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Research Article

Risk Assessment of Lung Cancer Caused by Indoor Radon Exposure in China during 2006–2016: A Multicity, Longitudinal Analysis

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Indoor radon is a well-documented environmental factor as a second cause of lung cancer. Based on the chronological data on indoor radon concentration, lung cancer incidence, and the distribution of sex-age-specific population, the risk of lung cancer caused by indoor radon exposure in a total of 15 cities in China was assessed by using the risk model developed by the U.S. Environmental Protection Agency (EPA) in this study. The estimate revealed that both the excess relative risk (ERR) and lifetime relative risk (LRR) have obviously increased after 2010. The population attributable risk (PAR) in 2016 was estimated in a range from 6.66% to 22.42%, with a median of 15.33% for the 15 cities. The lung cancer incidence attributed to indoor radon in 2016 ranged from 3.96 to 15.07 per 10,000 population in males and 1.21 to 8.27 per 10,000 population in females. Across age and sex, the risk of lung cancer caused by indoor radon was found more pronounced in males and 40-45 age groups. The chronological variation of radon concentrations was considered in this study; the estimate of lung cancer caused by indoor radon in China is considered more reasonable than ever before.

1. Introduction

Radon (^{222}Rn) is a naturally occurring radioactive gas that originates from uranium in rocks and soils. Radon can seep out of the ground or building materials, diffuse into buildings, and accumulate to relatively high levels in dwellings and buildings with poor ventilation [1, 2]. According to the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), the inhalation of radon and its progenies contributes to approximately 52% of the average radiation dose received by adults from natural sources [3, 4]. By emission of alpha particles, radon can decay into a series of radioactive progenies that can be inhaled into the lungs and deposited in the pulmonary epithelial lining. The alpha radiation emitted from deposited radon progenies can disrupt the DNA of lung cells and potentially initiate lung cancer [5, 6].

The relationship between radon and lung cancer can be dated back several hundred years. Miners in Eastern Europe were observed with a high lung cancer mortality rate as early as the fifteenth century, which could be attributed to radon exposure. In early research, evidence of radon-induced lung cancer mainly came from cohort studies on uranium and other underground miners [7–9]. Wide surveys of underground miners from different countries provided quantitative information on the exposure–response relationship between lung cancer risk and cumulative radon exposure [10–13]. Based on the results of experimental animal studies and epidemiologic studies, the International Agency for Research on Cancer (IARC) classified radon as a group I

human carcinogen [14]. In 2009, the World Health Organization (WHO) recognized radon as the second largest risk factor for lung cancer after smoking [15]. Therefore, studies on the health effects of radon are warranted due to the increasing concern of radon exposure.

Although radon concentrations in most buildings are much lower than those in mines, the potential risk of lung cancer caused by indoor radon has aroused widespread concern in the general population. To estimate the risk of residential radon to the public, several research groups had assessed the lung cancer risk from residential radon in many countries before 2000 [16–20]. In recent years, studies in North America indicated that the risk of lung cancer could increase by 11% with an increase of 100 Bq/m^3 in radon concentration, whereas European studies provided similar results of risk increase by 16% [21–24]. Pooled results of two case-control studies in Gansu and Shenyang in China also showed that the lung cancer risk increased significantly with higher radon concentration, and the odds ratio (OR) with 95% confidence intervals (CIs) at 100 Bq/m^3 was 1.33 (1.01, 1.36) [25]. All pooled results were consistent with extrapolations from miner data, suggesting that long-term radon exposure at concentrations found in many homes increases the risk of lung cancer for both smokers and nonsmokers, and the risk is linear with exposure without a threshold.

Compared with those residential radon studies, model-based estimation is more suitable for wide area assessment and prediction of the health impact caused by radon exposure. In recent decades, several risk models have been developed for the risk assessment of indoor radon. Most of the models are based on extrapolation of existing epidemiologic miner models [26–29]. Previous model-based estimations suggested that radon is responsible for 7% of lung cancers in Germany, 4% in the Netherlands, 20% in Sweden, 11% in Norway, and 10%–15% in the United States [30–33]. A recent study showed that the median population attributable risk (PAR) of lung cancer mortality from residential radon in 66 countries is approximately 13.6%–16.5% [34]. However, the variations in chronological radon exposure over time and its impact on risk estimation have not been fully discussed in most of the previous studies.

Our previous study established a database on indoor radon concentrations in China from 1980 to 2019 [35]. The results confirmed an obvious increasing trend in residential radon concentration in China during the past four decades. Although most countries reported declining trends of indoor radon concentration, China is one of the minority countries where radon concentration increased.

Considering the average concentration of indoor radon in the 2010s had approximately doubled from that in the 1980s, it was reasonable that the estimation of health risks should be based on variable historical data rather than the average in a specific time, especially for populations at different ages. Furthermore, our previous studies also indicated that the indoor radon concentration and its increasing tendency varied among cities in China. Thus, intercity differences in risk estimation should not be ignored. However, studies on risk assessment that focused on the historical variation and intercity differences of indoor radon were insufficient.

