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A Method to Estimate the Chronic Health Impact of Air Pollutants in U.S. Residences

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Abstract

Background: Indoor air pollutants (IAPs) cause multiple health impacts. Prioritizing mitigation options that differentially impact individual pollutants and comparing IAPs to other environmental health hazards requires a common metric of harm.

Objectives: The objective was to demonstrate a methodology to quantify and compare health impacts from IAPs. The methodology is needed to assess population health impacts of large-scale initiatives – including energy efficiency upgrades and ventilation standards – that affect indoor air quality (IAQ).

Methods: Available disease incidence and disease impact models for specific pollutant-disease combinations were synthesized with data on measured concentrations to estimate the chronic heath impact, in Disability Adjusted Life Years (DALYs), due to inhalation of a subset of IAPs in U.S. residences. Model results were compared to independent estimates of DALYs lost due to disease.

Results: $PM_{2.5}$, acrolein, and formaldehyde accounted for the vast majority of DALY losses caused by IAPs considered in this analysis, with impacts on par or greater than estimates for secondhand tobacco smoke and radon. Confidence intervals of DALYs lost derived from epidemiology-based response functions are tighter than those derived from toxicology-based, inter-species extrapolations. Statistics on disease incidence in the US indicate that the upper-bound confidence interval for aggregate IAP harm is implausibly high.

Conclusions: The demonstrated approach may be used to assess regional and national initiatives that impact IAQ at the population level. Cumulative health impacts from inhalation in U.S. residences of the IAPs assessed in this study are estimated at 400–1100 DALYs annually per 100,000 people.

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Abbreviations

BoD	Burden of Disease	CRA	Cumulative Risk Assessment
DALYs	Disability-Adjusted Life Years	HAPs	Hazardous Air Pollutants
IAPs	Indoor Air Pollutants	IAQ	Indoor Air Quality
ID	Intake-DALYs	IND	Intake-Incidence-DALYs
SHS	Second-Hand Smoke	WHO	World Health Organization

Introduction

Air pollutant concentrations in many homes exceed health-based standards for chronic and acute exposures (Logue et al. 2011). On average, Americans spend more than 65% of their time in residences (Klepeis et al. 2001), and numerous studies have noted the importance of the indoor environment to cumulative air pollutant intake (Samet 1993; Weisel et al. 2005). Impact assessment methods have been applied to estimate aggregate chronic health impacts for outdoor air pollution (EPA 1999; Muller and Mendelsohn 2007) and from pollutant inhalation in office buildings (Fisk et al. 2011). Yet, to our knowledge, no study has yet considered both disease incidence and severity to assess aggregate health impacts of air pollutant inhalation in residences.

Air pollutants known to be hazardous based on epidemiological and toxicological information include "criteria pollutants" specified in the 1970 Clean Air Act Amendments and hazardous air pollutants (HAPs) specified in the 1990 Clean Air Act Amendments. There is growing concern that some bio-accumulating semi-volatile organic compounds and ultrafine particles – both ubiquitously present in residences – may cause substantial adverse health effects at typical environmental levels. However, there is currently insufficient toxicological data to quantify that impact. The U.S. EPA and the California EPA each publish health standards or guidelines for long term exposure concentrations to protect against cancer and non-cancer chronic effects. The hazard associated with residential air pollutant exposure can be quantified as the percentage of homes that exceed specified non-cancer standards or as the incremental risk of cancer incidence across the population. These methods consider only disease potential for non-cancer endpoints and disease incidence for cancer; they do not incorporate disease severity. Quantitatively comparing impacts of individual residential indoor air pollutants, and comparing their estimated cumulative health impact to that of other environmental hazards requires a single metric that includes both disease incidence and severity. A comprehensive metric will facilitate the evaluation of residential indoor air quality (IAQ) interventions including source control measures and ventilation.

Epidemiological and toxicological research has contributed to the development of tools to bridge the gap from measured pollutant exposure levels to disease incidence and from disease incidence to health costs in Disability Adjusted Life Years (DALYs). Concentration-response relationships have been quantified for criteria pollutants. The U.S. EPA aggregated several of these disease incidence models for use in the cost benefit analysis of the Clean Air Act (1999). Several studies have estimated the health impact per incidence of specific diseases (Hong et al. 2010; Lvovsky et al. 2000; Melse et al. 2010). Huijbregts et al. (2005) published cumulative impact and effect factors for exposure to air pollutants including air toxics and ozone. These models provide the basis for performing a human health impact assessment for inhalation of indoor air pollutants.

In this study we combined disease incidence and DALY-based health impact models to develop a methodology to estimate the population average health costs due to chronic inhalation of a broad suite of air pollutants in U.S. residences. We first analyzed published data to calculate

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mean exposure concentrations and estimated age-dependent inhalation intakes. We used disease incidence and disease impact models to predict pollutant-specific impacts and total DALY-based health costs to identify the residential IAPs that have the greatest impacts on health in the U.S. As a check on the method, and the estimated aggregate impact of IAP, we compare our findings to independent estimates of DALY losses related to SHS, to diseases that could potentially result from air pollutant exposure, and to all non-communicable, non-psychiatric diseases in the U.S.

Methods

Indoor Air Pollutant Intake. To calculate pollutant inhalation in U.S. residences, we used a data compilation described by Logue et al. (2011) that includes summary statistics from 77 studies that reported residential air pollutant concentration measurements in the U.S. and other countries with similar lifestyles. The aggregate data were used to calculate concentrations relevant to assessing chronic residential exposures to 267 chemical air pollutants. Seventy of the pollutants had sufficient toxicological and epidemiological data to calculate chronic health impact using the methodology described below, and were included in this study. They are listed in Table 1. Our analysis did not extend to contaminants from biological sources such as molds and allergens. We thus refer to the suite of pollutants considered as "non-biological".

Determining Annual Population Health Impact. The annual health impact of residential indoor air pollutants was calculated by considering the total intake in residences as an increment adding to intake in other environments. The increment was calculated by considering in-home inhalation of air containing the population-mean exposure concentrations relative to the theoretical case of the population inhaling residential air containing no pollutants.

The DALY metric allows quantification and comparison of the health costs from varied disease endpoints that can result from various pollutants. As a measure of equivalent years of life lost due to illness or disease, the DALY quantifies overall disease costs (impacts) due to both mortality and morbidity. DALYs include years of life lost (YLL) to premature mortality and equivalent life years lost to reduced health or disability (YLD). For each disease, the DALYs lost per incidence are equal to:

$$DALY_{disease} = YLL_{disease} + YLD_{disease} \quad [1]$$

The equivalent life years lost to reduced health are weighted from 0 to 1 based on the severity of the disease. For example, a five-year illness that reduces quality of life to 4/5 that of a healthy year is valued at 1 DALY lost.

Several authors have determined the DALYs lost per incidence of specific diseases using the preeminent work of Murray and Lopez (Huijbregts et al. 2005; Lvovsky et al. 2000; Murray and Lopez 1996a, b; WHO 2009). Multiplying disease incidence by a "DALY factor" yields total DALYs lost per disease incidence.

$$DALYs = (\partial DALYs / \partial Disease\ Incidence) * Disease\ Incidence \quad [2]$$

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Equation 2 uses a partial derivative in recognition that DALY losses are incrementally impacted by causes other than disease. The total burden of disease in a community can be calculated as the aggregate, across all diseases, of DALY factors multiplied by disease incidence rates.

