individuals [32]. An RPE of 13 is considered to be approximately the lactate threshold

[33] so our subjects as well as those subjects from the other studies kept their self-selected paces below the lactate threshold. Further it was also observed that as subject fitness increased, as represented by VO_2 peak, there was an increase in the difference in energy expended between walking trials (Figure 2).

The workload selected in the moderate range may reduce CHD risk factors or all-cause mortality, but this workload may be inadequate for weight loss or to improve other health outcomes. From a meta-analysis conducted by Swain and Franklin [15] greater cardioprotective benefits were obtained with an increase in either the relative or absolute intensity of exercise. In fact, a faster walking speed was associated with a reduced CHD risk, independent of the total energy expenditure [15, 34]. One study found that only physical activities at intensities >4.5 METs were associated with a decreased incidence of hypertension and reduced all-cause mortality [35]. Our self-selected pace was below this MET value while the prescribed pace was above this value. Additionally more favorable risk profiles occur with vigorous activity compared to moderate-intensity physical activity. Swain and Franklin [15] reported that 3 studies indicated that groups exercising at the highest intensity (65-75% of VO₂ max) experienced a decrease in diastolic blood pressure, but the group that exercised at lower intensities (45-57% VO₂ max) did not. Likewise improvements in glucose control and insulin sensitivity only occurred at vigorous intensities (65-70% VO₂ max) but not at moderate intensities (40–55% VO₂ max). In general, greater relative intensities result in greater improvements in aerobic fitness and selected CHD risk factors. Other epidemiologic studies have shown that each 1-MET increases in exercise capacity confers an 8-17% reduction in cardiovascular and all-cause mortality [15, 36, 37]. A review of the literature has shown that in low-fit subjects a minimal intensity of 30% VO₂ reserve is needed, while in high-fit subjects exercise above 40% VO₂ reserve is needed to improve cardiovascular fitness [27]. In the present study only the prescribed workload was adequate to provide some of these health benefits and still many subjects could not complete this workload; this agrees with previous research [31].

In the self-selected walking pace, our subjects walked at a lower perceived exertion (~11) than when the exercise was prescribed. This RPE was slightly higher than that reported by Pintar et al. [38], who noted that both normal weight and overweight women (~20 yr old) selected a preferred walking intensity that resulted in the selection of a similar RPE (\sim 10) for a 15 minute trial. The women had a slightly higher RPE for the task than men, and more closely obtained the desirable exercise intensity. Mattsson et al. [14] reported that normal weight subjects use about 36% VO₂ max when walking at a self-selected comfortable pace. Similarly Huebschmann et al. [39] reported that during cycle ergometer exercise that individuals with type 2 diabetes perceived the work to be more difficult (higher RPE) than obese individuals, even when adjusted for the relative work intensity. The higher self-selected RPE in our study may be because the selfselected pace on the track was their first testing day and the subjects may have been slightly anxious. In contrast to our finding, Dasilva et al. reported a higher perceived exertion

and a less positive affective valence in treadmill walking than during over ground walking [40].

The best predictor of walking speed in the present study was fat mass which explained 32% of the variability in selfselected speed; fitness did not significantly explain the variability in walking speed. These data support an inverse relationship between adiposity and self-selected walking speed and suggested that obese individuals may choose lower walking speed in their daily exercise regimens as compared to lean individuals. Pintar [38] reported that fitness but not body weight influenced preferred exercise intensity. Discrepancies in these findings are most likely due to the fact that they used college-aged women, while our subject pool was more middle-aged and both men and women were included. Furthermore, it is well established in the literature that the addition of nonmetabolically active weight increases energy expenditure proportional to the weight [41]. Thus individuals carrying more body fat would most likely perceive the exercise to be more difficult.

A potential limitation of the present study is that visits two and three could not be randomized due to the learning effect that the prescribed exercise visit may have had on the self-selected walking pace. By allowing our subjects to walk on a track, this enabled the subject to have normal variance in speed and be blinded to pace by walking on the track during the second visit. All visits were separated by at least three days to avoid the influence of fatigue and muscle soreness; no subject was tested a second time if they still had muscle soreness following the first study day. There is also the possible confounding factor that the self-selected pace was measured on a track, while the prescribed pace was on the treadmill. We have previously shown that energy expenditure does not differ when walking on the track or treadmill at similar speeds [42]; however other investigators have found a higher metabolic cost of treadmill walking [40, 43] and a higher perceived exercise and a less positive affective valence [40].

In this paper we have only cited the ACSM and AHA guidelines of "150 minutes of moderate intensity aerobic activity every week" (p. vi), which would equal 30 minutes of walking five days a week. However, it should be acknowledged that there are other guidelines that have been published (e.g., the Institute of Medicine, World Health Organization, USDA, etc.) that recommend for the prevention of weight gain at least 60 min of moderate physical activity daily is needed and for weight loss/weight maintenance this may need to be as high as 60–90 min daily [7]. Individuals wishing to lose weight may have difficulty not only completing 30 minutes of exercise but also completing longer durations which have been shown to be necessary for successful weight loss [7]. Further it should be noted that although we collected the data as one exercise bout for 30 minutes, there has been considerable research that has indicated that the 30 minutes (or 60 minutes) can be accumulated using multiple short bouts of exercise [44], which may be a more efficient strategy for individuals who are incorporating higher intensity physical activity into their day or need to put in 60-90 minutes/day.

According to the recommendations from ACSM and the American Heart Association, individuals should participate

in at least 150 minutes a week of moderate intensity cardiovascular exercise or 75 minutes a week of vigorous-intensity aerobic physical activity. Previous literature has indicated that more vigorous exercise is needed for many of the health-related benefits. Despite "moderate exercise" being a nebulous term, which can be widely interpreted, these preliminary data show that subjects self-selected a workload in the middle of the moderate range, but less than the workload (>60% VO₂ peak-high end of the moderate intensity range) that may be necessary to confer health benefits beyond just weight loss [15]. Yet many individuals cannot maintain that workload for 30 minutes. This finding potentially has broad implications to practitioners in giving physical activity recommendations, as workload recommendations at the high end of the moderate range may result in a decreased in adherence. Further men and overweight subjects tended to select lower workloads when asked to exercise at a moderate pace.

References

- [1] R. R. Pate, M. Pratt, S. N. Blair et al., "Physical activity and public health: a recommendation from the centers for disease control and prevention and the american college of sports medicine," *Journal of the American Medical Association*, vol. 273, no. 5, pp. 402–407, 1995.
- [2] Centers for Disease Control and Prevention, "Physical Activity Guidelines for Americans," 2008, http://www.cdc.gov/physicalactivity/everyone/guidelines/adults.html.
- [3] K. Ohkawara, S. Tanaka, M. Miyachi, K. Ishikawa-Takata, and I. Tabata, "A dose-response relation between aerobic exercise and visceral fat reduction: systematic review of clinical trials," *International Journal of Obesity*, vol. 31, no. 12, pp. 1786–1797, 2007
- [4] Y. Goldberg, M. Boaz, Z. Matas, I. Goldberg, and M. Shargorodsky, "Weight loss induced by nutritional and exercise intervention decreases arterial stiffness in obese subjects," *Clinical Nutrition*, vol. 28, no. 1, pp. 21–25, 2009.
- [5] 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.
- [6] J. F. Brun, S. Bordenave, J. Mercier, A. Jaussent, M. C. Picot, and C. Préfaut, "Cost-sparing effect of twice-weekly targeted endurance training in type 2 diabetics: a one-year controlled randomized trial," *Diabetes and Metabolism*, vol. 34, no. 3, pp. 258–265, 2008.
- [7] J. M. Jakicic, B. H. Marcus, W. Lang, and C. Janney, "Effect of exercise on 24-month weight loss maintenance in overweight women," *Archives of Internal Medicine*, vol. 168, no. 14, pp. 1550–1559, 2008.
- [8] I. Giannopoulou, L. L. Ploutz-Snyder, R. Carhart et al., "Exercise is required for visceral fat loss in postmenopausal women with type 2 diabetes," *Journal of Clinical Endocrinology and Metabolism*, vol. 90, no. 3, pp. 1511–1518, 2005.
- [9] R. Ross, I. Janssen, J. Dawson et al., "Exercise-induced reduction in obesity and insulin resistance in women: a randomized controlled trial," *Obesity Research*, vol. 12, no. 5, pp. 789–798, 2004.
- [10] W. L. Haskell, I. M. Lee, R. R. Pate et al., "Physical activity and public health: updated recommendation for adults from the american college of sports medicine and the american

- heart association," *Circulation*, vol. 116, no. 9, pp. 1081–1093, 2007.
- [11] C. F. Fitzsimons, C. A. Greig, D. H. Saunders et al., "Responses to walking-speed instructions: implications for health promotion for older adults," *Journal of Aging and Physical Activity*, vol. 13, no. 2, pp. 172–183, 2005.
- [12] E. M. Murtagh, C. A. G. Boreham, and M. H. Murphy, "Speed and exercise intensity of recreational walkers," *Preventive Medicine*, vol. 35, no. 4, pp. 397–400, 2002.
- [13] E. Lind, R. R. Joens-Matre, and P. Ekkekakis, "What intensity of physical activity do previously sedentary middle-aged women select? Evidence of a coherent pattern from physiological, perceptual, and affective markers," *Preventive Medicine*, vol. 40, no. 4, pp. 407–419, 2005.
- [14] E. Mattsson, U. E. Larsson, and S. Rossner, "Is walking for exercise too exhausting for obese women?" *International Jour*nal of Obesity and Related Metabolic Disorders, vol. 21, no. 5, pp. 380–386, 1997.
- [15] D. P. Swain and B. A. Franklin, "Comparison of cardioprotective benefits of vigorous versus moderate intensity aerobic exercise," *American Journal of Cardiology*, vol. 97, no. 1, pp. 141–147, 2006.
- [16] G. E. Duncan, S. D. Anton, S. J. Sydeman et al., "Prescribing exercise at varied levels of intensity and frequency: a randomized trial," *Archives of Internal Medicine*, vol. 165, no. 20, pp. 2362–2369, 2005.
- [17] D. J. Harriss and G. Atkinson, "Ethical standards in sport and exercise science research," *International Journal of Sports Medicine*, vol. 30, no. 10, pp. 701–702, 2009.
- [18] T. Baynard, R. M. Franklin, S. Goulopoulou, R. Carhart, and J. A. Kanaley, "Effect of a single versus multiple bouts of exercise on glucose control in women with type 2 diabetes," *Meta-bolism*, vol. 54, no. 8, pp. 989–994, 2005.
- [19] J. A. Kanaley, S. Goulopoulou, R. M. Franklin et al., "Plasticity of heart rate signalling and complexity with exercise training in obese individuals with and without type 2 diabetes," *International Journal of Obesity*, vol. 33, no. 10, pp. 1198–1206, 2009.
- [20] G. A. Borg, "Psychological basis of physical exertion," *Medicine* & Science in Sports & Exercise, vol. 14, pp. 377–380, 1982.
- [21] A.C.o.S. Medicine, ACSM's Guidelines for Exercise Testing and Prescription, Lippencott Williams & Wilkens, Hagerstown, Md, USA, 8th edition, 2009.
- [22] American College of Sports Amedicine, *ACSM's Guidelines* for Exercise Testing and Prescription, Lippincott Williams & Williams, Philadelphia, Pa, USA, 2009.
- [23] Y. H. Chang and R. Kram, "Metabolic cost of generating horizontal forces during human running," *Journal of Applied Physiology*, vol. 86, no. 5, pp. 1657–1662, 1999.
- [24] C. T. Farley and T. A. McMahon, "Energetics of walking and running: insights from simulated reduced- gravity experiments," *Journal of Applied Physiology*, vol. 73, no. 6, pp. 2709– 2712, 1992.
- [25] R. Kram and C. R. Taylor, "Energetics of running: a new perspective," *Nature*, vol. 346, no. 6281, pp. 265–267, 1990.
- [26] T. M. Lejeune, P. A. Willems, and N. C. Heglund, "Mechanics and energetics of human locomotion on sand," *Journal of Experimental Biology*, vol. 201, no. 13, pp. 2071–2080, 1998.
- [27] D. P. Swain and B. A. Franklin, "VO₂ reserve and the minimal intensity for improving cardiorespiratory fitness," *Medicine* and Science in Sports and Exercise, vol. 34, no. 1, pp. 152–157, 2002.

[28] P. Ekkekakis and E. Lind, "Exercise does not feel the same when you are overweight: the impact of self-selected and imposed intensity on affect and exertion," *International Journal of Obesity*, vol. 30, no. 4, pp. 652–660, 2006.

- [29] S. Lee, J. L. Kuk, L. E. Davidson et al., "Exercise without weight loss is an effective strategy for obesity reduction in obese individuals with and without type 2 diabetes," *Journal of Applied Physiology*, vol. 99, no. 3, pp. 1220–1225, 2005.
- [30] R. Ross, I. Janssen, J. Dawson et al., "Exercise-induced reduction in obesity and insulin resistance in women: a randomized controlled trial," *Obesity Research*, vol. 12, no. 5, pp. 789–798, 2004.
- [31] P. Ekkekakis, "Let them roam free?: physiological and psychological evidence for the potential of self-selected exercise intensity in public health," *Sports Medicine*, vol. 39, no. 10, pp. 857–888, 2009.
- [32] W. McArdle, F. Katch, and V. Katch, *Exercise Physiology: Nutrition, Energy and Human Performance*, Lippincott, Williams & Williams, Philadelphia, Pa, USA, 7th edition, 2010.
- [33] J. J. Demello, K. J. Cureton, R. E. Boineau, and M. M. Singh, "Ratings of perceived exertion at the lactate threshold in trained and untrained men and women," *Medicine and Science in Sports and Exercise*, vol. 19, no. 4, pp. 354–362, 1987.
- [34] X. Z. He and D. W. Baker, "Changes in weight among a nationally representative cohort of adults aged 51 to 61, 1992 to 2000," *American Journal of Preventive Medicine*, vol. 27, no. 1, pp. 8–15, 2004.
- [35] B. E. Ainsworth, W. L. Haskell, A. S. Leon et al., "Compendium of physical activities: classification of energy costs of human physical activities," *Medicine and Science in Sports and Exercise*, vol. 25, no. 1, pp. 71–80, 1993.
- [36] S. N. Blair, H. W. Kohl, C. E. Barlow, R. S. Paffenbarger, L. W. Gibbons, and C. A. Macera, "Changes in physical fitness and all-cause mortality: a prospective study of healthy and unhealthy men," *Journal of the American Medical Association*, vol. 273, no. 14, pp. 1093–1098, 1995.
- [37] J. Myers, M. Prakash, V. Froelicher, D. Do, S. Partington, and J. Edwin Atwood, "Exercise capacity and mortality among men referred for exercise testing," *New England Journal of Medicine*, vol. 346, no. 11, pp. 793–801, 2002.
- [38] J. A. Pintar, R. J. Robertson, A. M. Kriska, E. Nagle, and F. L. Goss, "The influence of fitness and body weight on preferred exercise intensity," *Medicine and Science in Sports and Exercise*, vol. 38, no. 5, pp. 981–988, 2006.
- [39] A. G. Huebschmann, E. N. Reis, C. Emsermann et al., "Women with type 2 diabetes perceive harder effort during exercise than nondiabetic women," *Applied Physiology, Nutrition and Metabolism*, vol. 34, no. 5, pp. 851–857, 2009.
- [40] S. G. Dasilva, L. Guidetti, C. F. Buzzachera et al., "Psychophysiological responses to self-paced treadmill and overground exercise," *Medicine and Science in Sports and Exercise*, vol. 43, no. 6, pp. 1114–1124, 2011.
- [41] J. F. Miller and B. A. Stamford, "Intensity and energy cost of weighted walking versus running for men and women," *Jour*nal of Applied Physiology, vol. 62, no. 4, pp. 1497–1501, 1987.
- [42] C. Hall, A. Figueroa, B. Fernhall, and J. A. Kanaley, "Energy expenditure of walking and running: comparison with prediction equations," *Medicine and Science in Sports and Exercise*, vol. 36, no. 12, pp. 2128–2134, 2004.
- [43] K. Parvataneni, L. Ploeg, S. J. Olney, and B. Brouwer, "Kinematic, kinetic and metabolic parameters of treadmill versus

- overground walking in healthy older adults," *Clinical Biomechanics*, vol. 24, no. 1, pp. 95–100, 2009.
- [44] M. H. Murphy and A. E. Hardman, "Training effects of short and long bouts of brisk walking in sedentary women," *Medicine and Science in Sports and Exercise*, vol. 30, no. 1, pp. 152–157, 1998.

