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Breathing-rate adjusted population exposure to ozone and its oxidation products in 333 cities in China

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ABSTRACT

While PM2.5 (particles with aerodynamic diameter less than 2.5 µm) concentrations in China are beginning to decline because of pollution abatement measures, ozone (O3) concentrations continue to rise. In this study, we have used a Monte Carlo approach to estimate breathing-rate adjusted (BRA) population exposure to ozone and its oxidation products based on hourly O3 measurements collected in 2017 from monitoring stations in 333 Chinese cities. The median measured outdoor O3 concentration in these cities was 31 ppb, while the median calculated indoor concentrations of ozone and ozone-derived oxidation products were 7.5 ppb and 21 ppb, respectively. The median BRA O3 exposure concentration was 12 ppb, ranging from 2.2 ppb to 18 ppb among the cities. Eastern and central cities had higher exposure concentrations, while northeastern and western cities had lower. On average, the residents of these cities spent 88% of their time indoors. Consequently, even with breathing rate adjustments, indoor O3 exposure averaged 50% of the total O3 exposure nationwide. The median BRA exposure concentration for ozone-derived products was 18 ppb, ranging from 4.5 ppb to 32 ppb among the cities. On average, BRA exposure concentrations were 1.6 times larger for oxidation products than for ozone, while seasonal variations of exposure concentrations were smaller for oxidation products than for ozone. As many of the products of indoor ozone chemistry are toxic, the health consequences of exposure to such products should be further investigated.

1. Introduction

The evidence that ozone exposure results in significant adverse health effects continues to grow. Previous studies have found that ozone exposures correlate with both morbidity and mortality (Bell et al., 2004; Bell et al., 2005; Di et al., 2017; Sun et al., 2018; Yin et al., 2017). Ozone has been shown to contribute to the risk of death from respiratory and cardiovascular disease (Jerrett et al., 2009; Turner et al., 2016; Wang et al., 2017), including chronic obstructive pulmonary disease (Huang et al., 2018a) and emphysema (Wang et al., 2019). It has been found to influence platelet activation, vascular endothelial function, and even blood pressure (Day et al., 2017; Xia et al., 2018; Zeng et al., 2017).

People spend most of their time indoors, where surfaces often have a sorbed organic film whose constituents include organic compounds that react rapidly with ozone, such as squalene, unsaturated fatty acids and their esters, terpenoids and sesquiterpenes (Weschler, 2006; Weschler and Nazaroff, 2017; Wu et al., 2017). Humans themselves are covered with a thin layer of skin surface lipids containing squalene (~10%), sapienic acid (~6%), other unsaturated fatty acids, and unsaturated triglycerides (Nicolaides, 1974). Ozone reactions with these compounds generate oxidation products, some of which may adversely affect human health (Weschler, 2000; Weschler, 2004; Weschler, 2006; Weschler et al., 2006). Studies have found that the oxidation products of terpenes are potent contact allergens and might have inflammatory and respiratory sensitizing properties (Weschler, 2006; Wolkoff et al., 2013). The oxidation products of limonene increase the rate of blinking in human subjects, indicating stimulation of trigeminal nerves (Kleno and Wolkoff, 2004). Using a head out mouse bioassay, Wolkoff et al. (2013) derived a human reference value (RF) for sensory irritation of...
0.16 ppm for 3-isopropenyl-6-oxo-heptanal (IPOH), a product of O₃/limonene chemistry, and an RF for airflow limitation of 0.03 ppm for 4-oxopentanal (4-OPA), a product of O₃/skin oil chemistry. 4-OPA has also been identified as an irritant and sensitizer using a combined local lymph node assay in a mouse model (Anderson et al., 2012). 4-OPA has also been shown to be redox active (Fu et al., 2013), and to adversely impact cellular viability (Lipsa et al., 2016). Oxidation products indoors, such as acetaldehyde, acrolein, propionaldehyde and formaldehyde may contribute to or exacerbate asthma (Leikaf, 2002). Taken together, this evidence suggests that products of ozone-initiated chemistry, as well as ozone itself, contribute to adverse health effects.

Outdoor ozone concentrations have been increasing in urban China. From 2014 to 2016, the annual-average outdoor ozone concentration increased from 87.7 µg/m³ (44.7 ppb) to 98.6 µg/m³ (50.3 ppb), while at the same time the concentrations of other gaseous pollutants (SO₂, CO and NO₂) declined (Li et al., 2017; Huang et al., 2018b). Even outdoor PM₂.₅ concentrations have begun to decrease (Li et al., 2017), reflecting various pollutant abatement measures. Taking urban stations in Beijing as an example, from 2004 to 2015, 1-h average ozone concentration increased 58% and 8-h average concentration increased 51%. During the warm season, from May to Sep, the increase of 1-h and 8-h average ozone concentrations were 64% and 41% (Cheng et al., 2018). From 2013 to 2017, in the Yangtze River Delta, Pearl River Delta and Sichuan Basin, the maximum daily 8-h average outdoor ozone concentrations increased 60%, 19% and 8%, respectively, (Lu et al., 2018).

Ozone exposure, which is defined as the product of ozone concentration in a microenvironment and the time a person spends in that microenvironment, is a useful parameter for better understanding its adverse health effects, as well as designing control strategies for both ozone and its oxidation products. Since inhalation is the main pathway by which people are exposed to ozone, breathing-rate directly influences the dose of ozone that people receive. Adjusting ozone exposures for breathing-rate during the period of exposure, provides a more accurate indicator of inhalation exposure to ozone and its oxidation products.

A number of studies have estimated ozone exposure in China using outdoor ozone concentrations (Lu et al., 2018; Sun et al., 2018; Zhan et al., 2018; Zhao et al., 2018). However, outdoor ozone concentrations fail to properly account for indoor exposure to ozone and its oxidation products. Weschler (2006) calculated that, in selected US studies, daily inhalation of indoor ozone accounted for between 25% and 60% of total daily ozone exposures. Chen et al. (2012) found that 58% of the variability in ozone mortality coefficients among eighteen National Morbidity, Mortality, and Air Pollutions Study cities could be explained by accounting for differences in total ozone exposure, and these differences resulted in large part from differences in the outdoor-to-indoor transport of ozone in these eighteen cities. Niu et al. (2018a) measured personal ozone exposure (synonymous with ozone exposure concentration) for 43 university students in Shanghai between late-May and mid-October and found that indoor ozone exposures accounted for 54% to 87% of personal ozone exposure. As part of this study, Niu et al. (2018b) proceeded to examine correlations between personal ozone exposure and respiratory inflammatory response, as indicated by exhaled nitric oxide and several biomarkers in buccal samples. The results indicated that a 10 ppb increase in ozone was significantly associated with a 3.9% increase in fractional exhaled nitric oxide (a biomarker of airway inflammation), a 36% increase in inductive nitric oxide synthase (iNOS), and a decrease in the average DNA methylation of the NOS2A gene (which encodes the iNOS protein). Significantly, “… effects were much stronger when using personal exposure monitoring than fixed-site measurements”, demonstrating the value of accounting for indoor exposures as well as outdoor exposures. Zhang et al. (2019) measured personal ozone concentrations for subjects over 50 years of age in Nanjing from Oct 31 to Nov 3; individual ozone was negatively correlated to pulmonary function data, forced vital capacity and forced expiratory volume. Xiang et al. (2019) modelled the exposure concentration of ozone in 339 Chinese cities and found that indoor exposures accounted for 59% (95% confidence interval: 26–79%) of the total ozone exposure. However, none of these previous studies attempted to account for differences in breathing rates when humans are outdoors, indoors awake, or indoors asleep; nor did any estimate indoor exposures to products of ozone-initiated reactions (i.e., indoor ozone chemistry).

