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The impact of changing exposure to PM$_{2.5}$ on mortality for US diplomats with multiple international relocations: a modelling study

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Abstract

Background Current evidence linking long-term exposure to fine particulate matter (PM$_{2.5}$) exposure and mortality is primarily based on persons that live in the same residence, city and/or country throughout the study, with few residential moves or relocations. We propose a novel method to quantify the health impacts of PM$_{2.5}$ for United States (US) diplomats who regularly relocate to international cities with different PM$_{2.5}$ levels.

Methods Life table methods were applied at an individual-level to US mortality statistics using the World Health Organization's database of city-specific PM$_{2.5}$ annual mean concentrations. Global Burden of Disease concentration-response (C-R) functions were used to estimate cause-specific mortality and days of life lost (DLL) for a range of illustrative 20-year diplomatic assignments for three age groups. Time lags between exposure and exposure-related mortality risks were applied. Sensitivity analysis of baseline mortality, exposure level, C-R functions and lags was conducted. The effect of mitigation measures, including the addition of air purifiers, was examined.

Results DLL due to PM$_{2.5}$ exposure for a standard 20-year assignment ranged from 0.3 days for diplomats' children to 84.1 days for older diplomats. DLL decreased when assignments in high PM$_{2.5}$ cities were followed by assignments in low PM$_{2.5}$ cities: 162.5 DLL when spending 20 years in high PM$_{2.5}$ cities compared to 62.6 DLL when spending one of every four years (5 years total) in a high PM$_{2.5}$ city for older male diplomats. Use of air purifiers and improved home tightness in polluted cities may halve DLL due to PM$_{2.5}$ exposure. The results were highly sensitive to lag assumptions: DLL increased by 68% without inception lags and decreased by 59% without cessation lags for older male diplomats.

Conclusion We developed a model to quantify health impacts of changing PM$_{2.5}$ exposure for a population with frequent relocations. Our model suggests that alternating assignments in high and low PM$_{2.5}$ cities may help reduce PM$_{2.5}$-related mortality burdens. Adding exposure mitigation at home may help reduce PM$_{2.5}$ related mortality. Further research on outcome-specific lag structures is needed to improve the model.

Keywords Mortality, Death, Relocation, Movers, Diplomats, Model, Cessation lag, Particulate matter, PM$_{2.5}$, Mitigation

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Background

Strong evidence has been established that an increase in exposure to fine particulate matter air pollution equal to or smaller than 2.5 microns ($PM_{2.5}$) is associated with an increase in the risk of mortality [1, 2]. A comprehensive systematic review of cohort studies reported that a 10 µg/m^3 increase in long-term $PM_{2.5}$ exposure is associated with an 8% (95% CI: 6%, 9%) increase in natural-cause mortality and each cause of mortality evaluated in the review showed statistically significant associations with $PM_{2.5}$ exposure: 11% (9%, 14%) increase for circulatory disease, 16% (10%, 21%) increase for cerebrovascular disease, 10% (3%, 18%) increase for respiratory disease and 12% (7%, 16%) increase for lung cancer per the same increment in $PM_{2.5}$ [3]. Another extensive systematic review including more than 25 years of cohort studies reported a similar order of excess mortality risk due to $PM_{2.5}$ exposure: 8% (95% CI: 6%, 11%) increase in all-cause mortality, and cause-specific mortality including 11% (8%, 14%) for cardiopulmonary disease and 13% (7%, 20%) for lung cancer, per 10 µg/m^3 increase in $PM_{2.5}$, respectively [2]. Interestingly, the meta-analysis estimated indicated robust $PM_{2.5}$-mortality associations, but with heterogeneity in the magnitude of associations among geographic regions (North America, Europe, and Asia). The Health Effects Institute conducted a comprehensive systematic review of the associations between long-term exposure to traffic-related air pollution (TRAP) and a wider range of adverse health outcomes including birth and respiratory outcomes for children [4]. The findings have provided an overall high or moderate-to-high level of confidence in the association between long-term exposure to TRAP and adverse health outcomes including all-cause mortality, mortality due to circulatory disease, ischemic heart disease (IHD), and lung cancer as well as for asthma onset in both children and adults and acute lower respiratory infections (LRI) in children [4].

In addition to epidemiological studies, a range of health impact assessment approaches have been developed for quantifying the impact of long-term $PM_{2.5}$ exposure on mortality. In 2019, air pollution was estimated to contribute to 6.7 million deaths globally, including 4.1 million deaths attributable to ambient $PM_{2.5}$ and 2.2 million deaths attributable to household air pollution [5, 6]. The Global Burden of Disease (GBD) 2019 study estimated that ambient particulate matter was the seventh highest risk factor globally in terms of disability adjusted life years (DALYs) and household air pollution was the tenth highest [7]. While the association between long-term exposure to $PM_{2.5}$ and mortality has been well documented in the literature, little information is available about the impact of cycles of annual exposure increases and decreases occurring multiple times during a 20 year period, such as those due to multiple international relocations. Prior studies have used time-varying $PM_{2.5}$ levels estimated through exposure modelling, although these studies were limited to persons that resided in one country during the study and with annual $PM_{2.5}$ exposure levels typically below 20 µg/m^3 [8–10]. A recent literature review of the health effects of $PM_{2.5}$ on persons with frequent relocations identified 12 studies that reported a difference in health effects among persons who relocated and non-relocated persons [11]. These included a study that reported an increase in the hazard ratio (HR) for all-cause mortality per 10 µg/m^3 increase in $PM_{2.5}$ among persons who relocated within the United States, with greater effects noted among white persons (HR=1.21; 95% CI: 1.20, 1.22) than African American persons (1.12; 1.08, 1.15) [12]. The mean change in annual ambient $PM_{2.5}$ among movers was less than 1 µg/m^3. A reanalysis of the US Harvard Six Cities Study reported RRs for all-cause mortality per 18.6 µg/m^3 increment of $PM_{2.5}$ stratified by movers (1.08; 95% CI: 0.67, 1.76) and non-movers (1.30; 1.10, 1.54), although there was limited statistical power for the findings among movers [13]. Finally, a study of congestive heart disease (CHD) deaths according to proximity to highly trafficked roads among persons in western Canada reported a lower RR of CHD death among persons who moved further away from traffic (RR=1.14; 95% CI: 0.95, 1.37) than persons who moved closer to traffic (1.20; 1.00, 1.43) [14]. While these studies provide evidence of the impacts on mortality among persons with relocations, they do not address the impact of multiple relocations to areas with large changes in ambient $PM_{2.5}$ concentrations.

When frequent changes of exposure are involved, the lag between the time when exposure changes and the time when the resulting health effect is evident becomes crucial. The time lag between reduced exposure and its impact on mortality (the 'cessation lag') has been explored by the US Environmental Protection Agency (EPA) who proposed a biphasic lag [15] in which the mortality risk evolves in two distinct phases to reach a minimum level after 20 years. This lag was informed by prior studies including a study of air pollution reductions resulting from a ban on the sale of coal in Dublin, a model developed by Leksell and Rabi of the reduction in $PM_{2.5}$ and its impact on life expectancy based on several large cohort studies in the US and Europe, and other research on disease incidence including bronchitis, diminished lung functioning and lung cancer [16, 17]. The proposed 'inception lag' (i.e. the lag representing increased health risk following an increase in exposure) followed the inverse pattern of the cessation lag. Other lag functions varying in shape and duration, specifically with different non-linear slopes and timing, have been proposed for all-cause and individual cause-specific mortality [18]. They include a steep eight-year lag based on findings from the

Harvard Six Cities study for PM$_{2.5}$ exposure and all-cause mortality [19] and a 38-year triphasic lag based on smoking cessation studies and lung cancer [18, 20, 21].

The US diplomatic corps is a dynamic population with international relocations every one to six years, the timing of which depends on the diplomat's area(s) of expertise, their US government agency and related agency staffing policies. The location of each relocation is determined by the diplomat's agency and global staffing needs. Diplomats often have limited input into location decisions, particularly for career foreign service officers, however, preference may be given to diplomats with increasing seniority. Specific city assignments, or postings, are contingent upon medical clearance both for the employee and for their accompanying family members [22]. Time lags between exposure and exposure-related health effects are of particular concern among this population as the duration of assignment in each city is generally relatively short (often 2–3 years) and air pollution levels could vary dramatically from one assignment city to the next.

The health impacts of air pollution during international assignments for diplomats and their family members are of great concern to the US government and are one of many occupational health risks diplomats face. The US government has explored mitigation options to help reduced air pollution exposure for diplomats and their family members while working in cities with high PM$_{2.5}$ including the filtration of air with air purifiers (APs) in offices and residences and improving home airtightness through taping or caulking windows and doorways. A study of 21 US diplomats in Kathmandu, Nepal found that the ratio of personal/ambient (P/A) PM$_{2.5}$ was 0.32 when a moderate to high level of mitigation was already used at home. After additional high capacity APs were added in residences, the P/A ratio was halved to 0.16 [23]. There are a few other reports on the impact of using APs on indoor air quality in the US, Beijing, and Shanghai [24–26]. However, the results seem to vary among settings.

To fill the research gap on the health impact of air pollution exposures that vary over time, this study aims to develop a model to estimate the impact of frequent international relocations on mortality due to PM$_{2.5}$ and to apply the model to US diplomats in a variety of contrasting assignment scenarios. The model explores the effects of different assumptions regarding lags between exposure and health impacts and also examines the impact of exposure mitigation measures on the mortality estimates.