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Research Article

The Association of Obesity with Walking Independent of Knee Pain: The Multicenter Osteoarthritis Study

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Practice guidelines recommend addressing obesity for people with knee OA, however, the association of obesity with walking independent of pain is not known. We investigated this association within the Multicenter Osteoarthritis Study, a cohort of older adults who have or are at high risk of knee OA. Subjects wore a StepWatch to record steps taken over 7 days. We measured knee pain from a visual analogue scale and obesity by BMI. We examined the association of obesity with walking using linear regression adjusting for pain and covariates. Of 1788 subjects, the mean steps/day taken was 8872.9 \pm 3543.4. Subjects with a BMI \geq 35 took 3355 fewer steps per day independent of knee pain compared with those with a BMI \leq 25 (95% CI -3899, -2811). BMI accounted for 9.7% of the variability of walking while knee pain accounted for 2.9%. BMI was associated with walking independent of knee pain.

1. Introduction

Walking for exercise is promoted by the Arthritis Foundation and the American College of Rheumatology for people with knee osteoarthritis (OA) in order to promote healthy living [1, 2]. Walking, among other types of physical activity, results in a reduction in knee pain and improvement in functional ability [3, 4]. Moreover, meeting physical activity guidelines through activities like walking can reduce the risk of death [5]. This is particularly noteworthy given that knee OA is associated with an increased risk of all-cause death [6] and given the rising prevalence of knee OA [7], this mortality risk poses significant public health implications.

For older adults with knee OA, knee pain is associated with difficulty walking [8], and often considered the primary

culprit for low levels of physical activity and walking [9]. Obesity is also associated with difficulty walking and low levels of physical activity, and it is a primary risk factor for knee OA [10, 11]. About 1/3 of the United States population is obese with a BMI of at least 30 [12]. Furthermore, one in three obese adults have arthritis [13]. However, the association of obesity independent of knee pain with walking is not known. This is an important association to understand since weight loss is rarely prescribed to patients in practice [14] and instead clinicians typically focus on pharmacologic therapies for knee pain. While such practice is in contrast to treatment guidelines for OA, which recommend both weight loss intervention and pain management [15], evidence supporting these guidelines for an outcome of walking is sparse.

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Hence, the purpose of our study is to examine the association of obesity with walking independent of knee pain. We hypothesized that obesity would be associated with walking independent of knee pain.

2. Materials and Methods

2.1. Sample. The cross-sectional study sample consisted of participants in the Multicenter Osteoarthritis (MOST) Study, a large multicenter longitudinal cohort study of communitydwelling subjects who have or are at high risk of knee OA [16]. The MOST sample included adults aged 50 to 79 years were recruited from the communities in and surrounding Birmingham, Alabama and Iowa City, Iowa. Inclusion criteria, based on risk for knee OA, included the presence of known risk factors, including being age over 50 years, female gender, previous knee injury or operation, and body weight in excess of the median weight for each ageand sex-specific group based on data from the Framingham OA Study [17]. The MOST study protocol was approved by the institutional review boards at the University of Iowa in Iowa City, University of California San Francisco, University of Alabama in Birmingham, and Boston University Medical Center.

2.2. Walking Subsample. Information on walking, pain, and obesity were collected at the 60-month MOST follow-up exam between June of 2009 and January of 2011. We restricted the analysis sample to those participants who had a minimum of 3 days of walking data since previous studies have found this to be the minimum number of days needed for a reliable estimate of physical activity [18].

Of the 2330 MOST participants attending the 60-month follow-up visit, 16% (377) did not agree to wear the StepWatch, and 2% (58) had monitor malfunctions. Of the remaining 1895 participants who wore the StepWatch, 94% (1788/1895) wore it for at least 3 valid days and represent the study sample. The StepWatch was worn for 3, 4, 5, 6, and 7 days by 3%, 4%, 7%, 12%, and 74% of participants, respectively. In general, participants included in this analysis were more likely to have better health status (e.g., lower BMI, depressive symptoms, less muscular weakness, and fewer comorbidities) compared with those not included in the analysis (data not shown).

2.3. Outcome

2.3.1. Walking. Following collection of clinic data where pain and body mass index (BMI) were collected, trained research assistants followed a written protocol to fit the StepWatch to the ankle of each study participant, and provided written and verbal instructions for putting on the device each morning and taking off the device at bedtime for the next 7 days.

The StepWatch is a small ($70 \times 50 \times 20$ mm; 38 g), water-proof, self-contained device that is worn on the ankle and records the number of steps taken every minute while providing no feedback to the user. The StepWatch has high concurrent validity in comparison with several reference standard measures of step frequency in older adults, high convergent

validity in comparison with SF-36 scores among subjects with OA, and high test-retest reliability in adults [19, 20].

To determine if subjects wore the monitor long enough to be counted as a full day, we adopted a published method for processing accelerometry data [21], and defined ten hours of monitoring as the minimum amount of time needed to define a full day. The ten hour threshold represents more than 66% of waking hours and has been utilized as a threshold in studies of physical activity in the general adult population [22] and people with knee OA [21]. Time in use was counted from the first step recorded in the morning to the last step recorded in the evening. To exclude times subjects may have taken off the StepWatch during the day, we omitted times where the monitor registered no steps for 180 consecutive minutes during the day (see the appendix).

We quantified walking as the total number of steps taken per day on average as a continuous outcome. We calculated steps/day by totaling the number of steps taken each valid day of monitoring divided by the number of valid days.

2.4. Independent Variables

2.4.1. Knee Pain Severity. We measured knee pain severity as the average pain in the past 30 days on Visual Analogue Scale (VAS) ranging from 0 to 100. Subjects with two painful knees were categorized according to the VAS pain score of the more painful knee.

2.4.2. Obesity. We defined obesity using from BMI computed from standardized weight and height assessments. For analyses, we treated BMI as a continuous factor and a categorical factor by classified according to World Health Organization (WHO) categories [23].

2.5. Covariates. The following factors were treated as covariates based on existing literature linking them to function or physical activity [16, 24-27]: age, sex, living situation (alone or with someone), education (<college, ≥college), race (white, nonwhite), radiographic knee osteoarthritis (ROA) defined as a Kellgren and Lawrence score of ≥2 in the tibiofemoral joint, pain in the hip, ankle, or foot (present versus absent), comorbidities (0 versus ≥ 1) estimated with the modified Charlson comorbidity index [28], depressive symptoms (<16, ≥16) measured with the Center for Epidemiologic Studies Depression Scale (CES-D) [29], as well as knee strength in tertiles from the mean of four isokinetic knee extensor torque repetitions at 60 deg/sec measured in Newton-Meters (Cybex Inc. Medway, MA). All covariates were collected at the 60 month clinic visit except for living situation, which was collected at the baseline clinic visit.

2.6. Statistical Analysis. We examined levels of walking by employing descriptive statistics and plotted a histogram of the distribution of walking across our sample. Then, we examined the association of pain and BMI with walking. We first examined the independent effects of pain and BMI by calculating effects estimates and 95% confidence intervals (CI) using multiple linear regression adjusting for both pain and BMI (categorically) as well as for covariates. We also

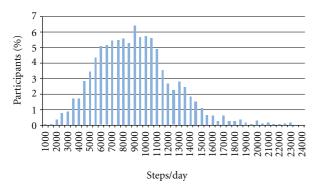


Figure 1: Histogram of steps taken per day. N = 1788.

adjusted for study site (Alabama or Iowa), to account for differences in data collection of StepWatch data and other study variables. We also examined the relative effect of pain and BMI with walking from partial correlation coefficients and standardized beta coefficients. We confirmed walking was normally distributed from visual inspection of Figure 1.

3. Results

The mean age the 1788 subjects included was 67.2 (sd = 7.7) years. Most participants' (36%) were overweight with a BMI \geq 25 and <30 followed by 29% being obese with a BMI \geq 30 and <35. The majority of subjects were women (60%) and white (90%). Table 1 lists characteristics of the subjects included in this study.

The average number of steps taken per day was 8872.9 (sd = 3543.4). Figure 1 shows the average step counts per day taken by participants.

More pain and higher BMI were associated with fewer steps taken per day (see Table 2). Each increase of 10 points on the VAS for pain was associated with taking 167 fewer steps per day. Subjects who were overweight (BMI \geq 25 and <30), in obese class I (BMI \geq 30 and <35), and in obese class II (BMI \geq 35) took 989, 2069, and 3355 fewer steps per day compared with those with a healthy weight (BMI <25).

Mutually adjusting for pain and BMI along with covariates explained 28% of the variability of walking. Pain accounted for 2.9% of the variability while BMI accounted for 9.7% of the variability of walking. Similarly, a one standard deviation increase in pain accounted for a decrease of 0.07 of a standard deviation of walking, while a one standard deviation increase in BMI accounted for a decrease of 0.28 of a standard deviation of walking (see Table 3).

4. Discussion

We found BMI to be strongly associated with walking independent of knee pain. In particular, we found BMI to account for 9.7% of the variability of walking in comparison to only 2.9% for pain. These findings suggest that obesity has an important association with low levels of walking in people with or at high risk of knee OA independent of knee pain.

Knee pain accounted for little of the variability of walking when considered along with the effect of obesity. To put the relative effect of pain into perspective, knee pain accounted for only 10% of the total variability accounted by our model (pain, BMI, and all covariates). In contrast, obesity accounted for 35% of the total variability of the same model. We found a similar trend from the standardized beta coefficients with a one standard deviation increase in BMI accounting for more change in walking than the same increase in pain. This difference is notable given that knee pain is a major cause of functional limitation in people with knee OA [30-33]. However, from a conceptual perspective, the performance of physical function, such as walking speed, is distinctly different than how much physical activity one performs on a daily basis. Furthermore, previous studies have reported a weak association between knee pain and physical activity [34-36]. One possible reason for this is that people may have avoided walking for different reasons. Those with low levels of knee pain did not walk for fear of increasing their knee pain, while those with high knee pain were unable to walk due to current pain levels. Disentangling these associations is needed for future longitudinal studies.

We found obesity to have a strong association with walking, which has been reported previously in adults who are normal weight and obese and general population studies [37, 38]. Subjects in the highest BMI category walked over 3000 steps less per day than those in the lowest BMI category. The magnitude of this difference is clinically meaningful as it approaches a one standard deviation difference for walking in our sample. Given that our study is cross-sectional, we cannot infer causal direction, and association between obesity and walking is likely bidirectional. For instance, low levels of walking or physical activity could result in obesity. Similarly, people who are obese could have difficulty walking and hence have low levels of walking. Irrespective of the directionality, we found obesity to be strongly associated with walking independent of pain, which underscores the obesity epidemic in the United States and the importance of addressing obesity to avoid future poor health outcomes.

Step counts collected in our study cannot be compared with previous studies utilizing pedometers. Pedometers are known underestimate the number of steps taken by older adults up to 33% compared with a StepWatch [39], hence step counts in our study are higher than pedometer based studies. However, the average step counts in our study are comparable with smaller studies that employed the StepWatch in people with knee or hip OA and older adults [40, 41]. For instance, Winter et al. reported 30 people with radiographic knee OA walked 9350 steps/day, which is similar to our finding of 9194 and 8598 steps/day for men and women, respectively.

Our study has several strengths. First, we report daily walking from a large cohort of people with or at high risk of knee OA who wore a validated walking monitor. Second, this is the first study to report the association of obesity with walking independent of knee pain in people with or at high risk of knee OA using a well-validated objective monitor. There are some limitations to our study. First, subjects may have changed walking habits with the knowledge that their habitual walking was being recorded. Previous study suggests this "testing effect" is greatest when subjects wear an unsealed

TABLE 1: Summar	v of baseline characteristics	across all subjects and	within BMI categories.
TABLE 1. Sullillial	v of baseinie characteristics	actoss all subjects affu	within Divil Categories.

	All subjects $(n = 1788)$	BMI <25 (n = 269)	BMI ≥ 25 and < 30 (n = 641)	BMI ≥ 30 and < 35 (n = 529)	BMI ≥ 35 $(n = 352)$
Knee pain (0–100) [Mean (sd)]	18.8 (20.9)	15.6 (18.3)	16.6 (19.5)	19.6 (20.8)	24.2 (23.8)
BMI [kg/m] [Mean (sd)]	30.7 (6.0)				
BMI <25 [%]	15				
BMI ≥25 and <30 [%]	36				
BMI ≥30 and <35 [%]	29				
BMI ≥35 [%]	20				
Age [Mean (sd)]	67.2 (7.7)	67.9 (7.8)	68.4 (8.0)	67.0 (7.6)	64.5 (6.7)
Sex [% women]	60	68	57	57	63
Living situation [% Lives alone]	17	16	16	17	18
Education [% ≥ College]	47	55	47	45	44
Race [% White]	90	96	92	89	85
Site [% Alabama]	38	37	38	37	39
Radiographic Knee Osteoarthritis [%]	54	41	52	55	71
Pain in the hip, ankle, or foot [%]	52	43	47	53	66
No Comorbidity [%]	59	66	61	59	51
Depressive Symptoms [Mean CES-D (sd)]	6.4 (6.8)	5.3 (5.8)	5.8 (6.1)	6.8 (7.3)	7.7 (7.6)
Knee extensor strength [N-M/kg] [Mean (sd)]	1.03 (0.41)	1.17 (0.41)	1.11 (0.43)	1.00 (0.37)	0.82 (0.33)

Table 2: Change in the number of daily steps attributed to pain and BMI after adjustment for covariates.

	Adjusted* beta [95% CI]
Knee Pain (10 unit increments on 0–100 VAS scale)	-166.8 [-245.5, -88.1]
BMI	
<25 "healthy weight"	Reference
≥25 and <30 "overweight"	-989.4 [-1437.0, -541.7]
≥30 and <35 "obese class I"	-2069.6 [-2540.1 , -1599.2]
≥35 "obese class II-III"	-3355.1 [-3899.4, -2810.8]

^{*} Mutually adjusted for pain and BMI as well as age, sex, living situation, education, race, study site, Radiographic Knee Osteoarthritis, pain in the hip, ankle, or foot, comorbidity, depressive symptoms, and isokinetic knee extensor strength.

monitor, that is, when subjects are aware of how many steps are being recorded [42–44]. We believe any increases in walking due to a testing effect were minimized since study participants did not know the number of steps that were recorded. Second, we acknowledge we employed few psychological measures as covariates in our analyses. We were limited to measures already collected in the MOST study and were not able to add measures of self efficacy or fear avoidance, which are likely associated with walking. Lastly, our sample consisted of people both with and at high risk of knee osteoarthritis, therefore, it is not clear if our findings are directly generalizable to those with knee OA. We performed a sensitivity analysis stratifying our sample by those with and without ROA and found similar effects for pain and obesity within each strata compared with our main findings.

5. Conclusion

We found BMI to be associated with walking independent of pain in the studied sample of people with or at high risk of knee OA. These findings support clinical practice guidelines that obesity is an important modifiable factor to intervene upon and particularly relevant for walking among people with or at high risk of knee OA. Future research should investigate the longitudinal association of BMI and knee pain with walking in order to better understand the temporal relationships between these factors.