In this study, we systematically analyzed chronological data on indoor radon and lung cancer and assessed the risk of lung cancer caused by indoor radon exposure in 15 cities in China based on existing models. By carefully discussing the temporal variation of the health risk, the results of this study could be beneficial for the estimate of the health and economic burden due to indoor radon exposure and help provide valuable information for the formulation of related national regulations.

2. Materials and Methods

2.1. Data Collection. In this study, the dataset consisted of indoor radon concentration, lung cancer incidence (ICD-10, C33-C34), sex-age-specific population data, and smoking prevalence. Historical data on indoor radon concentration were obtained from the database established in our previous study, which originally collected 129 surveys in 147 cities from 1980 to 2019 in China [35]. Data on the incidence of lung cancer from 2006 to 2016 were obtained from the National Central Cancer Registry (NCCR) [36–46]. The demographic structure was based on the Seventh National Census in 2020 and assumed no demographic changes with time [47]. The smoking prevalence in adults was cited from the Chinese Center for Disease Control and Prevention (CDC) [48]. Due to lack of detailed local smoking prevalence for different cities, the national average smoking prevalence was applied for risk assessment in this study.

Finally, a chronological database of indoor radon and lung cancer among different cities were constructed, covering every piece of information that included administrative divisions, date of investigation, mean value, sample number, sex-age-specific population, sex-age-specific incidence (mortality) rates, incidence (mortality) rates, and age-standardized rates.

2.2. Risk Models. In this study, we used the U.S. Environmental Protection Agency (EPA) risk assessment model, which was updated in 2003 [29]. The model was primarily based on the results of 11 cohort studies of radon-exposed miners in China, Canada, Europe, the USA, and Australia by the Biological Effects of Ionizing Radiation Committee (BEIR VI) [13]. Compared to the original version by BEIR VI, the EPA model had modified and extended its approach, in which the age-specific smoking prevalence was taken into consideration. Spline smoothing was used to estimate the excess relative risk (ERR) at ages of 55, 65, and 75 to avoid biologically implausible discontinuities in the curve. However, it would have potential uncertainty in this model when the extrapolating is taken in risk assessment from high radon exposure for miners to relatively low radon exposure for residents.

The EPA model is expressed as

$$\mathrm{ERR}(a) = \delta\beta(W_{5-14} + \theta_{15-24}W_{15-24} + \theta_{25+}W_{25+})\varphi_{\mathrm{age}}\gamma_z, \quad (1)$$

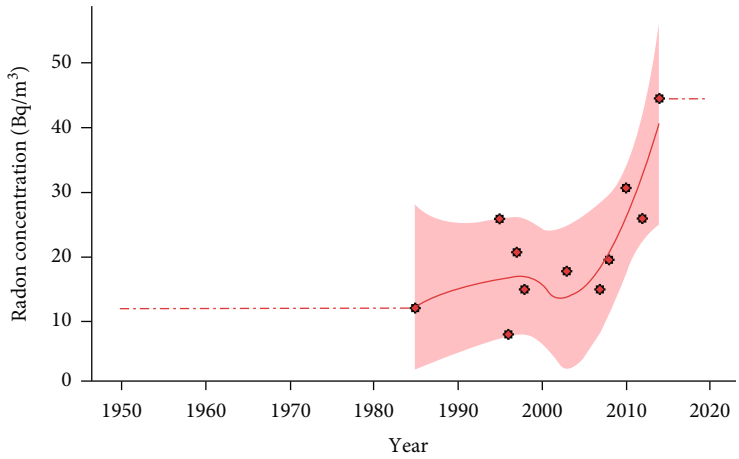

Figure 1: Indoor radon concentrations from different surveys in Shanghai. Lines with ribbons represent median estimates with 95% credible intervals.

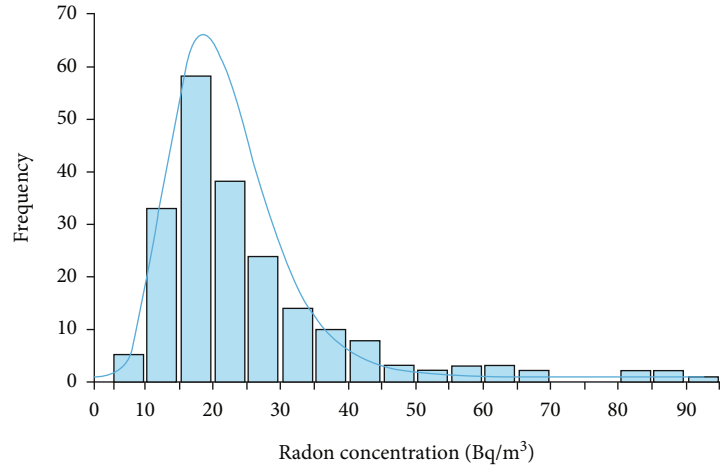

Figure 2: Logarithmic normal distribution of radon concentration from a survey in 2008.