Our analysis used two approaches to calculate DALY losses from estimated exposure concentrations. For criteria pollutants (ozone, NO_2, $PM_{2.5}$, SO_2, and CO) we used an Intake-Incidence-DALY (IND) method that uses epidemiology based concentration-response functions to quantify disease incidence rates; these are combined with estimates of DALYs per disease incidence reported in the literature. For non-criteria pollutants we used an Intake-DALY (ID) approach used the work of Huijbregts et al. (2005) to calculate the health impact associated with intake of non-criteria pollutants based on animal toxicity literature. The IND approach is preferred since it does not require interspecies extrapolations that generally involve larger uncertainties than the epidemiologically based concentration-response functions. However, the IND approach can only be used for pollutants with information on concentration-response functions in humans. Ozone was the only pollutant for which both the ID and IND approaches could be applied.

While the disease incidence relationships in the IND and ID approaches are accepted health impact models, they are, nevertheless, simplifications of population wide responses to chronic inhalation exposure. Our approaches use linear (IND) and nearly linear (ID) disease incidence models without effect thresholds. For these types of disease incidence models, only the mean of the concentration distribution is needed to estimate population impact. Existence of a threshold concentration for disease incidence, or a strongly non-linear disease to intake response would necessitate accurate determination of the shape of the population intake distribution. A discussion of the impact of threshold effects on our DALYs estimates is included in the Discussion section. The potential impacts of non-linear response functions are beyond the scope of the current study.

The Intake-Incidence-DALY (IND) Approach. The first step of the IND method comprised the application of Concentration-Response (C-R) functions to determine disease incidence. For almost all of the disease outcomes the C-R function follows the formula:

$$\Delta Incidence = -[y_o * (\exp(-\beta \Delta C_{exposure}) - 1] * population \quad [3]$$

where y_o is the baseline prevalence of illness per year, β is the coefficient of the concentration change, and *population* is the number of people exposed. For each pollutant and outcome, y_o and β vary. Respiratory illness due to long-term NO_2 intake uses a slightly different C-R functional form, but still relies on a β with specified uncertainty

When aggregating concentration response functions and DALY factors we tried to include all of the diseases with available established relationships between concentrations and disease incidence. We did not include diseases/outcomes that were negligible compared to the other diseases included. The health endpoints selected and DALYs per incidence of disease are summarized in Table 2.

Chronic $PM_{2.5}$ exposure affects both the respiratory and cardiovascular systems. The three outcomes that we included were all-cause mortality, chronic bronchitis, and stroke. Pope et

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al. (2002) predicted incidence rates of all-cause mortality and the average years of life lost per unit increase in $PM_{2.5}$ (Pope et al. 2009); we divided the former by the latter to get DALYs lost per incidence. The 95th percentile range of the DALYs per death was set to represent the span of values seen in the literature (Lvovsky et al. 2000; Pope et al. 2009). Recent studies have shown that chronic $PM_{2.5}$ exposure can lead to heart disease and thickening of arterial walls (Künzli et al. 2004). The total impact of $PM_{2.5}$ on cardiovascular health is not known. However, recent work by Miller et al. (2007) has shown associations between chronic $PM_{2.5}$ and stroke, an outcome of heart disease, in women. The endpoint of non-fatal stroke was included in the analysis using the hazard ratios derived by Miller et al. (2007) for both men and women. This is likely an underestimation of the total impact of $PM_{2.5}$ on heart disease. The DALYs lost per non-fatal stroke incidence were taken from Brook et al. (2010). The incidence of stroke predicted was split between 0, 1, and >1 complications and the percentage of stroke that resulted in death was determined based on the findings of Brook et al. (2010). Burnett et al. (1999) developed a concentration response function for hospital admissions associated with long term $PM_{2.5}$ exposure. However, since the impact was negligible compared to the impact of mortality, chronic bronchitis and stroke, we did not include this outcome. There is evidence that $PM_{2.5}$ exposure is associated with other health outcomes including diabetes and reduced lung function; however, these findings are relatively new and have not been included in this work.

For carbon monoxide and sulfur dioxide, the only outcomes relevant to chronic exposure appear to be hospital admissions. Chronic ozone and NO_2 exposure have been associated with early death and respiratory illness respectively. The input parameters into the C-R functions for these outcomes are the same as those used in the EPA cost benefit analysis of the Clean Air Act (EPA 1999). For hospital admission and respiratory illness, we used the DALYs/incidence values available in the literature. For ozone mortality, as with $PM_{2.5}$, it is unclear how much life is lost due to early death. Values in the literature range from a few weeks to 10 years. We chose a large range of values to represent this uncertainty (Levy et al. 2001; Lvovsky et al. 2000).

The C-R functions are formulated to calculate the increment of disease incidence per increment of exposure concentration, not total disease incidence for a given exposure concentration. According to population-weighted demographics (Klepeis et al. 2001; US Census Bureau 2010), summarized in Table 3, the 'average' American spends 70% of the time in residences. The chronic exposure-relevant concentration contributed from indoor exposure was therefore set to 70% of the indoor concentration.

$$\Delta C_{exposure} = 0.7 C_{indoors} \quad [4]$$

Incidence rates were combined with DALY factors to calculate total health impacts by pollutant (Equation 2). A Monte-Carlo approach was used to calculate impacts by pollutant by sampling with replacement from the available distributions of DALY factors and β. We assumed that all DALY factor distributions are log-normal.

The Intake-DALYs (ID) Approach. The ID approach extrapolated directly from indoor concentrations to total DALYs lost due to intake of specific pollutants. From this standpoint, it was convenient to rewrite Equation 2 as:

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$$DALYs=(\partial DALY/\partial Disease\ Incidence)*(\partial Disease\ Incidence/\partial intake)*intake \quad [5]$$

where *intake* is the mass of pollutant that an individual inhales over a given time frame. Huijbregts et al. (2005) computed expected ranges of human impact for cancer and non-cancer chronic effects of 1,192 substances, applying equal weightings for a year lost, independent of age (i.e. zero discounting). Using the values determined by Huijbregts et al. (2005), the DALYs lost for one year of breathing pollutant *i* is calculated using the following equations:

$$DALYs_i=(\partial DALY/\partial intake)*intake \quad [6]$$

$$DALYs_i=C_i*V*[(\partial DALY_{cancer}/\partial intake)_i*ADAF+(\partial DALY_{non\text{-}cancer}/\partial intake)_i] \quad [7]$$

where $\partial DALY/\partial intake_i$ are the cancer and non-cancer mass intake-based DALY factors, C_i is the indoor concentration, V is volume of air breathed in the residence each year, and ADAF is the age-dependent adjustment factor for cancer exposure as described below.