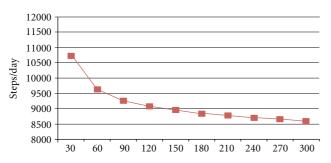
Appendix

Previous literature to distinguish periods of inactivity from nonwear have employed monitors designed to measure all types of physical activity (Actigraph). These studies have employed thresholds of 60 minutes to 90 minutes of no activity to represent nonwear. However, it is not known if these same thresholds can be generalized to a monitor only measuring step counts, as periods of physical inactivity likely differ from periods of not walking worn by people with or at high risk of knee OA. One previous study of adult bariatric surgical candidates found a threshold of 120 minutes of no

Table 3: Percentage of variability of walking attributed to pain, BMI, and covariates and expected change in walking for each one standard deviation increase in pain, BMI, and continuous covariates.

	D (1.1	C ₄ 1 1: 1
	Partial	Standardized
	R-square (%)	Beta*
Knee pain (0–100)	2.9	-0.08
BMI (continuous)	9.7	-0.28
Age	8.4	-0.28
Sex	0.3	
Living situation	0.1	
Education	0.0	
Race	0.0	
Site	2.1	
Radiographic Knee Ostearthritis	0.2	
Pain in the hip, ankle, or foot	0.0	
Comorbidity	2.4	
Depressive Symptoms	0.7	
Isokinetic knee extensor strength	1.6	0.17

^{*} Calculated for continuous variables only.



Nonwear threshold values: minimum consecutive minutes of no steps to classify nonwear time

Figure 2

steps was suggested as nonwear time [45]. We examined how increases in threshold values of nonwear time changed reporting of the daily average wear time, steps, and time walking at a moderate intensity.

As threshold values of nonwear time increased, the monitor was counted as being worn for a greater duration of time. Subsequently, the average number of steps/day decreased. However, changes in steps/day did not change appreciably using threshold values of nonwear greater than 180 minutes. As a result, we employed a 180 minute threshold of no steps to distinguish periods of inactivity from not wearing the monitor (for more details see Figure 2).

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References

- [1] "Walk with Ease," 2009, http://www.arthritis.org/programs .php.
- [2] R. D. Altman, M. C. Hochberg, R. W. Moskowitz, and T. J. Schnitzer, "Recommendations for the medical management of osteoarthritis of the hip and knee: 2000 update," *Arthritis and Rheumatism*, vol. 43, no. 9, pp. 1905–1915, 2000.
- [3] 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.
- [4] 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.
- [5] "Physical activity guidelines for Americans," US Department of Health and Human Services, 2008.
- [6] E. Nüesch, P. Dieppe, S. Reichenbach, S. Williams, S. Iff, and P. Jüni, "All cause and disease specific mortality in patients with knee or hip osteoarthritis: population based cohort study," *BMJ*, vol. 342, article d1165, 2011.
- [7] G. Spahn, R. Schiele, G. O. Hofmann et al., "The prevalence of radiological osteoarthritis in relation to age, gender, birth-year cohort, and ethnic origins," *Zeitschrift fur Orthopadie und Unfallchirurgie*, vol. 149, no. 2, pp. 145–152, 2011.
- [8] M. Shih, J. M. Hootman, J. Kruger, and C. G. Helmick, "Physical activity in men and women with arthritis National Health Interview Survey, 2002," *American Journal of Preventive Medicine*, vol. 30, no. 5, pp. 385–393, 2006.
- [9] D. D. Dunlop, J. Song, P. A. Semanik et al., "Objective physical activity measurement in the osteoarthritis initiative: are guidelines being met?" *Arthritis and Rheumatism*, vol. 63, no. 11, pp. 3372–3382, 2011.
- [10] D. T. Felson, Y. Zhang, J. M. Anthony, A. Naimark, and J. J. Anderson, "Weight loss reduces the risk for symptomatic knee osteoarthritis in women: the Framingham Study," *Annals of Internal Medicine*, vol. 116, no. 7, pp. 535–539, 1992.
- [11] S. J. Strath, R. G. Holleman, D. L. Ronis, A. M. Swartz, and C. R. Richardson, "Objective physical activity accumulation in bouts and nonbouts and relation to markers of obesity in US adults," *Preventing chronic disease*, vol. 5, no. 4, article A131, 2008.
- [12] "Obestiy and Overweight," 2011, http://www.cdc.gov/nchs/ fastats/overwt.htm.
- [13] "Prevalence of doctor-diagnosed arthritis and arthritisattributable activity limitation—United States, 2007–2009," *Morbidity and Mortality Weekly Report*, vol. 59, no. 39, pp. 1261–1265, 2010.
- [14] D. J. Hunter, T. Neogi, and M. C. Hochberg, "Quality of osteoarthritis management and the need for reform in the us," *Arthritis Care and Research*, vol. 63, no. 1, pp. 31–38, 2011.
- [15] W. Zhang, G. Nuki, R. W. Moskowitz et al., "OARSI recommendations for the management of hip and knee osteoarthritis. Part III: changes in evidence following systematic cumulative update of research published through January 2009," Osteoarthritis and Cartilage, vol. 18, no. 4, pp. 476–499, 2010.

[16] D. T. Felson, J. Niu, A. Guermazi et al., "Correlation of the development of knee pain with enlarging bone marrow lesions on magnetic resonance imaging," *Arthritis and Rheumatism*, vol. 56, no. 9, pp. 2986–2992, 2007.

- [17] D. T. Felson, J. J. Anderson, A. Naimark, A. M. Walker, and R. F. Meenan, "Obesity and knee osteoarthritis. The Framingham Study," *Annals of Internal Medicine*, vol. 109, no. 1, pp. 18–24, 1988.
- [18] S. G. Trost, K. L. Mciver, and R. R. Pate, "Conducting accelerometer-based activity assessments in field-based research," *Medicine and Science in Sports and Exercise*, vol. 37, no. 11, pp. S531–S543, 2005.
- [19] B. Resnick, E. S. Nahm, D. Orwig, S. S. Zimmerman, and J. Magaziner, "Measurement of activity in older adults: reliability and validity of the step activity monitor," *Journal of Nursing Measurement*, vol. 9, no. 3, pp. 275–290, 2001.
- [20] S. Mudge, D. Taylor, O. Chang, and R. Wong, "Test-retest reliability of the StepWatch Activity Monitor outputs in healthy adults," *Journal of Physical Activity & Health*, vol. 7, no. 5, pp. 671–676, 2010.
- [21] J. Song, P. Semanik, L. Sharma et al., "Assessing physical activity in persons with knee osteoarthritis using accelerometers: data from the osteoarthritis initiative," *Arthritis Care & Research*, vol. 62, no. 12, pp. 1724–1732, 2010.
- [22] R. P. Troiano, D. Berrigan, K. W. Dodd, L. C. M\u00e4sse, T. Tilert, and M. Mcdowell, "Physical activity in the United States measured by accelerometer," *Medicine and Science in Sports and Exercise*, vol. 40, no. 1, pp. 181–188, 2008.
- [23] WHO, Obesity: preventing and managing the global epidemic. Report of a WHO Consultation, WHO Technical Report Series 894, World Health Organization, Geneva, Switzerland, 2000.
- [24] A. A. Guccione, D. T. Felson, and J. J. Anderson, "Defining arthritis and measuring functional status in elders: methodological issues in the study of disease and physical disability," *American Journal of Public Health*, vol. 80, no. 8, pp. 945–949, 1990.
- [25] J. Jordan, G. Luta, J. Renner, A. Dragomir, M. Hochberg, and J. Fryer, "Knee pain and knee osteoarthritis severity in self-reported task specific disability: The Johnston County Osteoarthritis Project," *Journal of Rheumatology*, vol. 24, no. 7, pp. 1344–1349, 1997.
- [26] A. A. Eyler, R. C. Brownson, S. J. Bacak, and R. A. Housemann, "The epidemiology of walking for physical activity in the United States," *Medicine and Science in Sports and Exercise*, vol. 35, no. 9, pp. 1529–1536, 2003.
- [27] L. Sharma, S. Cahue, J. Song, K. Hayes, Y. C. Pai, and D. Dunlop, "Physical functioning over three years in knee osteoarthritis: role of psychosocial, local mechanical, and neuromuscular factors," *Arthritis and Rheumatism*, vol. 48, no. 12, pp. 3359–3370, 2003.
- [28] J. N. Katz, L. C. Chang, O. Sangha, A. H. Fossel, and D. W. Bates, "Can comorbidity be measured by questionnaire rather than medical record review?" *Medical Care*, vol. 34, no. 1, pp. 73–84, 1996.
- [29] L. Radloff, "The CES-D scale: a self-report depression scale for research in the general population," *Applied Psychological Measurement*, vol. 1, pp. 385–401, 1977.
- [30] C. D. Mallen, G. Peat, E. Thomas, R. Lacey, and P. Croft, "Predicting poor functional outcome in community-dwelling older adults with knee pain: prognostic value of generic indicators," *Annals of the Rheumatic Diseases*, vol. 66, no. 11, pp. 1456–1461, 2007.
- [31] E. Thomas, G. Peat, C. Mallen et al., "Predicting the course of functional limitation among older adults with knee pain:

- do local signs, symptoms and radiographs add anything to general indicators?" *Annals of the Rheumatic Diseases*, vol. 67, no. 10, pp. 1390–1398, 2008.
- [32] S. Ayis, S. Ebrahim, S. Williams, P. Jüni, and P. Dieppe, "Determinants of reduced walking speed in people with musculoskeletal pain," *Journal of Rheumatology*, vol. 34, no. 9, pp. 1905–1912, 2007.
- [33] C. Jinks, K. Jordan, and P. Croft, "Osteoarthritis as a public health problem: the impact of developing knee pain on physical function in adults living in the community: (KNEST 3)," *Rheumatology*, vol. 46, no. 5, pp. 877–881, 2007.
- [34] S. L. Murphy, D. M. Smith, D. J. Clauw, and N. B. Alexander, "The impact of momentary pain and fatigue on physical activity in women with osteoarthritis," *Arthritis Care and Research*, vol. 59, no. 6, pp. 849–856, 2008.
- [35] S. G. Thomas, S. M. C. Pagura, and D. Kennedy, "Physical activity and its relationship to physical performance in patients with end stage knee osteoarthritis," *Journal of Orthopaedic and Sports Physical Therapy*, vol. 33, no. 12, pp. 745–754, 2003.
- [36] C. C. Winter, M. Brandes, C. Müller et al., "Walking ability during daily life in patients with osteoarthritis of the knee or the hip and lumbar spinal stenosis: a cross sectional study," BMC Musculoskeletal Disorders, vol. 11, article no. 233, 2010.
- [37] J. N. Davis, V. A. Hodges, and M. B. Gillham, "Physical activity compliance: differences between overweight/obese and normal-weight adults," *Obesity*, vol. 14, no. 12, pp. 2259– 2265, 2006.
- [38] T. Dwyer, D. Hosmer, T. Hosmer et al., "The inverse relationship between number of steps per day and obesity in a population-based sample—the AusDiab study," *International Journal of Obesity*, vol. 31, no. 5, pp. 797–804, 2007.
- [39] B. M. Tyo, E. C. Fitzhugh, D. R. Bassett, D. John, Y. Feito, and D. L. Thompson, "Effects of body mass index and step rate on pedometer error in a free-living environment," *Medicine and Science in Sports and Exercise*, vol. 43, no. 2, pp. 350–356, 2011.
- [40] D. K. White, Y. Zhang, D. T. Felson et al., "The independent effect of pain in one versus two knees on the presence of low physical function in a multicenter knee osteoarthritis study," *Arthritis Care and Research*, vol. 62, no. 7, pp. 938–943, 2010.
- [41] C. C. Winter, M. Brandes, C. Müller et al., "Walking ability during daily life in patients with osteoarthritis of the knee or the hip and lumbar spinal stenosis: a cross sectional study," *BMC Musculoskeletal Disorders*, vol. 11, article no. 233, 2010.
- [42] S. A. Clemes and R. A. A. Parker, "Increasing our understanding of reactivity to pedometers in adults," *Medicine and Science in Sports and Exercise*, vol. 41, no. 3, pp. 674–680, 2009.
- [43] N. Wollmerstedt, U. Nöth, A. Ince, H. Ackermann, J. M. Martell, and C. Hendrich, "The daily activity questionnaire. A novel questionnaire to assess patient activity after total hip arthroplasty," *Journal of Arthroplasty*, vol. 25, no. 3, pp. 475–480.e3, 2010.
- [44] M. Brandes, M. Ringling, C. Winter, A. Hillmann, and D. Rosenbaum, "Changes in physical activity and health-related quality of life during the first year after total knee arthroplasty," *Arthritis Care & Research*, vol. 63, no. 3, pp. 328–334, 2011.
- [45] W. C. King, J. Li, K. Leisear, J. E. Mitchell, and S. H. Belle, "Determining activity monitor wear time: an influential decision rule," *Journal of Physical Activity and Health*, vol. 8, no. 4, pp. 566–580, 2011.

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Research Article

Sitting Time and Cardiometabolic Risk Factors in African American Overweight Women

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Findings from previous research linking sedentary time with cardiometabolic risk factors and body composition are inconsistent, and few studies address population groups most vulnerable to these compromising conditions. The purpose of this paper was to investigate the relationship of sitting time to cardiometabolic risk factors and body composition among African American women. A subsample of African American women (N=135) completed health and laboratory assessments, including measures of blood pressure, resting heart rate, cholesterol, triglycerides, glucose, body mass index, body fat, sitting time, and demographics. Simultaneous, adjusted regression models found a positive association between weekend sitting time and glucose and an inverse association between weekly sedentary time and cholesterol (ps < .05). There were no significant associations between sedentary behavior and body composition. The unexpected relationship between sedentary time and cholesterol suggests that the relationship of sedentary behavior to cardiometabolic risk factors may depend on existing characteristics of the population and measurement definition of sedentary behavior. Results suggest distinctly different relationships between weekend and weekday sitting time, implicating a need for careful measurement and intervention that reflects these differences.

1. Introduction

Diseases of the heart continue to be the leading cause of death in the United States [1, 2] and accounted for 24.6% of all deaths in 2009, down only 1.4% from 2006 despite several national campaigns and research strategies to reduce mortality [1, 2]. African Americans have poorer health outcomes compared to their white counterparts [1, 2], and African American women disproportionately suffer from heart disease, with nearly half (45%) of African American women having some type of cardiovascular disease compared to only 32% of white women [3, 4].

Cardiometabolic risk factors, such as high blood pressure and resting heart rate, elevated cholesterol and glucose levels, and high body fat percentage, are associated with cardiovascular diseases [5] and may result from lifestyle choices, such as physical inactivity and poor dietary habits [6, 7]. The prevalence of high blood pressure, or hypertension, among African Americans in the United States has increased from 35.8% to 41.4% between 1988 and 2002 and is particularly high among African American women (44.8% in 2006) compared to white women (31.1% in 2006) [8, 9]. Among

African Americans, the prevalence of high (\geq 200 mg/dL) and elevated (\geq 240 mg/dL) cholesterol is higher among women than men, with 54.9% of African American women having high or elevated cholesterol compared to 51.1% of African American men [8].

Body composition and obesity are also directly linked to cardiovascular diseases and other health compromising conditions, such as diabetes and cancer [10]. Over one-third (39.2%) of African American women are obese or have a body mass index (BMI) \geq 30 kg/m², compared to only 21.8% of White women, 25.4% of White men, and 31.6% of African American men [11]. African American women also have greater adiposity or body fat compared to Caucasians [12, 13].

Regularly performed physical activity improves body composition and nearly all known health conditions; yet, self-reported measures suggest nearly 50% of the adult population fails to meet minimum physical activity recommendations [14], while objective measurement shows only 5% meet recommendations [15], suggesting that sedentary time comprises the largest portion of most people's days. African American women are less physically active than

white women, putting them at greater risk for chronic health conditions related to physical inactivity, including cardiovascular diseases [16], and leading to rising health care costs, which exceeded \$11 billion among morbidly obese adults in 2000 [17].

