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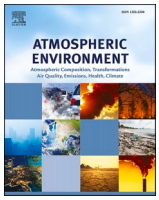

# Modelling the impact on mortality of using portable air purifiers to reduce $PM_{2.5}$ in UK homes

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## HIGHLIGHTS

- Portable air purifiers reduce indoor $PM_{2.5}$ by approximately 50%.
- Reductions in indoor $PM_{2.5}$ from air purifiers could add as much as 6 months of life expectancy in the UK.
- In residences with inadequate ventilation, air purifiers can be an effective strategy to improve health.

## ARTICLE INFO

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## ABSTRACT

This work assessed the potential impact on mortality and life expectancy that would occur due to reductions of indoor $PM_{2.5}$ in dwellings in the UK using portable air purifiers. Reductions in indoor $PM_{2.5}$ concentrations from air purifier use were modelled using findings from the literature for mean air purifier efficiency, mean indoor $PM_{2.5}$ concentrations associated with air purifier use, and the relative risks associated with exposure. Life-table models were used to estimate changes to mortality from the following $PM_{2.5}$-associated diseases: lung cancer, lower respiratory infection (LRI), chronic obstructive pulmonary disease (COPD), ischemic heart disease (IHD), and stroke. Different scenarios were modelled to represent a range of daily use patterns, the starting age of use and the duration of the intervention. The overall impact of the central scenario, in which air purifiers were used during all hours whilst at home (15.6 h) for the entirety of the modelled period (birth to 97 years), was to increase life expectancy in the birth cohort by, on average, 138 and 120 days for males and females, respectively, and to add more than 23 million years of life (YLG) to the UK population. When used at home, air purifiers reduced indoor $PM_{2.5}$ concentrations and prolonged life expectancy, but questions regarding feasibility of the intervention, occupant behaviour and social inequities remain. The estimation of the impact of use by the whole population is, however, important for informing policy and designing interventions.

## 1. Introduction

Outdoor air pollution is recognised as a significant risk to population health, and progress has been made in the United Kingdom (UK) (and elsewhere) to improve it. Recent improvements in ambient air quality have contributed to a greater focus on the quality of the air indoors. The contribution of indoor air pollutants to total exposure from our time spent at home is substantial, as people spend more than 65% of their time there (Klepeis, 2001). It is therefore important to understand ways in which airborne pollutant levels can be reduced and to assess the impact those reductions are estimated to have on health. Concentrations of indoor air pollution, including particulate matter, can exceed health-based guidelines developed by the World Health Organization (WHO) for both chronic and acute exposure (Logue et al., 2012). The contribution of indoor air pollution to total exposure, as well as the negative health impacts associated with exposure, has been demonstrated in past research (e.g., Weisel et al., 2005). Of noted concern, particulate matter less than 2.5 μm in aerodynamic diameter ($PM_{2.5}$) has been shown to impact multiple negative health outcomes including; cardiovascular diseases, asthma, bronchitis, premature mortality and lung cancer (Pope et al., 2020; Pope et al., 2002).

There is a growing body of evidence to support the important role

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that time at home plays in the total exposure to particulate matter. However, there remains considerable uncertainty in the findings (Bekö et al., 2013). In a recent study, Patel et al. (2020), using a test house and prescribed activities, reported that days with typical cooking activity had a 12-h average $PM_{2.5}$ concentration of approximately 15 $\mu g/m^3$, and that during days with substantial cooking activities (i.e., Thanksgiving Day) that average rose to approximately 60 $\mu g/m^3$, highlighting the importance of indoor activities in total exposure (Patel et al., 2020) In addition to specific human activities (e.g., cooking, cleaning, etc.), the indoor environment and the building envelope each play an important role in the chemical and physical properties of particulate matter indoors. Specifically, as PM from outdoor sources penetrate and deposit indoors, there is a meaningful shift in size (towards more $PM_{2.5}$) and chemical composition that may change, and worsen, the health impact of exposure indoors (Goldstein et al., 2020).

The adoption of technologies to mitigate indoor air pollution is increasingly common, and previous studies have considered the health benefits of different methods of particulate filtration (Batterman et al., 2012; Fisk, 2018; Fisk and Chan, 2017b). One of the most effective, and widely available, technologies to clean the surrounding air of $PM_{2.5}$ are portable air purifiers which utilise high efficiency particulate air (HEPA) filtration as the primary mechanism of air cleaning (Zhang et al., 2011). These devices have a number of advantages over other air cleaning methods, including that they can be located in rooms where people spend most of their time (such as bedrooms), are simple to install and operate, they do not produce potentially harmful secondary pollutants, and they do not require a central air handling system. Substantial reductions in $PM_{2.5}$ have been reported in indoor spaces using these devices; from a low of 29% (Barn et al., 2018) to as much as 82.7% (Zhan et al., 2018) with many studies reporting reductions of approximately 50% (e.g. McNamara et al., 2017; Shao et al., 2017). However, the impact on population health from reductions in exposure through interventions with air purifiers in homes has been minimally described in the literature. One recent study (Liu et al., 2021) found that the use of air purifiers in residences in China could be cost-effective at reducing $PM_{2.5}$-related mortality in China. The potential benefits, as well as any drawbacks, are important to understand if policymakers and designers are to respond appropriately. In the work presented here, measured reductions in mean $PM_{2.5}$ concentrations in bedrooms are used to quantify the change in (cause-specific) mortality in the UK population over time that would occur from the use of air purifiers in homes. The work aims to more explicitly estimate the potential impact to population health of reductions in long-term $PM_{2.5}$ exposure in homes.

## 2. Methods

### 2.1. Background

Quantitative health impact assessments involve estimating future rates of mortality and morbidity under different intervention scenarios compared to what is predicted without such interventions. A review of several health risk assessment tools for ambient air pollution are available in a paper by Anenberg et al. (2016). Anenberg et al. (2016) concluded that the choice of modelling tool depends on several factors, including the availability of data, the tolerance for technical complexity, and the ultimate use of the outcomes (e.g., for scoping purposes, or policy-making). For the work presented here, a model based on concentrations, rather than emissions, technically rigorous, previously peer-reviewed, and that allowed for direct user input was desirable. A commonly used approach to the assessment of changes in population mortality due to changes in the environment that met these criteria is life-table modelling (Miller and Hurley, 2003). Life tables can be used to predict survival patterns based on changes in age-specific death rates. The tables are used to estimate years of life lost (YLL) or gained (YLG), and changes in life expectancy in the population. This type of quantification of health impact has been used to assess air pollution at national scales (COMEAP, 2010), and to assess building level changes in exposure (Hamilton et al., 2015; Milner et al., 2015).