In this study, we have used an evaluated Monte Carlo method to investigate the breathing-rate adjusted (BRA) population exposure to ozone and its oxidation products for 333 cities in China based on hourly measurements made throughout the year 2017. Our aims have been to estimate: (1) the indoor concentrations of ozone and ozone-derived oxidation products in these cities; (2) BRA exposure concentrations of ozone and ozone-derived oxidation products; (3) the temporal, spatial, and age distribution of BRA exposure concentrations for ozone and its oxidation products; (4) the relationship between outdoor ozone and BRA exposure concentrations of ozone; (5) the contribution of BRA indoor ozone exposure to total BRA ozone exposure; and (6) the relationship between BRA exposure concentrations of ozone and its oxidation products.

2. Materials and Methods

2.1. Calculation of BRA exposure to ozone and its oxidation products

Exposure of ozone in a given microenvironment depends on the ozone concentration in that microenvironment and the time people spent in it. Ozone concentration in an indoor environment, Cᵢ, can be calculated using Eqs. (1) and (2):

\[ Cᵢ = F_{inf} \times C_{out} \]  (1)

\[ F_{inf} = \frac{(a_{inf}f_{inf} + a_{wo}f_{op})}{a_{inf}f_{inf} + a_{wo}f_{op} + k₁ + k₂} \]  (2)

where \( F_{inf} \) is the ozone infiltration factor, defined as ozone’s indoor/outdoor ratio in the absence of indoor sources (Xiang et al., 2019), and \( C_{out} \) is the outdoor ozone concentration (ppb). In Eq. (2), \( a_{inf} \) is the air exchange rate with windows closed and \( a_{wo} \) is the air exchange rate with windows open (h⁻¹); \( f_{inf} \) and \( f_{op} \) are the fractional time windows are closed and open, respectively; \( P \) is the penetration factor for ozone, set as 1 based on our field tests in actual residences; and \( k₁ \) and \( k₂ \) are the surface removal rate constants for exposed indoor surfaces and humans, respectively (h⁻¹). The surface removal rate constant to indoor surfaces, \( k₁ \), is the product of the average deposition velocity to indoor surfaces, \( v_{sb} \), and the indoor surface to volume ratio (\( A_{human}(IS × h) \)). The surface removal rate constant to human surfaces, \( k₂ \), is the product of the average deposition velocity to human surfaces, \( v_h \), and the human surface to volume ratio (\( A_{human}(IS × h) \)) (Weschler, 2016). The deposition velocity to either indoor surfaces or human surfaces reflects mass transport across the boundary layer of air adjacent to an indoor surface as well as reaction with alkenes on the indoor surface. Details regarding how mass transport and surface kinetics affect the deposition velocity can be found in Cano-Ruiz et al. (1993).

Given that ozone oxidation products have potential health effects, we have also calculated the indoor concentration of oxidation products, \( C_{pro} \). The ratio of oxidation products to indoor ozone can be estimated using the formation factor, \( F \), for gas-phase oxidation products and the ratio of ozone’s net 1st-order surface removal rate constant to the air exchange rate (Weschler, 2006):

\[ C_{pro} \times C_{out} = F(k₁ + k₂) \]  (3)

\[ \frac{C_{pro}}{C_{out}} = \frac{F(k₁ + k₂)}{a_{inf}f_{inf} + a_{wo}f_{op}} \]  (4)

Substituting “\( F_{inf} \times C_{out} \)” for “\( C_{pro} \)” in equation (3) and rearranging yields Eq. (4):
In Eqs. (3) and (4), the formation factor, $F$, is the ratio of the moles of gas-phase products produced to moles of ozone removed by indoor surface reactions. In other words, it is the sum of the yields for all gas-phase products formed by ozone-initiated indoor chemistry. When ozone reacts with a carbon–carbon double bond, a primary ozonide is formed. This primary ozonide is unstable and cleaves to form an (i) aldehyde or ketone and (ii) a Criegee intermediate. The Criegee intermediate reacts via different pathways, generating another oxidized product (Criegee, 1975). Hence, two oxidized products are generated for each ozone molecule consumed. These products can be in either the gas-phase or condensed phase. Our focus in the present study is on gas-phase products, since they are most likely to be inhaled. While several studies report yields for specific gas-phase oxidation products derived from simulated indoor conditions, there are few studies that attempt to measure the net yield of all gas-phase species (i.e., F). Rai et al. (2014) report yields for total volatile organic compounds resulting from ozone reacting with soiled t-shirts in chamber experiments. These ranged from 0.38 to 0.54. However, the analytical methods used—sorbent sampling with Tenax TA and DNPH cartridges—likely missed some of the resulting products. Based on continuous measurements using a PTR/MS in a simulated aircraft cabin with sixteen passengers, between 0.82 and 1.0 molecules of gas-phase ozone products were formed for each molecule of outdoor ozone removed by reactions within the cabin (Weschler et al., 2007). On these basis PTR/MS measurements, we have used a formation factor, $F$, equal to 0.9 in the present study.

Inhalation is the main pathway by which people are exposed to ozone. Breathing rate, which is dependent on daily activities and age, impacts the amount of ozone and its oxidation products that we inhale. Therefore, to better estimate intake of ozone and oxidation products, different breathing-rates during different activities for different age groups were considered in the present study. A person’s daily activities were divided into three categories: light exercise (for all time outdoors), sitting quietly (used for all time indoors except sleeping), and sleeping, each with a different breathing rate. As Eqs. (5)–(7) show, the indoor and outdoor exposures were adjusted by breathing rates ($Q_{in}$, $Q_{out}$, and $Q_{day}$). With these parameters and the outdoor ozone concentration, the indoor ozone exposure ($E_{in}$) and outdoor ozone exposure ($E_{out}$) were calculated using Eqs. (5)–(8):

$$E_{in} = \frac{C_{in}(Q_{in}T_{in} + Q_{o}T_{o})}{Q_{i}}$$  \tag{5}

$$E_{out} = \frac{C_{out}Q_{out}T_{out}}{Q_{i}}$$  \tag{6}

$$Q_{i} = \frac{Q_{in}T_{in} + Q_{o}T_{o} + Q_{day}T_{day}}{T_{day}}$$  \tag{7}

$$k_2 = \sum v_{th}A_{hi}$$  \tag{8}

where:
- $Q_{day}$, $Q_{in}$, and $Q_{out}$ are breathing-rate adjusted exposure concentrations (ppb)*h; $v_{th}$ is the deposition velocity of ozone onto human surfaces (m/cm); $A_{hi}$ is the surface area of occupant ($m^2$); $S$ is the floor area of the residence where occupants live ($m^2$); $h$ is the ceiling height of the residence, which was set to 2.6 m.

