**NOTICE: this is the author’s version of a work that was accepted for publication in Environment International. Changes resulting from the publishing process, such as peer review, editing, corrections, structural formatting, and other quality control mechanisms may not be reflected in this document. Changes may have been made to this work since it was submitted for publication. A definitive version was subsequently published in Environment International, VOL 81, (2015) DOI 10.1016/j.envint.2015.04.011**

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

James Milner1,\* james.milner@lshtm.ac.uk

Zaid Chalabi1 zaid.chalabi@lshtm.ac.uk

Sotiris Vardoulakis2,1 sotiris.vardoulakis@phe.gov.uk

Paul Wilkinson1 paul.wilkinson@lshtm.ac.uk

1 Department of Social & Environmental Health Research, London School of Hygiene & Tropical Medicine, 15-17 Tavistock Place, London, WC1H 9SH, UK

2 Centre for Radiation, Chemical & Environmental Hazards, Public Health England, Harwell Campus, OX11 0RQ, UK

\*Correspondence to:
Dr James Milner, Department of Social & Environmental Health Research, London School of Hygiene & Tropical Medicine, 15-17 Tavistock Place, London, WC1H 9SH, UK
Email: james.milner@lshtm.ac.uk. Tel: +44 (0)20 7927 2510. Fax: +44 (0)20 7580 4524

Word count: 3,500**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 PM2.5 exposures of 3 µg m-3. 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 PM2.5 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.

**Keywords** Indoor air pollution, Particulate matter, Health impact assessment, Quality of life, Disease recovery

**Highlights**

* Home energy efficiency interventions, if properly implemented, have the potential to improve indoor air quality
* Assessing the resulting health impact requires a model which accounts for effects on both mortality and morbidity
* This study demonstrates a new multi-state life table model which incorporates morbidity impacts
* The model includes disease recovery, which is important for some chronic conditions like asthma
* Including morbidity and recovery allows more thorough assessment of policies and interventions
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 (PM2.5), nitrogen dioxide (NO2) 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 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 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.

1. **METHODS**

Our analysis focuses on exposure to particulate air pollution with maximum aerodynamic diameter of 2.5 microns (PM2.5). 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 PM2.5 exposure (Wilkinson et al., 2009). In the study, changes in residential indoor PM2.5 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 PM2.5 indoor concentrations by 3.0 µg m-3 by 2050, compared with the 2010 baseline.

To assess the potential impact on both mortality and morbidity of this reduction in indoor PM2.5 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 PM2.5 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* (*hi,j,k*) is found from the number of individuals moving from *j* to *k* at age *i* (*ni*,*j*,*k*) divided by the population of state *j* at that age (*pi*,*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, *ni*,*j*,*k* is equal to zero and, hence, *hi*,*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 (*si+1,j,k*) can be shown to be

$$s\_{i+1,j,k}={\left(2-h\_{i,j,k}\right)}/{\left(2+h\_{i,j,k}\right)}$$

For example, in the case of movement between a given health state *j* and the death state *d*, *si*+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 (QALY), 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 (R2 = 1).

**2.3 Model parameterisation**

The multistate life table was used to assess the benefits for all-cause mortality, coronary heart disease (CHD), lung cancer, and asthma resulting from the 3.0 µg m-3 reduction in PM2.5 in the population of England and Wales. The model was parameterized for these health outcomes using age-specific data representing existing distributions of mortality and disease rates for England and Wales (Table 1). Given the limited evidence available on age-specific rates of asthma recovery/relapse, plausible approximations of the complex disease dynamics were assumed based on a longitudinal study of asthma incidence and prognosis (Strachan et al., 1996). Rates of relapse were based on rates of incidence, while for recovery simple age-specific functions were applied. These functions assumed an increase in the probability of recovery from birth until age 10, followed by a decrease to age 20, after which a constant level of recovery was assumed. The increasing and decreasing components of the recovery probability were specifically designed to maximise the correspondence of the modelled asthma prevalence to the observed prevalence in the population (to ensure that unrealistic increases or decreases in baseline prevalence did not occur over time).