The age at which carcinogens are inhaled has an appreciable effect on total toxicity; therefore the EPA has developed ADAFs to calculate cancer health impact as a function of exposure age (EPA 2005). To align with EPA-recommended ADAFs, we considered three age groups: under 2, 2-16, and over 16 years of age (EPA 2005). A population-weighted average annual air intake volume and ADAF were calculated by combining age distribution of the U.S. population, age-specific inhalation rates and time spent at home (Table 3).

Huijbregts et al. (2005) presented, for each chemical, both a central estimate (50th percentile value) and the estimated uncertainty of the DALYs per mass-intake of pollutant; uncertainty was assumed to be log-normal, characterized by a factor, k_i, equal to:

$$k_i=(97.5th\ percentile/2.5th\ percentile)^{0.5} \quad [8]$$

which includes the aggregated uncertainty of the rate of disease incidence as well as the uncertainty in the DALYs per incidence of disease. We used a Monte-Carlo approach to sample with replacement from uncertainty distributions of DALY factors derived from the central estimate of the DALY factor and the k_i value, to determine the central estimate and 95th percentile confidence interval (CI) for combined cancer and non-cancer DALYs for each pollutant. A Monte-Carlo approach was also used to determine the total DALYs lost from all of the pollutants analyzed using the IND and ID methods.

Despite the availability of a DALY factor for bromomethane, DALY-based impacts are not presented for this compound because the limited available concentration data (NYDOSH 2006) appear more indicative of a local outdoor source than general conditions in U.S. homes (Logue et al. 2011).

Radon, Second Hand Smoke (SHS), and Acute CO Poisoning Deaths. The population average DALYs lost to radon, SHS, and acute CO poisoning deaths were determined based on estimates of disease incidence from the literature. We include DALY loss estimates for these pollutants for two reasons: 1) to compare health impacts calculated for a subset of SHS pollutants using the IND and ID methodologies to independent estimates of overall DALY losses associated with SHS exposure (as described below), and 2) to compare estimated IAP-associated DALY losses calculated in the current study to estimates for these three established indoor health hazards.

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To estimate the health impact from radon, SHS and acute CO, we used Equation 2 with disease incidence estimates from the literature, summarized in Table 4. For radon and acute CO poisoning, only the endpoint of premature death was used to estimate DALY losses. The DALYs lost per incidence of various SHS outcomes and per early mortality due to acute CO poisoning and radon were taken from the literature and are also summarized in Table 4.

Comparison to DALYs Estimated by Other Methods. Results from this study were compared to three other estimates of population-wide DALYs for the U.S. While our study used an impact assessment approach, the studies used for comparison are Cumulative Risk Assessment (CRA) and Burden of Disease (BoD) studies (Ezzati and Lopez 2004; McKenna et al. 2005; WHO 2009). The BoD studies used available statistics to determine the disease incidence rate as a function of age, sex, and geographical location. They then assigned a DALY value based on the years of life lost and disability incurred. The CRA studies determined the fraction of disease or death attributable to a specific risk factor based on epidemiological studies of specific populations. This is similar to, though far more complex than, than our method of estimating health impact due to SHS and radon. If the disease rate and DALY factors were accurate and we used the same discount ratings and time weightings for the age at which years of life are lost, both methods should estimate the same number of DALYs lost associated with a specific risk factor. Indoor air, independent of the impact of household use of solid fuels, had not been studied in a CRA analysis thus far. We compare results from our methodology to CRA results with the caveat that the methods are far from equivalent and the comparison should be seen only as a point of reference. The comparison also provides a useful tool for bounding uncertainties for our impact assessment method.

The World Health Organization (WHO) compiled disease incidence data for all communicable and non-communicable diseases and injuries to determine the total number of DALYs lost per year for 192 countries (WHO 2009). McKenna et al. (2005) aggregated United States' mortality and morbidity data to determine the top 20 causes of DALY losses for men and women in 1996. Ezzati and Lopez (2004) estimated the total DALYs lost from smoking and tobacco use in industrialized nations by determining the impact of disease beyond what would be expected in non-smoking homes. The total DALYs that we estimated for all IAPs analyzed with the ID and IND methods were compared to estimates from these studies to discern whether the full CI of the aggregate IAP impact of indoor residential air is plausible. Additionally we used our ID and IND methodology to calculate health impact for a suite of measured SHS components and we compared the aggregate CI of the DALYs lost for these components to CRA-derived DALY estimates.

SHS is a complex mixture of chemicals. Nazaroff and Singer (2004) estimated increases in specific volatile organic compound concentrations (1,3-butadiene, 2-butanone, acetaldehyde, acetonitrile, acrolein, acrylonitrile, benzene, ethyl benzene, formaldehyde, naphthalene, phenol, styrene, toluene, and xylenes) expected for average smoking activity. Simons et al. (2007) found that homes with smokers on average had $PM_{2.5}$ concentrations that were 16 $\mu g/m^3$ higher than the homes of non-smokers. We applied the IND and ID modeling frameworks established here to determine the additional DALYs lost due to living in a household that had indoor concentrations

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elevated by the specified levels. We used the Monte-Carlo sampling to determine an aggregate CI for the DALYs lost due to exposure to this chemical mixture.

Results

Figure 1 shows the estimated number of DALYs lost due to indoor inhalation intake of HAPs and ozone based on the ID approach. Formaldehyde and acrolein had the largest estimated number of DALYs, 46 (95% CI 0.2-14,000) and 47 (95% CI 2.4-1050) respectively, higher than the upper-bound of the CI for all but two other pollutants, ozone and acetaldehyde. Of the 65 pollutants compared using the ID method, only 15 had 95% CIs that overlapped with the CI for formaldehyde.

Figure 2 plots the disease incidence and DALYs using the IND method for chronic exposure to criteria pollutants. The estimated DALYs lost due to incidences of stroke, chronic bronchitis, and premature death due to $PM_{2.5}$ contributed substantially to annual health impact. Mortality due to ozone is also a significant contributor to the total DALYs. The estimated number of DALY losses associated with hospitalization was relatively low for each pollutant. NO_2 is potentially a significant acute health hazard, but we did not consider acute effects in this analysis. Non-lethal chronic exposure to carbon monoxide (CO) and sulfur dioxide are not substantial contributors to DALYs from the outcomes we evaluated. There is concern that indoor concentrations of CO may have an adverse effect on certain susceptible populations (EPA 2010). Currently there is insufficient empirical evidence to reliably quantify the health impact from chronic CO exposure.

Figure 3 shows the estimated DALYs from exposure to the 12 analyzed IAPs with the highest DALYs per year per 100,000 people. Also shown in this figure are estimates of DALYs lost per 100,000 people per year attributed to SHS (51; 95% CI of 42 to 60), acute CO deaths (4.9; 95% CI of 4.7 to 5.1)), and radon exposure for smokers (79; 95% CI of 25 to 255) and non-smokers (13; 95% CI of 4 to 42). For smokers, we are overestimating the DALYs attributable to radon per se because a portion of the DALYs for smokers exposed to radon would result solely from smoking. For ozone, the ID and IND approaches estimated an annual DALYs per 100,000 people of 6.7 (95% CI: 0.3-160) and 2.3 (95% CI: 0.2-26) respectively. There is substantial overlap in the CIs for both approaches, though the IND CI is smaller. These results suggest that $PM_{2.5}$, acrolein, formaldehyde, radon, and SHS are the most harmful non-biological air pollutants in residences on a population basis. In addition, we calculated that intake of the subset of compounds in SHS noted above would cause an annual loss of 1000 DALYs (95% CI: 300–14000) per 100,000 individuals living in households with SHS, and a population averaged annual loss of 110 DALYs (95% CI: 40–1600) per 100,000 residents in all U.S. households.