Breathing-rate adjusted (BRA) exposure concentrations for ozone and its oxidation products can be expressed as Eqs. (9) and (10):

$$C_{exp,0} = \frac{E_{in} + E_{out}}{T_{day}} = \frac{Q_{in}T_{in} + Q_{day}T_{day} + Q_{out}T_{out}}{Q_{i}}$$  \tag{9}

$$C_{exp,p} = \frac{E_{in}(k_1 + k_2)F_{out}(Q_{in}T_{in} + Q_{day}T_{day} + Q_{out}T_{out})}{(v_{th}A_{hi} + v_{in}A_{hi})C_{out}T_{in} + Q_{day}T_{day} + Q_{out}T_{out}}$$  \tag{10}

$C_{exp,0}$ and $C_{exp,p}$ are breathing-rate adjusted exposure concentrations (ppb) of ozone and its oxidation products, respectively.

Note that the “breathing rate adjusted exposure concentrations”, $C_{exp,0}$ and $C_{exp,p}$, calculated using Eqs. (9) and (10) have units of ppb; these are essentially “breathing rate adjusted exposures”, $E_{in}$ and $E_{out}$, with units of ppb*h, that have been normalized by the number of hours in a day (24 h).

2.2. Monte Carlo framework and distribution of input parameters

A Monte Carlo simulation was used to estimate the BRA exposures to ozone and its oxidation products, the framework of which is shown in Fig. 1. Two types of input parameters, ten environmental parameters and three activity patterns, were included. The environmental parameters determined the ozone concentrations in different microenvironments where people were exposed. Activity patterns determined the time people spent and the volume of air people inhaled in different microenvironments. Each input parameter generates 2000 numbers based on its distribution. Vector operations with a scale of 2000 were performed, based on the equations described above, and resulted in output vectors with a scale of 2000.

The distributions for different input parameters were obtained from either previous studies or our own measurements, as shown in Table 1. The distribution of air infiltration rates was determined from published literature. Measurements had been made in residences in 4 cities (Harbin, Beijing, Shanghai and Guangzhou) located in 4 typical climate conditions.
zones of China, with 214, 277, 412 and 202 valid samples, respectively (Cheng, 2018). We had previously measured 312 natural ventilation rates in 55 different bedrooms in Beijing over four seasons and determined the distribution of these measurements (Yao and Zhao, 2019). Penetration factors (P) were set equal to unity based on our measurements in a student dormitory on the campus of Tsinghua University as part of this study. The lower quartile value was 0.83, indicating a narrow distribution. The distribution of ozone removal rates by indoor surfaces, including furniture and building material surfaces (k1), was based on our previous study, which included two repeated measurements in each of 15 bedrooms of 14 residences (Yao and Zhao, 2018). Given that the data was insufficient for statistical testing, a normal distribution was assumed, and the mean and standard deviation (SD) values were used as the distribution parameters. We determined the distribution of deposition velocities of ozone onto human surfaces from a review of the literature. Similar to k1, a normal distribution was assumed, and mean and SD values were used as the distribution parameters. Distributions of human surface areas, breathing rates, time spent outdoors and fraction of time spent outdoors for different age groups and genders in different provinces and geographic areas were taken from the Exposure factors handbook of Chinese population (Duan et al., 2013; Wang et al., 2016), which surveyed 91,121 adults and 75,490 children in mainland China. Within a province or geographic area, the distribution of these input parameters in different cities is considered to be the same. Hourly average outdoor ozone concentrations for different monitoring stations were obtained from the environmental monitoring stations. The mean and SD of daily average concentrations at different monitoring stations in a city were used as the distribution parameters for uniform distribution. Additional details regarding the distribution of all the input parameters are provided in the Supporting Information (SI).

2.3. Quality control and model evaluation

Days on which there was less than 12 h of outdoor ozone concentration data were eliminated. Daily average outdoor ozone concentrations for all monitoring stations were then calculated. In this way, we obtained daily average outdoor ozone concentrations for all cities for 357 days. For those cities within which there were at least two monitoring stations, the standard deviation for different monitoring stations was calculated. 1409 monitor stations in total were considered in this study. Among the 333 cities, two of them had only one monitoring station within the city. The standard deviation of the nearest cities was used to estimate the ozone distribution for these two cities. We selected Beijing as a representative city and, for 200 separate times, we calculated the daily exposure concentration for a random day with a loop of 2000. The Monte–Carlo error (MCE) is defined as the standard deviation of the mean values of the 2000 loops (Koehler et al., 2009). The minimum standard deviation of all of the 200 standard deviations of the 2000 loops is defined as SD. The number of loops is considered to be acceptable if the ratio of MCE to SD is less than 5% (Zhou and Zhao, 2012). The calculated MCE to SD ratio of the output (daily exposure concentrations) is 2.3%, indicating that 2000 loops are sufficient.

Personal ozone concentrations measured in Shanghai and Nanjing were used to evaluate the model. To be consistent with the study population and time of year of the field studies, in the Shanghai comparison we calculated the ozone exposure concentration for people 18–44 years old during the period from 29 May to 12 Oct; in the Nanjing comparison we calculated the ozone exposure concentration for people 45–79 years old during 31 Oct to 3 Nov. Further details regarding the cases used for model evaluation, as well as the parameter settings for the modeling are shown in Table S12. The results indicated that the modelled ozone exposure concentrations agreed reasonably well with the measured ones, as shown in Fig. S1 in the SI.