where δ is the smoking modification factor and set to be 2 or 0.9 for never-smokers or ever-smokers, respectively; β is the slope of the exposure–risk relationship ($\beta = 0.0634$); W_{5-14}, W_{15-24}, and W_{25+} represent cumulative radon exposures expressed in working level per month (WLM) received during time windows of 5–14, 15–24, and 25+ years or more prior to the attained age, respectively; θ_{15-24} and θ_{25+} are modification factors of W_{15-24} and W_{25+}, respectively; φ_{age} is the attained age-related modification factor; and γ_z represents the effect of exposure rate in the EAC model or exposure time in the EAD model.

2.3. Data Analysis. In this study, the data were analyzed in two-stage protocol. In the first stage, we focused on the local estimation of lung cancer risk in Shanghai, as the city had detailed historical data, including 11 surveys on indoor radon over the past four decades and lung cancer data for over 10 consecutive years. After interpolating and smoothing, the processed data of indoor radon concentrations were applied to evaluate the cumulative radon exposure for residents of different ages in each specific year from 2000 to 2021. The cumulative radon exposure was further converted into the unit of working level per month (WLM) in the risk assessment, assuming an equilibrium factor of 0.4 and a residence time of 7,000 h at home per year.

The EPA model was applied to assess lifetime radon exposure and ERR for each sex-age category, assuming a 5-year latency period. To estimate the ERR and its 95% CIs, we performed Monte Carlo simulation by sampling 10,000 replications of the concentration distributions for indoor radon and age distribution for the population (shown in Figure S1). The detailed instructions about the Monte Carlo simulation was provided in Table S1 and as supplementary materials. Figure S2 shows an example of the simulation results.

In contrast to most previous studies, indoor radon exposure data changing with year were used to estimate the ERR, and a time series correlation analysis between the estimated ERR and lung cancer age-standardized incidence rate (ASIR) was also performed in this study.

In the second stage, we selected 15 cities in China that had at least five years of available lung cancer data and at

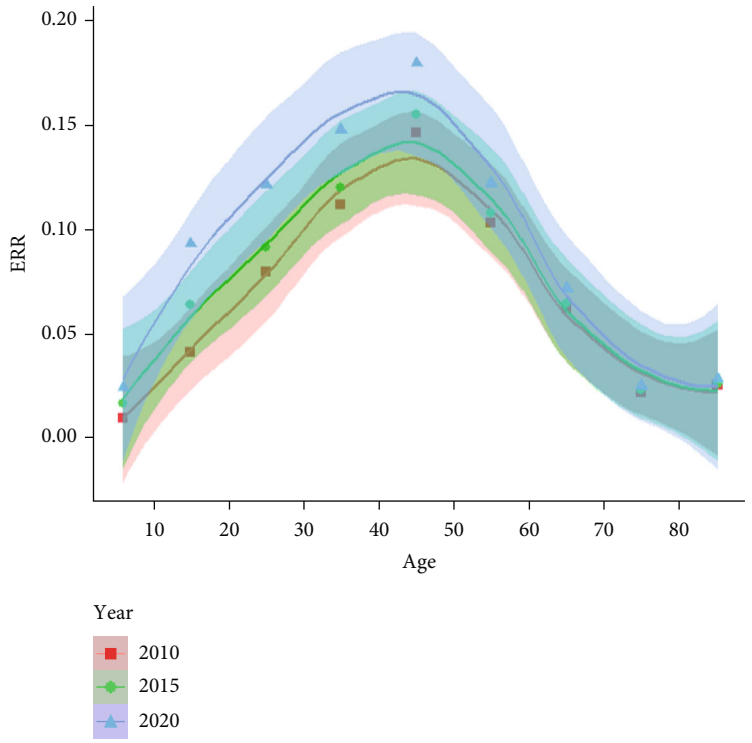

Figure 3: Comparison of estimated ERR for Shanghai in 2010, 2015, and 2020.