Methods

The model described in this exploratory analysis was applied to illustrative persons in three age groups (representing US diplomats and their families) in a variety of hypothetical 20-year assignment scenarios from 2000 to 2019 and was based on US mortality rates adjusted to account for time spent in locations with different levels of PM$_{2.5}$.

Diplomatic assignments

Ten hypothetical assignments were created for this analysis. The assignments and mean annual PM$_{2.5}$ concentrations in each assignment location are shown in Fig. 1 and a list of all assignment locations is included in Supplementary Table 1. The assignments were developed to demonstrate a range of patterns of air pollution exposure and were based on typical international postings for US diplomats with occasional rotations in Washington, DC. The first two assignments, Standard A and Standard B, were based on the actual assignment histories for two

Assignment	Assignment Description	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
													Year of Assignment								
1	Standard A	19	19	50	50	19	19	19	6	7	6	43	43	28	28	28	6	6	43	43	6
2	Standard B	49	49	30	30	40	40	40	86	86	86	9	9	9	9	8	23	28	28	6	6
3	Standard A x 2[1]	19	19	50	50	19	19	19	6	7	6	43	43	28	28	28	6	6	43	43	6
4	High 1 year & low 3 years cycle	50	12	12	13	43	12	11	10	49	10	9	9	43	9	8	8	50	8	8	6
5	High 2 years & low 2 years cycle	50	50	12	13	43	43	11	10	49	49	9	9	43	43	8	8	50	50	8	6
6	High 3 years & low 1 year cycle	50	50	50	13	43	43	43	10	49	49	49	9	43	43	43	8	50	50	50	6
7	High 4 years x 5	50	50	50	50	43	43	43	43	49	49	49	28	43	43	43	43	50	50	50	50
8	Very high 2 years & low 2 years cycle	97	97	12	13	97	97	11	10	120	120	9	9	120	119	8	8	115	101	8	6
9	Very high 20 years	97	97	97	97	97	97	120	120	120	120	142	120	120	119	116	123	115	101	121	105
10	Standard A with 50% mitigation[2]	19*	19*	50*	50*	19	19	19	6	7	6	43*	43*	28*	28*	28*	6	6	43*	43*	6

🟩 low (PM$_{2.5}$ ≤ 20 µg/m³)   🟨 moderate (PM$_{2.5}$ 21-40 µg/m³)   🟧 high (PM$_{2.5}$ 41-60 µg/m³)   🟥 very high (PM$_{2.5}$ > 60 µg/m³)
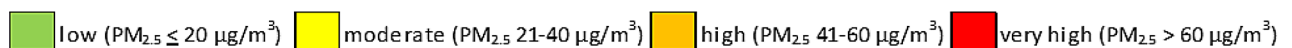
Fig. 1 Annual mean PM$_{2.5}$ concentration in each city for the ten assignment profiles. [1] The "Standard A x 2" assignment includes the Series A assignment repeated for a total duration of 40 years. [2] In this assignment, diplomats used air purifiers in their residences while working in cities in Africa and Asia during years indicated with an asterisk (*)

former diplomats. Further variations of the Standard A assignment were used as follows: a longer assignment of 40 years from ages 25 to 64 years (Assignment 3); a cycle with one year in a highly polluted city followed by a greater number of years in a less polluted city (Assignment 4), and vice versa (Assignment 6); the same number of years of postings in highly polluted and less polluted cities (Assignments 5 and 8); highly polluted cities only (Assignment 7 and 9); and with enhanced air pollution mitigation measures including air purifiers used in personal residences and in the workplace in highly polluted cities (Assignment 10). All assignments are for 20 years, except for Assignment 3.

Estimation of city specific PM$_{2.5}$ exposure
All assignments were modelled for the years 2000 to 2019, except for assignment 3 ("Standard A x 2") which covered 2000 to 2039. Each city was assigned an annual average PM$_{2.5}$ concentration based on published estimates. In most cases, the estimated ambient PM$_{2.5}$ concentration published in the World Health Organization's (WHO) ambient air pollution database was used [27]. In instances where the PM$_{2.5}$ level was not available in the WHO database for the relevant year, the annual mean PM$_{2.5}$ for the nearest year was used. For time spent in the United States, US national and Washington, D.C. observed annual mean PM$_{2.5}$ levels reported by the US Environmental Protection Agency were used for years 1999 to 2021 [28]. To represent previous exposure, US annual mean PM$_{2.5}$ figures for the years 1981 to 2000 were taken from estimates by Meng et al. [29] using a chemical transport model (GEOS-Chem) with geographically weighted regression (GWR) adjustment of satellite remote sensing and ground based PM$_{2.5}$, PM$_{10}$ and total suspended particle (TSP) measurements, when available [29]. For years 1955 to 1980, 22.1 μg/m^3 was used as the annual PM$_{2.5}$ for the US as this was the estimated annual mean for 1981 reported by Meng et al. For years 2001 to 2021, US annual mean PM$_{2.5}$ figures reported by the US Environmental Protection Agency were used in the model and for years 2022 to 2045, 8.0 μg/m^3 was used as the annual PM$_{2.5}$ for the US as this was the annual mean for 2021.

Quantification of the impact on mortality
Mortality rates based on routine US statistics (see Impacted mortality rate calculations including added risk due to PM$_{2.5}$) were used to represent those of diplomats in three age groups: (1) an older diplomat born in 1955 with assignments conducted between ages 45–64 years, (2) a young diplomat born in 1975 with assignments conducted between ages 25–44 years and (3) a child, who accompanied their diplomat parent(s) on assignments from birth to age 20 years. A standard life table method

was used to estimate life expectancy and days of life lost (DLL) for each age group following each of the 10 assignments using the method described below [30]. The results were compared to those assuming the individual lived exclusively in the US over the same period.

GBD concentration-response functions
For each year of each assignment, we applied the GBD's meta-regression, Bayesian, regularized, trimmed (MR-BRT) [7] concentration-response (C-R) functions to calculate the risk of PM$_{2.5}$-related mortality in relation to the theoretical minimum-risk exposure level (TMREL) estimated by the GBD study for six outcomes: IHD, stroke, chronic obstructive pulmonary disease (COPD), LRI, type 2 diabetes mellitus, and lung cancer. The C-R functions estimate the relationship between long-term exposure to PM$_{2.5}$ and mortality, using a non-linear exposure response model. The GBD project used data from numerous cohort studies from varied global locations with a wide variety of PM$_{2.5}$ levels and sources.

   Application: In the study, the CRFs were used to estimate the number of deaths and diseases that could be attributed to PM$_{2.5}$ exposure globally. By combining the CRFs with global air quality data, the researchers could estimate the health burden of air pollution in different regions and countries.

   In summary, the study used data-driven models to predict how air pollution impacts health worldwide, helping to understand the global burden of diseases linked to air quality.

   The GBD C-R functions are based on the ambient PM$_{2.5}$ concentration and, for IHD and stroke, vary by (5-year) age groups beginning at age 25 years. The IHD and stroke functions do not include any added risk for persons under age 25. Supplementary Fig. 1 shows the GBD C-R functions for each outcome.

Inception and cessation lags
As our central estimate of the lag between change in exposure and change in risk, the mortality risks calculated for each location using the GBD functions were lagged using the 20-year cessation lag published by the US Environmental Protection Agency (EPA) [31] and a 20-year inception lag assumed to be the inverse of the EPA cessation lag (Supplementary Fig. 2). The EPA 20-year cessation lag retains 70% of the risk associated with the difference in the previous year's elevated PM$_{2.5}$ concentration to account for short-term effects. During the next four years, the risk decreases by 12.5% per year, reaching a level of 20% risk distributed evenly over the course of the remaining 15 years with risk reduced by 1.3% per year to account for long-term effects of PM$_{2.5}$ exposure on lung cancer mortality [21, 32]. During the first year of the (inverse) EPA inception lag, 30% of the

risk is applied in the first year. During years two to five, the risk increases by 12.5% per year and during years six to twenty, the risk increases by 1.3% per year. After 20 years, the full risk is applied to the mortality rates.

To apply the lags in this analysis, we first calculated the RR of the risk in the prior location relative to the risk in the next location (i.e. the risk for the old location divided by the risk of the new location). When moving to a new location with an increase in $PM_{2.5}$, the inception lag was applied only to the RR for the new location (location B) divided by the RR of the prior location (location A) minus 1:

$$lagged\,risk,\ moved\,from\,location\,A\,(low\,PM_{2.5})$$
$$to\,B\,(high\,PM_{2.5}) = \frac{RR_B}{RR_A} - 1$$

When moving to a new location with a decrease in $PM_{2.5}$, the cessation lag was applied to the RR for the prior location (location A) divided by the RR of the new location (location B) minus 1:

$$lagged\,risk,\ moved\,from\,location\,A\,(high\,PM_{2.5})$$
$$to\,B\,(low\,PM_{2.5}) = \frac{RR_A}{RR_B} - 1$$

Risks were lagged when the ambient $PM_{2.5}$ mean changed by ≥ 5 μg/m³. Supplementary Table 2 presents example calculations using an inception lag and a cessation lag. The remaining portion of the inception lag was removed from the risk calculation when a diplomat moved to a location with lower ambient $PM_{2.5}$ while the (previous) inception lag was still evolving. Similarly, the remaining portion of the cessation lag was removed from the calculation when a diplomat moved to a location with higher ambient $PM_{2.5}$.