3. Results and discussion

3.1. Indoor concentrations of ozone and its oxidation products

Fig. 2(a) shows the distribution for annual average of daily indoor concentrations of ozone, Cin, and its gas-phase oxidation products, Cgas, for 333 Chinese cities in 2017. City-by-city results, including measured annual average outdoor concentrations, are listed in Table S13. Among these cities, the median (mean) value for outdoor O3 concentration was 31 ppb (30 ppb), while indoor ozone was 7.5 (7.2) ppb, and the median (mean) value for its products was 21.3 (20.8) ppb. The estimated concentration of oxidation products was approximately 2.8 times larger than the measured concentration of indoor ozone. Fig. 2(b) shows a scatter plot of the annual average of daily concentrations of indoor ozone and annual average of daily concentrations of oxidation products in the 333 cities. The scatter is large and bounded by $C_{gas}/C_{in}$ of 1.8 on the low end, 7.8 on the high end, with a central tendency of 2.8. The

### Table 1

Sources and distributions for different input parameters.

| Input parameter | Distribution | Distribution parameters | Source |
|-----------------|--------------|-------------------------|--------|
| **Environmental parameters** | αin | Log-normal | Median (Geometric standard deviation (GSD)): 1.8 (0.38) h^{-1} | Cheng (2018) |
| P | Fixed | 1 | | Field test by authors |
| $k_1$ | Normal | Mean (SD): 2.8 (1.1) h^{-1} | | Measured by authors (Yao and Zhao, 2018) |
| $\alpha_{in}$ | Log-normal | Median (GSD): 5.73 (0.87) h^{-1} | | Measured by authors (Yao and Zhao, 2019) |
| $\alpha_{wo}$ | Log-normal | Median (GSD): 2.44 (1.01) h^{-1} | | Measured by authors (Yao and Zhao, 2018) |
| $\alpha_{ww}$ | Log-normal | Median (GSD): 0.83 (0.87) h^{-1} | | Measured by authors (Yao and Zhao, 2019) |
| $\alpha_{wo}$ | Log-normal | Median (GSD): 5.73 (0.87) h^{-1} | | Measured by authors (Yao and Zhao, 2019) |
| $\alpha_{ww}$ | Log-normal | Median (GSD): 2.44 (1.01) h^{-1} | | Measured by authors (Yao and Zhao, 2018) |
| $\alpha_{ww}$ | Log-normal | Median (GSD): 0.83 (0.87) h^{-1} | | Measured by authors (Yao and Zhao, 2019) |

* Monte Carlo simulations were also conducted using a skew distribution for outdoor ozone concentrations. There was little difference between annual average exposure concentrations of ozone and its oxidation products calculated from uniform- and Gamma-distributed outdoor ozone concentrations (Fig. S3). See SI for further details.

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**Note:** The table provides a summary of the input parameters, their distributions, and the sources of these distributions. The distributions are specified for various environmental and activity parameters, with details on the methods used for determining these distributions. The table is part of a comprehensive study on the exposure concentrations of ozone and its oxidation products in indoor environments across China, considering both indoor and outdoor concentrations and the impacts of varying environmental conditions and activities.
scatter reflects substantial variation in certain input parameters (e.g., air exchange rate and rate constant for O₃ removal by occupants) from city to city.

The oxidation products are derived chiefly from surface reactions (Weschler, 2000; Yao and Zhao, 2018). As discussed in Section 2.1, we have assumed that 0.9 molecules of gas-phase products are formed for each molecule of outdoor ozone removed by reaction with indoor surfaces (i.e., \( F = 0.9 \)). As discussed in Weschler (2006) and shown in Eq. (3), the ratio of oxidation products to indoor ozone can be estimated as \( F \) times the ratio of ozone’s net 1st-order surface removal rate constant to the air exchange rate. The mean value for the surface removal rate constant, \( k_1 \), in Chinese residences is measured to be 2.8 h⁻¹ (Yao and Zhao, 2018), while the mean value for removal by the surfaces of one human body, \( k_2 \), as calculated for the different cities in this study, is 0.32 h⁻¹. The average air exchange rate when a residence has open windows or closed windows and the fraction of time windows are open or closed is listed in Table 1. In Chinese residences, the ratio of the rate constant for net surface removal of ozone (\( k_1 + k_2 \)) to average ventilation rate (\( a_{vent,O3} + a_{vent,op} \)) is large – typically between two and five. Hence, given a formation factor of 0.9, the ratio of the concentration of gas-phase products to that of indoor ozone is in the range of three, with considerable variability, as illustrated in Fig. 2(a) and (b).

3.2. Spatial distribution of BRA ozone and oxidation product exposure concentrations

The breathing-rate adjusted exposure concentrations for ozone, \( C_{exp,O3} \), and for its oxidation products, \( C_{exp,p} \), have been calculated for each of the 333 cities using Eqs. (9) and (10), respectively (Table S13). Fig. 3(a) shows the spatial distribution of annual average BRA exposure concentrations (ppb) for ozone, and Fig. 3(b) shows this for its oxidation products. Among the different cities, the annual average BRA ozone exposure concentration ranged from 2.2 ppb (Haixi Mongolian and Tibetan Autonomous Prefecture) to 18.0 ppb (Alxa League), with a median value of 12.0 ppb. Generally, northeastern and west China had relatively lower BRA ozone exposure concentrations, while eastern and central China had relatively higher BRA ozone exposure concentrations. The outdoor ozone concentration, infiltration factor and daily activity pattern were the factors that most influenced the ozone exposure.
concentrations. In this study, we used measured outdoor ozone concentrations for each city. In contrast, the infiltration factor, which is a function of air change rate, surface removal rates and penetration factor, was fixed for different cities within one province. The same was true for daily activity patterns. The strong influence of the outdoor ozone concentration on the ozone exposure concentration is illustrated in the case of Alxa League, a southern city in Inner Mongolia, and Hulun Buir, a northern city in Inner Mongolia (see the red stars in Fig. 3(a)). Although exposure concentrations for these two cities were calculated with the same infiltration factor and residents’ activity pattern, their annual average ozone exposure concentrations were substantially different, reflecting large differences in outdoor ozone concentrations. The influence of infiltration factor and residents’ activity pattern is illustrated by Jilin compared to Guangdong Province (see the areas marked with black circles). The annual average of daily outdoor ozone concentrations were quite similar in these two provinces, 29.7 and 29.1 ppb, but the annual average BRA ozone exposure concentrations were much higher in Guangdong. This was because the people in Guangdong Province tended to stay outdoors longer than people in Jilin Province, and the average ventilation rate in Guangdong Province (0.38 h⁻¹) was higher than that in Jilin Province (0.24 h⁻¹).

The annual average BRA exposure concentrations of ozone oxidation products ranged from 4.5 to 31.9 ppb, with a median value of 17.7 ppb. As was the case for ozone, Alxa League had the highest annual average (31.9 ppb) and Haixi Mongol and Tibetan Autonomous Prefecture had the lowest (4.5 ppb). Northeastern China tends to have higher oxidation product exposures than southeastern China, a trend that was not seen for ozone itself. BRA oxidation product exposure concentration is influenced most strongly by outdoor ozone concentration coupled with the fraction of time people spend indoors. The more time people spend indoors, the higher the BRA oxidation product exposure concentration, which is opposite the relationship for the ozone exposure concentration. Hence, time spent outdoors explains situations where, for some cities in Liaoning and Guangzhou Provinces, there was high BRA ozone exposure concentration and low BRA product exposure concentration. For cities with high outdoor ozone concentrations, outdoor ozone concentration had a large influence on product exposure concentration. In such cases, cities with higher BRA ozone exposure concentrations tended to have higher BRA product exposure concentrations, which is observed among certain cities in provinces such as Inner Mongolia, Gansu and Tibet (see the areas marked with red circles). BRA ozone exposure concentrations are compared to the BRA product exposure concentrations, city by city, in Table S13.