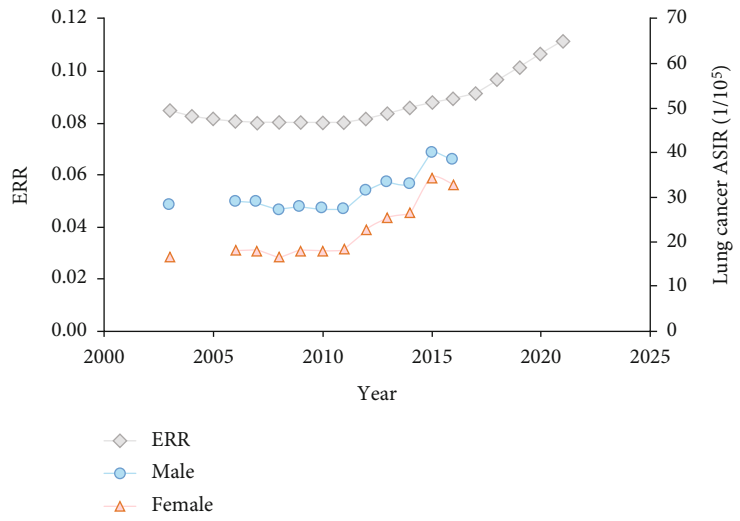

Figure 4: Age-standardized ERR and sex-specific lung cancer ASIR in 2003–2021 in Shanghai.

least five radon surveys. For these cities, the locally weighted regression (LOESS) was used to make up for missing data on radon concentrations from 2000 to 2021. The age-standardized ERR of each city was estimated using the same model and method used for the estimation in Shanghai. The PAR is calculated as

$$\mathrm{PAR}\% = \frac{p(\mathrm{ERR})}{p(\mathrm{ERR}+1)+1} \times 100\%, \quad (2)$$

where p indicates the proportion of radon exposure in the total population, and it is set to be 1 in this study.

Then, the PAR was multiplied by the number of lung cancer ASIR for each sex-age category to calculate the number of radon-attributable lung cancer incidence.

Due to the limited available data, the variations of the ERR and lung cancer ASIR by year were roughly linearly fitted to obtain the increasing or decreasing rate. It should be noted that these rates were not an accurate quantitative estimation but merely a rough result reflecting a general

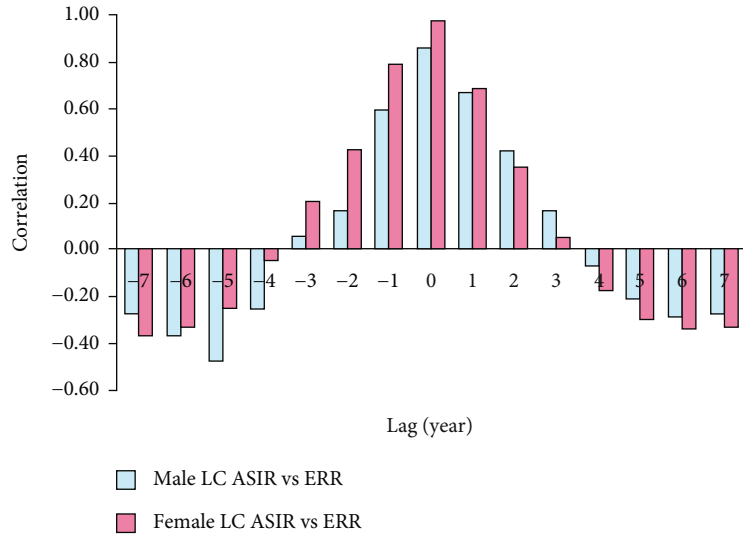

Figure 5: Comparison of time lag cross-correlation of sex-specific lung cancer ASIR with ERR in Shanghai.

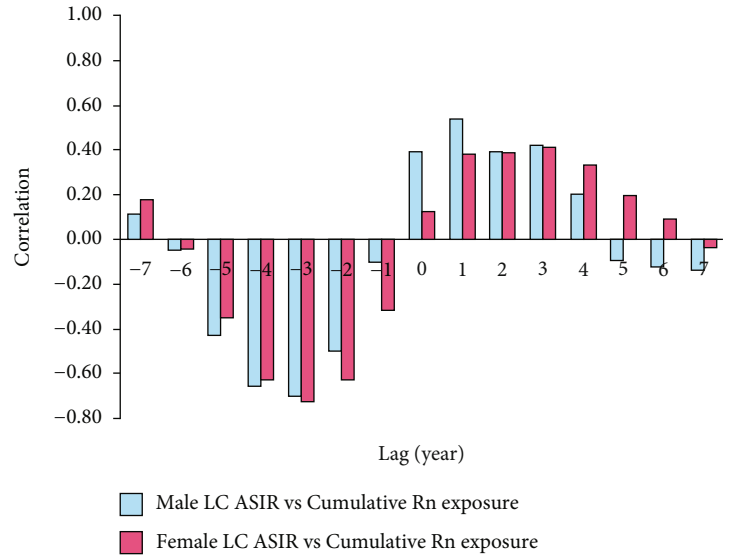

Figure 6: Comparison of time lag cross-correlation of sex-specific lung cancer ASIR with cumulative radon exposure in Shanghai.

trend. The intercity comparison was based on the increasing ERR and lung cancer ASIR from 2006 to 2016.