### 2.2. Model description

In the work presented here, life-table models were used to quantify the impacts on mortality from reductions in indoor $PM_{2.5}$ concentrations through the use of portable air purifiers at home.

A life table is a demographic model of the pattern of survival and mortality in a population. Life table methods have been used commonly for health impact assessment of environmental policies in recent years (Milner et al., 2014; Qi et al., 2020). Briefly, the method is based on age-specific mortality rates, which are used to calculate probabilities of survival by year-of-age and calendar year. To perform an impact assessment, these underlying mortality rates are adjusted to reflect changes in mortality risk from changes in exposure (in this case exposure to $PM_{2.5}$) by applying relative risks calculated using published exposure-response functions. Cumulative probabilities of survival over multiple years are calculated by multiplying together the individual single-year survival probabilities. Applying these cumulative survival probabilities to a population allows the calculation of life years lived by the population (where one life year is a full year of life lived by one person), which in turn can be used to estimated average remaining life expectancy per person by age. Calculations of changes in mortality and life-expectancy were estimated based upon the life table formulae from Miller and Hurley (Miller and Hurley, 2003; Miller, 2010). The full formulae and description can be found in the online Supplemental Information.

The model was implemented with the open source statistical software R (R Core Team, 2018). The same underlying mortality rates were assumed to apply in all future years, and birth rates were held the same as those in the starting year (2019). The use of unchanging mortality rates is a common approach given that changes in exposure are of interest. Results are robust to assumptions about change in future death rates, and it is not necessary to have correct or even realistic estimates of future 'baseline' death rates as long as the actual and assumed death rates follow the same log-linear shape as described in COMEAP (2010).

The IOMLIFET spreadsheet, a freely available open-source health risk assessment tool (Miller, 2010), was used to assess the validity of the life table model used in this work. This spreadsheet has previously been used to assess the impact from a host of different environmental changes (e.g., Williams et al., 2018; Manojkumar et al., 2020). IOMLIFET was reviewed in Anenberg et al. (2016), along with eleven other health assessment tools, and was chosen for this work because it is applicable to analyses ranging from local to global scale, $PM_{2.5}$ concentrations as well as population and incidence data are user defined, and it models the change in the risk of premature death over the life of a defined cohort. In practice, if mortality rates (all-cause and cause-specific) used in the comparison were the same, and quality-of-life weights are set to one, the life year and life expectancies of the two models should be the same. The two models showed excellent agreement ($R^2 \sim 1$). Similar model agreement was found in other work that compared IOMLIFET and similar models (Milner et al., 2015).

### 2.3. Model parameterisation

The life-table model was used to determine the benefit from the reduction of indoor $PM_{2.5}$ in residences in the UK from the use of portable air purifiers. The model was parameterised using population and age-specific disease and mortality data for 2019 from the Office for National Statistics (ONS, 2019). Calculation of mortality combine the relative risk, projected forward in time, to predict a survival curve. Due to demographic differences in the underlying mortality rates (e.g., cause of death) there is a gap between the survival curves of females and males. The difference in life expectancy between the sexes is equivalent to the area between the curves (COMEAP, 2010). Therefore, the impacts

on mortality are calculated and reported separately for women and men. Mortality rates for causes the Global Burden of Disease (GBD) found to be associated with $PM_{2.5}$ were included in the model; all-cause, lung cancer, chronic obstructive pulmonary disease (COPD), lower respiratory infection (LRI), stroke and ischemic heart disease (IHD). Age-specific all-cause and disease specific mortality rates were taken from the 2019 GBD study (data described in Murray et al., 2020).

The mean indoor pre-intervention $PM_{2.5}$ concentration in UK homes of 11.4 μg/m$^3$ used in the model was from a study in the UK by Lai et al. (2004). The percentage reduction of $PM_{2.5}$ used in the model was 52%. This percentage was the mean of the means of measured efficiencies of air purifiers found in the literature (Table 3). A percentage reduction was used rather than an absolute reduction because it better represents the actual operation of air purifiers in homes and allowed for sensitivity analysis of air purifier efficiencies. Additionally, this approach provides estimates of impacts that are not sensitive to pre-intervention $PM_{2.5}$ concentrations. Therefore, impacts can be modelled with a range of initial $PM_{2.5}$ concentrations. There is a growing body of evidence on people's personal exposures to $PM_{2.5}$ and the importance of location, and time and activity (e.g., Goldstein et al., 2020; Meng et al., 2009; Patel et al., 2020; Steinle et al., 2015) but, there remain few reports of continuously measured concentrations in residences in the UK. It is, therefore, useful to retain the flexibility of proportional reductions within the model to allow for assessments across a range of pre-intervention IAQ conditions.

The relative risks (RRs) for each cause of death (and all-cause) were from the GBD (WHO, 2019). The curves start at 0 μg/m$^3$ (mean RR = 1), and continue to 500 μg/m$^3$ (mean RR = 1.36). Although some studies indicate that the risk curves are supra-linear, steeper at lower concentrations, especially for specific causes of death, analyses by Henschel et al. (2013) suggested that it is reasonable to use a linear curve in Europe for all-cause mortality. It should be noted that this may lead to more conservative estimates of impact for cause-specific mortality at the lower $PM_{2.5}$ concentrations used in this study. The upper and lower confidence intervals of the RRs were calculated which allowed for the testing of impact across the range of potential risk (which is further discussed in the next section).