Several studies have looked at the relationship between sedentary behavior and disease risk, but few have looked at specific measured sedentary behavior, defining sedentary time as low physical activity during leisure time [18–20]. For example, increased sedentary time has been associated with increased BMI, mortality rates, high glucose levels, and insulin resistance, regardless of physical activity level among both men and women [21-25]. However, there have also been recent studies that did not demonstrate these relationships [26-28]. In addition, a recent literature review suggested that although there is not sufficient evidence to support significant relationships between sedentary behavior and body weight gain and sedentary behavior and cardiovascular disease biomarkers, there is evidence in the literature to support a strong relationship between sedentary behavior and mortality, suggesting that, while important, these relationships are not well documented or described

Inconsistencies in the reported relationships between sedentary behavior and cardiometabolic risk factors and body composition suggest that specific population or measurement characteristics of studies may be contributing to findings. For example, one study showed that 27.3% to 95.9% of the association between sedentary behavior and health outcomes (e.g., blood pressure, cholesterol) was explained by BMI or waist circumference [30]. Most of the literature has included a majority of white participants [22, 27, 28, 30], and the relationship of sedentary behavior to cardiometabolic risk factors and body composition in ethnic minority women remains unclear. One study suggested that decreased occupational sitting time may decrease BMI and promote healthy behaviors among women [25], but few have explored the direct relationship between sitting time that includes both weekday and weekend, occupational and leisure time, or have distinguished between occupational and leisure sitting time and its associations with cardiometabolic risk factors and body composition. The purpose of this study was to investigate the relationship of weekday and weekend sitting time to cardiometabolic risk factors, including blood pressure, resting heart rate, cholesterol, triglycerides and glucose, and body composition among overweight and obese African American women. We hypothesized that increased sitting time would be associated with higher rates of cardiometabolic risk factors, obesity, and increased body fat and explored the issue of whether weekday (occupational) sitting time or weekend (recreational) sitting time would be more important in contributing to cardiometabolic outcomes.

2. Materials and Method

2.1. Participants. The current study was a secondary analysis using data from the Health Is Power (HIP) study (1R01CA109403). Four hundred ten community dwelling,

African American (n = 263), and Hispanic or Latina (n = 263) 147) women participated in HIP, a multisite, longitudinal, community-based, randomized controlled trial to increase physical activity [31-42] in Houston and Austin, Texas. Eligible participants, self-identified as African American or Hispanic or Latina, were between the ages of 25 and 60 years old, able to read, speak, and write in English or Spanish, not pregnant or planning to become pregnant within the next 12 months, a Harris or Travis County resident, not planning on moving in the next 12 months, physically inactive or doing fewer than 30 minutes of physical activity per day on 3 or more days per week, and free from health conditions that would be aggravated by physical activity [43]. A subsample of Houston African American participants (n = 135) completed laboratory assessments at baseline, Time 1 (T1) [31, 41]. All HIP study assessments, measures and procedures were approved by the Committee for the Protection of Human Subjects at the University of Houston, and women provided written informed consent prior to participation.

2.2. Assessments. Women who met inclusionary criteria gave informed consent and completed a T1 health assessment, where they completed an interviewer administered questionnaire measuring physical activity and demographics, measures of blood pressure and resting heart rate, and anthropometric measures of BMI and body fat [36–38, 42].

The laboratory assessment included a venous blood sample and a whole body dual-energy X-ray absorptiometry (DXA) scan [31, 41]. Women completed these measures after fasting for 8 or more hours and wore metal-free clothing.

2.3. Cardiometabolic Risk Factors. Systolic and diastolic blood pressures were measured using manual aneroid sphygmomanometry by a trained research team member using established protocols. Participants were asked to sit quietly during measurement with their left arm bared and supported at heart level and their feet flat on the floor. Two readings were obtained, separated by two minutes, and averaged for use in analyses. If the first two readings differed by more than 5 mmHg, a third reading was obtained and averaged.

Resting heart rate was assessed after participants sat quietly for two minutes. A trained assessor measured their radial pulse at their left wrist [44]. Assessors counted beats for one full minute and repeated the procedure for accuracy. An average of the two measurements in beats per minute was used in analyses.

A venous blood sample was collected from a peripheral arm vein into Vacutainers pretreated with either sodium heparin or K2 EDTA (Vacutainer; Becton-Dickinson, Franklin Lakes, NJ) after 8 or more hours of fasting [31] and analyzed for plasma total cholesterol, high-density lipoprotein (HDL), low-density lipoprotein (LDL), triglyceride, and glucose concentrations using separate enzymatic assays in triplicate as described by the manufacturer (Pointe Scientific, Canton, MI). A ratio of total cholesterol to HDL (total cholesterol/HDL) was also used in analyses.

2.4. Body Composition. Anthropometric measures of BMI and body fat were collected by trained personnel using established protocols [36–38, 41, 42]. Individual height was measured using a standard stadiometer apparatus with participants' shoes being removed. Body weight and percent body fat were measured twice using bioelectrical impedance analysis (BIA) using a Tanita TBF-310 body composition analyzer (Tanita, Arlington Heights, Illinois). BMI was calculated using stadiometer heights and BIA body weights. All measures were collected twice, and the average of the two measurements was used in analyses.

Percent body fat was also measured by DXA. DXA measurements were completed by a trained staff member between 6:00 and 8:00 AM and took 10–15 minutes per participant. DXA scans were used to measure whole body fat mass, lean mass, bone mass and total percent body fat, as previously described [31, 41]. Only total percent body fat was used in current analyses.

- 2.5. Sitting Time. Sitting time was measured using items from the International Physical Activity Questionnaire (IPAQ) long form administered at the baseline health assessment. The IPAQ long form is typically used to measure work-related, transportation, domestic and leisure-time physical activity. In addition, the instrument measures time spent sitting over the last seven days by time spent sitting in a motor vehicle and time spent sitting during the week and weekend [18]. Sitting time was reported in terms of total minutes during the week and weekend. Adhering to the IPAQ protocol, data were cleaned and missing or spurious data were excluded from any analyses [36].
- 2.6. Sociodemographics. Items assessing ethnicity, household income, and education were adapted from the Maternal Infant Health Assessment (MIHA) survey [45], derived from the CDC's Pregnancy Risk Assessment Monitoring System (PRAMS) Questionnaire [46]. Items have shown good reliability and have been used with samples representing diverse ethnicities [47].
- 2.7. Statistical Analyses. All statistical analyses were conducted in SPSS version 19.0 (IBM SPSS Statistics for Windows, IBM Corporation, Somers, NY). The current study is limited to a subsample of African American women enrolled in Houston, TX, who were offered a laboratory assessment (n = 135) at baseline T1. Only participants with complete data for a particular measure were included in all analyses, which varied by assessment procedure/measure. Women in the subsample were slightly older (M = 46.6 years, SD = 8.9) than the total African American sample (M = 42.9years, SD = 9.6; t = -3.141, P = .002) but were similar in education, income, BMI, and percent body fat. Bivariable correlations were conducted among cardiometabolic risk factor variables and body composition variables and between cardiometabolic risk factor and body composition variables. Simultaneous linear regression models were used to estimate the effect of weekday and weekend sitting time on cardiometabolic risk factors, including systolic and diastolic

blood pressure, resting heart rate, total cholesterol, HDL, LDL, triglycerides, the ratio of total cholesterol to HDL, and glucose, and on body composition, including BMI, BIA percent body fat, and DXA percent body fat, controlling for age, education, and income. Significance for all analyses was set at P < .05.

3. Results

- 3.1. Descriptive Characteristics. African American women were middle aged (M=46.6 years, SD = 8.9) and obese (M BMI = 34.9 kg/m², SD = 9.5). Over half (52.7%) had graduated from college, and the majority (56.7%) reported an income 401% or greater above the Federal Poverty Level [48] or an income greater than \$82,807. Mean (and SD) cardiometabolic risk factors and body composition are presented in Table 1. Triglycerides varied by education (F(1,26)=5.650, P=.025); women who had not graduated from college had higher triglyceride values than women with a college education (M=73.2 versus 37.9). There were no other significant differences in cardiometabolic factors or body composition by education or income.
- 3.2. Bivariable Correlations. Age was significantly positively correlated with systolic blood pressure (r=.219, P=.011) and glucose (r=.288, P=.036). Correlations between cardiometabolic factors and body composition are shown in Table 2. Total cholesterol was significantly negatively correlated with weekend sitting time (r=-.374, P=.050) and total sitting time (r=-.376, P=.049). LDL was also significantly negatively correlated with weekend sitting time (r=-.425, P=.027). Sitting time was not correlated with any other cardiometabolic risk factors or body composition variables.
- 3.3. Regression Models. There were no significant linear associations between weekday sitting time and either cardiometabolic factors or body composition. In contrast, linear regression models suggest a moderate association between weekend sitting time and glucose ($\beta = .266$, t = 1.960, P = .056), which may be significant with increased power.

Linear regression models mimicked bivariable correlations and indicated a surprising negative linear association for both weekend ($\beta = -.374$, t = -2.058, P = .050) and total ($\beta = -.376$, t = -2.069, P = .049) sitting time to total cholesterol, suggesting that greater sitting time was associated with lower total cholesterol levels. Greater sitting time during the weekend was also associated with lower LDL levels ($\beta = -.425$, t = -2.347, P = .027). Also of interest, there were no significant associations between sitting time and body composition.

4. Discussion

Based on previous research, we expected to find that greater time spent sitting was associated with poorer cardiometabolic indicator values. We found some limited support for this hypothesis with glucose; however, reduced

Table 1: Descriptive information for samplecardiometabolic risk factors and body composition.

	N	Mean	SD	Normal Ranges
Cardiometabolic risk factors				
Systolic blood pressure (mmHg)	135	126.4	14.4	<120
Diastolic blood pressure (mmHg)	135	79.7	10.2	<80
Resting heart rate (beats/min)	135	71.9	8.4	60-100
Total cholesterol (mg/dL)	30	181.3	28.5	<200
HDL (mg/dL)	30	46.6	12.5	≥60
LDL (mg/dL)	29	123.0	25.9	<100
Triglycerides (mg/dL)	29	58.3	46.9	10-150
Total cholesterol/HDL	30	4.0	1.2	
Glucose (mg/dL)	53	86.0	27.3	≤100
Body composition				
Body mass index (kg/m ²)	135	34.9	9.5	18.5-24.9
BIA body fat (%)	134	42.8	7.7	23-35*
DXA total body fat (%)	125	41.7	6.0	23-35*
Sedentary time				
Weekday sedentary time (min)	128	425.0	274.2	
Weekend sedentary time (min)	128	370.4	269.0	
Total sedentary time (min/week)	128	795.5	477.5	

^{*} Normal range for body fat listed is not specific to measurement method and is for women 41 to 60 years old.

TABLE 2: Correlation coefficients between cardiometabolic risk factors and body composition.

	1	2	3	4	5	6	7	8	9	10	11	12
(1) Systolic blood pressure (mmHg)	1	.780**	.101	.111	010	.138	.020	.071	029	.381**	.313**	.220*
(2) Diastolic blood pressure (mmHg).780**	1	.201*	.163	082	.220	.091	.148	091	.423**	.370**	.267**
(3) Resting heart rate (beats/min)	.101	.201*	1	.367*	176	.393*	.352	.291	.016	.260**	.123	.027
(4) Total cholesterol	.111	.163	.367*	1	.258	.883**	.327	.283	.167	.113	.132	032
(5) HDL	010	082	176	.258	1	105	141	687**	144	396*	331	110
(6) LDL	.138	.220	.393*	.883**	105	1	.100	.454*	045	.327	.273	.028
(7) Triglycerides	.020	.091	.352	.327	141	.100	1	.589**	.712**	012	.069	164
(8) Total cholesterol/HDL	.071	.148	.291	.283	687**	.454*	.589**	1	.335	.389*	.282	079
(9) Glucose	029	091	.016	.167	144	045	.712**	.335	1	021	.120	.110
(10) Body mass index (kg/m²)	.381**	.423**	.260**	.113	396*	.327	012	.389*	021	1	.783**	.775**
(11) BIA body fat (%)	.313**	.370**	.123	.132	331	.273	.069	.282	.120	.783**	1	.756**
(12) DXA total body fat (%)	.220*	.267**	.027	032	110	.028	164	079	.110	.775**	.756**	1

^{**} Correlation is significant at the 0.01 level (2-tailed). *Correlation is significant at the 0.05 level (2-tailed).

sample size limited our power to detect this effect. We did not find expected relationships between sitting time and body composition. It may be that since this sample was already overweight and obese, time spent sitting had little impact on this outcome. In contrast to our hypotheses, we found that greater time spent sitting on the weekend was associated with better cholesterol levels, suggesting that in this sample, there is some advantage to time spent sitting that may counteract the sedentary nature of sitting.

The curious relationship that we found between sitting and improved cholesterol suggests that something unexpected is driving this relationship. Perhaps women who have more sitting time are more relaxed and have more leisure time in general. The variables used to measure sedentary time included time spent sitting while driving. In this sample, in sprawling Houston, we have found an inverse association between car ownership and physical activity (results not shown); perhaps more time spent sitting while driving led to more time doing physical activity either because the participant commuted to their physical activity destination or because the increased driving time led to a desire to be more active, which may have impacted these cardiometabolic factors. Another curious finding is that nearly all of the relationships reported here were between weekend sedentary time, rather than weekday time. During the week, perhaps time is more carefully scripted by work and family responsibilities, while weekends have more discretionary time. Future studies should continue to specify time spent during weekends and weekdays separately in terms of both measurement and intervention. In contrast, people

may feel that weekends are a time for rest and relaxation, that is, sedentary time. Thus, intervention strategies that decrease sedentary and sitting time during the week might be more sustainable, as they get integrated along with already ritualized weekday responsibilities.

Previous research exploring the relationship between sitting time and other cardiometabolic risk factors has yielded similar findings. For example, Yates et al. found that sitting time was positively associated with fasting insulin, C-reactive protein, and insulin resistance in women after adjusting for physical activity [49]. However, no other studies have found a relationship between sedentary time and cholesterol or lipoprotein measures in women [27, 50], warranting further investigation to elucidate study findings.

In our sample of community volunteers, we found slightly elevated blood pressure, with 60.1% of the sample exceeding normal ranges for both systolic and diastolic, somewhat higher than the national prevalence [8, 9]. This sample had relatively poor cholesterol levels, with most women having too low values of HDL and too high levels of LDL, similar to national samples [8]. Most of the sample was overweight or obese, which likely reflects not only very high prevalence of high body fat in the population [11–13] but also the nature of the study recruitment, which sought volunteers to enroll in a study to increase physical activity or improve dietary habits.

This study is among the first to investigate the relationship of weekday and weekend sitting time to cardiometabolic risk factors in African American women and includes a sizeable sample of African American women, who are most vulnerable to obesity and chronic health conditions. This study includes the use of validated and reliable measures for this population, including DXA- and BIA-measured percent body fat. However, this study was not without its limitations. A significant study limitation was the use of selfreported sedentary time. Accelerometry is considered the gold standard of physical activity and sedentary behavior measurement and may have enhanced study findings. In addition, this study was limited to the relationship between weekday and weekend sitting time and cardiometabolic risk factors and did not explore the relationship between physical activity and these risk factors, for which there is a known strong relationship. The use of cross-sectional versus longitudinal data limits us from making assumptions about causality in this study, and due to study population characteristics, findings may not be generalized to other non-African American populations. Missing data for laboratory assessments and measures also limits findings and may explain differences in findings between the current study and established literature.