We also evaluated the lifetime relative risk (LRR) of lung cancer according to the definition in the BEIR IV report (National Research Council (NRC), 1988). The LRR of lung cancer is given by the sum of the risks of lung cancer death for each year:

$$R_e = \sum_{i=1}^{75} \frac{h_i(1+e_i)}{h_i^* + h_i e_i} \prod_{k=1}^{i-1} \exp(-(h_k^* + h_k e_k))[1 - \exp(-(h_i^* + h_i e_i)], \quad (3)$$

where R_e is the lifetime risk of lung cancer for a given exposure period; h_i and h_i^* are the lung cancer and overall mortality rates for age i, respectively; e_i is the ERR due to exposure to radon for age i; $\exp(-(h_k^* + h_k e_k))$ is the probability of surviving year k; and $[1 - \exp(-(h_i^* + h_i e_i)]$ is the probability of death in year k. The lifetime probability of lung cancer mortality is then the summation of years from 1 to 75, as given in Equation (3). A lifespan of 75 years was assumed in this study.

3. Results

3.1. The ERR of Lung Cancer Caused by Indoor Radon in Shanghai. Figure 1 presents the historical data on indoor radon concentrations from different surveys in Shanghai. The arithmetic means of the radon concentration for each survey are plotted in dots, and the smoothed curve is used to calculate the cumulative indoor radon exposure between 1985 and 2015 by assuming that the concentration before 1985 was the same. It is observed that the radon concentration rapidly increased after 2000.

Table 1: ERR and PAR of lung cancer incidence in 2006.

City	C_{Rn} (Bq/m^3)	$ERR_{never-smoker}$	$ERR_{ever-smoker}$	ERR[a] (95% CIs)	PAR[a] (%)	Lung cancer incidence ($1/10^5$)	
						Male	Female
Beijing	33.9	0.182	0.082	0.156 (0.013, 0.326)	13.52	5.49	3.05
Shanghai	18.2	0.094	0.043	0.081 (0.044, 0.206)	7.48	3.22	1.46
Guangzhou	35.7	0.288	0.130	0.247 (0.122, 0.616)	19.79	17.01	7.68
Shijiazhuang	26.3	0.110	0.050	0.094 (0.012, 0.201)	8.59	NA	NA
Wuhan	31.1	0.145	0.065	0.124 (0.057, 0.304)	11.01	5.68	2.69
Lianyungang	17.9	0.070	0.032	0.060 (0.027, 0.147)	5.65	3.59	1.48
Suzhou	34.0	0.300	0.135	0.257 (0.118, 0.632)	20.43	11.65	4.05
Shenyang	80.0	0.216	0.097	0.185 (0.055, 0.425)	15.64	9.41	5.25
Qingdao	40.7	0.191	0.086	0.164 (0.071, 0.398)	14.06	NA	NA
Hangzhou	25.5	0.205	0.092	0.176 (0.060, 0.413)	14.98	7.76	2.95
Jiaxing	25.7	0.083	0.037	0.071 (0.026, 0.169)	6.66	3.50	1.18
Zhongshan	63.2	0.328	0.148	0.281 (0.127, 0.690)	21.95	14.08	5.72
Yinchuan	67.1	0.287	0.129	0.246 (0.107, 0.599)	19.72	NA	NA
Xi'ning	20.9	0.110	0.050	0.094 (0.042, 0.230)	8.60	NA	NA
Tianjin	41.3	0.314	0.141	0.269 (0.122, 0.660)	21.21	NA	NA

[a]Calculation was based on the national average smoking prevalence (about 26%).

Table 2: ERR and PAR of lung cancer incidence in 2016.

City	C_{Rn} (Bq/m^3)	$ERR_{never-smoker}$	$ERR_{ever-smoker}$	ERR[a] (95% CIs)	PAR[a] (%)	Lung cancer incidence ($1/10^5$)	
						Male	Female
Beijing	38.4	0.183	0.082	0.157 (0.076, 0.391)	13.61	5.98	3.95
Shanghai	25.8	0.104	0.047	0.089 (0.033, 0.212)	8.20	3.96	2.92
Guangzhou	33.7	0.262	0.118	0.225 (0.108, 0.557)	18.35	10.96	5.36
Shijiazhuang	32.1	0.132	0.059	0.113 (0.040, 0.266)	10.15	4.65	2.48
Wuhan	51.3	0.155	0.070	0.133 (0.051, 0.317)	11.75	6.81	2.70
Lianyungang	27.0	0.083	0.037	0.071 (0.027, 0.170)	6.66	3.10	1.21
Suzhou	29.9	0.248	0.112	0.213 (0.116, 0.542)	17.58	10.01	5.19
Shenyang	86.8	0.305	0.137	0.261 (0.108, 0.630)	20.71	15.07	7.92
Qingdao	45.7	0.211	0.095	0.181 (0.070, 0.432)	15.33	9.17	3.65
Hangzhou	30.2	0.223	0.100	0.191 (0.110, 0.491)	16.00	9.81	5.61
Jiaxing	37.7	0.126	0.057	0.108 (0.043, 0.259)	9.74	5.82	4.72
Zhongshan	57.7	0.316	0.142	0.271 (0.126, 0.669)	21.33	13.14	8.27
Yinchuan	77.7	0.337	0.152	0.289 (0.146, 0.725)	22.42	11.06	5.43
Xi'ning	23.1	0.113	0.051	0.097 (0.043, 0.236)	8.82	5.64	2.50
Tianjin	22.1	0.251	0.113	0.215 (0.125, 0.555)	17.70	11.13	6.21

[a]Calculation was based on the national average smoking prevalence (about 26%).