Four scenarios were defined to assess the changes in mortality under different conditions, a summary of these can be found in Table 1. The central scenario ('All at Home') was based on measured data from 18 London flats that participated in the Quasimodo study (Quality of Indoor Air on Sites Matched with Outdoor Air Quality Datasets to Improve Wellbeing Outcomes). The mean total hours of air purifier use by all participants in the Quasimodo study was 15.6 h/day (Cooper et al., 2021). Time spent outside of the home environment (indoors or outdoors) was excluded and did not contribute to the assessed changes in mortality. Health impacts were limited to what was attributable to time spent indoors at home. 'All at Home' examined the impacts on the current UK population, including all ages from birth upwards, for the 97-year study period. 97 years was chosen because it represents a reasonable long lifespan, captures almost every member of a birth cohort (mean life expectancy in the UK for birth cohort in 2009 was 78.8 years for males and 82.8 years for females), and is within the range of model periods found in other studies (50–105 years). Further scenarios were used to examine differences in impact that could result from different periods of daily and lifetime air purifier use. Two scenarios, 'All Sleep' and '65+ Sleep', modelled the use of air purifiers only during sleeping hours as use in bedrooms was described in Cooper et al. (2021). Night-time use assumed that the occupants were in the same room as the air purifier the entire time, thereby reducing some uncertainty from the model. The '65+ scenarios ('65+ at Home' and '65+ Sleep') selected only those in the population 65 and older to reflect evidence from another study that found the health benefits of air purifiers were highest, relative to the costs, for this age group (Fisk and Chan, 2017b).

Findings from COMEAP (2010) showed that the use of a lag between the intervention that reduces $PM_{2.5}$ concentrations and changes in health outcomes (i.e., cessation lag) made relatively little difference to the lifetable results over the long-term. Therefore, the model used in the work described here does not include a cessation lag.

**Table 1**
Summary of different modelled scenarios, including baseline $PM_{2.5}$ concentration, air purifier use and duration of intervention.

| | All at Home (central scenario) | All Sleep | 65+ Sleep | 65+ at Home |
|---|---|---|---|---|
| $PM_{2.5}$ concentration indoors (μg/m$^3$)[a] | 11.4 | 11.4 | 11.4 | 11.4 |
| Air purifier use (hours/day) | [b] 15.6 | 8 | 8 | 21.6 |
| Duration of use (years) | 97 | 97 | 33 | 33 |
| Starting age | birth | birth | ≥65y.o. | ≥65y.o. |

[a] Monitored mean indoor $PM_{2.5}$ concentration from (Lai et al., 2004).
[b] Monitored mean daily air purifier use from Cooper et al. (2021).

## 2.4. Uncertainty analysis

Three further analyses were run to assess key uncertainties in the model, and to gain a better understanding of the sensitivity of the model to parametric changes. A summary of these tests is shown in Table 2.

### 2.4.1. Test 1: air purifier efficiency

The first analysis (Test 1) tested the effect that varying the efficiency of air purifier had on the modelled impacts. The measured range of $PM_{2.5}$ reduction efficiencies of air purifiers in real-world conditions reported in the literature (Table 3) were used in all four modelled scenarios. These efficiencies ranged from a low of a 29% reduction in indoor $PM_{2.5}$ to a high of 82.7%. Although the range was relatively large, the majority of studies, and the results from the Quasimodo study, clustered around a 50% reduction.

The studies that are most directly comparative to the work presented here are those with relatively long study periods (12 weeks–5 months), indoor $PM_{2.5}$ levels in the range observed in monitoring studies in the UK (6.6–28.4 μg/m$^3$, see Table 4), air purifiers that used HEPA filtration, and located in areas with outdoor PM levels and, occupancy and activity patterns that are similar to those observed in the UK (i.e., studies conducted in western Europe, and North America). Using this criteria, four studies in particular suggest that the efficiencies modelled were representative of the actual conditions expected in residences in the UK (Cheng et al., 2016; McNamara et al., 2017; Park et al., 2017; Ward et al., 2017). These studies had an average percent reduction of 53.5% ranging from 37 to 68%.

**Table 2**
Summary of model inputs analysed for uncertainty and sensitivity.

| | Air purifier Efficiency | Relative Risk | Pre-intervention indoor $PM_{2.5}$ concentration annual mean (μg/m$^3$) |
|---|---|---|---|
| **Central Scenario** | Mean | Mean | [d] 11.4 |
| **Test 1**: Air purifier efficiency distribution | Mean, Min., Max.[a] | Mean | [d] 11.4 |
| **Test 2**: Coefficient of risk distribution | Mean | Mean, Min., Max.[b] | [d] 11.4 |
| **Test 3**: Pre-intervention indoor $PM_{2.5}$ concentration | Mean | Mean | 6.6[c] and 18.8d[e] |

[a] See Table 3 for a summary of air purifier efficiencies from the literature.
[b] Lower and upper confidence bounds, and means, of relative risk from (WHO, 2019).
[c] Measured mean in 18 east London flats (Cooper et al., 2021).
[d] Lai et al. (2004).
[e] Shrubsole et al. (2012).