The exposure-response functions used in the model to assess the impact of the intervention were based on various sources (Table 1). For mortality, evidence was obtained from the American Cancer Society (ACS) cohort study on associations between long-term exposure to PM2.5 and all-cause, cardiovascular, and lung cancer mortality (Pope et al., 2002; 2004). For morbidity associated with PM2.5, evidence for CHD was based on the Women’s Health Initiative study (Miller et al., 2007), evidence for lung cancer was based on the ACS study (Pope et al., 2002; 2004), and evidence for asthma was from the Dutch Prevention and Incidence of Asthma and Mite Allergy study (Gehring et al., 2010). Disease-specific utility (quality-of-life) weights were based on WHO Global Burden of Disease (GBD) analyses (WHO, 2008).

To account for the lagged effect of the intervention on changes in health status, time-dependent exponential functions were used to model the latency period between changes in exposure and changes in risk. For all-cause mortality, CHD and lung cancer impacts, the full reduction in risk was assumed to be experienced after 20 years, based on empirical evidence of the effect of smoking cessation over time and plausible assumptions about disease progression over time (Lin et al., 2008). Changes in asthma risk were assumed to occur without any lag.

The simulations were carried out over a follow up period of 90 years to allow the population alive at the time of the intervention to die out (including the 2009 birth cohort) and to reflect the full impact on the starting population. Simulations were performed both with and without asthma recovery to assess the impact of this on the model predictions of (1) baseline disease prevalence and (2) the impact of the intervention. Further details on model parameterisation and input data can be found in the Web Appendix.

**3. RESULTS**

Without recovery and relapse in the simulation, asthma prevalence is determined solely by the incidence and mortality rates, resulting in unrealistically high levels of the condition in the population (Fig. 2). In reality, many people with asthma will recover, particularly in adolescence and early adulthood; this movement from asthma to the recovered state is required in the model to replicate more closely the actual prevalence. On the other hand, for conditions such as CHD and lung cancer, which occur mostly in older ages and without appreciable recovery, the modelled levels in the population remain relatively constant into the future.

The model, including asthma recovery, suggests the intervention (3 µg m-3 reduction in PM2.5) would result in an increase of just over 13 million QALYs over the modelled 90 year follow up period (Table 2). There would also be an average increase in quality-adjusted life expectancy at birth of 101 and 68 days for males and females, respectively. The slightly larger gains for males represent the higher baseline mortality rates and prevalence of childhood asthma, coronary heart disease and lung cancer in males. Since the reduced PM2.5 exposure results in people living longer on average, the main benefit in life years lived is experienced at older ages. As Table 2 shows, the incorporation of asthma recovery has only a modest effect on the life table results, since the overall health impact is dominated by the higher severity health outcomes (mortality, CHD and lung cancer).

Benefits of the intervention for mortality (not plotted here) would generally increase with age, reaching a peak at around age 80 with over 300 fewer deaths in the population per year at this age by the end of the follow up period. Due to the resulting upward shift in the age structure of the population, at above age 80 the benefits begin to decrease. Approaching age 90 more deaths occur following the intervention than in the baseline (pre-intervention) scenario because the reduced exposure does not avert deaths but merely postpones them: the total number of deaths remains the same over the longer term, leading to an increase at older ages as the population at these ages becomes larger.

The multistate model also provides relevant morbidity information that cannot be obtained using a standard life table, demonstrating substantial reductions in the number of new disease cases due to the intervention and corresponding reductions in disease burdens over the follow up period (Fig. 3). Note that here the plots show the burden averted for each disease. After the full 90 year follow up period, approximately 260,000 fewer people in England and Wales would have asthma, 55,000 fewer would have CHD, and 3,000 fewer would have lung cancer. Again the plot demonstrates that, unlike the model predictions of baseline disease prevalence (Fig. 2), inclusion of asthma recovery in the intervention impact assessment has only a minor effect on the results.

Figure 4 shows the reduction in the number of cases of each disease at different ages by the end of the follow up period. For asthma, there is benefit across all ages. However, for CHD and lung cancer, benefits begin to accrue from approximately age 40 and increase until roughly age 70. Above this age, the relative benefit decreases and approaching age 90 there would be a greater number of people with each disease than at present. Again, this is due to the increased population at older ages resulting from increases in life expectancy due to the housing intervention.

