Inhalation exposure to ambient polycyclic aromatic hydrocarbons and lung cancer risk of Chinese population

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An Euler atmospheric transport model (Canadian Model for Environmental Transport of Organochlorine Pesticides, CanMETOP) was applied and validated to estimate polycyclic aromatic hydrocarbon (PAH) ambient air concentrations at ground level in China based on a high-resolution emission inventory. The results were used to evaluate lung cancer risk for the Chinese population caused by inhalation exposure to PAHs. The uncertainties of the transport model, exposure, and risk analysis were assessed by using Monte Carlo simulation, taking into consideration the variation in PAH emission, aerosol and OH radical concentrations, dry deposition, respiration rate, and genetic susceptibility. The average benzo[*a*]pyrene equivalent concentration (B[*a*]P_{eq}) was 2.43 [≈1.29–4.50 as interquartile range (IR)] ng/m^3. The population-weighted B[*a*]P_{eq} was 7.64 (IR, ≈4.05–14.1) ng/m^3 because of the spatial overlap of the emissions and population density. It was estimated that 5.8% (IR, ≈2.0–11%) of China's land area, where 30% (IR, ≈17–43%) of the population lives, exceeded the national ambient B[*a*]P_{eq} standard of 10 ng/m^3. Taking into consideration the variation in exposure concentration, respiration rate, and susceptibility, the overall population attributable fraction (PAF) for lung cancer caused by inhalation exposure to PAHs was 1.6% (IR, ≈0.91–2.6%), corresponding to an excess annual lung cancer incidence rate of 0.65×10^{-5}. Although the spatial variability was high, the lung cancer risk in eastern China was higher than in western China, and populations in major cities had a higher risk of lung cancer than rural areas. An extremely high PAF of >44% was estimated in isolated locations near small-scale coke oven operations.

China | respiration exposure | PAH | air pollution

Polycyclic aromatic hydrocarbons (PAHs) are included in the Convention on Long-Range Transboundary Air Pollution Protocol on Persistent Organic Pollutants and are among the most toxic organic pollutants of concern in China. High PAH air concentrations have been shown to induce heritable mutations in mice (1). In addition, an increased lung cancer risk in people who work in coke oven and aluminum production plants, where they are exposed to high levels of PAHs, has been documented (1).

The annual PAH emission in China was ≈114,000 tons in 2004, accounting for 29% of the global total (2). Consequently, the ambient air PAH concentrations in many parts of China are orders of magnitude higher than those in developed countries. For instance, it was reported that the mean particulate phase PAH concentration in the North China Plain, including both urban and rural areas, was 346 ng/m^3 in the winter of 2005 (3). This concentration is significantly higher than PAH concentrations measured in Birmingham, United Kingdom in 1992 (20.3 ng/m^3), London in 1997 (39 ng/m^3), and Chicago in 2004 (70 ng/m^3) (4–6). Because the combustion of biomass and coal for energy in China will not change significantly in the coming decades, severe PAH contamination in China is expected to continue. A number of studies have been conducted on the health risks caused by PAH exposure in China but those studies were limited to either occupational exposure or localized cases with extremely high PAH concentrations (7, 8). Lung cancer has been ranked as the fourth and fifth leading causes of cancer death in Chinese males and females, respectively (9). So far, however, a nationwide assessment of the potential health risks caused by PAH exposure in China has not been done.

Measured PAH concentrations in ambient air can be used to evaluate human exposure risk. However, in most cases, these measurements were conducted randomly at a limited number of sites. For a regional study, models, with satisfactory validation, are preferred for estimating the spatial distribution of ambient air PAH concentrations for population exposure assessment. For example, an Industrial Source Complex Long-Term model was used to map PAH concentrations at ground level in Tianjin, China and a relatively high exposure risk was found among urban residents (10).

Because atmospheric PAH concentrations vary from place to place because of variation in emissions and atmospheric transport, there is also a high degree of variability in human exposure to PAHs via inhalation. In addition, differences in respiration rate and genetic susceptibility among individuals can lead to a different risk at the same exposure level (11). To the best of our knowledge, however, genetic susceptibility has never been included in a probabilistic lung cancer risk assessment of PAH exposure. The individual respiration rate is body weight dependent (12), while genetic susceptibility for lung cancer risk is associated with the polymorphism of genes related to metabolism of carcinogens and DNA repair (1). Based on these relationships, the variability in respiration rate and susceptibility can be taken into account in a population-level exposure risk assessment.