**Table 3**
Summary of studies on the effects of portable air purifier use on the reduction on $PM_{2.5}$ in residences.

| First author (publication year) country | Study design, sample size, characteristics | Study duration | Indoor $PM_{2.5}$ concentration ($\mu g/m^3$); mean or median, SD during intervention and control; % reduction | % Reduction in $PM_{2.5}$ |
|---|---|---|---|---|
| Allen et al. (2011) Canada | Randomised crossover trial, 25 homes, non-smokers | 7 days | Mean ± SD (p-value): control: 11.2 ± 6.1 (<0.01) Intervention: 4.6 ± 2.6 (<0.01) % reduction: 58.9 | 58.9 |
| Barn et al. (2008) Canada | Randomised crossover trial, 32 homes, non-smokers | 2 days | Mean ± SD (p-value): control: 6.7 ± 20.7 (<0.01) Intervention: 4.2 ± 7.3 (<0.01) % reduction: 37.3 | 37.3 |
| Barn et al. (2018) Mongolia | Randomised controlled trial, 512 pregnant adults, non-smokers | 7 days | GM (95%CI): control: 24.5 (22.2, 27.0) Intervention: 17.3 (15.8, 18.8) %reduction 29.0 | 29.0 |
| Brauner et al. (2008) Denmark | Randomised crossover trial, 21 homes, non-smokers | 2 days | GM (95%CI): control: 12.6 (11.2, 14.1) Intervention: 4.6 (3.5,6) % reduction 63.5 | 63.5 |
| Brehmer et al. (2019) China | Randomised crossover trial, 43 children | 14 days | Mean ± SD (p-value): control: 34 ± 17 (<0.01) Intervention: 15 ± 9.6 (<0.01) % reduction: 63.5 | 63.5 |
| Brehmer et al. (2020) China | Randomised crossover trial, 43 children | 14 days | Median (IQR), (p-value): Control 30 (19) Intervention: 13 (15) (<0.05) % reduction: 55.9 | 55.9 |
| Butz and Breysse (2011) USA | Randomised 3-arm controlled trial, 126 children with asthma with smoker | 7 days | Mean ± SD (p-value): control: 38.9 ± 25.0 (<0.01) Intervention: 17.9 ± 15.2 (<0.01) % reduction: 54.0 | 54.0 |
| Cheng et al. (2016) USA | Randomised controlled trial, 8 homes, non-smokers | 12 weeks | 5- min aggregated median/mean (p-value): control: 5.2/6.1 Intervention: 2.6/4.0 (<0.001) % reduction: 37.0 | 37.0 |
| Cooper et al. (2021) UK | Randomised crossover trial, 18 households | 6 months | Median: 6.6 | 45.0 |
| Cox et al. (2018) USA | Randomised controlled crossover trial, 43 homes near major road | 4 weeks | Median (p-value): control baseline: 9.6 Control filter: 8.2 Intervention baseline: 7.6 Intervention filter: 3.4, (0.0125) % reduction: 58.5 | 58.5 |
| Eggleston et al. (2005) USA | Randomised controlled trial, 97 children with asthma | 72 h | Median (IQR), (p-value): Control 30 (20–45) Intervention: 24 (10–43) (<0.001) % reduction: 36.8 | 36.8 |
| Huang et al. (2020) USA | Randomised crossover trial, 6 homes, non-smokers | 21 days | Mean ± SD (p-value): control: 14.2 ± 20.9 (<0.01) Intervention: 8.5 ± 8.3 (<0.01) % reduction: 41.6 | 41.6 |
| James et al. (2019) USA | Randomised crossover trial, 37 homes near major road | 2 days | Median (range), (p-value): Control baseline: 10.4 (0.6–53.2) control filter: 7.8 (<LOD-37.9) intervention baseline: 12.0 (0.3–80.9) intervention filter: 4.5 (1.1–18.0) (<0.0125) % reduction 62.5 | 62.5 |
| Kajbafzadeh et al. (2015) Canada | Randomised controlled trial, 44 homes, non-smokers | 7 days | Median/mean ± SD: control: 7.5/7.1 ± 6.1 intervention: 3.7/4.3 ± 2.6 % reduction: 40.0 | 40.0 |
| Karottki et al. (2013) Denmark | Randomised controlled trial, 27 homes, non-smokers | 14 days | Median (5th-95th percentile): Living room: control: 8 (3.4, 20.7) intervention: 4.3 (0.2, 12.2) Bedroom control: 7.6 (1.4, 19.2) intervention: 3.7 (1, 14) % reduction: Living room:46.3 Bedroom: 51.3 | 51.3 |
| Liu et al. (2018) China | Randomised crossover trial, 20 homes, non-smokers | 14 days | Mean ± SD: control: 58.24 ± 52.74 Intervention: 37.99 ± 45.89 % reduction: 34.8 | 34.8 |
| Maestas et al. (2019) USA | Randomised crossover trial, 40 homes, non-smokers | 3 days | Mean ± SD, (range) (p-value): control: 17.5 ± 16.9 (4.1–117.5) LE: 8.4 ± 5.4 (1.3–39.5) HE: 7.0 ± 4.5 (1.1–30.8) (<0.001) % | Low efficiency: 52.0 High Efficiency: 60.0 |

(continued on next page)

**Table 3** (*continued*)

| First author (publication year) country | Study design, sample size, characteristics | Study duration | Indoor $PM_{2.5}$ concentration ($\mu g/m^3$); mean or median, SD during intervention and control; % reduction | % Reduction in $PM_{2.5}$ |
|---|---|---|---|---|
| | | | reduction: LE: 52.0 HE: 60.0 | |
| McNamara et al. (2017) USA | Randomised controlled trial, 48 homes, wood stones | 5 months | Medina (range): control baseline: 19.8 (6.0, 101.9) Control filter: 22.0 (2.4, 163.2) intervention baseline: 15.7 (6.1, 63.1) intervention filter: 5.7 (0.7, 65.6) % reduction: 66.0 | 66.0 |
| Morishita et al. (2018) USA | Randomised crossover trial, 40 homes, non-smokers | 3 days | Median/mean ± SD: control: 13.1/17.5 ± 13 LE: 7.8/8.4 ± 3.9 HE: 6.0/7.1 ± 3.5 % reduction: LE: 52.0 HE:60.0 | Low efficiency: 52.0 High Efficiency: 60.0 |
| Park et al. (2017) USA | Randomised crossover trial, 16 homes | 12 weeks | Mean ± SEM (p-value): Baseline: 7.42 ± 1.42 week 6 intervention: 4.76 ± 0.65 week 12 intervention: 4.28 ± 0.81 ($p < 0.001$) % reduction: 43.0 | 43.0 |
| Rice et al. (2018) USA | Unmasked trial, 82 participants, smoke in home | 5 weeks | Median (IQR), (p-value): pre-intervention: 31 (17, 63) post-intervention: 17 (10,35), (<0.001) % reduction: 45.0 | 45.0 |
| Shao et al. (2017) China | Randomised crossover trial. 20 homes, non-smokers | 14 days | Mean ± SD (p-value): 10-day average: control: 60 ± 45 intervention: 24 ± 15 (<0.01) % reduction: 10-day average: 60.0 | 60.0 |
| Spilak et al. (2014) Denmark | Randomised crossover trial, 28 homes | 14 days | Mean (95% CI): control bedroom: 8.33 (6.72–9.93) control living: 8.32 (6.95–9.69) intervention bedroom: 4.74 (3.53–6.68) intervention living: 4.48 (3.35–6.06) % reduction: 54.5 | 54.5 |
| Ward et al. (2017) USA | Randomised controlled crossover trial, 98 homes with wood stoves | 5 months (winter) | Median (range): Control baseline:16.1 (3.9, 508.2) control filter: 16.9 (2.4, 163.2) intervention baseline: 17.1 (6.1, 163.1) intervention filter: 6.5 (0.7, 65.6) % reduction: 68.0 | 68.0 |
| Weichenthal et al. (2013) Canada | Randomised crossover trial. 37 participants | 7 days | Median/mean ± SD: Control: 42.5/61.0 ± 64 intervention 22.0/30.0 ± 30 %reduction: 50.8 | 50.8 |
| Wheeler et al. (2014) Canada | Randomised crossover trial, 31 homes | 3 days | Gravimetric median (min-max): Control 3.87 (0.37–30.19) intervention: 1.92 (0.35–11.28) % reduction: 52.0 | 52.0 |
| Zhan et al. (2018) China | Randomised crossover trial, 6 participants | 4 weeks | Mean: control: 49.0 intervention: 8.47 % reduction: 82.7 | 82.7 |