### 3.3. BRA exposure concentrations for ozone and its oxidation products in different geographic areas and temperature periods; differences among age groups

To examine the impact of ambient temperature on exposure concentrations, we have divided the year into three periods: cool, air temperatures lower than 10 °C; transition, higher than 10 °C and lower than 22 °C; and warm, higher than 22 °C. To be classified as a cool period, transition period, or warm period, the criteria had to be met for five consecutive days; the first of these five days was viewed as the start of cool periods, warm periods or transition periods. Fig. 4(a) shows the BRA ozone exposure concentrations in different geographic areas during these three temperature periods. For all geographic areas, BRA ozone exposure concentrations were highest in warm periods (median ~ 15 ppb), followed by the transition periods (median ~ 10 ppb), and the cool periods (median ~ 5 ppb). The pattern reflected the pattern for the outdoor ozone concentrations. In warm periods, northern areas, such as the HB and DB areas, tended to have higher BRA ozone exposure concentrations than other areas. Although outdoor ozone concentrations in southern areas were higher than in northern areas (due to higher air temperatures and stronger sunlight), windows were opened more in northern areas compared to southern areas. During warm periods, air conditioners and cooling systems are widely used in southern China, and windows were closed more often than in the north. Furthermore, extremely hot weather in southern areas during the summer decreased the time spent outdoors. Hence, the difference between northern and southern areas during warm periods is reinforced by lower indoor ozone concentrations and less time outdoors in the south. During transition periods and cool periods, differences between geographic areas are less pronounced.

Fig. 4(b) shows the BRA product exposure concentrations in different geographic areas during the three temperature periods. Product exposure concentrations in the warm, cool, and transition periods did not vary from one another as much as they did for ozone. This is partially explained by the fact that the influence of high ozone concentrations in warm periods was somewhat neutralized by less time spent indoors. A similar counterbalance occurred in cool periods – although outdoor ozone concentrations were lower in cool periods, people spent more time indoors, increasing their exposure to ozone oxidation products. Such counterbalancing resulted in relatively stable
BRA product exposure concentrations from warm to transition to cool temperature periods. The median value of BRA exposure concentrations in the three periods was approximately 15 ppb. During warm periods, BRA product exposure concentrations were similar to BRA ozone exposure concentrations in Fig. 4(a) (~15 ppb on average). During cool periods, BRA product exposure concentrations were roughly three times larger than BRA ozone exposure concentrations (~15 ppb vs. ~5 ppb). During transition periods, BRA product exposure concentrations were roughly 1.5 times larger than BRA ozone exposure concentrations (~15 ppb vs. ~10 ppb).

The DB area had higher BRA product exposure concentrations than the other areas during the cool period. Given that outdoor ozone concentrations were low during cool periods, differences in outdoor ozone concentrations among different geographic areas were not as impactful as the differences in time spent outdoors. The extremely cold weather during “cool periods” in the DB area led to longer time spent indoors, which resulted in lower BRA exposure concentrations for ozone and higher BRA exposure concentrations for oxidation products than in the other areas.

To probe the differences in exposure concentrations among different age groups, we used Beijing as an example. Fig. 5 shows the distribution of BRA ozone exposure concentrations for different age groups in Beijing. The median value of the BRA ozone exposure concentration ranged from 6.0 ppb for 0–5 months to 9.4 ppb for 45–59 years; the maximum exposure concentration was 43.5 ppb (45–59 years old). People between 18 and 79 years of age had higher BRA exposure concentrations than other age groups. Time spent outdoors was the main reason for the difference. The breathing rate for different age groups also contributes to the difference. Age did not have as large an impact on exposure concentrations as did temperature period or geographic region.

### 3.4. The relationship between outdoor ozone concentrations and BRA ozone exposure concentrations

The ratio of the BRA ozone exposure concentration to the outdoor ozone concentration ($C_{\text{exp,O3}}/C_{\text{out}}$) can be expressed as:

$$\frac{C_{\text{exp,O3}}}{C_{\text{out}}} = \frac{T_{\text{day}}[F_{\text{out}}(Q_{\text{in}}, T_{\text{in}}) + Q_{\text{out}} T_{\text{out}}]}{Q_{\text{in}} T_{\text{in}} + Q_{\text{out}} T_{\text{out}}}$$

(11)

Fig. 6 shows a scatter plot of $C_{\text{exp,O3}}$ versus $C_{\text{out}}$. The slope of the least squares regression line (i.e., $C_{\text{exp,O3}}/C_{\text{out}}$) is 0.35 with $r^2 = 0.62$. The lower and upper bounds are 0.18 and 0.49. This finding is in remarkably good agreement with our own breathing rate adjusted results.

Another metric, related to the ratio of the ozone exposure concentration to the outdoor ozone concentration, is the percent contribution of indoor ozone exposure concentration to total daily ozone exposure concentration. Fig. 7 shows the fractional contribution of BRA ozone exposure concentration to total daily ozone exposure concentration for six large cities in 2017. For each city, breathing-rate adjusted averages are compared to non-breathing-rate adjusted averages. Also shown are the breathing-rate adjusted nationwide average for the fractional contribution calculated in the present study and the non-breathing-rate adjusted nationwide average for the fractional contribution calculated by Xiang et al. (2019).
indoor ozone exposure to total daily BRA ozone exposure both nationwide and for six typical cities, each representing one of the different climate regions of China used in this study. On average, the breathing-rate-adjusted indoor ozone exposure contributes 50.1% to total exposure for all 333 cities; among the six cities Xian had the lowest fractional contribution (43%) and Guangzhou the highest (55%). Indoor ozone exposure is substantial due to the large fraction of time people spend indoors, which roughly balances the fact that indoor ozone concentrations are much lower than outdoor ozone concentrations (due in large part to reactions on indoor surfaces).

Fig. 7 also compares breathing-rate adjusted and non-breathing rate adjusted estimates of the percent contributions of indoor ozone exposure to total ozone exposure for the six typical cities. The contributions of BRA indoor ozone exposure were roughly 7% lower than non-BRA indoor exposures, reflecting higher breathing-rates when people were outdoors. Finally, Fig. 7 compares the nationwide average of the indoor contribution estimated in the present study to the nationwide average of the indoor contribution estimated in the study by Xiang et al. (2019), which did not adjust for breathing rates in different environments. Nationwide, the percent contribution of BRA indoor ozone exposure to total daily BRA ozone exposure was 50.1% in the present study using measurements from 2017 compared to 59.0% in the study by Xiang et al. (2019) that did not adjust for breathing rates and used measurements from 2015 – reasonable agreement, given BRA versus non-BRA.