Impacted mortality rate calculations including added risk due to $PM_{2.5}$

US 2019 mortality rates (MRs) by age and gender were obtained for all-causes and each of the six pollution-sensitive conditions using International Classification of Diseases, 10th revision (ICD-10) codes specified in the GBD 2019 risk estimate publication [7, 33]. For each pollution sensitive condition, impacted mortality rates (iMRs) were calculated by multiplying the GBD-derived RRs by the US MRs.

The remaining, non-pollution sensitive (NPS) MR at each age was calculated by subtracting the US MR for each of the six pollution sensitive conditions from the US all-cause MR at that age. The iMRs for the six pollution sensitive conditions were added back to the NPS MR at each age to yield a new all-cause iMR.

Life expectancy, days of life lost and excess deaths

The probability at age ***a*** of surviving to the next year of age was calculated using the standard life table method, assuming that deaths occur at the mid-point of the year:

$$survival\,probability_a = \frac{2 - all\,cause\,iMR_a}{2 + all\,cause\,iMR_a}$$

Remaining life expectancy at birth, noted as *b*, was calculated by adding the life years from birth to 100 years' age (noted as *j* in the formula) divided by the cumulative survival during year *j*:

$$life\,expectancy_b = \frac{\sum life\,years_j}{cumulative\,survival_j}$$

Days of life lost (DLL) were calculated by subtracting the life expectancy under each assignment from the life expectancy calculated for the same diplomat living exclusively in the US. Deaths, expressed per one million population, were calculated by multiplying the impacted all-cause mortality rate for each year of age by one million and summing the number of deaths during *j*. Deaths were calculated for male and female diplomats in each of the three age groups as well as for persons of comparable age and sex diplomats that lived exclusively in the United States. Similarly, excess deaths, expressed per million population, were calculated as the additional deaths among diplomats compared to a person of the same age and sex who lived exclusively in the United States. All key assumptions for the modelling are presented in Supplementary Table 3.

Impact of mitigation at home and in workplace

In assignment 10, we explored the potential effects of using exposure mitigation strategies, including air purifiers and home sealing, to reduce the $PM_{2.5}$ concentration in the homes and workplaces of diplomats working in cities in Africa and Asia. The air in most US Embassy and Consulate buildings is highly filtered and the US government provides air purifiers to US diplomats working in many cities in Africa and Asia [34]. According to the results from our previous personal monitoring study among US diplomats in Kathmandu, Nepal, the ratio of mean *personal* $PM_{2.5}$ exposure to mean *ambient* $PM_{2.5}$ was reduced by 50% (from 0.32 to 0.16) following the addition of enhanced mitigation in diplomats' residences [23]. These findings were applied to the "Standard A with mitigation assignment" by using 50% of the city-year specific $PM_{2.5}$ exposure level during international assignments in Africa and Asia.

Sensitivity analysis

We conducted eight sensitivity analyses to examine the sensitivity of the model results to key assumptions/parameters using the Standard A assignment. Variation 1 used US mortality statistics from the top 5% of US counties according to total household income [35]) to reflect the fact that the baseline mortality for diplomats is likely to be lower than the US average population. To test the sensitivity of the model to the chosen lag structures, Variations 2 to 6 used alternate lag structures applied to the GBD C-R functions: Variation 2 used a steep eight-year lagapplied to COPD and LRI only (no change in other four causes); Variation 3 used a longer 38-year triphasic lag for lung cancer only (no change in other five causes); Variation 4 applied only the cessation lag (i.e. no inception lag, assuming the full effect from the first year in a new location with higher $PM_{2.5}$ level); Variation 5 applied only the inception lag (i.e. no cessation lag, assuming the increased mortality risk associated with time spent in a location with high $PM_{2.5}$ was discontinued upon leaving that location); and Variation 6 applied no lagged effects. Supplementary Fig. 2 includes plots of the lags used in variations 2 (short lag applied to COPD and LRI) and 3 (long lag applied to lung center). To assess the effects of uncertainty in the C-R functions, Variations 7 and 8 used the upper and lower limits of the 95% confidence intervals around the GBD functions.

Results

The cause-specific lagged increases in the relative risks (RR) of mortality due to stroke, IHD, COPD, lung cancer, LRI, and type 2 diabetes for an older diplomat who began the Standard A assignment at age 45 years are included in Fig. 2. There were no lags applied prior to the beginning of the diplomatic assignment and an inception lag was applied during years 1–4, 11–14, and 18–19 of the Standard A assignment when the diplomat lived in a city with ambient annual $PM_{2.5}$ that was at least 5 $\mu g/m^3$ greater than the prior city location and a cessation lag was applied during years 5–10, 15–17, and 20 when the diplomat moved to a city with ambient annual $PM_{2.5}$ at least 5 $\mu g/m^3$ less than the prior city's $PM_{2.5}$ annual mean. A cessation lag was also applied at the conclusion of the diplomatic assignment from age 65–84 years.

After the older diplomat began the Standard A assignment at age 45, among the six pollution-sensitive conditions, the greatest difference between RRs for the diplomat compared to the RR for the person who lived exclusively in the US was observed for stroke during the fifteenth year of the assignment, during the third and final year working in Bangkok, Thailand with an RR of 1.39 (95% CI: 1.23, 1.57) and mean annual $PM_{2.5}$ of 27 $\mu g/m^3$. During the same year, the RR of stroke for a person living in the US was 1.07 (1.0, 1.17) and mean annual

$PM_{2.5}$ of 9 $\mu g/m^3$. During the course of the 20-year Standard A assignment, the RRs of mortality for each of the six pollution sensitive conditions have three distinct peaks, each occurring at the conclusion of a series of years with consecutively increasing ambient $PM_{2.5}$ levels. The pollution sensitive condition with the second greatest annual difference in the RR of mortality during the Standard A assignment for a diplomat compared to a person living in the US was for IHD, with an RR of 1.29 (95% CI: 1.14, 1.49), during the third and final year working in Bangkok when the mean annual $PM_{2.5}$ was 27 $\mu g/m^3$ (Fig. 2A). During the same year, the RR of IHD for a person living in the US was 1.04 (95% CI: 1.00, 1.15) and mean annual $PM_{2.5}$ of 9 $\mu g/m^3$, and the confidence intervals (CIs) for the diplomat and the person of comparable age living in the US overlap. The RRs of mortality for IHD and stroke are elevated before the older diplomat began the Standard A assignment at age 45 and these RRs are reflective of the GBD C-R functions based on US $PM_{2.5}$ levels. Supplementary Fig. 3 shows the pollution sensitive condition iMRs for an older diplomat in the Standard A assignment.

Figure 3 shows the deaths per one million persons due to all-causes, that incorporates the individual pollution sensitive iMRs for female older diplomats in various assignments. The assignment "High 3 years & low 1 year cycle" has the highest impacted mortality rate each year compared to the other assignments for most of the 20-year assignment period, from ages 45–64, although the CIs for the three assignments overlap in 19 of the 20 years of diplomatic postings. The CIs for the "High 3 years & low 1 year cycle" and the "High 2 years & low 2 years cycle" assignments do not overlap with the CIs for the US iMRs. However, the CIs for the assignment with the least number of years in a location with high $PM_{2.5}$, "High 1 year & low 3 years cycle", overlap with the CIs for the US iMRs during 19 of the 20 years of the diplomatic assignments. Beginning at age 65 after the completion of the assignments, the iMRs for the three assignments gradually decline to the US MR.