Although this sample was generally representative of African American women in terms of health status, this sample was of higher socioeconomic status, as is often the case with community volunteers in health promotion studies. Future studies should investigate larger samples that represent the entire community and continue to account carefully for sedentary and sitting time. As others have suggested, these findings suggest that simply decreasing sedentary time may not be sufficient to improve cardiometabolic risk and body

composition [28]. These findings may have produced more questions than they answered but underscore the complexity of the relationships between sedentary behavior and health outcomes, particularly in more vulnerable groups in the population.

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References

- [1] M. Heron, D. L. Hoyert, S. L. Murphy, J. Xu, K. D. Kochanek, and B. Tejada-Vera, "Deaths: final data for 2006," *National Vital Statistics Reports*, vol. 57, no. 14, pp. 1–134, 2009.
- [2] K. D. Kochanek, J. Xu, S. L. Murphy et al., "Deaths: preliminary data for 2009," *National Vital Statistics Reports*, vol. 59, no. 4, pp. 1–51, 2011.
- [3] A. M. Minino, M. P. Heron, S. L. Murphy, and K. D. Kochanek, "Deaths: final data for 2004," *National Vital Statistics Reports*, vol. 55, no. 19, pp. 1–119, 2007.
- [4] R. A. Williams, "Cardiovascular disease in African American women: a health care disparities issue," *Journal of the National Medical Association*, vol. 101, no. 6, pp. 536–540, 2009.
- [5] Health, United States, 2010 With Special Feature on Death and Dying, National Center for Health Statistics, Hyattsville, Md, USA, 2011.
- [6] Centers for Disease Control and Prevention, Physical Activity, 2011, http://www.cdc.gov/healthyplaces/healthtopics/ physactivity.htm.
- [7] National Heart Lung and Blood Institute, Your Guide to Lowering High Blood Pressure—Prevention, 2011, http://www.nhlbi.nih.gov/hbp/prevent/prevent.htm.
- [8] 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.
- [9] S. J. Ventura, J. C. Abma, W. D. Mosher, and S. K. Henshaw, "Estimated pregnancy rates by outcome for the United States, 1990–2004.," *National Vital Statistics Reports*, vol. 56, no. 15, pp. 1–28, 2008.
- [10] National Heart Lung and Blood Institute, "Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: the evidence report," Tech. Rep. 98-4083, National Institutes of Health, Bethesda, Md, USA, 1998.
- [11] "Differences in prevalence of obesity among black, white, and Hispanic adults—United States, 2006–2008," *Morbidity and Mortality Weekly Report*, vol. 58, no. 27, pp. 740–744, 2009.
- [12] M. M. Fahlman, H. L. Hall, and R. Lock, "Ethnic and socioeconomic comparisons of fitness, activity levels, and barriers to exercise in high school females," *Journal of School Health*, vol. 76, no. 1, pp. 12–17, 2006.
- [13] G. Hu, C. Bouchard, G. A. Bray et al., "Trunk versus extremity adiposity and cardiometabolic risk factors in white and African American adults," *Diabetes Care*, vol. 34, no. 6, pp. 1415–1418, 2011.

- [14] Centers for Disease Control and Prevention, BRFSS Prevalence and Trends Nationwide (States, DC, Territories) 2009—Adults with 30+ minutes of moderate physical activity five or more days per week, or vigorous physical activity for 20+ minutes three or more days per week, 2009, http://apps.nccd.cdc.gov/BRFSS/display.asp?cat=PA&yr=2009&qkey=4418&state=US.
- [15] R. P. Troiano, "Large-scale applications of accelerometers: new frontiers and new questions," *Medicine and Science in Sports* and Exercise, vol. 39, no. 9, p. 1501, 2007.
- [16] R. C. Brownson, A. A. Eyler, A. C. King, D. R. Brown, Y. L. Shyu, and J. F. Sallis, "Patterns and correlates of physical activity among US women 40 years and older," *American Journal of Public Health*, vol. 90, no. 2, pp. 264–270, 2000.
- [17] D. E. Arterburn, M. L. Maciejewski, and J. Tsevat, "Impact of morbid obesity on medical expenditures in adults," *International Journal of Obesity*, vol. 29, no. 3, pp. 334–339, 2005.
- [18] M. Booth, "Assessment of physical activity: an international perspective," *Research Quarterly for Exercise and Sport*, vol. 71, no. 2, supplement, pp. S114–S120, 2000.
- [19] A. Bankoski, T. B. Harris, J. J. McClain et al., "Sedentary activity associated with metabolic syndrome independent of physical activity," *Diabetes Care*, vol. 34, no. 2, pp. 497–503, 2011.
- [20] K. Wijndaele, N. Duvigneaud, L. Matton et al., "Sedentary behaviour, physical activity and a continuous metabolic syndrome risk score in adults," *European Journal of Clinical Nutrition*, vol. 63, no. 3, pp. 421–429, 2009.
- [21] G. F. Dunton, D. Berrigan, R. Ballard-Barbash, B. Graubard, and A. A. Atienza, "Joint associations of physical activity and sedentary behaviors with body mass index: results from a time use survey of US adults," *International Journal of Obesity*, vol. 33, no. 12, pp. 1427–1436, 2009.
- [22] H. J. F. Helmerhorst, K. Wijndaele, S. Brage, N. J. Wareham, and U. Ekelund, "Objectively measured sedentary time may predict insulin resistance independent of moderate- and vigorous-intensity physical activity," *Diabetes*, vol. 58, no. 8, pp. 1776–1779, 2009.
- [23] A. V. Patel, L. Bernstein, A. Deka et al., "Leisure time spent sitting in relation to total mortality in a prospective cohort of US adults," *American Journal of Epidemiology*, vol. 172, no. 4, pp. 419–429, 2010.
- [24] R. Santos, L. Soares-Miranda, S. Vale, C. Moreira, A. I. Marques, and M. Jorge, "Sitting time and body mass index, in a portuguese sample of men: results from the azorean physical activity and health study (APAHS)," *International Journal of Environmental Research and Public Health*, vol. 7, no. 4, pp. 1500–1507, 2010.
- [25] C. Tudor-Locke, N. W. Burton, and W. J. Brown, "Leisure-time physical activity and occupational sitting: associations with steps/day and BMI in 54–59 year old Australian women," *Preventive Medicine*, vol. 48, no. 1, pp. 64–68, 2009.
- [26] U. Ekelund, S. Brage, S. J. Griffin, and N. J. Wareham, "Objectively measured moderate- and vigorous-intensity physical activity but not sedentary time predicts insulin resistance in high-risk individuals," *Diabetes Care*, vol. 32, no. 6, pp. 1081–1086, 2009.
- [27] K. A. McGuire and R. Ross, "Sedentary behavior is not associated with cardiometabolic risk in adults with abdominal obesity," *Plos ONE*, vol. 6, no. 6, Article ID e20503, 2011.
- [28] K. A. McGuire and R. Ross, "Incidental physical activity and sedentary behavior are not associated with abdominal adipose tissue in inactive adults," *Obesity*, vol. 20, no. 3, pp. 576–582, 2012.

[29] K. I. Proper, A. S. Singh, W. Van Mechelen, and M. J. M. Chinapaw, "Sedentary behaviors and health outcomes among adults: a systematic review of prospective studies," *American Journal of Preventive Medicine*, vol. 40, no. 2, pp. 174–182, 2011.

- [30] E. Stamatakis and M. Hamer, "The extent to which adiposity markers explain the association between sedentary behavior and cardiometabolic risk factors," *Obesity*, vol. 20, no. 1, pp. 229–232, 2012.
- [31] M. L. Kueht, B. K. McFarlin, and R. E. Lee, "Severely obese have greater LPS-stimulated TNF- α production than normal weight African-American women," *Obesity*, vol. 17, no. 3, pp. 447–451, 2009.
- [32] C. S. Layne, S. K. Mama, J. A. Banda, and R. E. Lee, "Development of an ecologically valid approach to assess moderate physical activity using accelerometry in community dwelling women of color: a cross-sectional study," *International Journal of Behavioral Nutrition and Physical Activity*, vol. 8, article 21, 2011
- [33] R. E. Lee and C. Cubbin, "Striding toward social justice: the ecologic milieuof physical activity," *Exercise and Sport Sciences Reviews*, vol. 37, no. 1, pp. 10–17, 2009.
- [34] R. E. Lee, S. K. Mama, K. P. McAlexander, H. Adamus, and A. V. Medina, "Neighborhood and PA: neighborhood factors and physical activity in African American public housing residents," *Journal of Physical Activity & Health*, vol. 8, supplement 1, pp. S83–S90, 2011.
- [35] R. E. Lee, S. K. Mama, A. V. Medina et al., "Neighborhood factors influence physical activity among community dwelling African American and Hispanic or Latina women," *Health & Place*, vol. 18, no. 1, pp. 63–70, 2012.
- [36] R. E. Lee, S. K. Mama, A. V. Medina et al., "Multiple measures of physical activity, dietary habits and weight status in African American and hispanic or latina women," *Journal of Community Health*, vol. 36, no. 6, pp. 1–13, 2011.
- [37] R. E. Lee, A. V. Medina, S. K. Mama et al., "Health is power: an ecological, theory-based health intervention for women of color," *Contemporary Clinical Trials*, vol. 32, no. 6, pp. 916–923, 2011.
- [38] R. E. Lee, D. P. O'Connor, R. S. Ray et al., "Effects and mediators of a group cohesion intervention to increase physical activity and improve dietary habits in women of color: health is power," *American Journal of Health Promotion*, vol. 26, no. 4, pp. e116–e125, 2012.
- [39] R. E. Lee et al., "Social cohesion increases fruit and vegetable consumption in obese ethnic minority women," in *Proceedings* of the 11th International Congress of Behavioral Medicine, Washington, DC, USA, 2010.
- [40] R. E. Lee et al., "Reach and retention of minority women in the Health Is Power (HIP) study," *Annals of Behavioral Medicine*, vol. 41, supplement, p. s127, 2011.
- [41] Y. Lopez III, D. P. O'Connor, T. A. Ledoux et al., "Analysis of body composition methods in a community sample of African American women," *Women Health*, vol. 51, no. 8, pp. 709–723, 2011.
- [42] S. K. Mama, B. E. Quill, M. E. Fernandez-Esquer et al., "Body image and physical activity among Latina and African American women," *Ethnicity and Disease*, vol. 21, no. 3, pp. 281–287, 2011.
- [43] S. Thomas, J. Reading, and R. J. Shephard, "Revision of the physical activity readiness questionnaire (PAR-Q)," *Canadian Journal of Sport Sciences*, vol. 17, no. 4, pp. 338–345, 1992.
- [44] D. Bernstein, "Pediatric heart and heart-lung transplantation," in Nelson Textbook of Pediatrics, R. M. Kliegman,

- R. E. Behrman, H. B. Jenson et al., Eds., Saunders Elsevier, Philadelphia, Pa, USA, 18th edition, 2007.
- [45] California Department of Public Health, Maternal and Infant Health Assessment (MIHA) Survey, 2006, http://apps.nccd.cdc.gov/BRFSS/display.asp?cat=PA&yr=2009&qkey=4418&state=US.
- [46] Centers for Disease Control and Prevention, PRAMS, 2006, http://www.cdc.gov/reproductivehealth/PrenatalSmkbk/data_sources_definitions.htm.
- [47] R. Sarnoff and D. Hughes, "Increasing health insurance coverage in the first year of life," *Maternal and Child Health Journal*, vol. 9, no. 4, pp. 343–350, 2005.
- [48] U.S. Department of Health and Human Services, *The 2007 HHS Poverty Guidelines*, 2007.
- [49] T. Yates, K. Khunti, E. G. Wilmot et al., "Self-reported sitting time and markers of inflammation, insulin resistance, and adiposity," *American Journal of Preventive Medicine*, vol. 42, no. 1, pp. 1–7, 2012.
- [50] A. A. Thorp, G. N. Healy, N. Owen et al., "Deleterious associations of sitting time and television viewing time with cardiometabolic risk biomarkers: Australian diabetes, obesity and lifestyle (AusDiab) study 2004-2005," *Diabetes Care*, vol. 33, no. 2, pp. 327–334, 2010.

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Research Article

Functional Movement Is Negatively Associated with Weight Status and Positively Associated with Physical Activity in British Primary School Children

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Although prior studies have suggested that overweight and obesity in childhood are associated with poorer functional movement performance, no study appears to have examined this issue in a pediatric population. The relations between BMI, ambulatory physical activity and functional movement screen (FMS) performance were compared in 58, 10-11-year-old children. Total FMS score was significantly, negatively correlated with BMI (P=.0001) and positively related to PA (P=.029). Normal weight children scored significantly better for total FMS score compared to children classified as overweight/obese (P=.0001). Mean \pm S.D. of FMS scores were 15.5 \pm 2.2 and 10.6 \pm 2.1 in normal weight and overweight/obese children, respectively. BMI and PA were also significant predictors of functional movement (P=.0001, Adjusted $R^2=.602$) with BMI and PA predicting 52.9% and 7.3% of the variance in total FMS score, respectively. The results of this study highlight that ambulatory physical activity and weight status are significant predictors of functional movement in British children. Scientists and practitioners therefore need to consider interventions which develop functional movement skills alongside physical activity and weight management strategies in children in order to reduce the risks of orthopaedic abnormality arising from suboptimal movement patterns in later life.

1. Introduction

Overweight and obesity in childhood are recognised as a major health problem worldwide [1] and while considerable data have been published relating to influences such as sedentary behaviour and nutritional habits on weight status, there is a dearth of information pertaining to the structural and functional limitations of excess weight in adults [2, 3], with even less data in children. This is despite authors noting that children display alterations to their functional movement as a consequence of excessive weight [2] and suboptimal movement patterns found in overweight and obesity can seriously impede daily physical activity level and limit functional performance [3, 4]. The concern here is that an inability to complete fundamental daily tasks coupled with excessive and prolonged loading of tissues in childhood may lead to orthopaedic abnormality in later life. Therefore, minimizing joint deterioration from excessive

joint loading or impaired movement patterns evident in obese and overweight children should be treated at the earliest opportunity [5]. However, no study appears to have examined these associations in children. Therefore, the aim of this study was to examine relations between habitual physical activity, functional movement patterns, and weight status in children.

2. Materials and Methods

2.1. Participants. Following institutional ethics approval, fifty-eight children (29 boys and 29 girls, 86% Caucasian) from a primary school in Central England volunteered and returned signed parental informed consent forms to participate in the study. Children were from school year 6 (aged 10-11years) and were predominantly Caucasian (81%). The mean age (SD) of the children was 10.7 (0.4) years. The school the children was drawn from was in an urban area

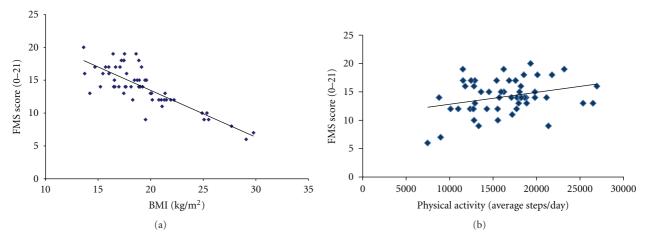


FIGURE 1: Scattergraph illustrating the relationship between; (a) FMS score and BMI and (b) FMS score and physical activity in British children.

of the city of Coventry and was located in the least deprived ward in the city with an average household income of £43,842 in comparison to £31,695 for the city average.

2.2. Procedures

2.2.1. Anthropometry. Body mass (kg) and height (m) were measured using a Stadiometer and weighing scales (Seca Instruments, Germany, Ltd) prior to the monitoring period. From this, body mass index was determined as kg/m². Children were classified as normal weight (68.4%) and overweight/obese (31.6%) according to IOTF criteria [6].

2.2.2. Physical Activity Assessment. Physical activity was assessed using a sealed, piezoelectric pedometer (New Lifestyles, NL2000, Montana, USA) worn over four days ($2 \times \text{weekdays}$ and $2 \times \text{weekend}$ days) in accordance with recommendations for the assessment of physical activity in children [7] and using protocols previously described [1]. Across the measurement period, the children completed a brief survey to verify that the pedometers were worn for the entire time of the study. Survey results were used to identify participants who reported removing the pedometer for $\geq 1 \text{ h}$, resulting in three exclusions from the data set, similar to other studies [8].