3.5. The products of ozone-initiated indoor chemistry

Fig. 8 shows that when BRA product exposure concentrations are plotted against BRA ozone concentrations, the slope ($C_{\text{exp,p}}/C_{\text{exp,O3}}$) has a central tendency of 1.6 and is bounded between 1.0 and 3.9. This slope indicates that, on average, for every 1 ppb of ozone that is inhaled, 1.6 ppb of ozone oxidation products are inhaled. Mathematically, the ratio of BRA exposure concentrations for products/ozone can be calculated as:

$$
C_{\text{exp,p}}/C_{\text{exp,O3}} = \frac{\text{(products inhaled indoors)}}{(O_3 \text{inhaled outdoors} + O_3 \text{inhaled indoors})}
$$

(12)

where the terms on the right side of this equation can be calculated from Eqs. (9) and (10). The sum of the concentrations of ozone oxidation products indoors is substantially larger than outdoors (Xiang et al., 2019). Eq. (12) captures the fact that ozone exposure occurs both outdoors and indoors, while product exposure is predominantly indoors. The ratio of product exposure concentration/ozone exposure concentration (central tendency $\sim 1.6$; Fig. 8) is lower than the ratio of indoor product concentration/ozone exposure concentration (central tendency $\sim 2.8$; Fig. 2), because time outdoors contributes to ozone exposure (nationwide average of 50%), but not to product exposure.

Fig. 8 drives home the point that, when considering the health impacts of exposure to ozone, we should also consider the health impacts of exposure to ozone-derived oxidation products. The reduction in ozone concentration that occurs as ozone is transported from outdoors to indoors is primarily due to reactions between ozone and chemicals on indoor surfaces. These reactions generate products, counter-balancing the decrease in the concentration of ozone itself (Weschler, 2000; Weschler and Carslaw, 2018). The chemicals on indoor surfaces that react fastest with ozone contain carbon–carbon double bonds. When ozone reacts with a double bond, a primary ozonide is formed. Subsequent reactions can produce aldehydes, ketones, carboxylic acids, hydroperoxides, $\alpha$-hydroxyhydroperoxides, and secondary ozonides (Criegee, 1975; Zhou et al., 2016a). A number of these resulting oxidation products may contribute to morbidity and mortality. In a 2006 paper, Weschler addressed this concern and included a table that listed numerous references reporting oxidation products resulting from ozone reacting with chemicals on indoor surfaces (Weschler, 2006). Since then, there have been significant advances in analytical methods that can detect ozone oxidation products (Farmer et al., 2019). In the period between 2007 and 2019, more than 35 additional studies have been published that identify products formed when ozone reacts with chemicals commonly found on indoor surfaces. (For a full listing of these papers, see Table S14). Worthy of special comment are the products formed close to the breathing zone when ozone reacts with surface lipids on occupants’ exposed skin, hair and clothing (Pandrangi and Morrison, 2008; Rim et al., 2009; Weschler et al., 2007; Wisthaler and Weschler, 2010). These products include carbonyls, dicarbonyls, hydroxycarbonyls, carboxylic acids, oxocarboxylic acids, and dicarbocyclic acids (Coleman et al., 2008; Lakey et al., 2016; Rai et al., 2014; Sun et al., 2012; Weschler, 2016; Zhou et al., 2016b). Squalene, a major constituent of human skin oil, has received special attention (Fu et al., 2013; Heine et al., 2017; Lakey et al., 2019; Petrick and Dubowski, 2009; Wells et al., 2008; Zhou et al., 2016a). In a 2012 paper, Anderson et al., identified 4-oxopentalan (4-OPA), a product of O$_3$/squalene chemistry, as an irritant; pulmonary exposure in mice was found to cause "... a significant elevation in nonspecific airway hyperreactivity, increased numbers of lung-associated lymphocytes and neutrophils, and increased interferon-g production by lung-associated lymph node cells". Lipsa et al. (2016) have shown that 4-OPA reduces the viability of human bronchial epithelial (16HBE14o-) cells. Using a bioassay, Fu et al. (2013) found that certain products of squalene oxidation were redox-active and concluded: "... it is conceivable that enhancements in hydrophilicity and redox activity from squalene upon ozone exposure could pose a higher health risk for human beings in an indoor environment."

While the toxicity of ozone has been extensively investigated, the net toxicity of the products of ozone-initiated indoor chemistry has received little attention. We know that certain oxidation products can be toxic (e.g., formaldehyde, acetaldehyde, methacrolein, 4-oxopentalan) and anticipate that others likely are (e.g., stabilized Criegee intermediates, secondary ozonides, hydroperoxides, ester hydroperoxides, epoxydes and epoxide diols). However, the health impacts of exposure to the total mixture of oxidation products, whose concentration increases when the concentration of outdoor ozone increases, are unknown. We consider additional investigation of this topic to be extremely important.

4. Conclusion

To our knowledge, this is the first study that has estimated exposure concentrations (i.e., personal exposure) for both ozone and its oxidation products.
products. Our Monte Carlo calculated exposures are based on hourly ozone measurements (year 2017) at the 1409 monitoring stations in 333 Chinese cities. The exposure concentrations have been adjusted for breathing rates while outdoors, indoors awake, and indoors asleep. Adjusting for breathing rate reduces the estimated total ozone inhaled by approximately 7% compared to estimations made without such adjustments. This difference reflects lower breathing rates when people are indoors, where urban residents spend about 88% of their days.

Our results indicate that breathing rate adjusted (BRA) ozone exposure, accounting for fractional time outdoors and indoors, was just 35% of exposures based solely on outdoor ozone concentrations. Ozone exposure varied with both season (i.e., temperature) and geographic region. Outdoor ozone concentrations and time spent outdoors (versus indoors) were significant drivers for these differences. Of special note is the observation that net ozone inhalation was largest during warm periods, followed by the transition periods, and smallest during the cold periods. In contrast, inhalation of ozone-derived products was similar during all three temperature periods.

Nationally, indoor inhalation of ozone accounts for approximately 50% of total ozone inhalation. The fractional contribution of indoor inhalation varied from city to city (e.g., 43% in Xian compared to 55% in Guangzhou) due to differences in air exchange rates and fraction of time spent outdoors. Inhalation of the sum of ozone-derived oxidation products was estimated to be, on average, 60% larger than inhalation of ozone. While ozone’s toxicity is well documented, toxicity information on its oxidation products is limited (see Introduction and Section 3.5).

The health consequences of exposure to products of indoor ozone chemistry should be more extensively investigated, since these products may be contributing to ozone’s documented impact on morbidity and mortality.

CRediT authorship contribution statement

Mingyao Yao: Conceptualization, Software, Data curation, Writing - original draft. Charles J. Weschler: Conceptualization, Formal analysis, Writing - review & editing. Bin Zhao: Conceptualization, Methodology, Supervision, Formal analysis, Funding acquisition, Writing - review & editing. Lin Zhang: Investigation. Rui Ma: Investigation.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2020.105617.