1. **DISCUSSION**

Our results suggest that home energy efficiency interventions in England and Wales, leading to a reduction in average indoor exposure to PM2.5 by 3.0 µg m-3, would increase quality adjusted average life expectancy by approximately three and a half months for males and two months females, and a greater proportion of life would spent be in good health. Over the modelled 90 year follow up period, the population would live an additional 13 million QALYs. By this time, there would be roughly 260,000 fewer people with asthma, 53,000 fewer with CHD and 3,000 fewer with lung cancer. As demonstrated, our multistate life table model can yield results of health impact with a greater level of detail than those of a conventional mortality-only life table, by allowing the accounting of changes in morbid states, including recovery from disease. This provides more realistic representation of disease dynamics which may be important for the accurate estimation of overall health burdens for diseases that have impacts on quality-of-life as well as the duration of life. Allowing modellers and policy-makers to study morbidity with recovery in addition to mortality may be important for many policy assessments, especially where, as with indoor air quality and respiratory illness, there may be frequent adverse effects but seldom fatal. The new multistate model is well suited to modelling such complexities, allowing the user to study the implications for various disease states in different age groups where the balance of morbidity and mortality may vary.

The results serve to highlight important benefits of the model. First, for ‘transient’ conditions like asthma where people may potentially recover and relapse from the disease, it is important to model these effects. Failure to do so may result in overestimation of baseline disease prevalence, though only modest errors in evaluations of the impacts of interventions. Second, for conditions which are prevalent in old age, the multistate model captures the ways in which changes in life expectancy can result in increases in the disease burden at old age (since there will be a greater number of people alive at these ages). Such subtle effects would not be captured explicitly by other methods, such as the Comparative Risk Assessment (CRA) method used by the WHO’s GBD studies (e.g. Lim et al., 2012) or DisMod (Barendregt et al., 2003). Although there are some similarities between our model and DisMod at the basic level in terms of population balance between health states, DisMod cannot be used easily for health impact assessment. There are other dynamic methods available for assessing the morbidity impacts of interventions, primarily microsimulation tools including the Dynamic Modeling for Health Impact Assessment (DYNAMO-HIA) model (Lhachimi et al., 2012), the OECD/WHO Chronic Disease Prevention (CDP) model (e.g. Cecchini et al., 2010), and the UK Health Forum’s Foresight model (McPherson et al., 2012). However, these are complex models requiring large input data requirements and high computing resources. Other HIA methods have been developed for specific diseases or determinants of health only, such as the smoking-related Quit Benefits Model (Hurley and Matthews, 2007). In comparison, alongside the aforementioned benefits related to incorporation of disease recovery, our model also has the advantages of (relative) simplicity and generality.

In its current form the multi-state model makes certain simplifying assumptions. First, it assumes that exposure has an impact on the incidence of disease but not on the course of the disease and, second, that disease incidence and mortality are mutually independent. It also relies on simplifying assumptions about changes over time, which will require further development of methods. For example, the model currently also assumes baseline mortality and morbidity rates remain constant into the future. Although this is unrealistic, previous work has demonstrated that the magnitudes of health impacts estimated using life tables are relatively insensitive to the baseline rates (Miller and Hurley, 2006). More realistic disease dynamics could also be incorporated, for instance accounting for the gradual changes in risk and health outcomes that result from the introduction over time of housing interventions into the stock and resulting changes in exposure (Ezzati and Lopez, 2004). Finally, the model only allows individuals to exist in one state at a time. As such, a further level of realism could be provided by allowing multiple simultaneous diseases (co-morbidities). However, as highlighted in previous work, data limitations provide a significant barrier to the development and implementation of quantitative models of health impact (Milner et al., 2011; Veerman et al., 2005). The development of this and similar modelling tools would benefit greatly from improved age-specific data on disease prevalence and incidence rates, disease duration and recovery/relapse patterns with age. Often such data are of very limited availability, meaning plausible patterns must be derived from incomplete evidence.

1. **CONCLUSIONS**

This paper has presented a modelling assessment of the benefits for mortality and morbidity of home energy efficiency interventions in England and Wales whose combined effects reduce average indoor PM2.5 exposures by 3.0 µg m-3 (based on Wilkinson et al., 2009). The analysis was performed using a newly developed multistate life table which is capable of accounting for disease recovery and relapse, providing a significant improvement on similar existing models. Inclusion of recovery and relapse was demonstrated to result in much better prediction of underlying disease prevalence for certain conditions, particularly ‘transient’ ones which are prevalent in earlier life.

Including disease recovery in health impact models is an important step towards quantifying the effects on population health of complex actions and interventions in a comprehensive manner, which requires knowledge of the time course of both mortality and morbidity impacts. This, in turn, enables more thorough policy appraisal to be undertaken, including analyses of the effects of targeting policies at specific groups and identification of unintended adverse consequences within the population (for example, based on their age or underlying health status). Such refinement in health impact modelling is particularly relevant for ageing populations, with the increased prevalence of many chronic conditions and the shifting of the disease burden to older ages likely to have major repercussions for the cost of health care in the future (Caley and Sidhu, 2011; Christensen et al., 2009).