**Table 4**
Summary of findings reported from modelling and monitoring studies of indoor $PM_{2.5}$ in UK domestic buildings.

| First author (publication year) | Study design, characteristics | Indoor $PM_{2.5}$ concentration ($\mu g/m^3$) |
|---|---|---|
| Shrubsole et al. (2012) | Modelled with CONTAM, non-smoking | AM[a]: 28.4 (present day outdoor $PM_{2.5}$ concentrations) AM[a]: 18.8 (2050 outdoor $PM_{2.5}$ projections) |
| Hamilton et al. (2015) | CONTAM, standardised indoor in England | AM[a]: 17.8 (SD: 0.7) |
| Lai et al. (2004) | Monitoring in Oxford, UK | GM[b]: 11.4 GSD[c]: 2.4 |
| Cooper et al. (2021) | Monitoring in London, UK | AM[a]: 6.6 |

[a] AM = Arithmetic mean.
[b] GM: Geometric mean.
[c] GSD: Geometric standard deviation.

### 2.4.2. Test 2: upper and lower 95% confidence interval limits of RR

Recognising that the exposure-response function per change in $PM_{2.5}$ could introduce uncertainty into the model, the second part of the testing (Test 2) examined the effect of using the upper and lower values from the 95% confidence intervals from the distribution of the RRs derived from the 2019 Global Burden of Disease. This test was in line with the recommendations for sensitivity analysis made by COMEAP (2010).

### 2.4.3. Test 3: mean pre-intervention indoor $PM_{2.5}$ concentration

Test 3 investigated the effect of the mean starting (i.e., pre-intervention) concentration of indoor $PM_{2.5}$ on changes in mortality estimates. The model used percentage reduction of $PM_{2.5}$ to measure efficiencies of air purifiers, rather than absolute reductions, as described

in the methods. Therefore, effects on mortality were expected to be approximately linearly proportional to the change in starting concentration. That is, a starting concentration of 9.4 μg/m$^3$ would generate roughly half the impact that would be seen with a starting concentration of 18.8 μg/m$^3$, all things being otherwise equal. Given this assumption, modelling different starting concentrations provided a reliable and simple means of testing the functionality of the model whilst also providing useful metrics to compare mortality across a range of indoor air quality (IAQ) conditions likely to be present in real dwellings.

Each scenario was modelled with three different pre-intervention indoor $PM_{2.5}$ concentrations. In addition to the concentration of 11.4 μg/m$^3$ used in the main analysis, a higher concentration of 18.8 μg/m$^3$ was used, based on modelling of the domestic stock in London using an ambient $PM_{2.5}$ concentration of 9.0 μg/m$^3$ (close to the current mean ambient levels in the UK) (Shrubsole et al., 2012). The low pre-intervention concentration of 6.6 μg/m$^3$ was the mean concentration measured in London flats in the Quasimodo study (Cooper et al., 2021). A summary of modelled and measured indoor $PM_{2.5}$ can be found in Table 4.

### 2.5. Model outputs

The life-table models described here provided estimations of the differences in mortality between a mean pre-intervention concentration of $PM_{2.5}$ indoors in homes in the UK of 11.4 μg/m$^3$, against alternative scenarios that utilised air purifiers to reduce indoor levels. The model calculated changes for all combinations of age (in 5-year increments), by gender, and calendar year. Changes to life expectancy at birth were estimated based upon the calculated YLG divided across the whole population. Permanent changes in hazards (i.e., reductions in indoor $PM_{2.5}$ exposure) are expected to confer benefits every year into the future. However, it is typical in health models to discontinue the accumulation of benefit at some point due to greater and greater uncertainties about future conditions. In the work presented here, that point is 97 years from the start (2019), at a time that most in the first birth cohort have reached zero survival.

## 3. Results

Results from the monitoring campaign in London (Quasimodo) of indoor $PM_{2.5}$ concentrations and typical daily aur purifier use that informed the parameterisation and testing of the health impact model are described in detail in Cooper et al. (2021). The estimated impact on mortality and life-expectancy is reported for each modelled scenario in the following section, followed by the findings of the sensitivity and uncertainty analyses.

### 3.1. Quantification of health impact

The central scenario, 'All at Home', modelled the use of air purifiers by the whole UK population for 15.6 h/day (time at home). This scenario increased the number of years of life (YLG) in the UK by roughly 23 million YLG over the modelled period (97 years beginning in 2019). This YLG translates to an additional 138 and 120 days of life expectancy for males and females, respectively. The 'All Sleep' scenario led to over 12 million YLG and 71 and 62 days gained for males and females, respectively. The 'All 65+ and '65+ Sleep' scenarios resulted in only about 25% and 10% of the YLG compared with the central scenario (5.8 and 2.2 million YLG), respectively. These findings are approximately representative of the portion of the population that is above age 65, and the shorter duration of the intervention compared to the central scenario. A summary of the findings for all scenarios can be found in Table 5.

