



# Housing interventions and health: Quantifying the impact of indoor particles on mortality and morbidity with disease recovery



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## ABSTRACT

Housing interventions for energy efficiency and greenhouse gas emission reduction have the potential to reduce exposure to indoor air pollution if they are implemented correctly. This work assessed the health impacts of home energy efficiency measures in England and Wales resulting in a reduction in average indoor PM<sub>2.5</sub> exposures of 3 µg m<sup>-3</sup>. The assessment was performed using a new multistate life table model which allows transition into and between multiple morbid states, including recovery to disease-free status and relapse, with transition rates informed by age- and cause-specific disease prevalence, incidence and mortality data. Such models have not previously included disease recovery. The results demonstrate that incorporation of recovery in the model is necessary for conditions such as asthma which have high incidence in early life but likelihood of recovery in adulthood. The impact assessment of the home energy efficiency intervention showed that the reduction in PM<sub>2.5</sub> exposure would be associated with substantial benefits for mortality and morbidity from asthma, coronary heart disease and lung cancer. The overall impact would be an increase in life expectancy of two to three months and approximately 13 million QALYs gained over the 90 year follow-up period. Substantial quality-of-life benefits were also observed, with a decrease in asthma over all age groups and larger benefits due to reduced coronary heart disease and lung cancer, particularly in older age groups. The multistate model with recovery provides important additional information for assessing the impact on health of environmental policies and interventions compared with mortality-only life tables, allowing more realistic representation of diseases with substantial non-mortality burdens.

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## 1. Introduction

There is evidence to suggest that current strategies designed to improve housing energy efficiency for greenhouse gas mitigation may affect levels of various contaminants in indoor air due to changes in the level of dwelling ventilation (Wilkinson et al., 2009). Modelling studies have demonstrated that, depending on the standard of implementation and provision of compensatory purpose-provided ventilation, there is the potential for increases or decreases in indoor concentrations (Milner et al., 2014; Shrubsole et al., 2012). Like many environmental exposures, indoor air quality may be important more for its impact on morbidity and quality-of-life than on mortality. Many of the affected indoor pollutants, including fine particulate matter (PM<sub>2.5</sub>), nitrogen dioxide (NO<sub>2</sub>) and mould, have been associated with reduced quality-of-life, primarily through adverse respiratory effects (Belanger et al., 2006; Fisk et al., 2007; Kattan et al., 2007; Simoni et al., 2004). For assessing health impacts resulting from housing interventions, preferred

methods of impact assessment should therefore incorporate morbidity as well as mortality impacts.

Methods for modelling changes in population mortality due to changes in chronic environmental risk factors are relatively well developed (Ballester et al., 2008; Röösli et al., 2005). A commonly used method has been the life table (e.g. Miller and Hurley, 2003), which estimates patterns of survival in a population over time. The approach has been used extensively in many fields of research to study impacts on population mortality and life expectancy, including assessments of environmental health risks at the national and local levels (e.g. COMEAP, 2010; Tonne et al., 2008). In contrast, morbidity impacts are often modelled using simplified methods with little or no consideration given to changes over time (Schram-Bijkerk et al., 2013). One method of accounting for morbidity impact is the multistate life table (Barendregt et al., 1998; Feenstra et al., 2001), an extension to the standard life table in which individuals in the population move between different health states, including death as a terminal state. Time spent with disease is weighted for the reduced quality-of-life. Such models have been used to study disease patterns in older age (Lubitz et al., 2003; Nusselder and Peeters, 2006) but there have been relatively few applications to the assessment of environmental hazards (McCarthy et al., 2002). Further, multistate life table models have not

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previously allowed for recovery from disease: this is potentially an important limitation for conditions such as childhood asthma, which are often transitory (Sears et al., 2003).

In this paper we present an assessment of the health impact of changes in indoor fine particle pollution that might arise under future energy efficiency improvements in UK housing. The work uses a newly developed multistate life table model which integrates morbidity into the standard life table method and incorporates transitions between disease states, including the potential to recover from (and relapse to) disease.

## 2. Methods

Our analysis focuses on exposure to particulate air pollution with a maximum aerodynamic diameter of  $2.5 \mu\text{m}$  ( $\text{PM}_{2.5}$ ). A study of the public health 'co-benefits' of household energy efficiency policies for climate change mitigation in the UK was used as the basis for an assessment of the impact on health of changes in indoor  $\text{PM}_{2.5}$  exposure (Wilkinson et al., 2009). In the study, changes in residential indoor  $\text{PM}_{2.5}$  exposures for the UK population were modelled using a multizone building model for four hypothetical greenhouse gas emission reduction strategies (building fabric improvements, improved ventilation, fuel switching, and occupant behaviour changes) whose net effect was to reduce annual average  $\text{PM}_{2.5}$  indoor concentrations by  $3.0 \mu\text{g m}^{-3}$  by 2050, compared with the 2010 baseline.