Population attributable fraction (PAF) represents the proportional reduction of a certain disease or death within a population that would occur if the exposure to a risk factor were removed or reduced to an alternative level (13). This concept was applied successfully to address the global disease burden caused by multiple risk factors (14, 15) and the relationship between residential radon exposure and lung cancer in Europe (13).

The objectives of this study were to model the ambient air PAH concentrations and evaluate the lung cancer risk caused by inhalation exposure to PAHs at the population level in China. The variability in the exposure risk was addressed by using the spatial variation in PAH exposure concentration and the inter-individual variation in respiration rate and genetic susceptibility. Sixteen parent PAHs on the United States Environmental

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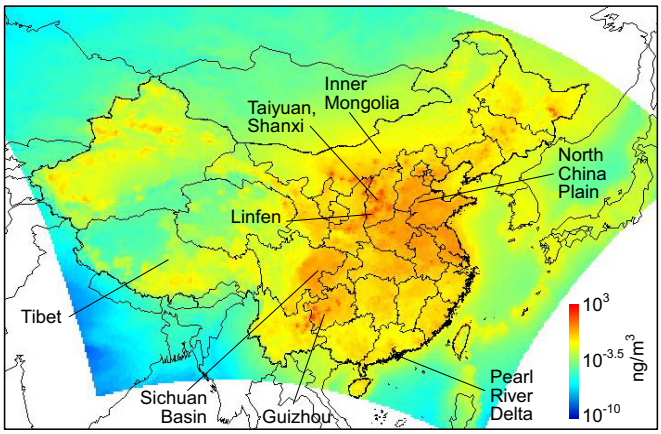

Fig. 1. Spatial distribution of annual mean air concentrations of B[*a*]P at 1.5 m above the ground in China (South China Sea not included) in 2003. The results are presented in log scale.

Protection Agency priority pollutant list (16) were included in this study.

Results and Discussion

Ambient Air PAH Concentrations. The calculated ambient air concentrations of benzo[*a*]pyrene (B[*a*]P), a representative carcinogenic PAH compound, at 1.5 m above ground surface are mapped in Fig. 1. The detailed results of the modeling for all PAHs are presented in *SI Appendix*. Given the relatively short atmospheric lifetime of gas-phase PAHs (17), the similarity in the spatial distributions of PAH concentrations and the PAH emission inventory is expected. In general, atmospheric PAH concentrations in east China were higher than those in the west. Particularly high PAH concentrations were estimated in the North China Plain, East Sichuan Basin, and part of Guizhou. Although the total area of these provinces accounts for only 12% of the land area of China, biomass consumption and coal usage for the coking industry in these provinces accounts for 48% and 66% of the national use, respectively. In addition, biomass and coke production generate >83% of the total PAH emission in China (18, 19). Extremely high PAH concentrations are estimated in Shanxi (from Taiyuan to Linfen) and the calculated ambient B[*a*]P concentrations in a number of model grid cells were as high as 400 ng/m^3. In fact, ≈1–2 μg/m^3 B[*a*]P has been measured at sites within this geographic area (20). Shanxi produced 300 million tons of coal in 2003 and ranked first in PAH emission among all Chinese provinces (18) because of thousands of small-scale coke ovens without any emission control measures (18). Relatively high PAH concentrations in west Guizhou can also be attributed to widespread small-scale coke production (18). In contrast, substantially lower B[*a*]P concentrations, ranging from 10^{-4} to 0.1 ng/m^3, were predicted in a vast area of western China, extending from Tibet to Inner Mongolia. PAHs emitted in eastern China are not transported to western China because air moves constantly eastward under the atmospheric westerly regime and the influence of the East Asian Monsoon in summer hardly extends beyond Shanxi and Sichuan (21).

Population Exposure. To emphasize the higher overall risk in higher population density areas, population-weighted B[*a*]P equivalent concentrations (B[*a*]P$_{eq}$) were calculated as products of the predicted B[*a*]P$_{eq}$ and population density divided by national average population density. The calculated population-weighted B[*a*]P$_{eq}$ over 1 × 1-km^2 grid cells are mapped in Fig. 2 *Left*. The population-weighted annual mean B[*a*]P$_{eq}$ was 7.64 [≈4.05–14.1 as interquartile range (IR)] ng/m^3 and was much higher than the value without population-weighting (2.43, ≈1.29–4.50 ng/m^3 as IR). The major PAHs that contributed to the high B[*a*]P$_{eq}$ were B[*a*]P (49.2%), dibenz(a,h)anthracene (15.9%), and benz[*a*]anthracene (11.5%), followed by benzo[*b*-]fluoranthene (4.9%) and benzo[*k*]fluoranthene (4.8%). Even though naphthalene (355 ng/m^3) and phenanthrene (94.5 ng/m^3) had the highest concentrations, they did not contribute significantly to the total B[*a*]P$_{eq}$ because of their relatively low toxicity.