During the Standard A and Standard B assignments, older diplomats had a greater number of excess deaths due to international assignments than young diplomats and children accompanying a parent on diplomatic assignments (Table 1). During the Standard A assignment, older diplomats (12,246.8 deaths per one million males, 6,640.7 deaths per one million females) had 8 times as many excess deaths as young diplomats (1,527.9 deaths per one million males, 708.6 deaths per one million females) and more than a 600-fold increase in excess deaths compared to a child accompanying their parent(s) on a diplomatic assignment (20.1 deaths per one million males, 12.1 deaths per one million females). Among the assignments examined, older diplomats living in one city
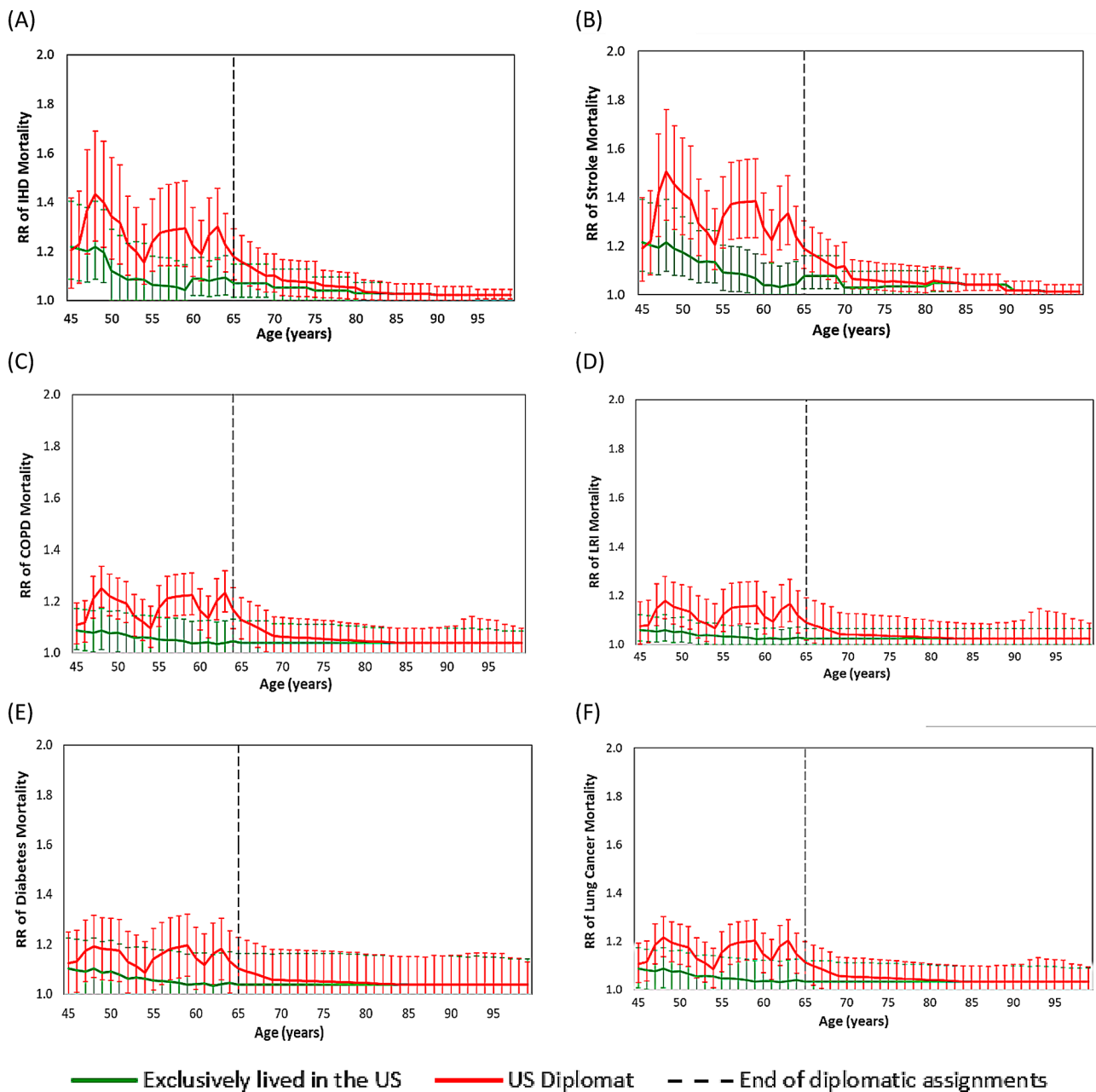
Fig. 2 Increase in relative risk of mortality for six pollution-sensitive conditions for an older diplomat in the Standard A assignment in comparison to an America living exclusively in the US during their lifetime. **A** IHD. **B** Stroke. **C** COPD. **D** LRI. **E** Type 2 diabetes mellitus. **F** Lung cancer.

with very high $PM_{2.5}$ for 20 years had the highest number of excess deaths due to international assignments (49,140.4 deaths per one million males, 28,758.3 deaths per one million females), followed by living in a series of high $PM_{2.5}$ cities in the assignment "High 4 years x 5" (29,658.2 deaths per one million males, 16,517.3 deaths per one million females). Among older diplomats in assignments with one, two or three of every four years living in locations with high $PM_{2.5}$ ($PM_{2.5}$ annual mean between 41 and 60 $\mu g/m^3$), the highest number of excess deaths were found during assignments with the

highest number of years in cities with high $PM_{2.5}$ locations, including the assignments "High 3 years & low 1 year cycle" (22,120.1 deaths per one million males, 12,329.7 deaths per one million females), "High 2 years & low 2 years cycle" (15,496.9 deaths per one million males, 8,555.1 deaths per one million females) and "High 1 year & 3 low years cycle" (10,009.2 deaths per one million males, 5,409.8 deaths per one million females).

Older diplomats had greater DLL than young diplomats in the Standard A assignment (80.4 DLL vs. 10.4 DLL) and in the Standard B assignment (84.1 DLL vs.
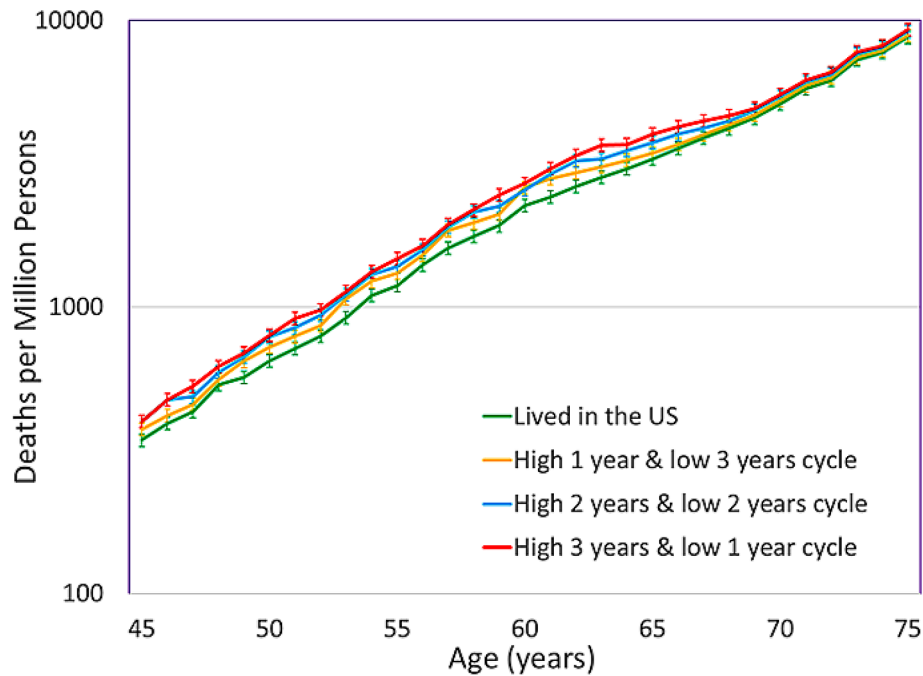
Fig. 3 Age-specific deaths per million for female older diplomats in three assignments

Table 1 Number of excess deaths[1] per one million persons for each diplomatic assignment[2]

Assignment	Assignment description[3]	Older diplomat		Young diplomat		Child	
		Excess deaths, male	Excess deaths, female	Excess deaths, male	Excess deaths, female	Excess deaths, male	Excess deaths, female
1	Standard A	12,246.8	6,640.7	1,527.9	708.6	20.1	12.1
2	Standard B	10,568.4	5,623.3	1,341.8	562.7	43.7	27.9
3	Standard A x 2	n/a	n/a	14,370.6	7,896.7	n/a	n/a
4	High 1 year & low 3 years cycle	10,009.2	5,409.8	1,171.2	536.3	40.7	26.7
5	High 2 years & low 2 years cycle	15,496.9	8,555.1	2,050.4	975.8	60.3	37.9
6	High 3 years & low 1 year cycle	22,120.1	12,329.7	3,007.8	1,466.6	75.7	44.4
7	High 4 years x 5	29,658.2	16,517.3	7,695.6	5,211.2	86.8	53.1
8	Very high 2 years & low 2 years cycle	24,988.7	14,514.8	3,304.8	1,678.9	108.5	70.7
9	Very high 20 years	49,140.4	28,758.3	6,332.1	3,321.5	163.5	103.8
10	Standard A with mitigation[4]	4,119.6	2,117.3	406.0	148.8	-1.8	-0.9

[1] The number of excess deaths per year *i* is calculated by subtracting the number of deaths during yar i per 1 million persons of the same age and sex living exclusively in the US from the number of deaths during ear i per 1 million diplomats completing a diplomatic assignment. The number of excess deaths listed on this table is the sum of excess deaths for each year from birth to age 100

[2] See Supplemental Table 1 for a list of cities and number of years in each city during the 10 assignments and see Fig. 1 for the annual mean $PM_{2.5}$ levels used in the calculations

[3] Ambient $PM_{2.5}$ level descriptions include low ($PM_{2.5}<20$ µg/m³), moderate ($PM_{2.5}$ 21–40 µg/m³), high ($PM_{2.5}$ 41–60 µg/m³) and very high ($PM_{2.5}>61$ µg/m³)

[4] In this assignment, diplomats used air purifiers in their residences while working in cities in Africa and Asia