Our analysis yielded a central estimate for the DALYs lost due to all IAPs analyzed using the ID and IND methods of 1100 DALYs per 100,000 people (95% CI: 400-13000) per year. For 80% of the Monte-Carlo samples, indoor $PM_{2.5}$ was associated with the largest number of DALYs, while acrolein and formaldehyde were the dominant contributors for 16% and 4% of the samples, respectively, and another IAP was the dominant contributor other than these three in less than 0.25% of the runs. For 90% of the samples, acrolein, formaldehyde, and $PM_{2.5}$

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contributed more than 80% of the total DALYs. This reinforces the finding that these three pollutants account for the majority of chronic health impact from intake of indoor air in non-smoking homes.

Discussion

Although there is large uncertainty in the number of DALYs estimated for each pollutant by the ID and IND models, several clear findings emerge. Our analysis demonstrates that in the majority of U.S. residences $PM_{2.5}$, acrolein, and formaldehyde dominate health impacts due to chronic exposures to non-biological air pollutants. The DALYs from these three pollutants appears to be much larger than the DALYs due to CO deaths from acute poisoning in homes. SHS and radon are also significant contributors to population-wide DALYs, but these exposures occur in a smaller fraction of homes.

Formaldehyde is primarily emitted from materials throughout the home. Acrolein is primarily emitted from materials and cooking (Seaman et al. 2007). $PM_{2.5}$ concentrations indoors, unlike acrolein and formaldehyde, are due to both indoor and outdoor sources and outdoor concentrations may exceed indoor levels in many locations (Weisel et al. 2005).

Our analysis yielded a central estimate of 1100 DALYs per 100,000 people (95% CI: 400-13000) per year for IAPs, excluding radon and SHS. For the United States overall, the World Health Organization (2009) estimated a total burden of 7700 DALYs per year per 100,000 for all non-communicable, non-psychiatric diseases combined. McKenna et al. (2005) identified the top 20 diseases that drive the health burden in the United States. Among diseases they identified, the ones that could be impacted by indoor air cause 3000 DALYs to be lost per 100,000 people per year. Ezzati and Lopez (2004) estimated that the population average burden of both firsthand (smokers) and secondhand tobacco smoke in industrialized nations is 12% of the annual DALYs lost, which we assume would represent 1700 DALYs per 100,000 people per year, i.e., 12% of the total DALYs estimated for the U.S. by the WHO (2009). Estimated DALYs due to indoor $PM_{2.5}$, acrolein, and formaldehyde combined (1100, 95% CI 700-13000) were substantially greater than DALYs due to the remaining 67 indoor air pollutants analyzed using the IND and ID methods combined (40, 95% CI 10-70).

Our estimate of DALYs lost to SHS components in the 11% of homes estimated to have SHS is of similar magnitude to the mean estimate of DALYs from indoor air pollutant inhalation in non-smoking homes. For the SHS analysis, pollutants that contribute the most to DALYs are again $PM_{2.5}$ and acrolein. Having a smoker in a residence, on average, doubles the concentrations of these two components relative to homes without smokers (Nazaroff and Singer 2004; Simons et al. 2007) , effectively doubling the DALY estimates. The complete chemical mixture of SHS should be more toxic than the limited subset of components examined here, but the DALYs estimate derived from the literature-reported health endpoints due to SHS is in the lower bound of the 95th percentile CI. This result suggests that the component-based method used in his paper may tend to overestimate DALYs or that an insufficient number of health endpoints is attributed to SHS.

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Both the IND and ID approaches rely on no-threshold disease incidence models that are linear or effectively linear over the concentration range of the analysis. The health impacts of $PM_{2.5}$ are broadly thought to be linear at low doses (Schwartz et al. 2002). Threshold effects may significantly reduce the actual health impacts due to formaldehyde (a cancer hazard) and acrolein exposure (a non-cancer hazard). While some studies have identified genotoxic effects for formaldehyde (Viegas et al. 2010), others have identified strong threshold effects (Salthammer and Bahadir 2009). Various thresholds for exposure to avoid cancer have been suggested ranging from 120 $\mu g\ m^{-3}$ suggested by the German Federal Institute for Risk Assessment and WHO to 12 $\mu g\ m^{-3}$ by Naya and Nakanishi (2005). Given the distribution of formaldehyde concentrations determined for residences, threshold effect levels of 12 $\mu g\ m^{-3}$ and 120 $\mu g\ m^{-3}$ would result in 32% and 87% reductions in predicted DALY losses, respectively. A high threshold would result in $PM_{2.5}$ and acrolein being the prime indoor pollutants of concern and formaldehyde being of lesser importance.

There is less available information on threshold levels for acrolein exposure. We determined the impact on the DALYs estimate for a threshold equal to the California EPA non-cancer reference exposure level (0.35 $\mu g\ m^{-3}$). Given the determined distribution of acrolein concentrations indoors, this would result in a 20% reduction in DALYs.

Conclusion

Using the methodology established here, we estimated that the total annual health impact of IAP inhalation in U.S. residences, excluding radon and SHS, is 1100 DALYs per 100,000 people (95% CI: 400-13000). The upper bound of the range was twice as high as the number of DALYs due to all non-communicable, non-psychiatric diseases as estimated by the WHO (2009) based on disease statistics. The upper-bound of the CI estimated for the DALYs lost due to exposure to a subset of pollutants in SHS using the methodology was also too high. The total annual DALYs lost due to IAPs is likely in the lower half of the calculated range, i.e. between the central estimate and lower bound of the 95th percentile CI: 400-1100 DALYs per 100,000 people.

Since the upper-bound CI for all IAP-related DALY losses is too high, the upper-bound for CIs of at least some of the individual pollutants included must also be too high. Since the vast majority of the total DALYs were due to acrolein, $PM_{2.5}$ and formaldehyde, further statistical analysis may be able to narrow the currently large CIs for impacts from these pollutants.

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References

Abbey, DE, Ostro, BE, Petersen, F, Burchette, RJ. 1995. Chronic respiratory symptoms associated with estimated long-term ambient concentrations of fine particulates less than 2.5 microns in aerodynamic diameter (PM2.5) and other air pollutants. Journal of Exposure Analysis and Environmental Epidemiology 5(2): 137-159.

Brook, RD, Rajagopalan, S, Pope, CA, III, Brook, JR, Bhatnagar, A, Diez-Roux, AV, et al. 2010. Particulate Matter Air Pollution and Cardiovascular Disease: An Update to the Scientific Statement From the American Heart Association. Circulation 121(21): 2331-2378.

Burnett, RT, Smith-Doiron, D, Stieb, D, Cakmak, S, Brook, JR. 1999. Effects of particulate and gaseous air pollution on cardiorespiratory hospitalizations. Archives Environmental Health 54(2): 130-139.