As expected, the geographic distribution of the population-weighted B[*a*]P$_{eq}$ was similar to that of ambient PAH concentrations. However, the population-weighted B[*a*]P$_{eq}$ values in the North China Plain, East Sichuan Basin, and Pearl River Delta were very high because of spatial overlap of the high PAH emission density and high population density. In addition, high population-weighted B[*a*]P$_{eq}$ values were identified in cities in northeast China (e.g., Shenyang and Harbin), southeast China (e.g., Guangzhou and Fuzhou), and western China (Urumuqi and Lhasa). In general, the population-weighted B[*a*]P$_{eq}$ values in urban areas, including small- and medium-sized cities and towns, were higher than those in rural areas. However, the difference between urban and rural areas in China was not as large as in developed countries. In developed countries, PAH emissions are primarily from traffic, waste incineration, and consumer product usage that occur primarily in urban areas (2). For example, the geometric mean concentration of 15 PAHs was 200 and 14 ng/m^3 in the urban center of Chicago and an adjacent rural site, respectively (22). In contrast, the most important PAH source in China is biomass combustion, which contributes 56% to the total PAH emissions in the country and occurs primarily

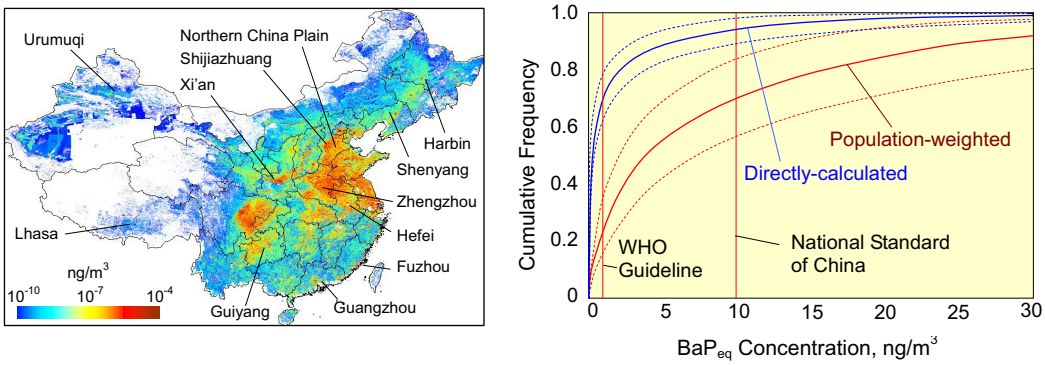

Fig. 2. Geographic distribution of population-weighted annual mean B[*a*]P$_{eq}$ in ambient air 1.5 m above the ground in China (South China Sea not included) in 2003 (*Left*, log scale) and the cumulative frequency distributions of annual mean B[*a*]P$_{eq}$ without (blue curves) and with (red curves) population weighting (*Right*). The dashed curves show the 25th and the 75th percentiles derived from the Monte Carlo simulation. The two red lines show the Chinese national standard (10 ng/m^3) and WHO guideline (1 ng/m^3) for B[*a*]P$_{eq}$ in ambient air.

Table 1. Relative contributions of various B[*a*]P emission sources to the total B[*a*]P emission and population B[*a*]P exposure concentration with and without population weighting

Source	Emission height. M	Emission* t/y	Emission* %	Direct-calculated exposure concentration† ng/m³	Direct-calculated exposure concentration† %	Population-weighted exposure concentration† ng/m³	Population-weighted exposure concentration† %	Intake fraction per million‡
Biomass burning	1.5	753	47	5.2×10^{-1}	43	1.8×10^{0}	48	27
Small-scale coke production	10	625	39	5.5×10^{-1}	46	1.3×10^{0}	36	24
Domestic coal combustion	1.5	133	8.3	9.7×10^{-2}	8.0	4.0×10^{-1}	11	35
Industrial sources	100	62.5	3.9	2.1×10^{-2}	1.7	1.1×10^{-1}	2.9	20
Open fire straw burning	10	11.9	0.74	1.3×10^{-2}	1.1	4.5×10^{-2}	1.2	43
Traffic gas combustion	1.5	4.01	0.25	4.4×10^{-3}	0.36	3.7×10^{-2}	1.0	106
Wild fires	10	1.12	0.07	1.2×10^{-3}	0.10	7.2×10^{-4}	0.02	7.4

*Annual emissions and the relative contributions to the total emission.
†Ambient air concentrations and the relative contributions to the total exposure from individual sources with or without population weighting, these values are arithmetic means of the all grids of the entire country.
‡Fraction of emission that is eventually inhaled.

in villages (18). Based on an extensive air monitoring campaign in the North China Plain in the winter of 2004–2005, PAH concentrations in rural areas were similar to, if not higher than, those in urban areas (23). If indoor air pollution is taken into consideration, the respiration risk in rural dwellings could be even higher than that in ruban dwellings because of the widespread use of biomass and coal-burning stoves.