**Acknowledgements**

This work, including development of the health impact model, was conducted as part of the project “Pollutants in the Urban Environment: An Integrated Framework for Improving Sustainability of the Indoor Environment (PUrE Intrawise)”, funded by EPSRC (Grant no. EP/F007132/1). The authors gratefully acknowledge this funding and the support of other project partners. The research leading to these results has also in part received funding from the European Union Seventh Framework Programme FP7/2007-2013 under grant agreement no 265325 (“PURGE”).

**REFERENCES**

1. Ballester F, Medina S, Boldo E, et al., 2008. Reducing ambient levels of fine particulates could substantially improve health: a mortality impact assessment for 26 European cities. *J Epidemiol Community Health* **62**, 98‒105.
2. Barendregt JJ, Van Oortmarssen GJ, Van Hout BA, et al., 1998. Coping with multiple morbidity in a life table. *Math Popul Stud* **7**, 29‒49.
3. Barendregt JJ, Van Oortmarssen GJ, Vos T, et al., 2003. A generic model for the assessment of disease epidemiology: the computational basis of DisMod II. *Popul Health Metr* **1**, 4.
4. Belanger K, Gent JF, Triche EW, et al., 2006. Association of indoor nitrogen dioxide exposure with respiratory symptoms in children with asthma. *Am J Respir Crit Care Med* **173**, 297‒303.
5. Bradford Hill A, 1977. *A Short Textbook of Medical Statistics*. Hodder & Stoughton Ltd, London, UK.
6. Caley M, Sidhu K, 2011. Estimating the future healthcare costs of an aging population in the UK: expansion of morbidity and the need for preventative care. *J Public Health* **33**, 117‒22.
7. Cecchini M, Sassi F, Lauer JA, et al., 2010. Tackling of unhealthy diets, physical inactivity, and obesity: health effects and cost-effectiveness. *Lancet* **376**, 1775‒84.
8. Christensen K, Doblhammer G, Rau R, Vaupel JW, 2009. Ageing populations: the challenges ahead. *Lancet* **374**, 1196‒1208.
9. COMEAP, 2009. *Long-Term Exposure to Air Pollution: Effect on Mortality*. Committee on the Medical Effects of Air Pollutants, Chilton, UK.
10. COMEAP, 2010. *The Mortality Effects of Long-Term Exposure to Particulate Air Pollution in the United Kingdom*. Committee on the Medical Effects of Air Pollutants, Chilton, UK.
11. Ezzati M, Lopez AD, 2004. Smoking and oral tobacco use. In: Ezzati M, Lopez AD, Rodgers A, et al. (Eds), *Comparative Quantification of Health Risks: Global and Regional Burden of Disease Attributable to Selected Major Risk Factors*. World Health Organization, Geneva, Switzerland.
12. Feenstra TL, van Genugten MLL, Hoogenveen RT, et al., 2001. The impact of aging and smoking on the future burden of chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* **164**, 590‒6.
13. Fisk WJ, Lei-Gomez Q, Mendell MJ, 2007. Meta-analyses of the associations of respiratory health effects with dampness and mold in homes. *Indoor Air* **17**, 284–96.
14. Forman D, Stockton D, Møller H, et al., 2003. Cancer prevalence in the UK: results from the EUROPREVAL study. *Ann Oncol* **14**, 648‒54.
15. Gehring U, Wijga AH, Brauer M, et al., 2010. Traffic-related air pollution and the development of asthma and allergies during the first 8 years of life. *Am J Respir Crit Care Med* **181**, 596‒603.
16. Hurley S, Matthews J, 2007. The Quit Benefits Model: a Markov model for assessing the health benefits and health care cost savings of quitting smoking. *Cost Eff Resour Alloc* **5**, 2.
17. Kattan MK, Gergen PJ, Eggleston P, et al., 2007. Health effects of indoor nitrogen dioxide and passive smoking on urban asthmatic children. *J Allergy Clin Immunol* **120**, 618‒24.
18. Lhachimi SK, Nusselder WJ, Smit HA, et al., 2012. DYNAMO-HIA–a dynamic tool modeling for generic health impact assessments. *PLOS One* **7**, e33317.
19. Lim SS, Vos T, Flaxman AD, et al., 2012. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet* **380**, 2224-60.
20. Lin HH, Murray M, Cohen T, et al., 2008. Effects of smoking and solid-fuel use on COPD, lung cancer, and tuberculosis in China: a time-based, multiple risk factor, modelling study. *Lancet* **372**, 1473-83.