Figure 2 shows an example of the distribution of indoor radon concentrations from a survey in 2008. The results of most surveys revealed that the distribution of indoor radon concentration roughly followed a lognormal distribution. The geometric standard deviations (GSDs) reported by 6 of the 11 surveys were between 1.31 and 1.61. In the model calculation in this study, the distribution of indoor radon concentration was assumed to be a lognormal distribution with a fixed GSD of 1.5, while the mean values varied from year to year.

Based on the age distribution in Shanghai, Figure 3 plots the curves of the estimated age-specific ERR in 2010, 2015, and 2020. The curves show that the ERR significantly increased in the 0-45 age group and then switched to decline in the over 45 age group. Compared with that of the older age, the increase of the estimated ERR for residents younger than 45 seems more pronounced. The ERR at age 40–45 was increased by 20.0% from 2010 to 2020.

The estimated age-standardized ERR and the reported lung cancer ASIR by sex in Shanghai are plotted in

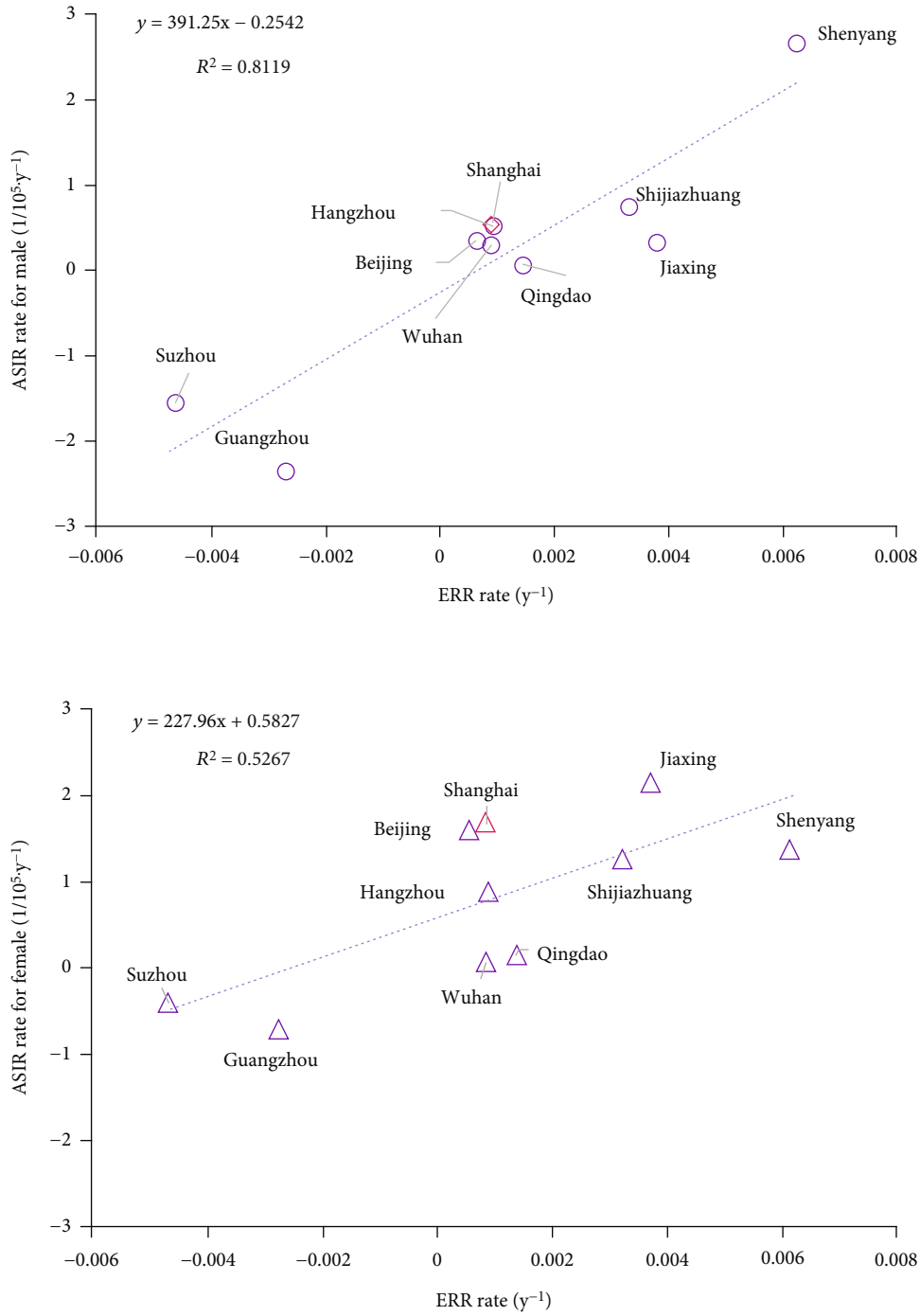

Figure 7: Correlation of the rate of change between ERR and sex-specific lung cancer ASIR in 2006–2016.

Figure 4. As shown in Figure 4, both the ERR and lung cancer ASIR increased in the late of 2000s. Compared with the trend of indoor radon concentrations in Figure 1, a time lag of the increase of ERR and ASIR can also be seen in Figure 4. The phenomenon could be explained as the delayed effect on radon-induced carcinogenesis in most previous studies [49–51].

The time series correlation analysis confirmed the correlation between the age-standardized ERR and lung cancer ASIR, as well as the delayed effect of indoor radon exposure on lung cancer. As shown in Figure 5, the correlation factors between lung cancer ASIR and age-standardized ERR are 0.862 and 0.978 for men and women, respectively. The time series correlation analysis revealed that there was no clearly delayed effect. It is thought that the delayed biological effect has already been considered in the EPA model, whereas it can be observed in Figure 6 that the lung cancer ASIR has a 3-year delay compared to the cumulative radon exposure.