The cumulative frequency distributions of the model predicted annual mean B[*a*]P_{eq} concentrations across China, with and without population-weighting, are plotted in Fig. 2 *Right*. The Chinese national standard is 10 ng/m³ [Indoor Air Quality Standards (GB/T 18883-2002, 2003-03-01), Ministry of Environmental Protection of China: www.zhb.gov.cn] and the WHO guideline is 1 ng/m³ (24) (red lines in Fig. 2). Approximately 5.8% (IR, 2.0 ≈11%) of the land area and 30% (IR, 17 ≈43%) of the population, equivalent to ≈0.56 (IR, ≈0.19–1.1) million km² with 400 million (IR, ≈230–570) people, are predicted to be exposed to B[*a*]P_{eq} concentrations >10 ng/m³. If the 1 ng/m³ B[*a*]P_{eq} WHO guideline is applied, the exceedence rates would be 30% (IR, ≈22–38%) of the land area and 77% (IR, ≈66–85%) of the population.

The population exposure to PAHs from different emission sources was also calculated, and the relative contributions were significantly different from their contributions to the total PAH emissions. Table 1 shows these differences using B[*a*]P as a representative PAH. Intake fractions (IFs), defined as the fraction of the emission that is eventually inhaled, for various sources are also presented in Table 1 (25, 26). The relative contribution of each emission source to the PAH exposure depends on the location and height of the emission. A typical example is the difference between high industrial sources (100 m) and small-scale coke ovens (10 m). The contribution of the latter increased from 39% of the total emission to 46% of the total exposure, whereas the contributions of the former decreased from 3.9% to 1.7%. When PAHs are emitted lower in the atmosphere, they are transported to ground level in the surrounding area. However, when PAHs are emitted at ground level, their transport to the surrounding area is limited. As a result, the relative contribution of biomass burning PAH emissions at ground level to the exposure (43%) was less than that to the total PAH emissions (47%). The relative contribution of an emission source to the population-weighted exposure was also affected by the location of the source. For example, the contribution of wild fires, which occurred in remote areas, to the population-weighted PAH exposure (0.02%) was significantly lower than its contribution to the total emission (0.07%). The overall IF was ≈27 per million for B[*a*]P emitted in China, and varied from 7.4 (wild fires) to 106 (traffic) per million, depending on the spatial distribution of the PAH emissions and population and the height of emission sources.

PAH Exposure Risk. To evaluate the potential lung cancer risk caused by inhalation exposure to PAHs, the overall PAF was calculated. Based on the average population-weighted B[*a*]P concentration, the calculated PAF was 0.53% (IR, ≈0.29–0.95%). However, when the variation in the asymmetrically distributed PAH exposure concentration, respiration rate, and genetic susceptibility were taken into account, the recalculated arithmetic mean PAF increased to 1.6% (IR, ≈0.91–2.6%), which suggests that the risk is underestimated when variability is not included. Fig. 3 shows the ccumulative frequency of PAFs with and without the variabilities. The underestimation of risk is particularly evident for high-risk populations when variability is not included. Because the cumulative frequency curve is the steepest between 0.1% and 1% and more than half of the Chinese population has PAF values within this range, 0.1% and 1% were used as the threshold levels for evaluating the risk. By including the variabilities in the model, the percentage of the population with PAF >1% increased from 13.7% (IR, ≈4.5–27.5%) to 24.5% (IR, ≈16.4–34.1%), while the percentage of the population with PAF <0.1% did not change significantly. Because China's lung cancer incidence rate was 40.7×10^{-5} in 2003 (27), the excess annual lung cancer incidence rate induced by