2.2.3. Functional Movement Assessment. The Functional Movement Screen (FMS) is a preparticipation screening tool which evaluates the fundamental movement patterns that underpin performance of all movement [9, 10]. The FMS consists of seven tests which challenge an individual's ability to perform basic movement patterns that reflect combinations of muscle strength, flexibility, range of motion, coordination, balance, and proprioception [9, 10]. The FMS was administered by a trained rater using standardised procedures, instructions, and scoring processes [9, 10]. Each participant was given 3 trials on each of the seven tests (deep squat, hurdle step, in-line lunge, shoulder mobility, active straight leg raise, trunk-stability push-up, and rotary

stability) in accordance with recommended guidelines [9, 10]. Each trial was scored from 0 to 3 with higher scores reflecting better functional movement. For each test, the highest score from the three trials was recorded and used to generate an overall composite FMS score with a maximum value of 21 and in accordance with recommended protocols [9, 11].

2.3. Data Analysis. Relationships between total FMS score, BMI and PA (average steps/day) were analysed using Pearson's product moment correlations. A 2 (Gender) \times 2 (weight status: normal weight versus overweight/obese) \times 2 (PA: children meeting/not meeting the health related steps/day cut off (8)) analysis of variance (ANOVA), using backwards elimination to achieve a parsimonious solution, was employed to examine any differences in functional movement (FMS score) according to gender, weight status, and physical activity. Where any significant differences were detected, Bonferroni post-hoc multiple comparisons were used to detect where these differences lay. Multiple linear regression was also employed to predict functional movement (FMS score) from BMI and Physical activity. Statistical significance was set a priori as P = .05.

3. Results

Total FMS score was significantly, negatively correlated with BMI (r=-.806, P=.0001, See Figure 1(a)) and positively related to PA (r=.301, P=.029, see Figure 1(b)). Furthermore, ANOVA found a significant main effect for weight status (F 1, 53 = 50.4, P=.0001) for total FMS score with normal weight children scoring significantly better than their overweight and obese peers (Mean Diff = 4.941, P=.0001, see Figure 2). Mean \pm S.D. of FMS scores were 15.5 \pm 2.2 and 10.6 \pm 2.1 in normal weight and overweight/obese children, respectively. Multiple linear regression also revealed a significant predictor model (F 2, 53 = 40.369, P=.0001, Adjusted $R^2=.602$) whereby BMI and physical activity (average steps/day) predicted 60.2%

	Total FMS score (0–21)		Dhyeica	Physical activity		BMI	
			,	,	(kg/m^2)		
			(Avg st	eps/day)			
	Mean	S.D.	Mean	S.D.	Mean	S.D.	
Boys $(n = 29)$	13.5	3.4	17480	4818	19.9	4.7	
Girls $(n = 29)$	14.5	2.8	15259	3585	19.1	2.9	
Normal weight $(n = 39)$	15.5	2.2	17078	4009	17.5	1.7	
Overweight/obese $(n = 19)$	10.6	2.1	14522	4602	23.3	0.9	

Table 1: Mean ± S.D. of total FMS score, physical activity (average steps/day), and BMI between gender and weight status groups.

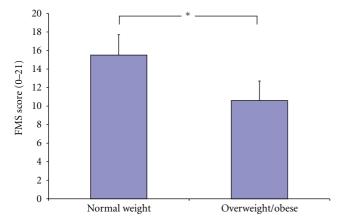


FIGURE 2: Mean \pm S.D. of total FMS score between children classified as normal weight and overweight/obese according to IOTF criteria (*P = .0001).

of the variance in functional movement score. However, BMI was a stronger predictor ($\beta = -.712$, P = .0001) of functional movement predicting 52.9% of the variance in total FMS score in comparison to physical activity ($\beta = 1200.0$, P = .03) where average steps/day predicted 7.3% of the variance in FMS score. Mean \pm S.D. of total FMS score, PA (average steps/day), and BMI between gender and weight status groups are presented in Table 1.

4. Discussion

Results of this study suggest that functional movement is related to weight status and habitual physical activity, with weight status having a stronger association with functional movement in this population. In this case although BMI and physical activity were both significant predictors of functional movement predicting 60.2% of the variance in FMS score, BMI alone predicted 52.9% of this variance in comparison to physical activity, where average steps/day predicted 7.3% of the variance in FMS score. This study supports prior research in adults which has suggested that excess weight results in functional limitations [2, 3] and assertions that children display altered functional movement as a consequence of excess weight [2]. The positive association seen between FMS score and habitual physical activity in the present study supports prior assertions that the lack of physical activity is associated with the lack of functional movement skills in adults [4]. However, in the context of the present study and population examined, it is possible that functional limitation may have existed prior to overweight/obesity. As such, excess weight and/or functional prowess are the result of natural selection since children who are functionally limited will remain inactive and will not develop the fundamental movement patterns that underpin performance to the same level of mastery as children who do not have a functional limitation. In the same way, children who are not functionally limited may more likely enjoy physical activity and thus, engage in more regular practise of the fundamental movement patterns that underpin performance. These children will consequently become more proficient in performance of fundamental movement patterns resulting in greater physical self efficacy and increased likelihood of participation in physical activity. To the authors knowledge this is the first study to examine associations between weight status, physical activity, and FMS in a pediatric population. The data presented here are however, important as they highlight those children who are overweight/obese present maladaptive movement patterns needed to accomplish tasks of daily life. Over time, these movement patterns coupled with the effect of excess weight on joint loading are likely to lead to orthopaedic abnormality in later life [4]. Moreover, such suboptimal movement patterns can prevent individuals from undertaking health enhancing physical activity [4].

There are several proposed mechanisms for the impact of overweight and obesity on functional movement. However, these suggestions have tended to be based on studies in adult populations with few studies examining this issue in children and adolescents. One suggestion is that overweight and obesity lead to alterations in the musculoskeletal system that place overweight individuals at higher risk of musculoskeletal pain [4, 12]. Such pain may then result in restricted range of movement and/or lower levels of physical activity. One study by Messier et al. [12] showed that a weight loss of approximately 5% following a combined diet and exercise programme improved physical function, mobility, and reduced pain in overweight and obese adults with osteoarthritis. However, musculoskeletal pain is considered a chronic condition that is less likely to affect children. Some studies in children and adolescents [13, 14] have evidenced changes in foot structure and plantar pressure distribution, as a result of structural adaptation to excess weight, which was suggested to lead to functional movement complications. Likewise, research by Zoico et al. [15], with older adults, has

concluded that a BMI of 30 kg m² or greater is significantly associated with functional limitation. However, Stenholm et al. [16] has further suggested that it is the ratio of fat free mass (FFM) to body mass which is important as individuals with higher FFM are more likely to be functionally proficient than those with lower FFM. However, while these studies were conducted with older adults, there is a lack of data examining the association between BMI, FFM, muscle strength, and functional movement in children [4]. Future research would therefore be welcomed in this area.

Similarly, only one other study to date [17] has reported specifically on the use of the Functional Movement Screen in a pediatric population. Burton et al. [17] reported that, in a sample of 39 middle school children, there were significantly higher total FMS scores in girls compared to boys, principally due to better performance in the deep squat, in line lunge, straight leg raise, and shoulder rotation. They concluded that girls exhibit superior quality in functional movement compared to boys but suggested that it was important to develop musculoskeletal interventions to improve functional movement in children and adolescents. However, although Burton et al. [17] did not account or control for variation in weight status in their study making it difficult to draw parallels with the current study, the range of total FMS scores reported in their study [17] is similar to those documented in this study. However, there was no gender difference found for FMS score in the present study but as Burton et al. [17] did not assess or control for weight status it is difficult to draw parallels between the two studies.

Irrespective of whether it is functional limitation that results in lack of physical activity and increases in overweight/obesity or whether lack of physical activity and overweight/obesity leads to suboptimal movement, the data present here support the need for interventions to increase habitual physical activity and improve functional movement in British children generally but those who are overweight and obese specifically. It is also important to note that there may be other factors which influence performance of the functional movement screen in children such as motivation to perform the movement patterns and particularly motor learning. Although the children in the present study had been familiarized with the movements involved in the FMS, it may be that there are practice effects in the FMS. No studies to date have examined this issue and the recommended protocols were followed when administering the FMS into the current study. However, future research examining the effects of motor learning on performance of the FMS would be useful in elucidating this area further.

In respect to the clinical significance of the current study, the results present here do not necessarily suggest a clinical need for those children exhibiting poor functional movement at their present age. The worry, however, is that if suboptimal movement patterns, present in children who are overweight/obese, persist into adolescence and adulthood, this will lead to a range of musculoskeletal problems of clinical significance. This includes a range of factors including knee osteoarthritis, early need for hip replacement and chronic pain, in addition to limitations in tasks of daily living (see [4] for a review). Thus, in overweight/obese children

who exhibit suboptimal functional movement, interventions aimed at improving the quality of functional movement via weight loss and/or increases in physical activity energy expenditure may offset more severe clinical implications in later life.

Considering the association between physical activity and FMS found in this study, it may be that the relationship between physical activity and weight status is magnified as children become older if their weight is not managed, resulting in further reduction in physical activity, functional movement, or both. This suggestion is, however, speculative and further research is needed to verify this claim. This exploratory study is also limited by a small sample size and larger scale studies would be welcomed to verify the claims made here. In addition, cause and effect in relation to physical activity and functional movement could not be determined in the present sample.

5. Conclusion

The results of this study do, however, highlight that ambulatory physical activity and weight status are significant predictors of functional movement in British children. In the current study weight status had a greater influence on functional movement compared to physical activity and children who were classified as overweight/obese demonstrated significantly poorer functional movement than children classified as normal weight.

Conflict of Interests

The authors declare they have no conflict of interests.

References

- [1] M. J. Duncan, A. Nevill, L. Woodfield, and Y. Al-Nakeeb, "The relationship between pedometer-determined physical activity, body mass index and lean body mass index in children," *International Journal of Pediatric Obesity*, vol. 5, no. 5, pp. 445– 450, 2010.
- [2] A. P. Hills, E. M. Hennig, N. M. Byrne, and J. R. Steele, "The biomechanics of adiposity—structural and functional limitations of obesity and implications for movement," *Obesity Reviews*, vol. 3, no. 1, pp. 35–43, 2002.
- [3] S. C. Wearing, E. M. Hennig, N. M. Byrne, J. R. Steele, and A. P. Hills, "The biomechanics of restricted movement in adult obesity," *Obesity Reviews*, vol. 7, no. 1, pp. 13–24, 2006.
- [4] J. Nantel, M.-E. Mathieu, and F. Prince, "Physical activity and obesity: biomechancial and physiological key concepts," *Journal of Obesity*, vol. 2011, Article ID 650230, 10 pages, 2011.
- [5] R. H. Rozendal, "Biomechanics of standing and walking," in Disorders of Gait and Posture, W. Bles and T. Brandt, Eds., pp. 3–18, Elsevier Scientific, Amsterdam, The Netherlands, 1986.
- [6] T. J. Cole, M. C. Bellizzi, K. M. Flegal, and W. H. Dietz, "Establishing a standard definition for child overweight and obesity worldwide: international survey," *British Medical Journal*, vol. 320, no. 7244, pp. 1240–1243, 2000.
- [7] S. G. Trost, "Objective measurement of physical activity in youth: current issues, future directions," *Exercise and Sport Sciences Reviews*, vol. 29, no. 1, pp. 32–36, 2001.

[8] C. Tudor-Locke, R. P. Pangrazi, C. B. Corbin et al., "BMI-referenced standards for recommended pedometer-determined steps/day in children," *Preventive Medicine*, vol. 38, no. 6, pp. 857–864, 2004.

- [9] G. Cook, L. Burton, and B. Hoogenboom, "Pre-participation screening: the use of fundamental movements as an assessment of function—part 1," *North American Journal of Sports Physical Therapy*, vol. 1, pp. 62–72, 2006.
- [10] G. Cook, L. Burton, and B. Hoogenboom, "Pre-participation screening: the use of fundamental movements as an assessment of function—part 2," *North American Journal of Sports Physical Therapy*, vol. 1, pp. 132–139, 2006.
- [11] A. G. Schneiders, A. Davidsson, E. Horman, and J. S. Sullivan, "Functional Movement Screen Normative values in a young active population," *International Journal of Sports Physical Therapy*, vol. 6, pp. 75–82, 2011.
- [12] S. P. Messier, R. F. Loeser, M. N. Mitchell et al., "Exercise and weight loss in obese older adults with knee osteoarthritis: a preliminary study," *Journal of the American Geriatrics Society*, vol. 48, no. 9, pp. 1062–1072, 2000.
- [13] D. L. Riddiford-Harland, J. R. Steele, and L. H. Storlien, "Does obesity influence foot structure in prepubescent children?" *International Journal of Obesity*, vol. 24, no. 5, pp. 541–544, 2000.
- [14] A. M. Dowling, J. R. Steele, and L. A. Baur, "What are the effects of obesity in children on plantar pressure distributions?" *International Journal of Obesity*, vol. 28, no. 11, pp. 1514–1519, 2004.
- [15] E. Zoico, V. Di Francesco, J. M. Guralnik et al., "Physical disability and muscular strength in relation to obesity and different body composition indexes in a sample of healthy elderly women," *International Journal of Obesity*, vol. 28, no. 2, pp. 234–241, 2004.
- [16] S. Stenholm, D. Alley, S. Bandinelli et al., "The effect of obesity combined with low muscle strength on decline in mobility in older persons: results from the InCHIANTI Study," *International Journal of Obesity*, vol. 33, no. 6, pp. 635–644, 2009.
- [17] R. Burton, K. Elkins, K. B. Kiesel, and P. J. Plisky, "Gender differences in functional movement screen and Y-balance test scores in middle aged school children," *Medicine & Science in Sports & Exercise*, vol. 41, p. 183, 2009.

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Clinical Study

Independent Benefits of Meeting the 2008 Physical Activity Guidelines to Insulin Resistance in Obese Latino Children

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We examined the independent association between moderate-to-vigorous physical activity (MVPA) and insulin resistance (IR) among obese Latino children (N=113; 7–15 years) who were enrolled in a community-based obesity intervention. Baseline information on physical activity was gathered by self-report. Clinical assessments of body composition, resting energy expenditure (REE), as well as glucose and insulin responses to an oral glucose tolerance test (OGTT) were performed after an overnight fast. Insulin resistance was defined as a 2 h insulin concentration >57 μ U·mL⁻¹. We observed that those obese children who met the 2008 Guidelines for MVPA (\geq 60 min/day) experienced a significantly lower odds of IR compared with those not meeting the Guidelines (OR = 0.29; 95% CI: (0.10–0.92)) and these findings were independent of age, sex, pubertal stage, acculturation, fasting insulin, and 2 h glucose concentrations. Efforts to promote 60 min or more of daily MVPA among children from ethnic minority and high-risk communities should assume primary public health importance.

1. Introduction

Childhood obesity has reached epidemic proportions in the United States, with 32 percent of children and adolescents defined as either overweight or obese [1]. Among Latino American children and adolescents, the prevalence and complications of obesity are even higher compared with children of other racial and ethnic identity [1]. This issue has substantial public health relevance as Hispanics are the largest and fastest growing ethnic minority in the United States, and data suggest that 85% of overweight youth will remain overweight as adults [2]. The reason behind this observed racial/ethnic disparity in obesity is not clear. Potential explanations include: (1) biological differences in the susceptibility or resistance to weight gain; (2) differences in metabolic/mechanical efficiency; (3) sociocultural differences in lifestyle factors (such as dietary composition or participation in physical activities); or (4) environmental factors such as access to preventive or treatment programs [3–6].