References

Anderson, S.E., Franco, J., Jackson, L.G., Wells, J.R., Ham, J.E., Meade, B.J., 2012. Irritancy and allergic responses induced by exposure to the indoor air chemical 4-oxopentanal. Toxicol. Sci. 127, 371–381.
Bell, M.L., Dominici, F., Samet, J.M., 2005. A meta-analysis of time-series studies of ozone and mortality with comparison to the national morbidity, mortality, and air pollution study. Epidemiology 16, 436–445.
Bell, M.L., McDermott, A., Zeger, S.L., Samet, J.M., Dominici, F., 2004. Ozone and short-term mortality in 95 US urban communities, 1987–2000. JAMA 292, 2372–2378.
Cano-Ruiz, J.A., Kong, D., Balas, R.B., Nazaroff, W.W., 1993. Removal of reactive gases at indoor surfaces - combining mass-transf and surface kinetics. Atmos Environ. A-Gen. 27, 2093–2050.
Chen, C., Zhao, B., Weschler, C.J., 2012. Assessing the influence of indoor exposure to “outdoor ozone” on the relationship between ozone and short-term mortality in U.S. communities. Environ. Health Perspect. 120, 235–240.
Cheng, N., Chen, Z., Sun, F., Sun, R., Dong, X., Xie, X., Xu, C., 2018. Ground ozone concentrations over Beijing from 2004 to 2015: Variance patterns, indicative precursors and effects of emission-reduction. Environ. Pollut. 237, 262–274.
Cheng, P.L., 2018. Natural Ventilation Rate Distribution in Dwellings in China’s 4 Major Cities. Tsinghua University.
Coleman, B.K., Destaillets, H., Hodgson, A.T., Nazaroff, W.W., 2008. Ozone consumption and volatile byproduct formation from surface reactions with aircraft cabin materials and clothing fabrics. Atmos. Environ. 42, 642–654.
Criegee, R., 1975. Mechanism of ozonolysis. Angew. Chem. Int. Ed. 14, 745–752.
Day, D.R., Xiang, J.B., Mo, J.H., Li, F., Chung, M.K., Gong, J.C., Weschler, C.J., Ohman-Strickland, P.A., Sundell, J., Weng, W.G., Zhang, Y.P., Zhang, J.F., 2017. Association of ozone exposure with cardiorespiratory pathophysiological mechanisms in healthy adults. JAMA Intern. Med. 177, 1344–1353.
Di, Q., Wang, Y., Zanobetti, A., Wang, Y., Koutrakis, P., Choirat, C., Dominici, F., Schwartz, J.D., 2017. Air pollution and mortality in the medicare population. N. Engl. J. Med. 376, 2513–2522.
Duan, X., Zhao, X., Wang, B., Chen, Y., Cao, S., 2013. Exposure Factors Handbook of Chinese Population (Adults) (in Chinese). China Environmental Science Press, Beijing.
Fadey, M.O., Weschler, C.J., Tham, K.W., Wu, W.Y., Sultan, Z.M., 2013. Impact of human prevailed organic aerosol derived from ozone-initiated chemistry in a simulated office environment. Environ. Sci. Technol. 47, 9933–9941.
Farmer, D.K., Vance, M.E., Abbatt, J.P.D., Abeleira, A., Alves, M.R., Arata, C., Boedicker, E., Bourne, S., Cardoso-Saldana, F., Corsi, R., DeCarlos, F.P., Goldstein, A.H., Grassian, V.H., Raat, J.H., Jimer, J.H., Kohan, T.F., Kate, E.P., Mattila, J.M., Nazaroff, W.W., Novoselov, A., Olbrien, R.E., Or, V.W., Patel, S., Sankhyan, S., Stevens, P.S., Tian, Y., Wade, M., Wang, C., Zhou, S., Zhou, Y., 2019. Overview of HOMChem: House observations of microbial and environmental chemistry. Environ. Sci.-Process. & Impact. 21, 1280–1300.
Fischer, A., Liangstrom, E., Langer, S., 2013. Ozone removal by occupants in a classroom. Atmos. Environ. 81, 11–17.
Fu, D., Leng, C., Kelley, J., Zeng, G., Zhang, Y., Liu, Y., 2013. ATR-IR Study of ozone initiated heterogeneous oxidation of squalene in an indoor environment. Environ. Sci. Technol. 47, 10611–10618.
Heine, N., Houle, F.A., Wilson, K.R., 2017. Connecting the elementary reaction pathways of Criegee intermediates to the chemical erosion of squalene interfaces during ozonolysis. Environ. Sci. Technol. 51, 13740–13746.
Huang, J., Li, G., Xu, G., Qian, X., Zhao, Y., Pan, X., Huang, J., Cen, Z., Liu, Q., He, T., Guo, X., 2018a. The burden of ozone pollution on years of life lost from chronic obstructive pulmonary disease in a city in Yangtze River Delta, China. Environ. Pollut. 242, 1266–1273.
Huang, J., Pan, X., Guo, X., Li, G., 2018b. Health impact of China’s Air Pollution Prevention and Control Action Plan: an analysis of national air quality monitoring data. Lancet Planet. Health 2, e313–e323.
Jerrett, M., Burnett, R.T., Pope, C.A., Ito 3rd, K., Thurston, G., Krewski, D., Shi, Y., Calle, E., Thun, M., 2009. Long-term ozone exposure and mortality. N. Engl. J. Med. 360, 1085–1095.
Kleno, J., Wolkoff, P., 2004. Changes in eye blink frequency as a measure of trigeminal stimulation by exposure to limonene oxidation products, isoprene oxidation products and nitrate radicals. Int. Arch. Occup. Environ. Health. 77, 235–243.
Koehler, E., Brown, E., Haneuse, S.J., 2009. On the assessment of Monte Carlo error in simulation-based statistical analyses. Am. Statist. 63, 155–162.
Lakey, P.S., Wishalter, A., Berkemeier, T., Mikoviny, T., Poschl, U., Shiraiwa, M., 2016. Chemical kinetics of multiphase reactions between ozone and human skin lipids: implications for indoor air quality and health effects. Indoor Air 27, 816–828.
Lakrye, P.S.J., Morrison, G.C., Won, Y., Parry, K.M., van Damoors, M., Tobias, D.J., Rim, D., Shiraiwa, M., 2019. The impact of clothing on ozone and squalene ozonolysis products in indoor environments. Commun. Chem. 2, 56.
Leikauf, G.D., 2002. Hazardous air pollutants and asthma. Environ. Health Perspect. 110, 505–526.
Li, R., Cai, L., Li, J., Zhao, A., Fu, H., Wu, Y., Zhang, L., Kong, L., Chen, J., 2017. Spatial and temporal variation of particulate matter and gaseous pollutants in China during 2014–2016. Atmos. Environ. 161, 235–246.
Lipso, D., Leva, P., Barrero-Moreno, J., Gedhan, M., 2016. Inflammatory effects induced by selected limonene oxidation products: 4-OPA, IPOH, 4-AMCH in human bronchial (16HBE14-) and alveolar (A549) epithelial cell lines. Toxicol. Lett. 262, 70–79.
Lu, X., Hong, J., Zhang, L., Cooper, O.R., Schultz, M.G., Xu, X., Wang, T., Gao, M., Zhao, Y., Zhang, Y., 2018. Severe Surface Ozone Pollution in China: A Global Perspective. Environment Intern. 138, 2019. 105617.
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Pandrangi, L.S., Morrison, G.C., 2008. Ozone interactions with human hair: Ozone uptake rates and product formation. Atmos. Environ. 42, 5079–5089.