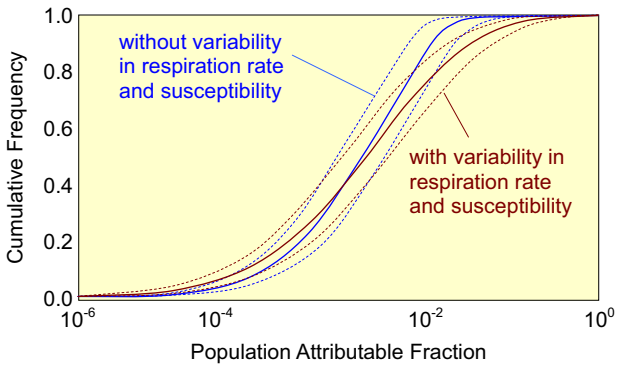

Fig. 3. The cumulated frequency distributions of PAF for lung cancer induced by inhalation exposure to PAHs in China (South China Sea not included). The results were derived with (brown curves) and without (blue curves) taking into consideration the variation in respiration rate and susceptibility. The 25th and 75th percentiles are presented as dashed curves.

PAH exposure was estimated to be 0.65×10^{-5}. The geographic distribution of PAF was similar to that of the PAH exposure concentration (*SI Appendix*).

Although the overall mean PAF for China was 1.6%, extremely high PAFs of >44% were predicted in a number of isolated areas, including Shanxi and Guizhou, where small-scale coke ovens had operated for years. The annual mean ambient B[*a*]P concentration in these areas can reach >400 ng/m^3, which is comparable with the occupational exposure of coke-oven workers (28). According to a survey in suburban Yuncheng, Shanxi, where a large number of coke production facilities are located, the annual lung cancer incidence was as high as 117.6×10^{-5} (29), significantly higher than the national average of 40.7×10^{-5} (30). Such a high localized risk is of particular concern for vulnerable populations, including children and those with high lung cancer susceptibility. Relatively high PAFs are predicted in major cities such as Tianjin, Shijiazhuang, Xi'an, Zhengzhou, Hefei, and Harbin where PAF values ranged from 1% to 10%. Over the entire eastern China, mean PAF values were ≈3%, which was much higher than mean PAF of 0.2% in western China. Zhang et al. (30) summarized the annual lung cancer incidence rate for 35 registries in 2003 and came up with rates between 10.1×10^{-5} and 69.9×10^{-5} (*SI Appendix*). This survey also revealed that the lung cancer incidence rates in large cities (45.9×10^{-5}) were significantly higher than those in small cities (26.6×10^{-5}) and that the lung cancer incidence rate in eastern China (36.1×10^{-5}) was higher than that in western China (17.5×10^{-5}) (30).

The PAFs induced by inhalation exposure to PAHs were also calculated for the registries involved in the epidemiological survey conducted by Zhang et al. (30), and the relatively low PAFs (≈0.08–8.33%) caused by PAH exposure implies the existence of other potential causes of lung cancer. Cigarette smoking, inhalation system disease, and exposure to other atmospheric pollutants are among the major causes of lung cancer (9, 27). For example, a nationwide survey revealed that cigarette smoking accounted for 50% of male deaths caused by lung cancer and 35% of female deaths caused by lung cancer (9). Although the risk caused by exposure from ambient PAHs was much less than that from cigarette smoking, the former exposure was unintentional to the entire population. It should also be pointed out that the calculated PAF assumed a lifelong inhalation exposure. Because PAH concentrations were much lower before the 1980s, the model prediction of lung cancer rate may be an overestimation. Because biomass and domestic coal combustion account for 63% of the total PAH emissions in China and these fuels are used indoors for heating and cooking (18), it is very likely that inhalation exposure to PAHs indoors is higher than outdoors in rural China. In an extreme case, it was reported that the average B[*a*]P concentration in kitchens of rural families in Xuanwei, Yunnan was as high as 14.7 μg/m^3 as a result of using smoky coal for heating and cooking (31). The higher lung cancer death rate of nonsmoking females compared with nonsmoking males in rural China is likely caused by exposure to smoke from biomass and coal combustion in kitchens (29). Because the current risk assessment is based on PAH concentrations in outdoor air, more information on rural indoor air PAH concentrations is urgently needed before an overall assessment of PAH inhalation risk can be conducted. In addition to respiration exposure, ingestion is an important pathway of human PAH exposure. Ingestion exposure to PAHs in China includes direct contamination of foods (32, 33) and contamination during the cooking process (33). The potential risk of PAH exposure caused by ingestion should also be evaluated in China.