To assess the potential impact on both mortality and morbidity of this reduction in indoor  $\text{PM}_{2.5}$  exposure in England and Wales, we have developed a multistate life table model which allows individuals in the population to exist in, and move between, a *good health* state, a number of *disease* states, a *recovered* state and *death*. A simplifying assumption is that individuals may have only one form of disease at a time. Inclusion of a recovered state is important to allow the rate at which individuals relapse to potentially differ from the rate at which individuals acquire disease from good health. The model was implemented using the open source statistical software R (R Core Team, 2012). In this work, the impact of  $\text{PM}_{2.5}$  changes was calculated on all-cause mortality, cardiovascular (coronary heart disease), lung cancer, and asthma mortality and morbidity (US EPA, 2009) (Fig. 1).

### 2.1. Model description

The multistate life table calculations are based on relatively simple population balance arithmetic extended to disease recovery and relapse. That is, the population leaving any state each year must be balanced by an equivalent movement of individuals into other states (which

may include death). The starting point is to calculate the probability of movement between every permissible combination of health states at each age. These probabilities are derived from age-specific population, all-cause and disease-specific mortality, and disease prevalence and incidence data. The probability of movement to state  $k$  from state  $j$  at age  $i$  ( $h_{i,j,k}$ ) is found from the number of individuals moving from  $j$  to  $k$  at age  $i$  ( $n_{i,j,k}$ ) divided by the population of state  $j$  at that age ( $p_{i,j}$ )

$$h_{i,j,k} = n_{i,j,k}/p_{i,j}.$$

Depending on the starting ( $j$ ) and finishing ( $k$ ) states at age  $i$ , movement between health states may represent either new cases of disease, recovery from disease, relapse to disease, or death. Movement between some health states is not permitted (e.g. there is no movement from death to any of the other states). In such situations,  $n_{i,j,k}$  is equal to zero and, hence,  $h_{i,j,k}$  becomes zero also. Assuming that deaths, new disease cases, disease recovery, and disease relapse all occur at a constant rate over a year of age (a standard life table assumption, e.g. Bradford Hill (1977)), the probability of remaining in state  $j$  by not moving to state  $k$  from age  $i$  to  $i + 1$  ( $s_{i+1,j,k}$ ) can be shown to be

$$s_{i+1,j,k} = (2 - h_{i,j,k}) / (2 + h_{i,j,k}).$$

For example, in the case of movement between a given health state  $j$  and the death state  $d$ ,  $s_{i+1,j,d}$  represents the probability of not dying (i.e. the survival probability) in that state from age  $i$  to  $i + 1$ , conditional on surviving to age  $i$ . It is then possible to calculate probabilities of individuals not moving to another state from birth to age  $i + 1$  using the cumulative probability of survival in that state from age 0 to  $i + 1$ , the probability of remaining in a given state (from birth to age  $i + 1$ ) and the probability of moving to each state (again, from birth to  $i + 1$ ). It is then straightforward to estimate the expected number of deaths and new disease cases in the population at a particular year of age. The proportions of the cohort in each health state at the end of a given year of age are found by multiplying together the appropriate probabilities described above (e.g. remaining in good health requires *not* moving to any disease state *and not* dying) and then summing the movements into and out of each state. The population in each health state is the result of survival and movement between states in the previous year. The populations in each state are then used to determine the fraction of a life year (LY) lived by these different groups, which may be weighted in relation to the reduced quality-of-life experienced by individuals. Finally, combining the resulting fractions of life years lived in the various health states leads to a quality-adjusted total number of life years (QALYs), from which the quality-adjusted life expectancy (QALE) remaining at each age is calculated. More detailed model equations can be found in a Web Appendix to this paper.

### 2.2. Model testing

The output from the multi-morbid state model should theoretically match that of a standard (mortality only) life table model if (1) the all-cause mortality rates used in the two models are the same, (2) the disease-specific mortality rates in all disease states are the same as the all-cause rates (i.e. diseases do not increase or decrease the risk of mortality) and (3) all quality-of-life weights are set to one (i.e. no reduction in quality-of-life due to disease). This is true irrespective of the number of disease states modelled and the disease incidence/prevalence rates. As a boundary test, therefore, the results of the steady-state multistate model with recovery were compared against the widely-used IOMLIFET standard life table model (Miller and Hurley, 2003) for up to three disease states and reproduced exactly the life year and life expectancy outputs of the standard model ( $R^2 = 1$ ).

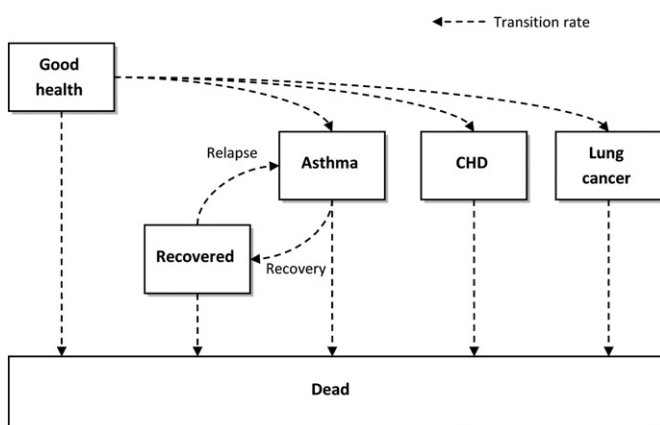


Fig. 1. Health states and pathways used in multistate life table to model impact of intervention.

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