Petrick, L., Dubowski, Y., 2009. Heterogeneous oxidation of squalene film by ozone under various indoor conditions. Indoor Air 19, 381–391.

Rai, A.C., Guo, B., Lin, C.H., Zhang, J., Pei, J., Chen, Q., 2014. Ozone reaction with clothing and its initiated VOC emissions in an environmental chamber. Indoor Air 24, 49–58.

Rim, D., Novoselec, A., Morrison, G., 2009. The influence of chemical interactions at the human surface on breathing zone levels of reactants and products. Indoor Air 19, 324–334.

Sun, C., Zhao, Y.-Y., Curtis, J.M., 2012. A study of the ozonolysis of model lipids by electrospay ionization mass spectrometry: Rapid Commun. Mass Spectrom. 26, 921–930.

Sun, Q., Wang, W., Chen, C., Ban, J., Xu, D., Zhu, P., He, M.Z., Li, T., 2018. Acute effect of multiple ozone metrics on mortality by season in 34 Chinese counties in 2013–2015. J. Intern. Med. 283, 481–488.

Tamas, G., Weschler, C.J., Bako-Biro, Z., Wyon, D.P., Strom-Tejsen, P., 2006. Factors affecting ozone removal rates in a simulated aircraft cabin environment. Atmos. Environ. 40, 6122–6133.

Turner, M.C., Jerrett, M., Pope, C.A., Krewski 3rd, D., Gapstur, S.M., Diver, W.R., Beckerman, B.S., Marshall, J.D., Su, J., Crouse, D.L., Burnett, R.T., 2016. Long-term ozone exposure and mortality in a large prospective study. Am. J. Respir. Crit. Care Med. 193, 1134–1142.

Wang, B., Duan, X., Zhao, X., 2016. Exposure Factors Handbook of Chinese Population Exposures to Ozone and ProductsofOzone-initiatedchemistry. Environ. Health Perspect. 114, 1489–1496.

Weschler, C.J., 2006. Ozone’s impact on public health: contributions from indoor carbonyls, dicarbonyls, and hydroxycarbonyls in indoor air. Proc. Natl. Acad. Sci. USA 107, 6568–6575.

Wolkoff, P., Larsen, S.T., Hammer, M., Kofod-Sorensen, V., Clausen, P.A., Nielsen, G.D., 2013. Human reference values for acute airway effects of five common ozone-initiated terpene reaction products in indoor air. Toxicol. Lett. 216, 54–64.

Wu, Y., Eichler, C.M., Leng, W., Cox, S.S., Marr, I.C., Little, J.C., 2017. Adsorption of Phthalates on Impervious Indoor Surfaces. Environ. Sci. Technol. 51, 2907–2913.

Xiu, Y., Niu, Y., Cai, J., Liu, Z., Liu, C., Li, H., Chen, C., Song, W., Zhao, Z., Chen, R., Kan, H., 2018. Effects of personal short-term exposure to ambient ozone on blood pressure and vascular endothelial function: a mechanistic study based on DNA methylation and metabolomics. Environ. Sci. Technol. 52, 12774–12782.

Xiang, J., Weschler, C.J., Zhang, J., Zhang, L., Sun, Z., Duan, X., Zhang, Y., 2019. Ozone in urban China: Impact on mortalities and approaches for establishing indoor guideline concentrations. Indoor Air 29, 604–615.

Yao, M., Zhao, B., 2019. Distribution of air change rates in residential buildings in Beijing, China. Proc. Eng (in press).

Yao, M.Y., Zhao, B., 2018. Surface removal rate of ozone in residences in China. Build. Environ. 142, 101–106.

Yin, P., Chen, R., Wang, L., Meng, X., Liu, C., Niu, Y., Lin, Z., Liu, Y., Liu, J., Qi, J., You, J., Zhou, M., Kan, H., 2017. Ambient ozone pollution and daily mortality: a nationwide study in 272 Chinese cities. Environ. Health Perspect. 125.

Zeng, X.-W., Qian, Z., Vaughan, M.G., Nelson, E.J., Dharmage, S.C., Bowatte, G., Perret, J., Chen, D.-H., Ma, H., Lin, S., de Foy, B., Hu, L.-W., Yang, B.Y., Xu, S.-L., Zhang, C., Tian, Y.-P., Nian, M., Wang, J., Xiao, X., Sao, W.-W., Zhang, Y.-Z., Dong, G.-H., 2017. Positive association between short-term ambient air pollution exposure and children blood pressure in China Result from the Seven Northeast Cities (SNEC) study. Environ. Pollution. 224, 698–705.

Zhan, Y., Luo, Y., Deng, X., Griesenmei, M.L., Zhang, M., Di, B., 2018. Spatiotemporal prediction of daily ambient ozone levels across China using random forest for human exposure assessment. Environ. Pollut. 233, 464–473.

Zhang, J., Sun, H., Chen, Q., Gu, J., Ding, Z., Xu, Y., 2019. Effects of individual ozone exposure on lung function in the elderly: a cross-sectional study in China. Environ. Sci. Pollut. Res. Int. 26, 11690–11695.

Zhao, X., Cheng, H., He, S., Cui, X., Pu, X., Lu, L., 2018. Spatial associations between social groups and ozone air pollution exposure in the Beijing urban area. Environ. Res. 164, 173–183.

Zheng, B., Lin, L., Yu, C., Lyu, J., Guo, Y., Bian, Z., Tan, Y., Pei, P., Chen, J., Chen, Z., Li, L., 2017. Distributions and associations between duration of sleep, daytime naps and insomnia symptoms among Chinese adults. Chin. J. Epidemiol. 38, 452–456.

Zhou, B., Zhao, 2012. Population inhalation exposure to polycyclic aromatic hydrocarbons and associated lung cancer risk in Beijing region: contributions of indoor and outdoor sources and exposures. Atmos. Environ. 62, 472–480.

Zhou, S., Forbes, M.W., Abbott, J.P., 2016a. Kinetics and products from heterogeneous oxidation of squalene with ozone. Environ. Sci. Technol. 50, 11688–11697.

Zhou, S., Forbes, M.W., Katrith, Y., Abbott, J.P., 2016b. Rapid oxidation of skin oil by ozone. Environ. Sci. Technol. Lett. 3, 170–174.