25.0 DLL) (Fig. 4). Similar trends were found for females, although the magnitude of impact was smaller than that of males. Among older male diplomats, the greatest DLL were observed during the assignment in one city with

very high $PM_{2.5}$ for 20 years, "Very high 20 years" (287.8 DLL), followed by serving in four cities with high $PM_{2.5}$ for a total of 20 years, "High 4 years x 5" (176.9 DLL), and an equal number of years in cities with very high
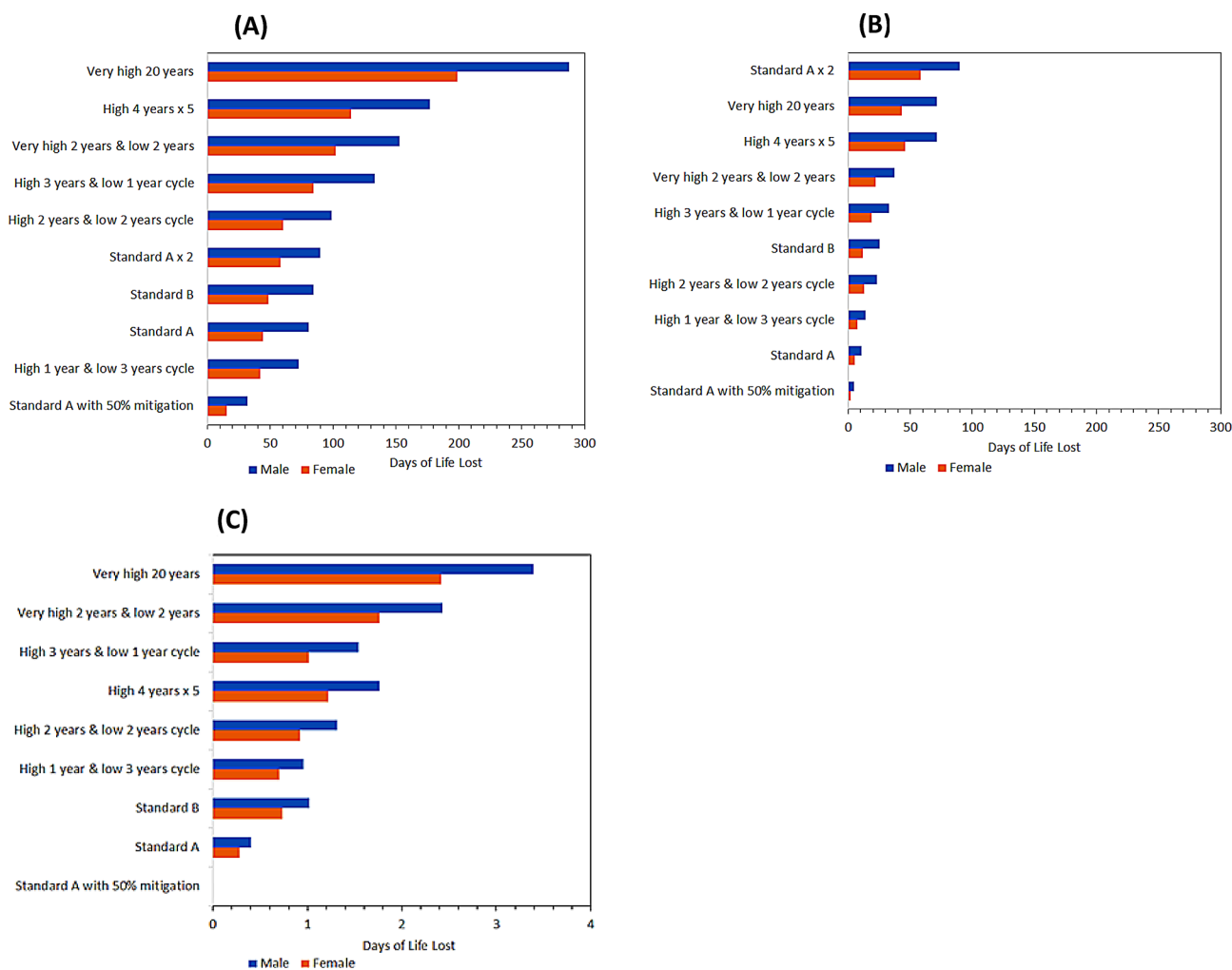
(A)

(B)

(C)

Fig. 4 Days of life lost associated with assignments for diplomats in three age groups. **A** Older diplomat. **B** Young diploma©. **C** Child

$PM_{2.5}$ and low $PM_{2.5}$, "Very high 2 years & low 2 years" (152.8 DLL). Among older male diplomats completing assignments with one, two or three of every four years in a city with high $PM_{2.5}$, the "High 3 years & 1 low year cycle" (132.9 DLL) assignment had the highest number of DLL among older male diplomats. The magnitude of DLL decreased as the number of years in cities with high $PM_{2.5}$ per four year cycle decreased to a low of 72.4 DLL while serving in the assignment with one year in a city with high $PM_{2.5}$, "High 1 year & low 3 years cycle". A male completing a 40-year assignment, "Standard A x 2" from ages 25–64 years (89.7 DLL) had only slightly higher DLL than a male completing the Standard A assignment for 20 years from ages 45–64 (80.4 DLL).

The RRs of cause-specific mortality for an older diplomat in the Standard A assignment with mitigation (Fig. 5) were much closer to that of a person of the same age and sex living in the US than in the Standard A assignment without mitigation. Similarly, the number of deaths per million older female diplomats in the Standard A

assignment with mitigation (Fig. 6) were much closer to that of a person of the same age and sex living in the US than in the Standard A assignment without mitigation. Examining the number of excess deaths among male diplomats for the Standard A assignment with mitigation compared to the Standard A assignment without mitigation, excess deaths decreased by 66% for older diplomats (from 12,246.8 to 4,119.7 deaths per million), 73% for young diplomats (from 1,527.9 to 406.0 deaths per million) and 49% for children of diplomats (from 3.5 to 1.8 deaths per million) (Table 1). Among males the DLL decreased by 60% for older diplomats (from 80.4 to 32.0 DLL), 55% for young diplomats (from 10.4 to 4.7 DLL) and results were similar in both scenarios for children (0.4 and −0.1 DLL).

The differences in DLL for the specified sensitivity analysis options were quantified by subtracting the DLL from the main model reported above: the differences in DLL ranged from −48.0 to +55.1 for older diplomats, −9.7 to +15.7 for young diplomats and −1.2 to +2.4 for
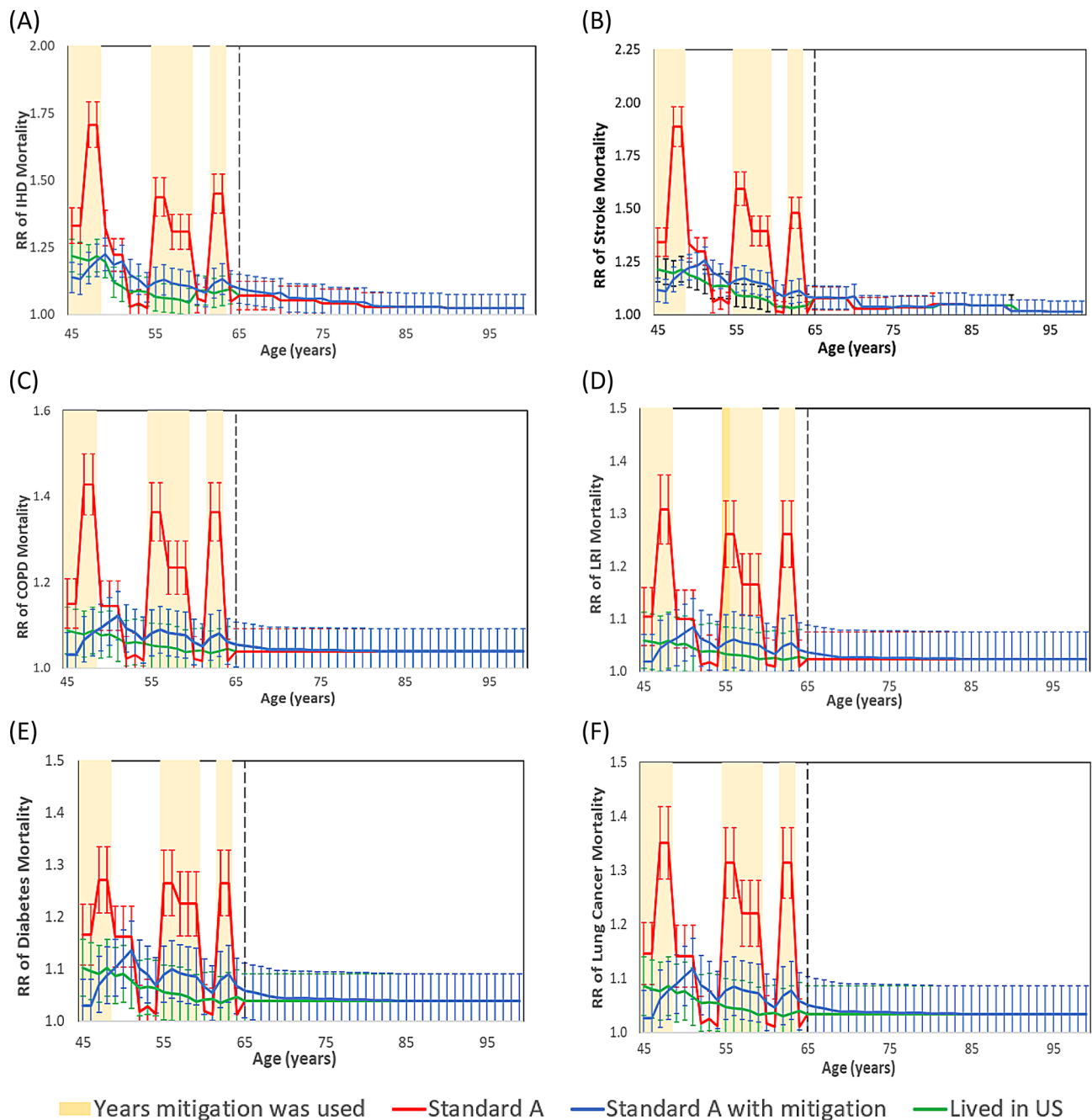
Fig. 5 RR of mortality due to PM$_{2.5}$ for the Standard A assignment with and without mitigation. (**A**) IHD, (**B**) Stroke, (**C**) COPD, (**D**) LRI, (**E**) Type 2 Diabetes and (**F**) Lung Cancer.

children (Fig. 7). Removing the inception lag had the largest impact on the DLL among the investigated model variations for older diplomats (+55.2 (+68%) DLL male, +36.4 (+76%) DLL female) and younger diplomats (+15.7 (+151%) DLL male, +9.4 (+90%) DLL female). Using the upper CI of the C-R function also resulted in an increase in DLL both among older diplomats (+22.5 (+28%) DLL male, +15.6 (+33%) DLL female) and young diplomats (+12.0 (+115%) DLL male, +7.3 (+70%) DLL female).