Cal EPA. 2005. Proposed Identification of environmental Tobacco Smoke as a Toxic Air Contaminant. Part B: Health Effects.California Environmental Protection Agency, Office of Environemental Health Hazard Assessment, Air Toxicology and Epidemiology Branch.

CDC. 2007. Carbon Monoxide--Related Deaths --- United States, 1999--2004. MMWR 56(50): 1309-1312.

de Hollander, AEM, Melse, JM, Lebret, E, Kramers, PGN. 1999. An aggregate public health indicator to represent the impact of multiple environmental exposures. Epidemiology 10(5): 606-617.

EPA. 1999. The Benefits and Costs of the Clean Air Act 1990 to 2010. Washington, D.C.:Environmental Protection Agency.

EPA. 2003. EPA Asessment of Risks from Radon in Homes. Washington, DC:Office of Radiation and Indoor Air, US Environmental Protection Agency.

EPA. 2005. Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens. Washington, DC:United States Environmental Protection Agency.

EPA. 2009. Exposure Factors Handbook: 2009 Update.US Environmental Protection Agency.

Logue JM, Price PN, Sherman MH, Singer BC, 2011 A Method to Estimate the Chronic Health Impact of Air Pollutants in U.S. Residences. Environ Health Perspectives doi:10.1289/ehp.1104035 LBNL- 5267E

EPA. 2010. Quantitative Risk and Exposure Assessment for Carbon Monoxide -Amended. Research Triangle Park, NC:US Environmental Protection Agency, Office of Air Quality Planning and Standards, Health and Environmental Impacts Division.

Ezzati, M, Lopez, AD. 2004. Smoking and Oral Tobacco Use. In: Comparative quantification of health risks: Global and Regional Burden of Disease Attributable to Selected Major Risk Factors, Vol. 1, (Ezzati M, Lopez AD, Rodgers A, Murray CJL, eds). Geneva:World Health Organization, 76.

Fisk, WJ, Black, D, Brunner, G. 2011. Benefits and costs of improved IEQ in U.S. offices. Indoor Air 21(5): 357-367.

Hasselblad, V, Eddy, DM, Kotchmar, DJ. 1992. Synthesis of Environmental Evidence - Nitrogen Dioxide Epidemiology Studies. Journal of Air and Waste Management 42(5): 662-671.

Hong, K-S, Saver, JL, Kang, D-W, Bae, H-J, Yu, K-H, Koo, J, et al. 2010. Years of Optimum Health Lost Due to Complications After Acute Ischemic Stroke: Disability-Adjusted Life-Years Analysis. Stroke 41(8): 1758-1765.

Huijbregts, MAJ, Rombouts, LJA, Ragas, AMJ, van de Meent, D. 2005. Human-toxicological effect and damage factors of carcinogenic and noncarcinogenic chemicals for life cycle impact assessment. Integrated Environmental Assessment and Management 1(3): 181-244.

Jerrett, M, Burnett, RT, Pope, CA, III, Ito, K, Thurston, G, Krewski, D, et al. 2010. Long-Term Ozone Exposure and Mortality. The New England Journal of Medicine 360(11): 1085-1095.

Klepeis, NE, Nelson, WC, Ott, WR, Robinson, JP, Tsang, AM, Switzer, P, et al. 2001. The National Human Activity Pattern Survey (NHAPS): A Resource for Assessing Exposure to Environmental Pollutants. Journal of Exposure Analysis and Environmental Epidemiology 11: 231-252.

Künzli, N, Jerrett, M, Mack, WJ, Beckerman, B, LaBree, L, Gilliland, F, et al. 2004. Ambient Air Pollution and Atherosclerosis in Los Angeles. Environ Health Perspect 113(2).

Levy, JI, Carrothers, TJ, Tuomisto, JT, Hammitt, JK, Evans, JS. 2001. Assessing the Public Health Benefits of Reduced Ozone Concentrations. Environ Health Perspect 109(12).

Logue JM, Price PN, Sherman MH, Singer BC, 2011 A Method to Estimate the Chronic Health Impact of Air Pollutants in U.S. Residences. Environ Health Perspectives doi:10.1289/ehp.1104035 LBNL- 5267E

Logue, JM, McKone, TE, Sherman, MH, Singer, BC. 2011. Hazard assessment of chemical air contaminants measured in residences. Indoor Air 21(2): 92-109.

Lvovsky, K, Huges, G, Maddison, D, Ostro, B, Pearce, D. 2000. Environmental costs of fossil fuels: a rapid assessment method with application to six cities. Washington, D.C.:The World Bank Environment Department.

McKenna, MT, Michaud, CM, Murray, CJL, Mards, JS. 2005. Asssessing the Burden of Disease in the Unites States Using Disability-Adjusted Life Years. American Journal of Preventative Medicine 28(5): 9.

Melse, JM, Essink-Bot, ML, Kramers, PGN, Hoeymans, N. 2010. A National Burden of Disease Calculation: Dutch Disability-Adjusted Life-Years. American Journal of Public Health 90(8): 1241-1247.

Miller, KA, Siscovick, DS, Sheppard, L, Shepherd, K, Sullivan, JH, Anderson, GL, et al. 2007. Long-Term Exposure to Air Pollution and Incidence of Cardiovascular Events in Women. New England Journal of Medicine 356(5): 447-458.

Muller, NZ, Mendelsohn, R. 2007. Measuring the damages of air pollution in the United States. Journal of Environmental Economics and Management 54(1): 1-14.

Murray, CL, Lopez, AD. 1996a. The global buden of disease: a comprehensive assessment of rmortality and disability from diseasees, injuries, and risk factors in 1990 and projected to 2020. Cambridge, MA: Harvard University.

Murray, CL, Lopez, AD. 1996b. Global health statistics: A compendium of incidence, prevalence, and mortality estimates for over 200 conditions. In: Global burden of disease and injury series, Vol. II. Cambridge, MA:Harvard University, 906.

Naya, M, Nakanishi, J. 2005. Risk assessment of formaldehyde for the general population in Japan. Regul Toxicol Pharmacol 43(3): 232-248.

Nazaroff, WW, Singer, BC. 2004. Inhalation of hazardous air pollutants from environmental tobacco smoke in US residences. Journal of Exposure Analysis and Environmental Epidemiology 14: S71-S77.

Logue JM, Price PN, Sherman MH, Singer BC, 2011 A Method to Estimate the Chronic Health Impact of Air Pollutants in U.S. Residences. Environ Health Perspectives doi:10.1289/ehp.1104035 LBNL- 5267E

NYDOSH. 2006. Guidance for Evaluating Soil Vapor Intrusion in the State of New York Appendix C. Troy, New York:Ney York State Department of Health, Center for Environmental Exposure, Bureau of Environmental Exposure Investigation.

Pope, CA. 2007. Mortality effects of longer term exposures to fine particulate air pollution: Review of recent epidemiological evidence. Inhalation Toxicology 19: 33-38.

Pope, CA, Burnett, RT, Thun, MJ. 2002. Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution. Journal of the American Medical Association 287(9): 1132-1141.