Obesity is commonly accompanied by insulin resistance, which precedes and plays a major role in the etiology of the metabolic syndrome, type 2 diabetes mellitus and subsequent cardiovascular disease [7–11]. Among US adults and adolescents, Latinos have the highest prevalence of the metabolic syndrome [12], and more recent reports indicate an increasing incidence of type 2 diabetes among obese Latino American, African American, and Native American adolescents [13].

Sedentary behavior is associated with both the risk of obesity and of insulin resistance [14–16], and there are data to suggest that the prevalence of sedentary behaviors is higher among children from less-represented minority groups [3, 4, 17–19]. In an effort to reduce physical inactivity in children, the United States Public Health Service (USPHS) 2008 Physical Activity Guidelines for Americans recommends a minimum of 60 min·day⁻¹ of moderate-to-vigorous physical activity (MVPA: e.g., bicycle riding, skate boarding, soccer, jump roping). Unfortunately, the majority

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of children and adolescents do not meet these recommendations [20]. Furthermore, there are now data in adults confirming the distinct contributions of physical activity and sedentary behavior to the development of obesity and metabolic impairments [21]; whether this remains so among children, however, is not clear.

Few studies have examined the associations among physical activity, sedentary behavior, body composition, and metabolic markers in defined samples of Latino children. Moreover, determining the interrelatedness of these factors is complicated by the fact that obesity lies in the biologic pathway between MVPA or sedentary behavior and metabolic risk, thereby minimizing the true magnitude of contribution from these lifestyle behaviors. We studied cross-sectionally a sample of obese Latino children in order to determine the independent contributions of MVPA and sedentary behaviors on the odds of insulin resistance (IR) over and above that of obesity. We hypothesized that obese children reporting more MVPA will have a lower odds of IR compared with their less active counterparts; however, the magnitude of this association would be mediated by obesity and other risk factors in the biological pathway between obesity and insulin resistance (e.g., sedentary behavior, glucose, and fasting insulin concentrations).

2. Methods

2.1. Subjects. Participants were 113 Latino youth (58 boys and 55 girls), ages of 7-15 years enrolled in a comprehensive community-based obesity intervention program. Participants were recruited through advertisements placed at community facilities such as clinics, schools, and churches in Washington, DC, USA. To avoid selection biases from large families, as well as correlated reporting biases within families, only one child from each family was permitted to participate in the study. Eligibility requirements included Latino ethnicity (as determined by the parent's identifying themselves, their spouses, and both sets of grandparents within the Hispanic or Latino cultural group), a body mass index (BMI) \geq 95th percentile for age and sex, and otherwise good health. Children who were chronically ill and/or with known medical conditions such as type 2 diabetes, pervasive development delay, cerebral palsy, severe asthma, Cushing syndrome, obesity-associated genetic syndrome (e.g., Prader-Willi syndrome), or untreated hypothyroidism were excluded from study. All study procedures were approved by the Institutional Review Board of Children's National Medical Center (CNMC), and parents provided informed written consent, while the children provided signed assent prior to their participation. All data were collected at one baseline visit prior to participation in the comprehensive treatment program. On the day of testing, participants were admitted to the General Clinical Research Center (GCRC) at CNMC at 07:00 following a 12 hr overnight fast.

2.2. Anthropometry. Body weight was measured on a digital scale (Healthometer, Bridgeview, IL, USA) with the participant in underwear and a hospital gown, while height was measured using a wall-mounted stadiometer (SECA 216,

Hanover, MD, USA). The body mass index (BMI) was then calculated as weight (kg)·height (m)⁻². Waist circumference was measured in triplicate at the umbilicus using a nonelastic tape measure to the nearest 0.1 centimeter. Total body fat mass (kg; %) and fat-free mass (kg) were determined by air displacement plethysmography (Life Measurement Inc., Concord, CA, USA) according to methods previously described [22]. Pubertal stage was determined by a pediatrician separately for boys and girls, and children were classified as prepubertal (Tanner stage 1); mid prepubertal (Tanner stage 2); or late prepubertal (Tanner stage 3).

2.3. Indirect Calorimetry. Resting energy expenditure (REE) was measured using an open-circuit indirect computerized calorimeter equipped with a mask (Sensor Medics, Yorba Linda, CA, and Ultima CardiO2 system, Medical Graphics Corporation, St. Paul, MN, USA). All calibration procedures were performed to manufacturer specifications prior to testing each morning, including manual flow calibrations using a 3 L syringe and gas calibration with gases of known concentration. The REE measurement was performed in a quiet, thermoneutral room. All measurements were done after a 20-minute rest period. Participants were measured at rest in a supine position for a total of 30 minutes including a 5-minute acclimation period. Data collected during the initial five minutes of testing were excluded from the total 30-minute test period to allow for an adjustment period to the test conditions. Measurements were recorded at 1 minute intervals and then averaged over the remainder of the test period to obtain a measure of REE that was adjusted for body weight (kg). The respiratory exchange ratio (RER = vCO₂/vO₂) was also determined at 1-min intervals and averaged over the test period. The RER under fasting conditions provides an indirect measure of metabolic flexibility. Values of the RER closer to 0.70 indicate a greater reliance on fat (relative to glucose) oxidation, while values closer to 1.00 indicate greater glucose oxidation.

2.4. Oral Glucose Tolerance Test. A 2-hour oral glucose tolerance test was performed using the protocol described by the World Health Organization [23]. Fasting blood samples were collected from an antecubital vein for the determination of basal glucose, insulin, and free fatty acids (FFAs) concentrations. Participants were then given oral glucose drink (1.75 gm⋅kg⁻¹ (body weight) of glucose with a maximum of 75 gm) to consume within 2-3 min. At 120 min after challenge, the blood sampling was repeated. Blood samples were placed in prechilled tubes and then were centrifuged at 4°C. Plasma was stored at -70°C until analyzed in the Core Laboratory of CNMC. Serum insulin concentrations were determined by a solid-phase, 2-site chemiluminescent immunometric assay (Immulite 2000 Analyzer, Diagnostic Products, Los Angeles, CA, USA). Plasma glucose levels were determined using the hexokinase-glucose-6-phosphate dehydrogenase method (Dade Behring Inc, Deerfield, IL, USA). Plasma concentrations of FFF were determined using standard microflourimetric procedures (Sigma; St. Louis, MO, USA).

Insulin sensitivity was calculated using the whole-body insulin sensitivity index (WBISI = 10,000/\(\sqrt{}\) ((fasting glucose $(mg \cdot dL^{-1}) \times fasting insulin (\mu U \cdot mL^{-1})) \times (average$ glucose × average insulin))), which has been validated in children and adolescents [24]. The WBISI has demonstrated excellent correlation with clamp-derived insulin sensitivity estimates in children and adolescents and with intramyocellular lipid content, a tissue marker for insulin resistance [24]. We also considered the 2 h postchallenge insulin concentration as an indicator of insulin action. Postchallenge insulin concentrations correlated strongly with the WBISI (r =-0.81; P < 0.0001) and is easier to interpret compared with the WBISI. There currently exist no evidence-based cutpoints for defining insulin resistance in children. Therefore, in order to determine the odds of insulin resistance (IR) associated with physical activity in these obese children, those with a 2 hr postchallenge insulin concentration > $57 \,\mu\text{U}\cdot\text{mL}^{-1}$ were characterized as IR; otherwise they were considered not insulin resistant. This postchallenge insulin concentration corresponds to a Homeostatic Model Assessment (HOMA-IR = (fasting glucose \times 0.056) \times (fasting insulin/22.1)) [25] value of >2.5, which is a clinical cut-point often used to characterize insulin resistance in children [26].

2.5. Physical Activity and Sedentary Behavior. Children completed the Activity and Sedentary Behavior Questionnaire (ASBQ), which comprised questions from the validated 2001 Youth Risk Behavior Survey and the World Health Organization Study of Health Behavior of School Children Survey [5]. The ASBQ queried both the frequency and duration of moderate-to-vigorous physical activity acquired through organized sport, informal recreational pursuits, and physical education class. In addition, the ASBQ contained questions about time per day watching television and at the computer. All questions contained categorical responses, which were scored on an ordinal scale. Two indices were then calculated by multiplying the frequency score by the duration score for these activities: (1) moderate-vigorous physical activity (MVPA) Index, with scores that ranged from 0 to 150; (2) sedentary index (SI)—which was derived from questions pertaining to TV viewing and computer time with scores ranging from 0 to 150. Physical education (PE) volume was derived by multiplying reported frequency by duration of weekly PE class resulting in scores ranging from 0 to 300 min·wk $^{-1}$. To address the aims of the 2008 Physical Activity Guidelines, the MVPA index was then dichotomized based on a volume of MVPA that did or did not meet those Guidelines for moderate-to-vigorous physical activity (every day for 60 min or more).

2.6. Acculturation. Level of acculturation is an important confounder of the associations among physical activity, obesity and diabetes risk [27]. To assess this important study variable, mothers answered questions (administered in Spanish by a fluently bilingual trained research assistant) derived from the Bicultural Involvement Questionnaire [28]. Acculturation was measured via 17 items assessing constructs of linguistic fluency and comfort level in Spanish and in English; language use at home, and with work and friends;

preferences in music, books, magazines and visual media. Each of the items was scored on a scale with a maximum score of 4 for 14 items, of 3 for two items, and of 5 for one item. Individual scores were then summed over all items to create a total score. Total scale scores ranged from 17 to 67, with higher scores reflecting greater acculturation to American culture. In the current study, the acculturation scale demonstrated adequate internal consistency ($\alpha = .77$).

2.7. Statistical Analyses. Descriptive statistics (mean \pm SD; frequencies (%)) were generated on all study variables according to those who were and who were not IR. Betweengroup differences in mean levels of the study variables were tested using t-tests for independent samples, while the chisquare statistic tested associations between categorical variable frequencies and IR status. Study variables demonstrating a statistically significant association with IR status in the bivariable analysis were then entered into a multivariable logistic regression model in order to determine their independent contribution to the odds of insulin resistance. The variables age, sex, pubertal status, and acculturation were forced into this model. The regression model was then run backward, with those variables demonstrating an alpha level of 0.10 or greater eliminated from the model.

Parameter estimates, as well as adjusted odds ratios (ORs) and 95% confidence intervals (CI) are reported for the final regression model. Because our primary study variables (MVPA and sedentary behavior) were ordinal indices, the OR represents the increase (or decrease) in the odds of IR for each *unit* increase of the given index. The odds of IR among children meeting the 2008 MVPA Guidelines were compared with the odds of children who did not (the referent group).

3. Results

Study characteristics according to IR status are presented in Table 1. As indicated, subjects with and without IR were similar with regard to age, BMI, fat mass, and percent body fat and Tanner score. Girls tended to have a slightly higher prevalence of IR (0.56) compared with boys (0.44) (P = 0.13), and those with IR also tended to have a higher Z-score for waist circumference (0.33). Fasting RER was comparable between the groups; however, it is worth noting that it was high in both groups, suggesting some degree of metabolic inflexibility among these obese children. Weight-adjusted REE was significantly lower (P < 0.01) and acculturation score was significantly higher (P < 0.05) in those children with IR, compared with those without IR.

Fasting glucose and FFA concentrations were no different between study groups; however, fasting insulin and 2 h glucose levels were significantly greater (P < 0.01) in the children with IR compared with those without it (Table 2). As expected, insulin sensitivity, based on the WBISI, was markedly lower (P < 0.001) and 2 h insulin concentrations substantially higher (P < 0.001) in those children who were defined as IR compared with those who were not. Of note is the observation that both groups of children were able to maintain normal glucose tolerance based on a fasting glucose concentration criterion of $<100\,\mathrm{mg}\cdot\mathrm{dL}^{-1}$.

TABLE 1: Subject characteristics.

	Insulin sensitive $(n = 53)$	Insulin resistant* $(n = 61)$		
Age (y)	11 ± 2	12 ± 2		
Sex (% male)	58	44		
BMI (kg/m ²)	29.3 ± 5.45	31.0 ± 4.9		
Fat mass (kg)	30.0 ± 11.6	32.2 ± 10.0		
Body fat (%)	42.2 ± 5.7	43.3 ± 5.5		
Waist circ. (Z score)	1.46 ± 0.65	1.58 ± 0.64		
Tanner score	2	2		
REE (kcal/kg)	35.7 ± 16.4	29.2 ± 11.7^{a}		
Fasting RER	0.86 ± 0.07	0.88 ± 0.07		
Acculturation score	32 ± 7	$35 \pm 7^{\mathrm{b}}$		

^{*} Based on a 2 h postchallenge insulin concentration greater than $64 \,\mu\text{U/mL}$ (median value); $^aP < 0.01$; $^bP < 0.05$. BMI: body mass index; body fat determined by DXA; A Tanner score of 2: mid prepubertal; REE: resting energy expenditure; RER: respiratory exchange ratio.

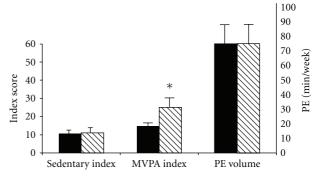
TABLE 2: Metabolic response characteristics to the OGTT.

	Insulin sensitive $(n = 53)$	Insulin resistant $(n = 61)$
Fasting glucose (mg·dL ⁻¹)	83.5 ± 5.8	85.7 ± 12.1
2 h glucose (mg \cdot dL ⁻¹)	102.1 ± 19.7	$118.4 \pm 30.2^*$
Fasting insulin $(\mu U \cdot mL^{-1})$	12.0 ± 8.9	$21.5 \pm 13.5**$
Fasting FFA (μ mol·L ⁻¹)	0.72 ± 0.28	0.67 ± 0.28
WBISI	10.6 ± 8.2	$3.7 \pm 2.2**$
2 h insulin ($\mu U \cdot mL^{-1}$)	29.8 ± 16.1	$133.4 \pm 83.8**$

OGTT: oral glucose tolerance test; *P < 0.01; **P < 0.001; WBISI: whole-body insulin sensitivity index.

Reported sedentary behavior was similar between groups (SI = 10.5 ± 6.2 versus 10.9 ± 6.5 for those with and without IR, resp.; P = 0.75; Figure 1). In contrast, the reported MVPA index was significantly lower among those characterized as insulin resistant compared with who were not (MVPA index = 14.8 ± 18.4 versus 24.9 ± 32.1 , resp.; P < 0.05). Participation in physical education tended to be lower among those children with IR compared with those without it (PE volume = $73.7 \pm 74.3 \text{ versus } 80.0 \pm 79.6 \text{ min} \cdot \text{wk}^{-1}$, resp.; P = 0.06). Only 18% of the participants met the USPHA guidelines for 60 min or more of MVPA on most days of the week. Those who met the guidelines had an MVPA index score of 58.6 \pm 31.7, compared with a score of 8.3 \pm 6.9 among those who did not (P < 0.001). We observed a strong unadjusted association between meeting those guidelines and protection from insulin resistance (OR = 0.28; P <0.01).

Study variables that were statistically significant in the bivariable analysis were then entered in a stepwise multivariable logistic regression model to determine their independent contribution to the odds of IR. These variables were MVPA, as well as fasting insulin, 2 h glucose concentrations, REE, and acculturation score. The variables age, sex, pubertal status, and waist circumference were also included due to their reported association with IR in the literature. The



*P < 0.05, based on t-test for independent samples

FIGURE 1: Mean (±SEM) values for the sedentary and MVPA indices and PE volume between obese children with (solid bars) and without (striped bars) insulin resistance.

Table 3: Parameter estimates* from the final logistic regression modeling (N = 113).