Policy Issues. Energy consumption in China is expected to increase rapidly in the coming decades. However, PAH emissions may not follow this same trend because most of the additional energy consumption will be in the form of power generation and industrial activities that have relatively low PAH emission factors (18). PAH emissions will decline in China in the future because existing small-scale coke ovens, which account for 39% of the total current PAH emissions, will be closed under the Coal Law. Over the past decade, hundreds of thousands of coal-burning family stoves in urban districts of Beijing have been replaced by natural gas stoves or centralized heating facilities, leading to a dramatic reduction in PAH emission (34). In fact, there is a trend to replace family stoves with centralized heating facilities in northern Chinese cities (18). As a result, despite the rapid growth in industry and fast increase in the number of automobiles, the projected PAH emissions in China for 2010 is ≈73% of the emissions in 2003 (18). If these PAH emission reduction measures are fully realized and meteorological conditions do not change significantly, we predict that the percentage of the Chinese population exposed to B[*a*]P_{eq} concentrations that exceed the national standard would be reduced from 30% (IR, ≈22–38%) in 2003 to 22% (IR, ≈10–37%) in 2010. The PAF would decrease from 1.6% (IR, ≈0.91–2.6%) to 1.2% (IR, ≈0.70–2.0%) during the same period.

Although the total PAH emission will decrease in the future, the emission from indoor biomass burning in rural areas, which contributes a large portion of the PAH exposure in China (48% in 2003), will increase slightly. Biomass will remain to be the major energy source in rural China for many years to come. However, how the biomass is burned will make a big difference in the PAH emissions (18). From 1980 to the mid-1990s, a program was carried out to promote improved biomass burning stoves in rural China (35). Although coal-burning stoves were not highlighted in the program and the achievement of the program was not as promising as planned (36), the potential for saving energy and reducing emissions by applying simple, inexpensive, practical, but effective low-technologies was well demonstrated. In addition to improved stoves, the emission from biomass burning can be reduced substantially by converting it to biogas or biomass briquette (18). In fact, the number of household biogas digesters increased rapidly in recent years (35). It was reported that the government subsidy for biogas digesters has increased 25% and 5 million new digesters will soon be in place (The Central Government of China: http://english.gov.cn). Under the increasing pressure of energy shortage, environmental pollution, and emission reduction obligation, the wide application of these new technologies appears to have a bright future. However, the access to continuous technical, financial, and administrative support required to apply these technologies continue to be a challenge.

Methodology

The model framework applied in the present study is a combination of three models including an atmospheric transport model, a population exposure model, and a lung cancer risk assessment model. These three models are briefly introduced below. More details are presented in *SI Appendix*.

Transport Modeling. The atmospheric transport of PAHs in China was predicted for the reference year of 2003 by using the CanMETOP (Canadian Model for Environmental Transport of Organochlorine Pesticides) model (37) based on a high-resolution emission inventory (18) and meteorological data for the same year. Given that the spatial resolution of the model was 24×24 km^2, the detailed variation in air concentration and deposition of PAHs at the subgrid scale was averaged and smoothed. Consequently, the ambient air concentrations at an urban site, where population density and emission rate were much higher than those in the surrounding suburban and rural areas within the same grid cell, were underestimated. Such a scaling effect was rectified by interpolating the calculated concentrations into 1×1-km^2 grids based on the 1×1-km^2 emission inventory (18). The model predictions, both originally calculated and spatially interpolated, were validated by comparing them with the literature reported mean concentrations at 33 sites over China. The interpolated results are generally acceptable. Monte Carlo simulation was applied to evaluate the total uncertainty. The detailed information on model

description, parameterization, down-scaling interpolation, validation, and uncertainty analysis is provided in *SI Appendix*.

Exposure Modeling. Human inhalation exposure to PAHs was evaluated based on B[*a*]P_{eq}, which was calculated by using the model predicted PAH concentrations at 1.5 m above ground surface and toxicity equivalent factors. The calculated B[*a*]P_{eq} values at 1 × 1-km^2 resolution were population-weighted to address the overall carcinogenic risk. The detailed information on BP_{eq} calculation and population-weighting are presented in *SI Appendix*.

Lung Cancer Risk Assessment. The lung cancer risk in each 1 × 1-km^2 grid cell was assessed by calculating a PAF of inhalation exposure to PAHs after Menzler et al. (13):

$$PAF = \frac{rr(c_{bap}) - 1}{rr(C_{bap})} \text{ and } rr(c_{bap}) = [URR_{cum.exp=100}]^{c_{bap}\times 70/100)}$$

where *rr* and *URR* are the relative risk associated with a given B[*a*]P concentration (c_{bap}) and unit relative risk, respectively (38). The *rr* was calculated by using a dose–response relationship between *rr* and cumulative exposure to B[*a*]P (38). A lifelong exposure of 70 years was assumed. The *URR* value of 4.49, at a benchmark of 100 $\mu g/m^3$ years of B[*a*]P exposure, was adopted based on the result of an epidemical study conducted in Xuanwei, China, where high lung cancer incidence rate was associated with indoor burning of smoky coal (39).