Pope, CA, III., Ezzati, M, Dockery, DW. 2009. Fine-Particulate Air Pollution and Life Expectancy in the United States. The New England Journal of Medicine 360(4): 376-386.

Salthammer, T, Bahadir, M. 2009. Occurrence, dynamics and reactions of organic pollutants in the indoor environment. Clean-Soil Air Water 37(6): 417-435.

Samet, JM. 1993. Indoor Air Pollution: A Public Health Perspective. Indoor Air 3(4): 219-226.

Samet, JM, Zeger, SL, Kelsall, JE, Xu, J, Kalkstein, LS. 1997. Air pollution, weather, and mortality in Philadelphia 1973-1988. Health Effects Institute

Schwartz, J, Laden, F, Zanobetti, A. 2002. The concentration-response relation between PM2.5 and daily deaths. Environ Health Perspect 110(10).

Seaman, VY, Bennett, DH, Cahill, TM. 2007. Origin, occurrence, and source emission rate of acrolein in residential indoor air. Environmental Science & Technology 41: 6940-6946.

Simons, E, Curtin-Brosnan, J, Buckley, T, Breysse, P, Eggleston, PA. 2007. Indoor Environmental Differences between Inner City and Suburban Homes of Children with Asthma. Journal of Urban Health: Bulletin of the New York Academy of Medicine 84(4).

US Census Bureau. 2010. Current Population Survey, Annual Social and Economic Supplement, 2010.: US census Bureau.

Logue JM, Price PN, Sherman MH, Singer BC, 2011 A Method to Estimate the Chronic Health Impact of Air Pollutants in U.S. Residences. Environ Health Perspectives doi:10.1289/ehp.1104035 LBNL- 5267E

Viegas, S, Ladeira, C, Nunes, C, Malta-Vacas, J, Gomes, M, Brito, M, et al. 2010. Genotoxic effects in occupational exposure to formaldehyde: A study in anatomy and pathology laboratories and formaldehyde-resins production. Journal of Occupational Medicine and Toxicology 5(25).

Weisel, CP, Zhang, J, Turpin, BJ, Morandi, M, Colome, S, Stock, T, et al. 2005. Relationships of Indoor, Outdoor, and Personal Air (RIOPA) Part I. Collection Methods.Health Effects Institute, Mickely Leland National Urban Air Toxics Research Center and Descriptive Analysis.

WHO. 2009. Global Health Risks: Mortality and burden of disease attributable to selected major risks. Geneva, Switzerland:World Health Organization.

Xu, J, Kochanek, KD, Murphy, SL, Tejada-Vera, B. 2010. Deaths: Final Data for 2007. (National Vital Statistics Reports).U.S. Department of Health and Human Services, Centers for Disease Control and Prevention.

Table 1: Pollutants included in analysis and assumed population average concentration (Conc.) ($\mu g/m^3$)

Pollutant	Conc.	Pollutant	Conc.	Pollutant	Conc.
1,1,2,2-tetrachloroethane	0.42	bromoform	0.39	mercury	1.6E-04
1,1,2-trichloroethane	0.46	cadmium	2.6E-03	methyl methacrylate	0.27
1,1-dichloroethene	1.2	carbon disulfide	0.34	methylene chloride	8.2
1,2-dibromoethane	0.14	carbon monoxide	810	methyl isobutyl ketone	1.2
1,2-dichloroethane	0.34	carbon tetrachloride	0.68	methyl tert butyl ether	12
1,3-butadiene	0.46	chlorobenzene	0.68	naphthalene	1.2
1,4-dichlorobenzene	50	chloroethane	0.26	nitrogen dioxide	13.1
2-butoxyethanol	2.6	chloroform	1.5	o-phenylphenol	0.13
2-ehtylhexanol	3.7	chloromethane	1.8	ozone	17.2
2-ethoxyethanol	0.43	chromium	2.2E-03	pentachlorophenol	2.9E-03
2-methoxyethanol	0.12	crotonaldehyde	4.7	PM2.5	15.9
acetaldehyde	22	cyclohexane	5.2	styrene	5.9
acrolein	2.3	di(2-ethylhexyl)adipate	1.6E-02	sulfur dioxide	2.9
acrylonitrile	0.27	dibenzo[a,c+a,h]anthracene	1.4E-05	tetrachlorothene	1.7
ammonia (NH3)	28	dibromochloromethane	0.44	tetrahydrofuran	15
arsenic	9.8E-04	d-limonine	23	toluene	2.3
atrazine	5.9E-04	ethanol	860	trichloroethene	0.16
benzaldehyde	2.5	ethylbenzene	3.9	vinyl chloride	1.7
benzene	2.5	formaldehyde	69	xylene, o	8.2

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benzo[a]pyrene	9.1E-05	hexachlorobutadiene	1.7	xylene,m/p	9.7
benzyl chloride	0.5	hexane	7.3	xylenes	7.4
beryllium	1.6E-06	Isopropylbenzene	0.4		
bis (2etylhexyl) phthalate	0.14	manganese	3.3E-03		
bromodichloromethane	0.49	methyl ethyl ketone	7.4		

The table includes the pollutants identified by Logue et al. (2011) that had sufficient toxicological and epidemiological data to calculate their health impact.

Table 2: Criteria pollutant concentration-response function outcomes and disability adjusted life years (DALYs) lost per incidence.

Pollutant	Outcome	β	yo	DALYs per Incidence
PM2.5	Total Mortality (Pope et al. 2002)	0.058 (0.002-0.010)	0.0074	1.4 (.14-14) (Pope 2007; Pope et al. 2002; Pope et al. 2009)
	Chronic Bronchitis (Abbey et al. 1995)	0.091 (0.078-0.105)	0.004	1.2 (0.12-12) (Lvovsky et al. 2000; Melse et al. 2010)
	Non-Fatal Stroke (Brook et al. 2010)	0.025 (0.002-0.048)	0.002	0 complications: 9.5 (9.25-9.75) 1 complication: 11.7 (11.1-12.4) >1 complication: 13.1(12.2-14.0) (Hong et al. 2010)
Carbon Monoxide	Hospital Admissions (Burnett et al. 1999)			4E-4 (Lvovsky et al. 2000)
	Asthma:	0.033 (0.016-0.050)	1.8E-3	
	Lung Disease	0.025 (0.000-0.057)	2.1E-3	
	Dysrhythmias	0.058 (0.012-0.102)	2.4E-3	
	Heart Failure	0.034 (0.002-0.066)	3.4E-3	
Nitrogen Dioxide	Hospital Admissions (Burnett et al. 1999)			4E-4 (Lvovsky et al. 2000)
	Respiratory Issues	0.004 (0.000-0.008)	9.5E-3	
	Congestive Heart Failure	0.003 (0.001-0.004)	3.4E-3	
	Ischemic Heart	0.003 (0.002-0.004)	8.0E-3	

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	Disease			
	Respiratory Illness, indicated by symptoms (Hasselblad et al. 1992)	0.028 (0.002-0.053)	N/A	4E-4 (Lvovsky et al. 2000)
Ozone	Mortality (Jerrett et al. 2010; Samet et al. 1997)	0.001 (0.000-0.002)	0.0077	1.0 (0.1-10) (Levy et al. 2001; Lvovsky et al. 2000)
	Hospital Admission (Burnett et al. 1999)			4E-4 (Lvovsky et al. 2000)
	Asthma	0.003 (0.001-0.004)	1.8E-3	
	Lung Disease	0.003 (0.001-0.005)	2.1E-3	
	Respiratory Infection	0.002 (0.001-0.003)	5.8E-3	
	Dysrhthmias	0.002 (0.000-0.004)	2.4E-3	
Sulfur Dioxide:	Hospital admissions (Burnett et al. 1999)	0.002 (0.000-0.003)	8.0E-3	4E-4 (Lvovsky et al. 2000)

y_o is the baseline prevalence of illness per year and β is the coefficient of the concentration change used for inputs into Eq. 3.