Irrespective of the scenario, the distribution of deaths amongst the five causes of death remains unchanged, differing only slightly between males and females, but remaining proportional to the differences in the disease-specific mortality rates between the sexes within the UK population. The contribution of each disease outcome to total all-cause ($PM_{2.5}$ attributable) deaths is presented in Table 6.

### 3.2. Sensitivity and uncertainty analyses

#### 3.2.1. Test 1: air purifier efficiency

Several tests were run to assess the sensitivity of the parameters defined in the scenarios. In the first test the $PM_{2.5}$ reduction efficiency of the air purifiers was tested, all other scenario parameters remained unchanged from the baseline model. Two reduction efficiencies were modelled, a low efficiency air purifier (29%) and a high efficiency air purifier (82.7%). The effect on mortality from the low efficiency air purifier was approximately 12 million YLG for the baseline scenario, compared to a maximum of more than 34 million YLG for the high efficiency air purifier. These model results translate to average days gained in the low efficiency situation of 75 for males and 65 for females. In contrast, the high efficiency air purifiers would add 201 days for males and 175 days for females. The test suggests that the relationship between reduction efficiency, or absolute reduction in $PM_{2.5}$ was nearly, but not quite, linear. A summary of Test 1 results for all scenarios is shown in Table 7 and Table 8.

#### 3.2.2. Test 2: upper and lower 95% confidence interval limits of RR

Due to the, often large, differences between the upper and lower confidence limits of the GBD RRs, this parameter has a substantial impact on mortality effects (Table 9). In the case of scenario 'All at Home', the difference between the lower and upper limits of the RR for all-cause mortality is more than 26 million YLG, twice the results of the central finding (23 million). This translates to a difference in the average additional life expectancy for males in the UK of 58 days vs. 211 days for the lower and upper limits, respectively. While for females the lower

**Table 6**
Distribution of disease-specific deaths for males and females in the UK.

| Cause of death | Percentage of attributable deaths (Males) | Percentage of attributable deaths (Females) |
|---|---|---|
| Lung cancer | 3% | 1% |
| LRI | 28% | 27% |
| COPD | 7% | 9% |
| IHD | 32% | 28% |
| Stroke | 29% | 35% |
| Total | 100% | 100% |

**Table 5**
Summary of life-table model results for the baseline case (mean RRs, mean air purifier efficiency, starting $PM_{2.5}$ concentration 11.4 μg/m$^3$).

| Outcome | Population | All at Home (central scenario) | All Sleep | 65+ Sleep | 65+ at Home |
|---|---|---|---|---|---|
| Average years of life gained (YLG) | Male | 12,427,646 | 6,385,418 | 1,150,070 | 3,023,585 |
| | Female | 11,140,862 | 5,725,708 | 1,066,704 | 2,801,626 |
| | Total | 23,568,509 | 12,111,126 | 2,216,774 | 5,825,211 |
| YLG per 100,000 | Total | 35,284 | 18,131 | 3319 | 8721 |
| Average days gained | Male | 138 | 71 | 13 | 34 |
| | Female | 120 | 62 | 12 | 30 |

**Table 7**
Test 1, sensitivity to a reduction in air purifier efficiency modelled for all scenarios using the baseline starting concentration and RRs with a **low** air purifier reduction efficiency of 29%.

| Test 1: Low air purifier efficiency (29%), Mean RRs, all-cause mortality | | | | | |
|---|---|---|---|---|---|
| Outcome | Population | All at Home | All Sleep | 65+ Sleep | 65+ at Home |
| Average years of life gained (YLG) | male | 6,732,391 | 3,340,993 | 601,379 | 1,778,332 |
| | female | 6,036,729 | 2,996,454 | 557,911 | 1,651,856 |
| | Total | 12,769,120 | 6,337,447 | 1,159,290 | 3,430,187 |
| Average days gained | male | 75 | 37 | 7 | 20 |
| | female | 65 | 32 | 6 | 18 |

**Table 8**
Test 1, sensitivity to an increase in air purifier efficiency modelled for all scenarios using the baseline starting concentration and RRs with a **high** air purifier reduction efficiency of 82.7%.

| Test 1: High air purifier efficiency (82.7%), Mean RRs, all-cause mortality | | | | | |
|---|---|---|---|---|---|
| Outcome | Population | All at Home | All Sleep | 65+ Sleep | 65+ at Home |
| Average years of life gained (YLG) | male | 18,122,902 | 9,429,842 | 1,698,762 | 4,268,839 |
| | female | 16,244,995 | 8,454,962 | 1,575,496 | 3,951,396 |
| | Total | 34,367,897 | 17,884,804 | 3,274,258 | 8,220,234 |
| Average days gained | male | 201 | 104 | 19 | 47 |
| | female | 175 | 91 | 17 | 43 |

**Table 9**
Test 2: effect of changes in relative risks using the upper and lower 95% CIs from the GBD.

| Scenario | RR (95% CI upper and lower) | LYG male | LYG female | LYG total pop. |
|---|---|---|---|---|
| All at Home | lower | 5,199,315 | 4,766,868 | 9,966,183 |
| | mean | 12,427,646 | 11,140,862.2 | 23,568,509 |
| | upper | 19,101,247 | 17,011,072 | 36,112,318.9 |
| All Sleep | lower | 3,488,054 | 3,215,593 | 6,703,646 |
| | mean | 6,385,418 | 5,725,708 | 12,111,126 |
| | upper | 9,499,523 | 8,466,178 | 17,965,700 |
| 65+ Sleep | lower | 634,899 | 604,546 | 1,239,444 |
| | mean | 1,150,070 | 1,066,704 | 2,216,774 |
| | upper | 1,702,346 | 1,570,852 | 3,273,198 |
| 65+ at Home | lower | 1,079,418 | 1,010,350 | 2,089,769 |
| | mean | 3,023,585 | 2,801,626 | 5,825,211 |
| | upper | 4,755,453 | 4,381,854 | 9,137,307 |

limit of the 95% CI of RRs results in 51 days extra average life expectancy and more than 183 days for the upper limit.