	Estimate	P value	OR	95% CI
MVPA	-1.247	0.035	0.29	(0.10-0.92)
Fasting insulin $(\mu U \cdot mL^{-1})$	0.086	0.001	1.09	(1.04-1.15)
2 hr glucose (mg⋅dL ⁻¹)	0.037	0.004	1.04	(1.01-1.06)

^{*} Estimates represent the decrease in the log odds of IR in those children meeting the 2008 Guidelines for MVPA (≥60 min/day) compared with those who did not, and the increase in the log odds of IR with each unit increase in fasting insulin or in 2 hr glucose concentrations. Model was adjusted for age, sex, pubertal stage, and acculturation.

MVPA index maintained marginal statistical significance in the multivariable model. Each unit increases in the MVPA index lowered the odds of IR by 3% (OR = 0.97; 95% (CI: (0.95-1.00)). We then repeated the analysis and substituted the dichotomous MVPA variable for the MVPA index. Parameter estimates from the final logistic regression model are presented in Table 3. In the presence of two very powerful correlates of insulin resistance: 2 hr postchallenge glucose (P < 0.01) and fasting insulin (P < 0.001) concentrations, MVPA maintained a significant association with IR as those obese children who reported enough MVPA to meet the 2008 Guidelines experienced a substantially lower odds of IR, compared with the children who did not ((OR = 0.29)) 95% CI: (0.10-0.92))). Age, sex, puberty status, REE, and acculturation score did not remain statistically significant in the multivariable model.

4. Discussion

Since obesity lies along the biological pathway between MVPA and insulin resistance, it would seem as though the contribution of MVPA to IR would be diminished in the presence of obesity. Similarly, the association among the weight-adjusted REE, the RER, the waist circumference, and IR appeared blunted in these obese children. That we observed MVPA to be associated with the odds of IR (independent of fasting insulin and 2 h postchallenge glucose levels) underscores the distinct relation of muscle contraction to insulin action (via improvements in mitochondrial

function, insulin signaling, and glucose transport) from that of obesity, as well as the continued importance of MVPA in mitigating the risk of metabolic impairments with obesity.

Other research has described the benefits of vigorous activity or MVPA to IR among diverse samples of children, and these reported benefits were also independent of total or central adiposity [29-31]. Importantly, these studies relied on both self-report and objective measures of physical activity. Pubertal status is often implicated as an effect modifier in the pathway toward pediatric diabetes. Previous studies consistently report that puberty is associated with about a 25-30% reduction in insulin sensitivity, with peak reduction occurring at Tanner stage 3 [32]; whether this functional impairment operates independent of accelerated fat deposition during puberty is not clear [7]. An ample body of evidence also supports the complicated relation that acculturation has with physical activity, obesity, and diabetes risk [27]. In general, a high level of acculturation is associated with greater amounts of reported physical activity and exercise; however, acculturation also increases the risk of obesity and diabetes risk, presumably through dietary changes. We did not observe an association between acculturation and MVPA, but those with IR had a significantly higher acculturation score compared with those without IR. This later association did not maintain statistical significance in the presence of fasting insulin and 2 hr glucose concentrations, however.

Adolescents watching greater than two hours of television per day engage in significantly less vigorous physical activity than those who spend less time in front of the screen [33]; however, very few studies report a strong correlation between television viewing and total physical activity, indicating that time engaging in one behavior does not necessarily displace time in the other. Moreover, the small positive correlation often reported between television viewing per se and obesity in children has been attributed to higher caloric intake with television viewing, rather than to lower energy expenditure [33, 34]. We observed that reported sedentary behavior (TV viewing and computer use time) was similar between those with and without IR in our obese pediatric sample, and similar to other studies [33-35], our measure of sedentary behavior demonstrated no association with the MVPA index (r = -0.02; P = 0.83), to BMI (r = 0.13; P = 0.16), or to body fat% (r = 0.17; P = 0.11). This assumes that sedentary behavior is not sufficiently biologically relevant to IR in the face of pediatric obesity, or that sedentary behavior was not measured accurately. Studies that used objective measures of sedentary time in children [30, 35, 36] report associations with metabolic outcomes that are distinct from those using measures of MVPA—thereby supporting the notion that sedentary time and MVPA are two separate behavioral and physiologic constructs.

Among the strengths of this study was the reliance on several clinical measures of metabolic control under both fasting and postprandial conditions. In addition, we believe the 2-h insulin response to an actual physiologic challenge (the OGTT) to be a more valid indicator of insulin resistance than a simple fasting value or the HOMA. We note, however, the limitations inherent to a cross-sectional

analysis in that the temporal sequencing between physical activity, attained obesity, and insulin resistance could not be established. Indeed, there is now evidence that children with the greatest impairments in insulin sensitivity have the greatest susceptibility to weight gain over time [16, 37, 38] and the greatest resistance to weight loss interventions [39, 40]. Second, we relied on a valid and widely used selfreported measure of physical activity and sedentary behavior; yet the information bias associated with any self-reported measure is well known. We assume, however, that the tendency to over- or underreport MVPA was nondifferential between those with and without IR, since participants were not aware of their 2 hr postchallenge insulin values when answering the physical activity questions. Therefore, we are confident that no systematic reporting bias occurred which would have falsely inflated the independent contribution of MVPA to IR risk.

These findings support the notion that even in the presence of frank pediatric obesity, moderate-to-vigorous physical activity conveys important benefits to insulin sensitivity—although further longitudinal evidence is necessary to infer a causal relation with confidence. Nonetheless, home-, school-, and community-based programs for promoting MVPA through physical education, sports, and/or culturally appropriate physical activities should be of primary public health importance in order to protect the health of children at risk for obesity and insulin resistance.

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References

- [1] C. L. Ogden, M. D. Carroll, L. R. Curtin, M. M. Lamb, and K. M. Flegal, "Prevalence of high body mass index in US children and adolescents, 2007-2008," *JAMA*, vol. 303, no. 3, pp. 242–249, 2010.
- [2] W. H. Dietz and S. L. Gortmaker, "Preventing obesity in children and adolescents," *Annual Review of Public Health*, vol. 22, pp. 337–353, 2001.
- [3] A. M. Fontvieille, J. Dwyer, and E. Ravussin, "Resting metabolic rate and body composition of Pima Indian and Caucasian children," *International Journal of Obesity*, vol. 16, no. 8, pp. 535–542, 1992.
- [4] M. S. Treuth, N. F. Butte, and W. W. Wong, "Effects of familial predisposition to obesity on energy expenditure in multiethnic prepubertal girls," *American Journal of Clinical Nutrition*, vol. 71, no. 4, pp. 893–900, 2000.
- [5] World Health Organization, "Health behavior in school-aged children: a WHO cross- national survey (HBSC). Research protocol for the 1997-98 study," in *Research Unit in Health and*

Behavioural Change, pp. 1–101, Department of Community Health Sciences. University of Edinburgh Medical School, Edinburgh, UK, 1998.

- [6] S. Z. Yanovski, "Resting metabolic rate in African-American and caucasian girls," *Obesity Research*, vol. 5, no. 4, pp. 321– 325, 1997.
- [7] M. I. Goran, G. D. C. Ball, and M. L. Cruz, "Cardiovascular endocrinology 2: obesity and risk of type 2 diabetes and cardiovascular disease in children and adolescents," *Journal* of Clinical Endocrinology and Metabolism, vol. 88, no. 4, pp. 1417–1427, 2003.
- [8] M. I. Goran, C. Lane, C. Toledo-Corral, and M. J. Weigensberg, "Persistence of pre-diabetes in overweight and obese hispanic children; Association with progressive insulin resistance, Poor β-cell function, and increasing visceral fat," *Diabetes*, vol. 57, no. 11, pp. 3007–3012, 2008.
- [9] J. A. Morrison, L. A. Friedman, P. Wang, and C. J. Glueck, "Metabolic syndrome in childhood predicts adult metabolic syndrome and type 2 diabetes mellitus 25 to 30 years later," *Journal of Pediatrics*, vol. 152, no. 2, pp. 201–206, 2008.
- [10] M. S. Treuth, N. F. Butte, K. J. Ellis, L. J. Martin, and A. G. Comuzzie, "Familial resemblance of body composition in prepubertal girls and their biological parents," *American Journal of Clinical Nutrition*, vol. 74, no. 4, pp. 529–533, 2001.
- [11] E. E. Ventura, C. J. Lane, M. J. Weigensberg, C. M. Toledo-Corral, J. N. Davis, and M. I. Goran, "Persistence of the metabolic syndrome over 3 annual visits in overweight hispanic children: association with progressive risk for type 2 diabetes," *Journal of Pediatrics*, vol. 155, no. 4, pp. 535–e1, 2009.
- [12] S. Cook, M. Weitzman, P. Auinger, M. Nguyen, and W. H. Dietz, "Prevalence of a metabolic syndrome phenotype in adolescents: findings from the Third National Health and Nutrition Examination Survey, 1988–1994," Archives of Pediatrics and Adolescent Medicine, vol. 157, no. 8, pp. 821–827, 2003.
- [13] American Diabetes Association, "Type 2 diabetes in children and adolescents," *Pediatrics*, vol. 105, pp. 671–680, 2000.
- [14] C. S. Berkey, H. R. H. Rockett, M. W. Gillman, and G. A. Colditz, "One-year changes in activity and in inactivity among 10- to 15-year-old boys and girls: relationship to change in body mass index," *Pediatrics*, vol. 111, no. 4, pp. 836–843, 2003.
- [15] J. M. McGavock, T. J. Anderson, and R. Z. Lewanczuk, "Sedentary lifestyle and antecedents of cardiovascular disease in young adults," *American Journal of Hypertension*, vol. 19, no. 7, pp. 701–707, 2006.
- [16] T. N. Robinson, "Reducing children's television viewing to prevent obesity: a randomized controlled trial," *JAMA*, vol. 282, no. 16, pp. 1561–1567, 1999.
- [17] N. F. Butte, G. Cai, S. A. Cole et al., "Metabolic and behavioral predictors of weight gain in Hispanic children: the Viva la Familia Study," *American Journal of Clinical Nutrition*, vol. 85, no. 6, pp. 1478–1485, 2007.
- [18] L. R. Dugas, K. Ebersole, D. Schoeller et al., "Very low levels of energy expenditure among pre-adolescent Mexican-American girls," *International Journal of Pediatric Obesity*, vol. 3, no. 2, pp. 123–126, 2008.
- [19] P. Gordon-Larsen, L. S. Adair, M. C. Nelson, and B. M. Popkin, "Five-year obesity incidence in the transition period between adolescence and adulthood: the National Longitudinal Study of Adolescent Health," *American Journal of Clinical Nutrition*, vol. 80, no. 3, pp. 569–575, 2004.

[20] Youth Risk Behavior Survey. National Center for Chronic Diseases Prevention and Health Promotion. Adolescent and School Health, 2001, http://www.cdc.gov/nccdphp/dash/yrbs.

- [21] G. N. Healy, D. W. Dunstan, J. Salmon et al., "Breaks in sedentary time: beneficial associations with metabolic risk," *Diabetes Care*, vol. 31, no. 4, pp. 661–666, 2008.
- [22] J. C. Nicholson, J. R. McDuffie, S. H. Bonat et al., "Estimation of body fatness by air displacement plethysmography in African American and white children," *Pediatric Research*, vol. 50, no. 4, pp. 467–473, 2001.
- [23] World Health Organization, *Definition, Diagnosis and Classification of Diabetes Mellitus and Its complications*, Department of Non-communicable Disease Surveillance. World Health Organization, Geneva, Switzerland, 1999.
- [24] C. W. Yeckel, R. Weiss, J. Dziura et al., "Validation of insulin sensitivity indices from oral glucose tolerance test parameters in obese children and adolescents," *Journal of Clinical Endocrinology and Metabolism*, vol. 89, no. 3, pp. 1096–1101, 2004.
- [25] J. C. Levy, D. R. Matthews, and M. P. Hermans, "Correct homeostasis model assessment (HOMA) evaluation uses the computer program," *Diabetes Care*, vol. 21, no. 12, pp. 2191– 2192, 1998.
- [26] R. C. Q. Da Silva, W. L. Miranda, A. R. Chacra, and S. A. Dib, "Metabolic syndrome and insulin resistance in normal glucose tolerant Brazilian adolescents with family history of type 2 diabetes," *Diabetes Care*, vol. 28, no. 3, pp. 716–718, 2005.
- [27] R. Pérez-Escamilla and P. Putnik, "The role of acculturation in nutrition, lifestyle, and incidence of type 2 diabetes among Latinos," *Journal of Nutrition*, vol. 137, no. 4, pp. 860–870, 2007.
- [28] J. Szapocznik, W. M. Kurtines, and T. Fernandez, "Bicultural involvement and adjustment in Hispanic-American youths," *International Journal of Intercultural Relations*, vol. 4, no. 3-4, pp. 353–365, 1980.
- [29] C. Y. Ku, B. A. Gower, G. R. Hunter, and M. I. Goran, "Racial differences in insulin secretion and sensitivity in prepubertal children: role of physical fitness and physical activity," *Obesity Research*, vol. 8, no. 7, pp. 506–515, 2000.
- [30] L. B. Saudinha, L. B. O. Andersen, S. A. Anderssen et al., "Objectively measured time spent sedentary is associated with insulin resistance independent of overall and central body fat in 9- To 10-year-old Portuguese children," *Diabetes Care*, vol. 31, no. 3, pp. 569–575, 2008.
- [31] C. Platat, A. Wagner, T. Klumpp, B. Schweitzer, and C. Simon, "Relationships of physical activity with metabolic syndrome features and low-grade inflammation in adolescents," *Diabetologia*, vol. 49, no. 9, pp. 2078–2085, 2006.
- [32] A. Moran, D. R. Jacobs, J. Steinberger et al., "Insulin resistance during puberty: results from clamp studies in 357 children," *Diabetes*, vol. 48, no. 10, pp. 2039–2044, 1999.
- [33] S. J. Marshall, S. J. H. Biddle, T. Gorely, N. Cameron, and I. Murdey, "Relationships between media use, body fatness and physical activity in children and youth: a meta-analysis," *International Journal of Obesity*, vol. 28, no. 10, pp. 1238–1246, 2004
- [34] U. Ekelund, S. Brage, K. Froberg et al., "TV viewing and physical activity are independently associated with metabolic risk in children: the European youth heart study," *PLoS Medicine*, vol. 3, no. 12, article e488, 2006.
- [35] H. J. F. Helmerhorst, K. Wijndaele, S. Brage, N. J. Wareham, and U. Ekelund, "Objectively measured sedentary time may predict insulin resistance independent of moderate- and

- vigorous-intensity physical activity," *Diabetes*, vol. 58, no. 8, pp. 1776–1779, 2009.
- [36] K. F. Janz, T. L. Burns, and S. M. Levy, "Tracking of activity and sedentary behaviors in childhood: the Iowa bone development study," *American Journal of Preventive Medicine*, vol. 29, no. 3, pp. 171–178, 2005.
- [37] M. S. Johnson, R. Figueroa-Colon, T. T. K. Huang, J. H. Dwyer, and M. I. Goran, "Longitudinal changes in body fat in African American and Caucasian children: influence of fasting insulin and insulin sensitivity," *Journal of Clinical Endocrinology and Metabolism*, vol. 86, no. 7, pp. 3182–3187, 2001.
- [38] O. E. Odeleye, M. De Courten, D. J. Pettitt, and E. Ravussin, "Fasting hyperinsulinemia is a predictor of increased body weight gain and obesity in Pima Indian children," *Diabetes*, vol. 46, no. 8, pp. 1341–1345, 1997.
- [39] O. Pinhas-Hamiel, L. Lerner-Geva, N. Copperman, and M. S. Jacobson, "Insulin resistance and parental obesity as predictors to response to therapeutic life style change in obese children and adolescents 10–18 years old," *Journal of Adolescent Health*, vol. 43, no. 5, pp. 437–443, 2008.
- [40] T. Reinehr, W. Kiess, T. Kapellen, and W. Andler, "Insulin sensitivity among obese children and adolescents, according to degree of weight loss," *Pediatrics*, vol. 114, no. 6, pp. 1569– 1573, 2004.