Table 3: Residential occupancy characteristics.

Age	Fraction of population	Cancer ADAF	Fraction of day at home	Air intake (m^3/day)
<2	3%	10	75%	7
2-16	19%	3	75%	13
≥16	78%	1	69%	15
Population Average		1.6	70%	14.4

The percentage of the population in each of these age groups was determined from the US census(2010). The percentage of time each age groups spends at home was determined from The National Human Activity Pattern Study (Klepeis et al. 2001). The age-dependent inhalation rate was taken from the US Environmental Protection Agency (EPA) (2009).

Table 4. Health outcomes attributable to second hand smoke (SHS), Radon and acute CO poisoning in the United States and the DALYs lost per incidence of each health outcome

Outcome	Annual Excess incidences due to SHS in US	DALYs per Incidence

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Asthma Episodes	202,300 (Cal EPA 2005)	40/1,000 cases (Lvovsky et al. 2000)
Otitis Media Visits	790,000 (Cal EPA 2005)	22/1,000 cases (de Hollander et al. 1999)
SIDS	430 (Cal EPA 2005)	78 (current US life expectancy)/case (Xu et al. 2010)
Cardiac Death	46,000 (95th% CI: 22,7000-69,600) (Cal EPA 2005)	1/case (de Hollander et al. 1999)
Lung Cancer Death	3400 (Cal EPA 2005)	14/case (de Hollander et al. 1999; Melse et al. 2010)
Outcome	**Annual Excess incidences due to Radon in US**	**DALYs per Incidence**
Lung Cancer Deaths (smokers)	18,000 (95th% CI: 5,600-58,000) (EPA 2003)	14/case (de Hollander et al. 1999; Melse et al. 2010)
Lung Cancer Deaths (non-smokers)	3,000 (95th% CI: 950-96,000) (EPA 2003)	14/case (de Hollander et al. 1999; Melse et al. 2010)
Outcome	**Annual Excess incidences due to carbon monoxide in US**	**DALYs per Incidence**
Acute Poisoning Deaths	1.53 deaths per million persons (95% CI of 1.47–1.59) (CDC 2007)	32 (Xu et al. 2010)

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Figures

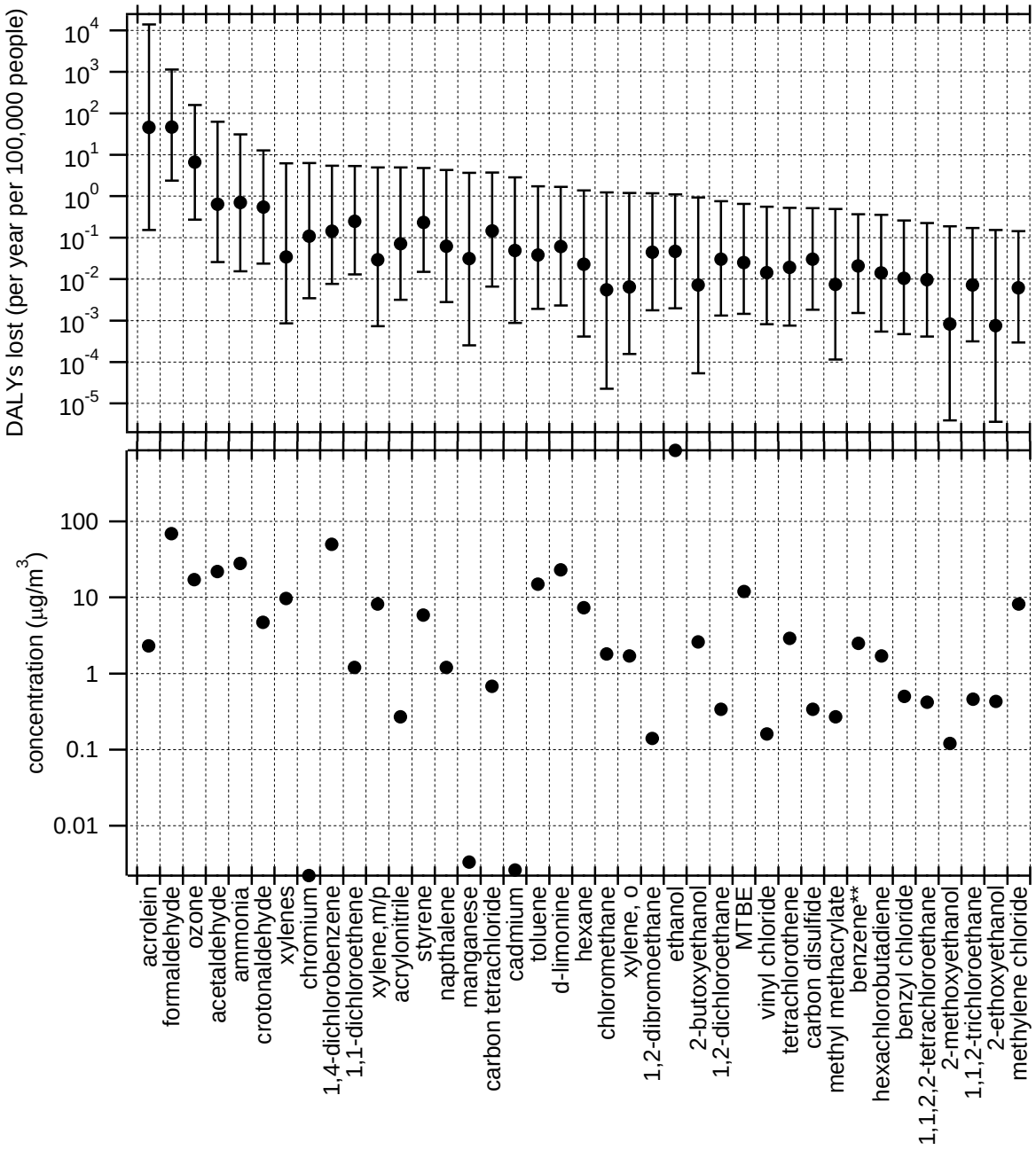

Figure 1: Estimated mean chronic exposure concentrations (lower panel) and health impacts associated with intake of indoor air, in disability adjusted life years (DALYs, upper panel), calculated with the Intake-DALYs (ID) model. The dots in the upper panel represent the central estimate of the DALYs and the whiskers extend to the 95th percentile CI of the DALYs lost.