21. Lubitz J, Cai L, Kramarow E, et al., 2003. Health, life expectancy, and health care spending among the elderly. *NEJM* **349**, 1048‒55.
22. McCarthy M, Biddulph JP, Utley M, et al., 2002. A health impact assessment model for environmental changes attributable to development projects. *J Epidemiol Community Health* **56**, 611‒6.
23. McPherson K, Marsh T, Rtveladze K, et al., 2012. Future modelling of chronic diseases: foresight and beyond. *Lancet* **380**, S9.
24. Miller BG, Hurley JF, 2003. Life table methods for quantitative impact assessments in chronic mortality. *J Epidemiol Community Health* **57**, 200‒6.
25. Miller BG, Hurley JF, 2006. *Comparing estimated risks for air pollution with risks for other health effects*. IOM Research Report TM/06/01. Institute of Occupational Medicine, Edinburgh, UK.
26. Miller KA, Siscovick DS, Lianne Sheppard MPH, et al., 2007. Long-term exposure to air pollution and incidence of cardiovascular effects in women. *NEJM* **356**, 447‒58.
27. Milner J, Shrubsole C, Das P, et al., 2014. Home energy efficiency and radon related risk of lung cancer: modelling study. *BMJ* **348**, f7493.
28. Milner J, Vardoulakis S, Chalabi Z, Wilkinson P, 2011. Modelling inhalation exposure to combustion-related air pollutants in residential buildings: application to health impact assessment. *Environ Int* **37**, 268–79.
29. Nusselder WJ, Peeters A, 2006. Successful aging: measuring the years lived with functional loss. *J Epidemiol Community Health* **60**, 448‒55.
30. Pope CA III, Burnett RT, Thun MJ, et al., 2002. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *JAMA* **287**, 1132‒41.
31. Pope CA III, Burnett RT, Thurston GD, et al., 2004. Cardiovascular mortality and long-term exposure to particulate air pollution. *Circulation* **109**, 71‒7.
32. R Core Team, 2012. *R: A language and Environment for Statistical Computing*. R Foundation for Statistical Computing, Vienna, Austria. http://www.R-project.org/
33. Röösli M, Künzli N, Braun-Fahrländer C, Egger M, 2005. Years of life lost attributable to air pollution in Switzerland: dynamic exposure-response model. *Int J Epidemiol* **34**, 1029‒35.
34. Schram-Bijkerk D, van Kempen EEMM, Knol AB, 2013. The burden of disease related to indoor air in the Netherlands: do different methods lead to different results? *Occup Environ Med* **70**, 126‒32.
35. Sears MR, Greene JM, Willan AR, et al., 2003. A longitudinal, population-based, cohort study of childhood asthma followed to adulthood. *NEJM* **349**, 1414‒22.
36. Shrubsole C, Ridley I, Biddulph P, et al., 2012. Indoor PM2.5 exposure in London’s domestic stock: modeling current and future exposures following energy efficient refurbishment. *Atmos Environ* **62**, 336‒43.
37. Simoni M, Scognamiglio A, Carrozzi L, et al., 2004. Indoor exposures and acute respiratory effects in two general population samples from a rural and an urban area in Italy. *J Expo Anal Environ Epidemiol* **14**, 144‒52.
38. Strachan DP, Butland BK, Anderson HR, 1996. Incidence and prognosis of asthma and wheezing illness from early childhood to age 33 in a national British cohort. *BMJ* **312**,1195‒9.
39. Sutcliffe SJ, Fox KF, Wood DA, et al., 2003. Incidence of coronary heart disease in a health authority in London: review of a community register. *BMJ* **326**, 320.
40. Tonne C, Beevers S, Armstrong BG, et al., 2008. Air pollution and mortality benefits of the London Congestion Charge: spatial and socioeconomic inequalities. *Occup Environ Med* **65**, 620‒7.
41. US EPA, 2009. *Integrated Science Assessment for Particulate Matter*. United States Environmental Protection Agency, Research Triangle Park, NC, USA.
42. Veerman JL, Barendregt JJ, Mackenbach JP, 2005. Quantitative health impact assessment: current practice and future directions. *J Epidemiol Community Health* **59**,361‒70.
43. WHO, 2008. *Global Burden of Disease 2004 Update: Disability Weights for Diseases and Conditions*. World Health Organization, Geneva, Switzerland.
44. Wilkinson P, Smith KR, Davies M, et al., 2009. Public health benefits of strategies to reduce greenhouse-gas emissions: household energy. *Lancet* **374**, 1917‒29.