The uncertainty and variability of the predicted exposure risk were evaluated by using Monte Carol simulation. The three distribution functions for exposure concentration (f_c), respiration rate (f_{ir}, relative to the population mean), and genetic susceptibility (f_{sus}, relative to the population mean) were adopted for describing the variabilities of the model input. The f_c was generated from the transport modeling. The f_{ir} was derived from a nationwide survey conducted from 2001 to 2004 and covered persons randomly chosen from four provinces in China and data on gender, age, body weight, and the minute ventilation were all collected (40). For developing f_{sus}, the quantitative information on polymorphism of human enzyme system responding to PAH metabolism and DNA repair was collected. Metastudies with large enough sample sizes to establish statistically significant results were selected. The frequencies of at-risk alleles of 13 types of polymorphism on eight lung cancer susceptibility-related genes of Chinese or Asian populations were collected from the literature. These data were not based on age or gender. Detailed information on the derivations of the respiration rate, calculation of the susceptibility functions, and the Monte Carlo simulation are provided in *SI Appendix*.

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1. Bostrom CE, et al. (2002) Cancer risk assessment, indicators, and guidelines for polycyclic aromatic hydrocarbons in the ambient air. *Environ Health Perspect* 110:451–488.
2. Zhang Y, Tao S (2009) Global atmospheric emission inventory of polycyclic aromatic hydrocarbons (PAHs) for 2004. *Atmos Environ* 43:812–819.
3. Liu SZ, et al. (2007) Atmospheric polycyclic aromatic hydrocarbons in north China: A wintertime study. *Environ Sci Technol* 41:8256–8261.
4. Harrison RM, Smith DJT, Luhana L (1996) Source apportionment of atmospheric polycyclic aromatic hydrocarbons collected from an urban location in Birmingham, UK. *Environ Sci Technol* 30:825–832.
5. Dimashki M, Lim LH, Harrison RM, Harrad S (2001) Temporal trends, temperature dependence, and relative reactivity of atmospheric polycyclic aromatic hydrocarbons. *Environ Sci Technol* 35:2264–2267.
6. Sun P, Blanchard P, Brice KA, Hites RA (2006) Trends in polycyclic aromatic hydrocarbon concentrations in the Great Lakes atmosphere. *Environ Sci Technol* 40:6221–6227.
7. Cheng JP, et al. (2007) PM10-bound polycyclic aromatic hydrocarbons (PAHs) and cancer risk estimation in the atmosphere surrounding an industrial area of Shanghai, China. *Water Air Soil Pollut* 183:437–446.
8. Tan JH, et al. (2006) Seasonal variation of particulate polycyclic aromatic hydrocarbons associated with PM10 in Guangzhou, China. *Atmos Res* 80:250–262.
9. Jiang JM, et al. (2008) Age-related effects of smoking on lung cancer mortality: A nationwide case-control comparison in 103 population centers in China. *Ann Epidem* 18:484–491.
10. Tao S, et al. (2006) Dispersion modeling of polycyclic aromatic hydrocarbons from combustion of biomass and fossil fuels and production of coke in Tianjin, China. *Environ Sci Technol* 40:4586–4591.
11. Kiyohara C, Otsu A, Shirakawa T, Fukuda S, Hopkin JM (2002) Genetic polymorphisms and lung cancer susceptibility: A review. *Lung Cancer* 37:241–256.
12. Layton DW (1993) Metabolically consistent breathing rates for use in dose assessments. *Health Phys* 64:23–36.
13. Menzler S, et al. (2008) Population attributable fraction for lung cancer due to residential radon in Switzerland and Germany. *Health Phys* 95:179–189.
14. Ezzati M, et al. (2003) Estimates of global and regional potential health gains from reducing multiple major risk factors. *Lancet* 362:271–280.
15. Lopez AD, Mathers CD, Ezzati M, Jamison DT, Murray CJL (2006) Global and regional burden of disease and risk factors, 2001: Systematic analysis of population health data. *Lancet* 367:1747–1757.
16. Keith LH, Telliard WA (1979) Priority pollutants: A perspective view. *Environ Sci Technol* 13:416–423.