Using the short lag on COPD and LRI and, separately, the long lag on lung cancer, both had small impacts on the DLL for older and young diplomats. Model variations applied to older diplomats resulted in a decrease in DLL: removing lags from the model (-16.3 (-20%) DLL male, -13.9 (-29%) DLL female), using the minimum C-R function (-17.1 (-21%) DLL male, -14.2 (-30%) DLL female), using high income mortality rates (-28.2 (-35%) DLL male and female) and removing the cessation lag (-48.0
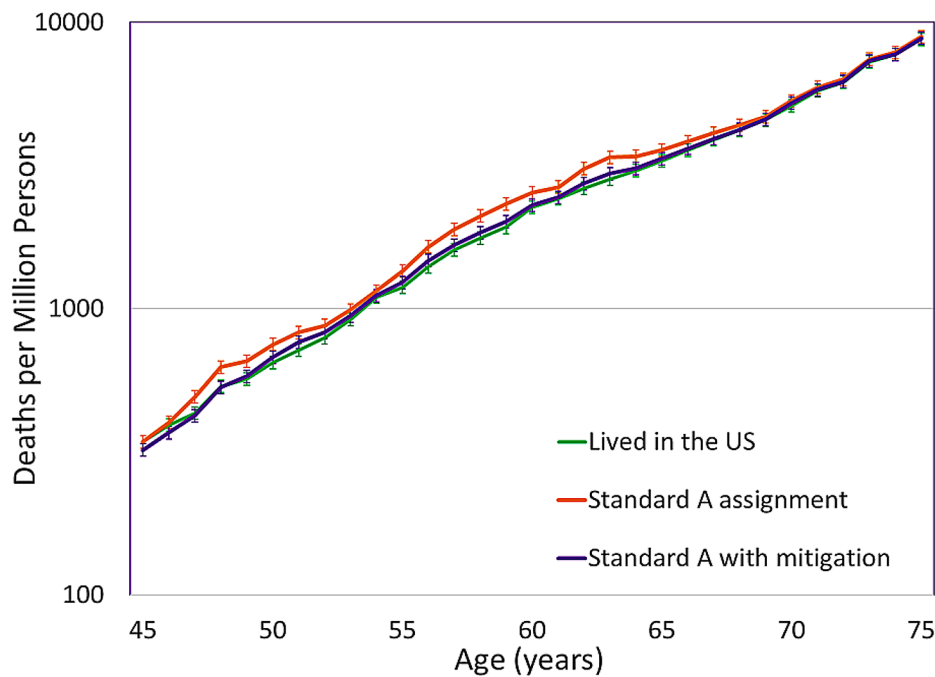
Fig. 6 Deaths per million female older diplomats in the Standard A assignment without mitigation
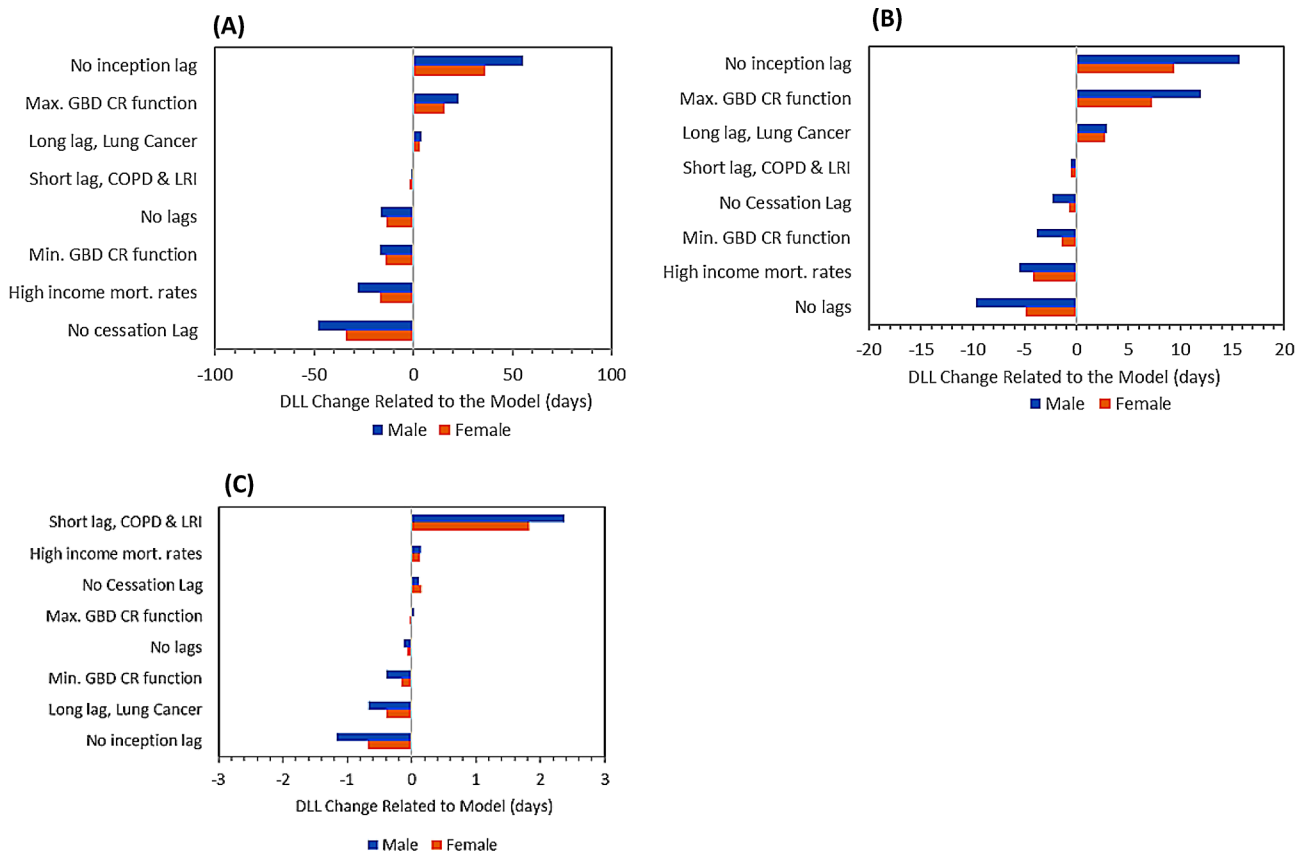

Fig. 7 Change in the DLL in sensitivity analysis for diplomats in three age groups. (**A**) Older diplomat, (**B**) Young diplomat and (**C**) Child.

(-59%) DLL male, -34.0 (-71%) DLL female). Application of the short lag to COPD and LRI suggested the largest possible impacts on DLL for children (+2.9 (+431%) DLL male, female +1.8 (+465%) DLL female). All other model variations resulted in less than 1 DLL gained or lost. Use of high income US baseline mortality statistics, instead of mortality statistics for the entire US population, resulted in a slight gain in DLL (+0.7 (+103%) DLL male, (+0.4 (+95%) DLL female) as did removal of the cessation lag for all six pollution sensitive conditions (+0.7 (+100%) DLL male, +0.5 (+100%) DLL female). Use of the 20-year inception and cessation lags yielded results that were roughly midway between the results when other elements of the model were changed.

Discussion

We developed a novel approach to estimate the impact on mortality due to local $PM_{2.5}$ concentrations given assignments with frequent relocations to areas of varying $PM_{2.5}$. A set of systematically designed 20-year diplomatic assignments were used to demonstrate the impacts of the assignment patterns of city-specific annual mean $PM_{2.5}$ on the health of US diplomats. The key findings include that age at the time of relocation is critically important in determining the impact of air pollution on mortality, with greater impacts found for relocations amongst older diplomats than for young diplomats or the child of a diplomat (due to greater underlying mortality rates in older age groups). The mortality impacts decreased when assignments in high $PM_{2.5}$ cities were followed by assignments in low $PM_{2.5}$ cities and using room APs and improving home airtightness in polluted cities may have the potential to reduce the impacts considerably. According to the sensitivity analysis, the estimated health impacts were more sensitive to the choice of inception and cessation lags than to the $PM_{2.5}$ exposure level, C-R functions, or baseline mortality. Removal of the inception lag increased the DLL by 68% and 76% for male and female older diplomats compared to the original model including both lags, while removal of the cessation lag reduced the DLL by 59% and 71%.

This study demonstrated a new indirect method using WHO air quality data and GBD C-R functions to quantify the lifetime health impacts of air pollution exposure that frequently changes for a highly mobile population with relocations between global cities. There are several notable features in the model. First, comparison with baseline mortality using the life table analysis enables calculation of DLL considering the time when death occurs during the life course. This adds value to estimates based on the number of deaths which are commonly reported by previous quantitative risk assessments such as the GBD study [7]. Second, the model enables incorporation of various time lag functions in terms of inception

and cessation, depending on the target health outcome. The dramatic changes in $PM_{2.5}$ exposure among the US diplomatic population highlight a paucity of evidence on the cause-specific delayed effects of changes in $PM_{2.5}$ exposure.