#### 3.2.3. Test 3: mean pre-intervention indoor $PM_{2.5}$ concentration

The final test of the model generated results based on different starting (pre-intervention) concentrations of indoor $PM_{2.5}$. The lowest starting concentration modelled was 6.6 μg/m3 and the highest was 18.8 μg/m3. The YLG for the pre-intervention concentration of 6.6 μg/$m^3$ was just under 11 million, whilst for 18.8 μg/$m^3$ the YLG was almost 37 million. A summary of all the scenarios modelled with these pre-intervention $PM_{2.5}$ concentrations can be seen in Table 10.

## 4. Discussion

The work presented here provides new insights into the potential effects on mortality from the widespread use of air purifiers in UK homes. Although the focus of this work is UK homes, the results are expected to be representative of other developed countries in Europe, where housing type and ambient $PM_{2.5}$ levels are similar. The relevance of the findings of this study to other situations and populations is one of the advantages of life-table modelling, as described in detail in COMEAP (2010). Absolute levels of death rates vary between locations, between the sexes and temporally. However, for a given percentage reduction in mortality hazards, the gains in life expectancy and in life-years per 100, 000 are similar in different populations, even when underlying hazard rates are different, which allows for transferability of results between countries (with appropriate scaling for population size).

Given what is currently known about the efficiency of air purifiers, (see Table 4 for a list and descriptions of some of the relevant studies), it is reasonable to expect that when they are operated and maintained properly, reductions in indoor $PM_{2.5}$ of approximately 50% can be achieved. A reduction in exposure of this scale would have considerable impact on $PM_{2.5}$-related mortality, and lead to meaningful increases in life expectancy. For the central scenario, the reduction in $PM_{2.5}$ led to over 23 million YLG over the 97-year study period, and 138 additional days of life expectancy for males and 120 for females from the birth cohort. If $PM_{2.5}$ removal efficiency was increased to the highest reported (82.7%), the mortality effect was over 34 million YLG (over 97 years), and an additional 200 and 175 days of life expectancy for males and females, respectively. When the upper limits of the RRs were used in the model, the total YLG during the study period for 'All at Home' rose to over 36 million, illustrating the significance of these exposure-response functions in accurate estimations of effect. Perhaps unsurprisingly, the benefits of air purifier use are proportional to several factors including, the pre-intervention concentration, the total years used and duration of daily use.

The magnitude of the modelled impacts on mortality presented here are in general agreement with work that achieved reductions in $PM_{2.5}$ in

**Table 10**
Summary of findings from different pre-intervention $PM_{2.5}$ concentrations (6.6 μg/$m^3$ top and 18.8 μg/$m^3$ bottom).

| Baseline PM2.5 concentration 6.6 μg/m3 | | | | | |
|---|---|---|---|---|---|
| Outcome | Population | All at Home | All Sleep | 65+ Sleep | 65+ at Home |
| **Average years of life gained (YLG)** | male | 5,733,909 | 3,226,968 | 582,763 | 1,292,599 |
| | female | 5,111,198 | 2,883,609 | 538,704 | 1,188,735 |
| | Total | 10,845,107 | 6,110,577 | 1,121,467 | 2,481,334 |
| **Average days gained** | male | 63 | 36 | 6 | 14 |
| | female | 55 | 31 | 6 | 13 |
| **Baseline PM2.5 concentration 18.8 μg/m3** | | | | | |
| **Outcome** | **Population** | **All at Home** | **All Sleep** | **65+ Sleep** | **65+ at Home** |
| **Average years of life gained (YLG)** | male | 19,350,912 | 9,677,814 | 1,737,871 | 4,880,103 |
| | female | 17,416,358 | 8,723,828 | 1,620,836 | 4,539,975 |
| | Total | 36,767,270 | 18,401,643 | ,358,707 | 9,420,078 |
| **Average days gained** | male | 214 | 107 | 19 | 54 |
| | female | 188 | 94 | 17 | 49 |

other ways (e.g., mechanical ventilation with filtration, or sealing of the building envelope). One such study estimated the overall impact of energy efficiency upgrades in UK homes and found that for an average $PM_{2.5}$ reduction of 3 μg/m$^3$ there was an increase in life expectancy of two to three months (Milner et al., 2015). Another study of improved energy efficiency and ventilation of homes in England found that with a 53% reduction in $PM_{2.5}$ (−4.8 μg/m$^3$ mean) the net health impact was an increase of over 2000 quality adjusted life years (QALYs) per 10,000 persons over 50 years of follow-up (Hamilton et al., 2015).

This is the first study to estimate the potential health benefits of air purifier use in residences at the national scale. Although other studies have examined the impact of reductions in $PM_{2.5}$ in other building use types, using other filtration strategies, and for specific populations (e.g., Bekö et al., 2008; Fisk and Chan, 2017a). We used a widely applied method for health impact quantification and parameterised our models with data from the best available sources:

- Indoor $PM_{2.5}$ exposures and air purifier efficiencies obtained from reviewing the literature and selecting the most appropriate studies for the setting.
- Exposure-response functions from the GBD. The evidence on the mortality impact of long-term $PM_{2.5}$ exposure is robust and widely accepted as causal. Exposure-response functions are regularly updated as additional data becomes available, and therefore some uncertainty can be introduced. However, the GBD functions include one of the largest datasets from a diversity of settings and populations.

There are limitations in the work presented here. Health modelling provides an attractive and useful method of evaluating the impact of interventions on population health. However, the reliability of the results is subject to the accuracy of available sources of information, and the ability to add scientific credibility when those sources are uncertain. For this work, one source of uncertainty was the mean residential indoor $PM_{2.5}$ level in the UK. Average concentrations are likely to vary widely across the housing stock due to several, poorly characterised, factors, such as occupant behaviours and ventilation type. The mean indoor $PM_{2.5}$ pre-intervention concentrations used in the model were from monitoring by Lai et al. (2004) completed in Oxford, UK. The measured mean annual outdoor $PM_{2.5}$ concentration in that study was 6.2 μg/m$^3$, lower than the annual UK mean (8.1 μg/m$^3$) (Department for Environment Food and Rural Affairs, 2021). Therefore, the measured indoor concentration may not be fully representative of the entire UK housing stock. However, modelling of both higher and lower pre-intervention concentrations provided reasonable bounds for potentially variable conditions across the UK, and elsewhere.