17. Hafner WD, Carlson DL, Hites RA (2005) Influence of local human population on atmospheric polycyclic aromatic hydrocarbon concentrations. *Environ Sci Technol* 39:7374–7379.
18. Zhang YX, et al. (2008) Emission of polycyclic aromatic hydrocarbons from indoor straw burning and emission inventory updating in China. *Ann NY Acad Sci* 1140:218–227.
19. Zhang YX, Tao S, Cao J, Coveney RM (2007) Emission of polycyclic aromatic hydrocarbons in China by county. *Environ Sci Technol* 41:683–687.
20. Huangpu G, Qiu M, Liang XP, Li YE (2002) Relationship between atmospheric polycyclic aromatic hydrocarbons pollution and lung cancer incidence in Datong City, China. *Shanxi Pre Med* 11:132–134.
21. Lin ZG, Zhang JC (1991) in *China Climate*, ed Zhang WD (China Meteorological Press, Beijing), pp 257–292.
22. Cotham WE, Bidleman TF (1995) Polycyclic aromatic-hydrocarbons and polychlorinated-biphenyls in air at an urban and a rural site near Lake Michigan. *Environ Sci Technol* 29:2782–2789.
23. Liu SZ, et al. (2008) Seasonal and spatial occurrence and distribution of atmospheric polycyclic aromatic hydrocarbons (PAHs) in rural and urban areas of the North Chinese Plain. *Environ Pollut* 156:651–656.
24. Ravindra K, Sokhi R, Van Grieken R (2008) Atmospheric polycyclic aromatic hydrocarbons: Source attribution, emission factors, and regulation. *Atmos Environ* 42:2895–2921.
25. Bennett DH, et al. (2002) Defining intake fraction. *Environ Sci Technol* 36:206A–211A.
26. Tainio M, et al. (2009) Evaluation of the European population intake fractions for European and Finnish anthropogenic primary fine particulate matter emissions. *Atmos Environ* 43:3052–3059.
27. Zhao Y, Wang SX, Aunan K, Seip HM, Hao JM (2006) Air pollution and lung cancer risks in China: A meta-analysis. *Sci Total Environ* 366:500–513.
28. Armstrong B, Tremblay C, Baris D, Theriault G (1994) Lung cancer mortality and polynuclear aromatic hydrocarbons: A case-cohort study of aluminum production workers in Arvida, Quebec, Canada. *Am J Epidem* 139:250–262.
29. Zhang SN, Li X (1998) Survey of lung cancer incidence in suburban areas. *J Pract Med Tech* 5:982–982.
30. Zhang SW, Chen WQ, Kong LZ, Li GL, Zhao P (2007) An annual report: Cancer incidence in 35 cancer registries in China, 2003. *Chin Tumor* 16:494–507.
31. Slooff W, Janus JA, Matthijsen AJCM, Montizaan GK, Ros JPM (1989) *Integrated Criteria Document PAHs* (National Institute of Public Health and Environmental Protection, Bilthoven, The Netherlands).
32. Tao S, et al. (2004) Polycyclic aromatic hydrocarbons (PAHs) in agricultural soil and vegetables from Tianjin. *Sci Total Environ* 320:11–24.
33. Li SG, Pan DH, Wang GX (1991) Analysis of the Bap and DahA content in food oil before and after cooking process. *Shanghai Environ Sci* 10:35–36.
34. Xu SS, Liu WX, Tao S (2006) Emission of polycyclic aromatic hydrocarbons in China. *Environ Sci Technol* 40:702–708.
35. Zeng XY, Ma YT, Ma LR (2007) Utilization of straw in biomass energy in China. *Renew Sust Energy Rev* 11:976–987.
36. Zhang JF, Smith KR (2007) Household air pollution from coal and biomass fuels in China: Measurements, health impacts, and interventions. *Environ Health Perspect* 115:848–855.
37. Ma JM, Daggupaty S, Harner T, Li YF (2003) Impacts of lindane usage in the Canadian prairies on the Great Lakes ecosystem. 1. Coupled atmospheric transport model and modeled concentrations in air and soil. *Environ Sci Technol* 37:3774–3781.
38. Armstrong B, Hutchinson E, Unwin J, Fletcher T (2004) Lung cancer risk after exposure to polycyclic aromatic hydrocarbons: A review and meta-analysis. *Environ Health Perspect* 112:970–978.
39. Gibbs GW (1997) Estimating residential polycyclic aromatic hydrocarbon (PAH) related lung cancer risks using occupational data. *Ann Occup Hyg* 41:49–53.
40. Zhu GJ (2006) *Physical and Psychological Status of Chinese Population*, (Peking Union Medical College Press, Beijing), pp 205–248.