3.2. Estimation of the ERR and PAR of Lung Cancer Incidence in Other Cities. The estimates of the age-standardized ERR and radon PAR of lung cancer incidence

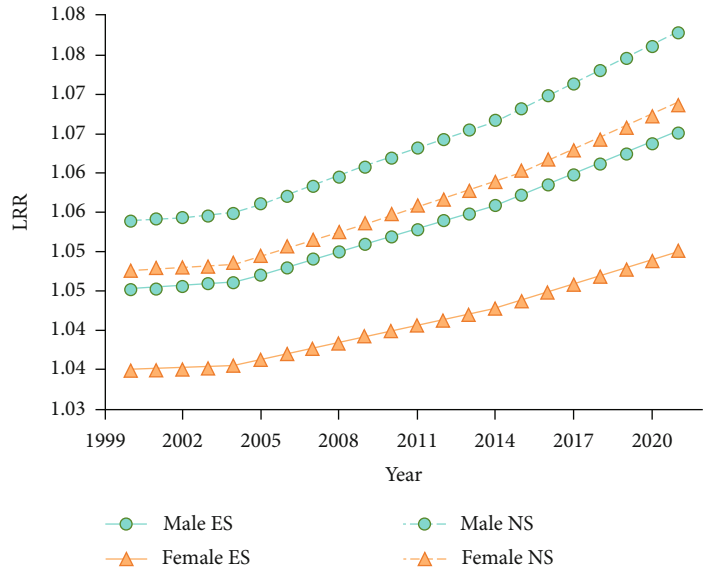

Figure 8: Changing trends of national lifetime relative risk by sex and smoking categories.

for the 15 cities are listed in Tables 1 and 2. As the collected data for the radon survey in most of the cities were not as detailed as those in Shanghai, Tables 1 and 2 simply compares the age-standardized ERR and radon PAR of lung cancer incidence for each city in 2006 and 2016, respectively. The results show that both the ERR and PAR of lung cancer incidence increase in both genders. The age-standardized ERR in the cities using the EPA model ranged from 0.060 (95% CI: 0.027, 0.147) to 0.281 (95% CI: 0.127, 0.690) in 2006 and from 0.071 (95% CI: 0.027, 0.170) to 0.289 (95% CI: 0.146, 0.725) in 2016, respectively. The PAR in 2006 ranged from 5.65% to 21.95%, with a median of 14.06% among the 15 cities, and the PAR in 2016 ranged from 6.66% to 22.42% with a median of 15.33%. Compared with the values in 2006, both the ERR and PAR increased in the 13 cities. The lung cancer incidence attributed to indoor radon in 2016 ranged from 3.96 to 15.07 per 10,000 population in males and 1.21 to 8.27 per 10,000 population in females. The results could be considered as the consequence of the rapid increase of indoor radon concentration which has been found in previous studies in China [52].

Due to the limited data on lung cancer ASIR, only the changing rates of ERR and lung cancer ASIR in 10 cities are plotted in Figure 7 for the intercity comparisons. Although these cities differed in radon concentrations, climates, and lifestyle, the increase of ASIR has an obviously positive correlation with the increase of ERR as shown in Figure 7. The R^2 values are 0.8119 and 0.5267 for men and women, respectively. This indirectly reflects that the increase in radon exposure is an important cause for the increase in lung cancer incidence.