Our exploratory study also has several limitations and methodological challenges. First, there was limited availability of annual mean $PM_{2.5}$ data for cities used in the assignment scenarios, particularly for years prior to 2010. The use of $PM_{2.5}$ annual mean data from several years before or after the year of interest may result in misclassification of exposure. This could underestimate the exposure in many cities because $PM_{2.5}$ was generally higher in earlier years when it was not commonly measured. Future development of the method would therefore benefit from consistent, routine ambient $PM_{2.5}$ monitoring across global cities, particularly in low and middle income countries. However, the accuracy of the $PM_{2.5}$ data is a lesser concern as the purpose of this study was to demonstrate changes in mortality associated with illustrative variations in $PM_{2.5}$ levels and not to directly attribute it to time spent in any particular city. Second, the model did not take into account the different, but highly uncertain, nature of $PM_{2.5}$ sources in each global location and the impact that $PM_{2.5}$ from different sources may have on mortality [36]. Indeed, our study included cities where the majority of $PM_{2.5}$ can be attributed to emissions from soils, plants, and dust (Riyadh and Dakar) and cities with high levels of $PM_{2.5}$ due to burning biofuels for domestic energy use (Bangkok and Kathmandu). Third, using estimates of city-wide $PM_{2.5}$ exposure may not provide an accurate representation of exposure for diplomats as they may spend more time in less polluted areas in the city as well as using Aps in their workplace and homes. Fourth, our method likely underestimates impacts in children because the GBD C-R functions were not designed to be used to estimate children's health risks and, in particular, the GBD 2019 study did not include C-R functions for IHD or stroke occurring in persons under 25 years age because the incidence of IHD and stroke in persons under 25 years age is very low and unlikely to be impact by $PM_{2.5}$. It should also be noted that our model included only mortality and the effects of air pollution on children may be more pronounced in terms of morbidity, such as exacerbation of asthma. Fifth, we used baseline mortality risks for the US general population to represent the baseline mortality risks for US diplomats which may not be entirely representative for diplomats' health conditions, as diplomats are likely to be healthier than the general population due to better nutrition and increased access to medical care. In order to address this issue, we explored the effects of limiting the baseline mortality rates to those with higher socioeconomic status (SES) in the sensitivity analysis. However, C-R functions may

also be different for the higher SES population as modification of $PM_{2.5}$ effects on mortality by SES is very often observed in cohort studies [37–39]. Sixth, the set of 10 illustrative assignments used in this study does not cover the wide range of realistic assignment patterns for US diplomats. Application of this model to a greater number of assignments using actual human resources data for US diplomats would provide realistic total mortality burdens among the US diplomatic population. Seventh, the use of mitigation activities including APs at home and improved home tightness in cities with high $PM_{2.5}$ assumed a 50% reduction in $PM_{2.5}$ exposure in those cities, based on results of our previous Kathmandu study [23]. As the reduction of the outdoor $PM_{2.5}$ level depends on the housing structure in each local setting in a different city, this is a very rough estimate and a universal assumption applied to a range of cities. While the results associated with mitigation suggested beneficial effects for all age groups, the reported DLLs should be interpreted with caution. Nonetheless, this example illustrates the possible health benefits, in terms of the reduction of DLLs, through the use of mitigation at home and in the workplace in highly polluted cities. An additional limitation of the analysis is the exclusive reliance on one set of exposure functions, notably the GBD's C-R functions for $PM_{2.5}$. While the sensitivity analysis examined the impact on mortality based on the upper and lower confidence intervals of the C-R functions, a tenth sensitivity analysis could have included the use of an alternative exposure function, such as 1.21 hazard ratio for all-cause mortality per 10 μg/m^3 increase in $PM_{2.5}$, identified by Awad et al. among white Americans who relocated within the US [12]. Finally, this research did not examine the impact on mortality of other factors that change when diplomats relocate including changes in their diet, access to clean potable water and food sources, nor changes in mortality due to motor vehicle accidents, violent crimes, or natural disasters [40–42]. Future research could benefit from including the impact of these exposures by location on diplomats' mortality estimations, in addition to examining the $PM_{2.5}$-related impacts on mortality.

The modelling strategy proposed here is unique and very few prior studies are directly comparable to the modeling strategy proposed here. A study conducted among US military families who experienced frequent relocations within the US reported that respiratory hospitalizations among children aged 2–5 years increased among those living in cities with high ozone (O_3) levels, although the study suffered from limited power when the analysis was confined to movers [43]. The study focused on US domestic relocations and did not involve dramatic changes in air pollution exposure. Several studies have quantified the health impacts of mitigation measures to reduce air pollution exposure. A UK modeling study found that lifetime use of Aps at home may increase life expectancy by 138 and 120 days or more for males and females, respectively [44]. Like ours, the study used life table methods and GBD C-R functions for IHD, stroke, COPD, LRI and lung cancer, although neither inception nor cessation lags were applied in the model, which is less crucial for minimal changes in exposure. A US modeling study evaluated the mortality-related benefits and costs of improvements in particle filtration in US homes and commercial buildings accounting for time spent in various environments as well as activity levels and associated breathing rates. The results indicated that the use of portable Aps in homes in the US could be a cost-effective strategy to reduce particle-related mortality [45]. While our results with mitigation measures were encouraging, the model did not address any detrimental effects of improved home airtightness, including possibly increased indoor $PM_{2.5}$, carbon dioxide and other indoor pollutants [46, 47].

The findings from this study provide useful information to support decision making to reduce health risks for people with frequent international relocations including the diplomatic corps and other professional groups whose $PM_{2.5}$-related mortality risk and life expectancy may be affected by overseas assignments. Our results suggest that, where possible, hiring agencies and employees may want to consider scheduling work in cities with high $PM_{2.5}$ at the beginning of the career rather than at older ages, when baseline mortality rates increase steeply with age. After completion of a posting in a city with high $PM_{2.5}$, hiring agencies and employees may also want to request that their next posting be in a city with low $PM_{2.5}$, if possible. It should be noted that there are many important factors in determining staffing assignments including the suitability for available positions, amount of training and expertise needed in various positions, as well as other personal and family health and safety considerations. Although ambient $PM_{2.5}$ could impact diplomats' health, it is only one of many important factors that employers may want to consider when determining global staffing assignments and the duration of assignments. Our previous study in Kathmandu suggested that the use of high capacity air purifiers and improvement of building airtightness greatly reduced $PM_{2.5}$ personal exposure [23]. Additional research is needed to better understand the association between personal and ambient $PM_{2.5}$ in different global settings to estimate the impact of mitigation more precisely on $PM_{2.5}$-related mortality. The application in this model of an indicative reduction rate of $PM_{2.5}$ exposure from the Kathmandu study suggested that potential health benefits could be achieved from these mitigation measures. In reality, there are inequalities in access to such expensive mitigation measures across the globe, especially in

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LMICs. In addition, the use of Aps may not be an ideal solution given concerns about climate change and planetary health.

Conclusions

We developed a novel health impact model to estimate the effect of lifetime exposure to changing $PM_{2.5}$ levels on mortality for individuals with regular international relocations, by applying published C-R functions with inception and cessation lags. The application of the model to US diplomats in various assignments suggested that an increased number of years living in high $PM_{2.5}$ cities resulted in an elevated mortality risk, with greater health impacts in older diplomats than young diplomats or their children because of their greater underlying mortality rates for conditions sensitive to air pollution. Alternating assignments, when possible spending a few years in a high $PM_{2.5}$ city followed by a year or more in a city with a lower $PM_{2.5}$ concentration may help to reduce the additional risk of mortality due to $PM_{2.5}$. Our results also suggest that the use of air purifiers and improved home airtightness may help mitigate health burdens due to exposure to ambient $PM_{2.5}$. The choice of inception and cessation lags is critical for the magnitude of the estimated mortality burdens and, as such, further research on the delayed effects of $PM_{2.5}$ exposure on cause-specific mortality is required to improve model estimates.

Abbreviations

AP	Air purifier
CHD	Congestive heart disease
COPD	Chronic obstructive pulmonary disease
C-R	Concentration response function
DALY	Disability adjusted life year
DLL	Days of life lost
EPA	US Environmental Protection Agency
GBD	Global Burden of Disease
HR	Hazard ratio
IHD	Ischemic heart disease
I/O	Ratio of indoor $PM_{2.5}$ to outdoor $PM_{2.5}$
LRI	Lower respiratory infection
MR-BRT	Meta-regression, Bayesian, regularized, trimmed
P/A	Ratio of personal $PM_{2.5}$ to ambient $PM_{2.5}$
$PM_{2.5}$	Particulate matter smaller than 2.5 μm
RR	Relative risk
TRAP	Traffic-related air pollution
TMREL	Theoretical minimum-risk exposure level
YLL	Years of life lost

Supplementary Information

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Supplementary Material 1

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Author contributions
LE, JM, PW, and AM conceived of the study. LE, JM and PW developed the methodology for the study. JM, PW, and AM supervised the study. LE wrote the initial draft of the manuscript and the other authors contributed to its development. LE, JM and AM reviewed and approved the final manuscript.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