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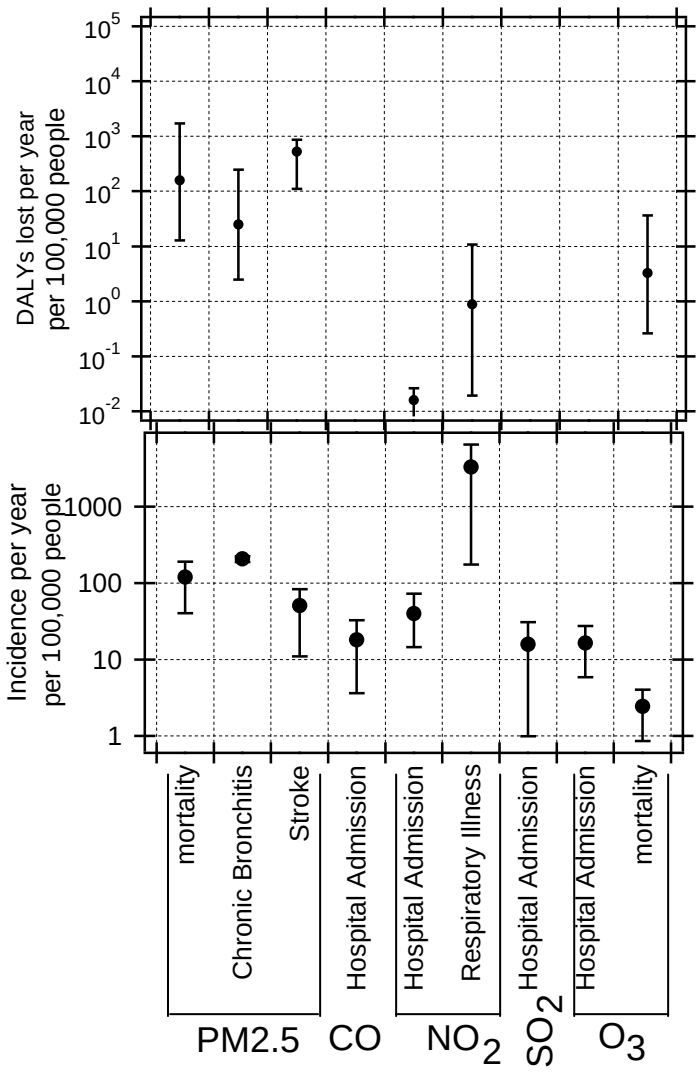

Figure 2: Incidence of disease and disability adjusted life years (DALYs) lost due to criteria air pollutant intake in residences. The lower panel shows the incidence of disease estimated using the concentration-response functions used in the IND approach. The upper panel shows the annual DALYs lost due to each disease outcome using the IND approach.

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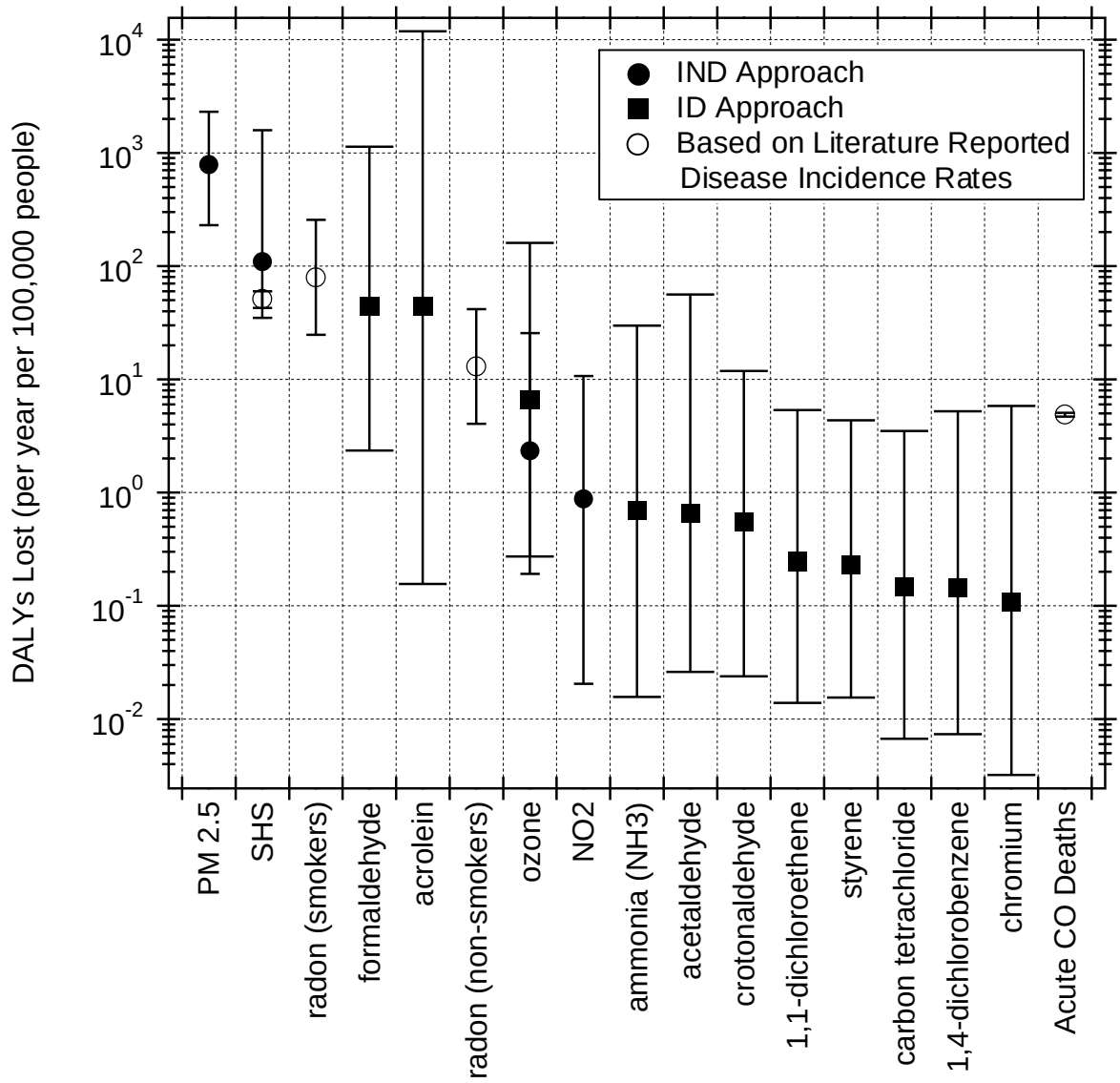

Figure 3: Estimated population averaged annual cost, in DALYs, of chronic air pollutant inhalation in U.S. residences; results for the 12 pollutants with highest median DALY estimates. The markers represent the central estimate and the whiskers extend to the 95th percentile CI. The square marker indicates pollutant DALYs calculated using the Intake-DALYs (ID) approach. The circle markers indicate the DALYs calculated using the Intake-Incidence-DALYs (IND) approach. Radon, acute CO deaths, and SHS DALYs were calculated using disease incidence rates attributed to them in the literature.

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