3.3. Estimation of the LRR Caused by Indoor Radon in China. By considering the smoking prevalence, the LRR caused by indoor radon exposure for the whole nation was estimated for different sex and smoking categories using the EPA model. The population-weighted average of indoor radon concentration was used in the calculations. As shown in Figure 8, the LRR of nonsmokers (NS) was significantly higher than that of ever-smokers (ES) in both men and women. As the smoking prevalence of women (2.10%) is much lower than that of men (50.50%), the gap between female nonsmokers and ever-smokers is larger than that for males. The curves in Figure 8 also indicate that the LRR has a significant growth rate in the recent thirty years. In 2021, the estimated LRR were 1.078, 1.065, 1.069, and 1.050 for male nonsmokers, male ever-smokers, female nonsmokers, and female ever-smokers, respectively.

4. Discussion

This study found that the ERR and PAR of lung cancer incidence attributable to indoor radon exposure increased in most Chinese cities. The local rate of increase in ERR and lung cancer ASIR showed a significantly positive. This may be explained by the rapid development of economic and great changes in lifestyle, which has resulted in the increase in indoor radon concentrations. Across age and sex, the risk of lung cancer attributable to indoor radon exposure was found more pronounced in males and 40-45 age groups. Besides, our results also indicated that the ERR and lung cancer ASIR present an overall increasing trend from 2010 to 2020 and the LRR remain constantly growing over the past two decades. The PAR evaluated in this study was comparable with that in other previous studies [53].

Lung cancer is the most common cause of cancer death in China, which is responsible for nearly 25.9% of all male cancer deaths and 20.6% of all female cancer deaths [54], and China is one of the minority countries where radon concentration significantly increased [3, 4]. Indoor radon should be emphasized, and the authority should strengthen its implementation of effective public policies and other interventions.

Smoking as the leading cause of lung cancer accounted for approximately 56.8% of lung cancer in men and 12.5% in women [55]. Hence, it is important to consider how smoking might modify risks due to radon. For a given smoking category, there is much difference in LRR between males and females. This is because the smoking category-specific baseline rates differ between males and females. Under the EPA model, the LRR of radon-induced lung cancer was much higher in never-smokers compared to that in long-term smokers; this is because the baseline risk of lung cancer among long-term smokers was much higher than (more than twice) among never-smokers [56–59].

Besides smoking, the fine particulate matter with a diameter less than 2.5 μm (PM2.5) is also associated with lung cancer. The national PM2.5 levels experienced a rapid increase in the early 21st century because of rapid industrialization and high energy consumption in China. In general, PM 2.5 concentrations present an overall decreasing trend until 2018 that have showing signs of improvement [60, 61]. The metaestimate for lung cancer risk associated with PM2.5 was 1.11 for mortality (95% CI: 1.05, 1.18) and 1.08 (95% CI: 1.03, 1.12) for incidence [62]. However, the combined effect of radon and PM2.5 had not been included in this study, which is necessary to be discussed in further study.

In this study, we assessed the lung cancer risk attributable to indoor radon among different cities and identified high-risk groups based on different population characteristics. However, several limitations still existed in our study. Firstly, there have been limited available studies on systematically designed survey on indoor radon concentration during the past decade from China. Secondly, human exposure to radon is multisource, but data limitations prevented the assessment of radon exposure from different source, such as underground space, occupational space, and outdoor space. Thirdly, we have only analyzed the lung cancer risk attributable to indoor radon exposure, and the joint effects of other indoor pollutants including gaseous contaminants were not considered during the study. Further studies are needed to be taken for estimation of the health effect and burden of disease not only by radon exposure but also by a greater range of indoor contaminants.

5. Conclusion

This study estimated spatial and temporal trends in lung cancer risk caused by indoor radon exposure in a total of 15 cities in China from 2006 to 2016 based on the established database. In Shanghai, the ERR had significantly increased after 2010, where indoor exposure is more harmful among younger residents. Overall, in most Chinese cities, the increase in indoor radon concentrations is very similar to the overall trend of the growth of ERR and PAR of lung cancer incidence. The local rate of increase in ERR and lung cancer ASIR showed a significantly positive correlation. Besides, the LRR remained high in nonsmokers.

This study preliminarily assessed the health effects of indoor radon exposure on lung cancer by using ERR, LRR, and PAR in 15 cities that have not been provided in previous studies in China. Limited by the available data, detailed discussions cannot be conducted in many cities. This study was expected to be beneficial in improving radon-related health risk assessments, policy making, and resource allocation in prevention of lung cancer cause by indoor radon.

Data Availability

Data is available on request.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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Supplementary Materials

The Monte Carlo method for ERR estimation was described in detail as the supplementary materials. *(Supplementary Materials)*

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