**Figure captions**

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

**Fig. 2.** Modelled disease prevalence for asthma, coronary heart disease (CHD) and lung cancer in England and Wales. Also includes comparison of modelled asthma prevalence with and without recovery/relapse included in the simulation.

**Fig. 3.** Impact of intervention on population with asthma (with and without recovery/relapse), coronary heart disease and lung cancer prevalence over time in England and Wales.

**Fig. 4.** Changes in population with (a) asthma (with and without recovery/relapse), (b) coronary heart disease and (c) lung cancer due to intervention in England and Wales in final year of follow up.

**Table 1.** Input data for home energy efficiency health impact assessment.

|  |  |  |  |
| --- | --- | --- | --- |
| Parameter | Health states | Source | Comments |
| Population | All | Office for National Statistics*a* | 2009 estimated mid-year resident population in England and Wales (26.98 million male, 27.83 million female) |
| Mortality | All | Office for National Statistics | 2009 deaths in England and Wales (238,062 male, 252,286 female) |
| Disease prevalence | Asthma | Office for National Statistics | 1998 prevalence of treated asthma in England and Wales |
|  | CHDLung cancer | Office for National StatisticsEUROPREVAL study (Forman et al., 2003) | 1994-98 prevalence of treated CHD in England and Wales1992 prevalence of lung cancer in UK (estimated using incidence and survival trends) |
| Disease incidence | Asthma | NHS Hospital Episode Statistics*b* | 2007-08 primary diagnosis of asthma in England |
|  | CHDLung cancer | Bromley Coronary Heart Disease Register (Sutcliffe et al., 2003)Cancer Research UK*c* | 1996-98 incidence of CHD in Bromley (England)2007 new cases of lung cancer in UK |
| Disease quality-of-life weights | AsthmaCHDLung cancer | WHO Global Burden of Disease 2004 (WHO, 2008)WHO Global Burden of Disease 2004 (WHO, 2008)WHO Global Burden of Disease 2004 (WHO, 2008) | 0.957 (asthma cases)0.8 (estimate based on acute myocardial infarction, angina pectoris, congestive heart failure)0.25 (metastasis stage for all cancers) |
| Exposure-response (mortality) | All | American Cancer Society study (Pope et al., 2002; 2004) | 1.06 increase in all-other and asthma mortality, 1.18 increase in cardiovascular mortality and 1.14 increase in lung cancer mortality per 10 µg m-3 (applied to ages 30+) based on US cohort study of outdoor air PM2.5.d Recommended as appropriate for use in UK (COMEAP, 2009). |
| Exposure-response (morbidity) | Asthma | Dutch Prevention and Incidenceof Asthma and Mite Allergy study (Gehring et al., 2010) | 1.28 increase in asthma incidence per interquartile range based on cohort study of children in Netherlands |
|  | CHDLung cancer | Women’s Health Initiative study (Miller et al., 2007)American Cancer Society study (Pope et al., 2002; 2004) | 1.21 increase in CHD per 10 µg m-3 based on cohort study of women in US.1.14 increase in lung cancer per 10 µg m-3 based on American Cancer Society lung-cancer mortality coefficient |

ahttp://www.ons.gov.uk
bhttp://www.hesonline.nhs.uk
chttp://www.cancerresearchuk.org
dBased on ambient PM2.5 given lack of equivalent epidemiologic evidence for indoor PM2.5 exposure

**Table 2.** Summary results of home energy efficiency health impact assessment.

|  |  |
| --- | --- |
| Health outcome | Increase due to intervention |
| With asthma recovery | Without asthma recovery |
| Quality-adjusted life years (QALY) lived over 90 yearsa | 13,155,000 | 13,245,000 |
| Quality-adjusted life expectancy (QALE) at birthb |  |  |
| Males | 101 days | 101 days |
| Females | 68 days | 69 days |

aResults rounded to nearest 1,000
bFor 2009 birth cohort