Although based on standard demographic methods, the life table provides only a statistical representation of the average survival experience for a population. In reality, mortality risks vary across populations, with lower socioeconomic groups in particular having higher underlying mortality risks (and therefore shorter life expectancies) at a given age. The method used in this work also quantified only mortality and as such it represents a conservative estimate of the total impact of air purifiers on health, which would also include substantial impact on morbidity. Compared to the commonly used comparative risk assessment approach (used, for example, by the GBD study) the life table has the advantage of accounting for dynamic changes in survival over time, making it more appropriate for assessing the impacts of policies and interventions.

In addition to temporal and contextual changes, occupant behaviour is also likely to be one of the most significant factors in both the potential for the generation of, and exposure to, indoor $PM_{2.5}$. Time-activity patterns remain poorly characterised and are expected to vary widely by age, location, SES, etc. (Steinle et al., 2013) In addition to the potential impacts from occupant behaviours such as cooking or smoking in homes, actual air purifier operating behaviours are not well studied, and improper or inadequate use could have a substantial effect on the ability of the device to reduce $PM_{2.5}$. Future research investigating how, when, and why, air purifiers are used in actual homes would help close some of these gaps in our knowledge. Our central scenario represents an ambitious level of air purifier implementation in the UK. It is unlikely that the entire population of the UK (or of any country) could own, and properly operate, air purifiers whilst at home for the entirety of their lives. However, the estimation of the impact of use by the whole population for a lifetime is important for establishing a baseline that can inform policymakers and designers.

Although it is widely recognised that there exists $PM_{2.5}$-associated mortality and morbidity, there is debate about the distribution of severity and mechanism of impact. The modelling carried out for this work was based upon averages, and therefore cannot provide information on specific impacts and associated inequalities. Additionally, whether $PM_{2.5}$ from different sources and, therefore, in different locations, has different impacts on health outcomes is still largely unknown. The relative risks used in this work were from the 2019 GBD which are based upon ambient and household (primarily solid-fuel burning) $PM_{2.5}$ levels, which remains a limitation of this work. These gaps in our understanding bring additional uncertainties to the health impact modelling.

As noted earlier, this work does not consider morbidity associated with diseases linked to $PM_{2.5}$ exposure, although this is likely to be considerable as many of these disease (e.g., COPD) can have effects years before death. The work presented here focusses solely on the mortality effects as this provides critical information for assessing risk. However, future work that captures the wider impacts to health should be undertaken. Asthma, especially in children, is of significant concern, and a recent asthma death attribution lawsuit in London (Dyer, 2020) could have implications for policy around $PM_{2.5}$. The potential benefits from the use of air purifiers in homes on asthma incidence should be explored. In addition to asthma, morbidities associated with $PM_{2.5}$ exposure should be included in future modelling. The total impact to quality of life, as well as the economic implications, due to mortality and morbidity effects of indoor $PM_{2.5}$ are important tools for policymakers to determine the appropriate levels and types of interventions. This work was not aiming to provide a comprehensive economic analysis, but rather it aimed to put into perspective the relative benefits to mortality (for which we have the strongest evidence for a causal link to $PM_{2.5}$) from reductions in $PM_{2.5}$ indoors at home from air purifier use.

Recent research into risks of exposure to poor indoor air quality indicates that the people who may benefit the most from interventions with air purifiers, those with vulnerabilities related to age, pre-existing health conditions, housing conditions, access to interventions, etc., may be those least likely to have the economic means to afford air purifiers (Ferguson et al., 2020, 2021). The examination of social inequities with regard to environmental exposures is critical to the effective management of risk and should be explored further in the context of air purifiers. Understanding these inequities in countries where the ambient air quality is good, or improving, and where the economic means exist that could address inadequacies in the domestic building stock is important. Of equal importance is research in places where ambient levels of $PM_{2.5}$ are much higher, air quality regulations may be less stringent (or non-existent), the quality of housing is poor, and where the distribution of wealth is grossly uneven. Investigating issues of just and equitable access to technologies that can improve IAQ, and reduce $PM_{2.5}$-associated mortality is an important area of future work.

Another issue that is worth considering is if the reliance on individual households of air purifiers to address population-level $PM_{2.5}$ exposures could lead to mitigation deterrence. That is, laying the burden of improving IAQ at the level of the individual could disincentivise structural changes that need to be made in policy and at scale to reduce indoor exposures for all people. For example, the provision of housing with adequate ventilation, or improvements to ambient air quality. A parallel can be made with the issues of excess winter deaths from cold

and how that led to paying for extra fuel rather than solving the underlying housing problems (Balfour and Allen, 2014).

## 5. Conclusion

The results presented here indicate that using air purifiers at scale could provide considerable health benefits by reducing indoor $PM_{2.5}$ exposure in the UK. The higher the pre-intervention indoor concentration of $PM_{2.5}$ the more substantial the benefit to life expectancy (and potentially other measures of health and quality of life). Recommendations for interventions to reduce $PM_{2.5}$ should be targeted at those homes most likely to have the highest indoor $PM_{2.5}$ levels due to location and building characteristics (e.g., older and/or poorly maintained structures) to impart the greatest benefit to population health and mortality. In places where concerted and collaborative efforts by policymakers, designers, industry and environmental agencies cannot alone reduce indoor $PM_{2.5}$, air purifier use at home is an effective strategy to reduce mortality from $PM_{2.5}$ exposure and increase life expectancy.

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## CRediT authorship contribution statement

**Elizabeth Cooper:** Conceptualization, Investigation, Writing – original draft, Writing – review & editing, Project administration. **James Milner:** Writing – review & editing, Methodology, softwaresoftwarre. **Yan Wang:** Software, Investigation, Writing – review & editing. **Samuel Stamp:** Investigation, Writing – review & editing. **Dejan Mumovic:** Supervision, Funding acquisition, Writing – review & editing.

## Declaration of competing interest

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

## Data availability

Data will be made available on request.

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## Appendix A. Supplementary data

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