References

1. Cohen AJ, Brauer M, Burnett R, Anderson HR, Frostad J, Estep K, Balakrishnan K, Brunekreef B, Dandona L, Dandona R, et al. Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution: an analysis of data from the Global Burden of diseases Study 2015. Lancet. 2017;389(10082):1907–18.
2. Pope CA 3rd, Coleman N, Pond ZA, Burnett RT. Fine particulate air pollution and human mortality: 25 + years of cohort studies. Environ Res. 2020;183:108924.
3. Chen J, Hoek G. Long-term exposure to PM and all-cause and cause-specific mortality: a systematic review and meta-analysis. Environ Int. 2020;143:105974.
4. Health Effects Institute. Systematic Review and Meta-analysis of selected Health effects of Long-Term exposure to Traffic-Related Air Pollution. In.; 2022.
5. Fuller R, Landrigan PJ, Balakrishnan K, Bathan G, Bose-O'Reilly S, Brauer M, Caravanos J, Chiles T, Cohen A, Corra L, et al. Pollution and health: a progress update. Lancet Planet Health. 2022;6(6):e535–47.
6. Health Effects Institute. State of Global Air 2023. 2023.
7. GBD Risk Factors Collaborators. Global burden of 87 risk factors in 204 countries and territories, 1990–2019: a systematic analysis for the global burden of Disease Study 2019. Lancet. 2020;396(10258):1223–49.
8. Di Q, Dominici F, Schwartz JD. Air Pollution and Mortality in the Medicare Population. N Engl J Med. 2017;377(15):1498–9.
9. Boogaard H, Samoli E, Patton AP, Atkinson RW, Brook JR, Chang HH, Hoffmann B, Kutlar Joss M, Sagiv SK, Smargiassi A, et al. Long-term exposure to traffic-related air pollution and non-accidental mortality: a systematic review and meta-analysis. Environ Int. 2023;176:107916.
10. Karimi B, Samadi S. Long-term exposure to air pollution on cardio-respiratory, and lung cancer mortality: a systematic review and meta-analysis. J Environ Health Sci Eng. 2024;22(1):75–95.
11. Edwards L, Wilkinson P, Rutter G, Milojevic A. Health effects in people relocating between environments of differing ambient air pollution concentrations: a literature review. Environ Pollut. 2022;292(Pt A):118314.
12. Awad YA, Di Q, Wang Y, Choirat C, Coull BA, Zanobetti A, Schwartz J. Change in PM2.5 exposure and mortality among Medicare recipients: combining a semi-randomized approach and inverse probability weights in a low exposure population. Environ Epidemiol. 2019;3(4):e054.
13. Krewski D, Burnett RT, Goldberg MS, Hoover BK, Siemiatycki J, Jerrett M, Abrahamowicz M, White WH. Overview of the reanalysis of the Harvard Six Cities Study and American Cancer Society Study of Particulate Air Pollution and Mortality. J Toxicol Environ Health A. 2003;66(16–19):1507–51.

14. Gan WQ, Tamburic L, Davies HW, Demers PA, Koehoorn M, Brauer M. Changes in residential proximity to road traffic and the risk of death from coronary heart disease. Epidemiology. 2010;21(5):642–9.

15. United States Environmental Protection Agency. Advisory Council on Clean Air Compliance Analysis Response to Agency request on Cessation lag. In.; 2004.

16. Clancy L, Goodman P, Sinclair H, Dockery DW. Effect of air-pollution control on death rates in Dublin, Ireland: an intervention study. Lancet. 2002;360(9341):1210–4.

17. Leksell I, Rabl A. Air pollution and mortality: quantification and valuation of years of life lost. Risk Anal. 2001;21(5):843–57.

18. Walton H. Supporting paper to COMEAP 2010 report the Mortality effects of Long term exposure to Particulate Air Pollution in the United Kingdom. In.; 2010.

19. Laden F, Schwartz J, Speizer FE, Dockery DW. Reduction in fine particulate air pollution and mortality: extended follow-up of the Harvard Six Cities study. Am J Respir Crit Care Med. 2006;173(6):667–72.

20. Ben-Shlomo Y, Smith GD, Shipley MJ, Marmot MG. What determines mortality risk in male former cigarette smokers? Am J Public Health. 1994;84(8):1235–42.

21. Knoke JD, Burns DM, Thun MJ. The change in excess risk of lung cancer attributable to smoking following smoking cessation: an examination of different analytic approaches using CPS-I data. Cancer Causes Control. 2008;19(2):207–19.

22. United States Department of State. Foreign Affairs Manual. In.; 2022.

23. Edwards L, Wilkinson P, Rutter G, Iverson L, Milojevic A. Impact of mitigation measures to improve home indoor air quality in Kathmandu, Nepal. Environ Research: Health 2023, 1.

24. Barkjohn KK, Norris C, Cui X, Fang L, Zheng T, Schauer JJ, Li Z, Zhang Y, Black M, Zhang JJ, et al. Real-time measurements of PM2.5 and ozone to assess the effectiveness of residential indoor air filtration in Shanghai homes. Indoor Air. 2021;31(1):74–87.

25. Padro-Martinez LT, Owusu E, Reisner E, Zamore W, Simon MC, Mwamburi M, et al. A randomized cross-over air filtration intervention trial for reducing cardiovascular health risks in residents of public housing near a highway. Int J Environ Res Public Health. 2015;12(7):7814–38.

26. Deng GL, Wang Z, Gao J, Xu Z, Li J. Indoor/outdoor relationship of PM2.5 concentration in typical buildings with and without air cleaning in Beijing. Indoor and Built Environment. 2017;26:60–8.

27. Air. quality database [https://www.who.int/data/gho/data/themes/air-pollution/who-air-quality-database]

28. Air Quality Trends. (2001–2018) [https://www3.epa.gov/region9/air/trends/pm25-annual.html]

29. Meng J, Li C, Martin RV, van Donkelaar A, Hystad P, Brauer M. Estimated long-term (1981–2016) concentrations of ambient fine particulate matter across North America from Chemical Transport Modeling, Satellite Remote Sensing, and Ground-based measurements. Environ Sci Technol. 2019;53(9):5071–9.

30. Miller BG, Hurley JF. Life table methods for quantitative impact assessments in chronic mortality. J Epidemiol Community Health. 2003;57(3):200–6.

31. Leavitt M. Advisory Council on Clean Air Compliance Analysis Response to Agency request on Cessation lag. In.; 2004.

32. Kenfield SA, Wei EK, Stampfer MJ, Rosner BA, Colditz GA. Comparison of aspects of smoking among the four histological types of lung cancer. Tob Control. 2008;17(3):198–204.

33. Centers for Disease Control and Prevention. National Vital Statistics System, Mortality 1999–2020 on CDC WONDER Online Database. In.; 2021.

34. United States Department of State. Survey data from posts, unpublished data. In.; 2018.

35. United States Census Bureau. American Community Survey 5-Year Data (2009–2021). In.; 2021.

36. Chowdhury S, Pozzer A, Haines A, Klingmuller K, Munzel T, Paasonen P, Sharma A, Venkataraman C, Lelieveld J. Global health burden of ambient PM(2.5) and the contribution of anthropogenic black carbon and organic aerosols. Environ Int. 2022;159:107020.

37. Christidis T, Erickson AC, Pappin AJ, Crouse DL, Pinault LL, Weichenthal SA, Brook JR, van Donkelaar A, Hystad P, Martin RV, et al. Low concentrations of fine particle air pollution and mortality in the Canadian Community Health Survey cohort. Environ Health. 2019;18(1):84.

38. Keidel D, Anto JM, Basagana X, Bono R, Burte E, Carsin AE, Forsberg B, Fuertes E, Galobardes B, Heinrich J et al. The role of Socioeconomic Status in the Association of Lung Function and Air Pollution-A Pooled Analysis of three adult ESCAPE cohorts. Int J Environ Res Public Health 2019, 16(11).

39. Krewski D, Jerrett M, Burnett RT, Ma R, Hughes E, Shi Y, Turner MC, Pope CA 3rd, Thurston G, Calle EE et al. Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality. Res Rep Health Eff Inst 2009(140):5–114; discussion 115–136.

40. Khan MAB, Grivna M, Nauman J, Soteriades ES, Cevik AA, Hashim MJ, Govender R, Al Azeezi SR. Global incidence and mortality patterns of Pedestrian Road Traffic injuries by Sociodemographic Index, with forecasting: findings from the Global Burden of Diseases, injuries, and risk factors 2017 study. Int J Environ Res Public Health 2020, 17(6).

41. World Health Organization. World health statistics 2023: monitoring health for the SDGs, sustainable development goals. In.; 2023.

42. Global Buden of Disease. Global, regional, and national incidence, prevalence, and years lived with disability for 354 diseases and injuries for 195 countries and territories, 1990–2017: a systematic analysis for the global burden of Disease Study 2017. Lancet. 2018;392(10159):1789–858.

43. Lleras-Muney A. The needs of the Army: using Compulsory Relocation in the military to Estimate the Effect of Air pollutants on children's Health. J Hum Resour 2010, 45.

44. Cooper E, Milner J, Wang Y, Stamp S, Mumovic D. Modelling the impact on mortality of using portable air purifiers in UK homes. Atmos Environ 2022, 289.

45. Fisk WJ, Chan WR. Effectiveness and cost of reducing particle-related mortality with particle filtration. Indoor Air. 2017;27(5):909–20.

46. Mannan M, Al-Ghamdi SG. Indoor air quality in buildings: a Comprehensive Review on the factors Influencing Air Pollution in Residential and Commercial structure. Int J Environ Res Public Health 2021, 18(6).

47. Kelly F, Fussell JC. Improving indoor air quality, health and performance within environments where people live, travel, learn and work. Atmos Environ. 2019;200:90–109.

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