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Health effects of home energy efficiency interventions in England: a modelling study

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ABSTRACT

Objective: To assess potential public health impacts of changes to indoor air quality and temperature due to energy efficiency retrofits in English dwellings to meet 2030 carbon reduction targets.

Design: Health impact modelling study.

Setting: England.

Participants: English household population.

Intervention: Three retrofit scenarios were modelled: (1) fabric and ventilation retrofits installed assuming building regulations are met; (2) as with scenario (1) but with additional ventilation for homes at risk of poor ventilation; (3) as with scenario (1) but with no additional ventilation to illustrate the potential risk of weak regulations and non-compliance.

Main outcome: Primary outcomes were changes in quality adjusted life years (QALYs) over 50 years from cardiorespiratory diseases, lung cancer, asthma and common mental disorders due to changes in indoor air pollutants, including secondhand tobacco smoke, $PM_{2.5}$ from indoor and outdoor sources, radon, mould, and indoor winter temperatures.

Results: The modelling study estimates showed that scenario (1) resulted in positive effects on net mortality and morbidity of 2241 (95% credible intervals (CI) 2085 to 2397) QALYs per 10 000 persons over 50 years follow-up due to improved temperatures and reduced exposure to indoor pollutants, despite an increase in exposure to outdoor-generated particulate matter with a diameter of 2.5 μm or less ($PM_{2.5}$). Scenario (2) resulted in a negative impact of −728 (95% CI −864 to −592) QALYs per 10 000 persons over 50 years due to an overall increase in indoor pollutant exposures. Scenario (3) resulted in −539 (95% CI −678 to -399) QALYs per 10 000 persons over 50 years follow-up due to an increase in indoor exposures despite the targeting of pollutants.

Conclusions: If properly implemented alongside ventilation, energy efficiency retrofits in housing can improve health by reducing exposure to cold and air pollutants. Maximising the health benefits requires careful understanding of the balance of changes in pollutant exposures, highlighting the importance of ventilation to mitigate the risk of poor indoor air quality.

Strengths and limitations of this study

- The epidemiological evidence about health effects associated with indoor air pollutants and thermal stress is of varying certainty, though more evidence exists for exposure to outdoor pollution and temperature; therefore, only exposures with strong evidence were used.
- This study uses advanced validated building physics models to determine the change in indoor pollutant and thermal exposures related to energy efficiency retrofits.
- The uncertainty in the exposure responses on estimates of health impacts, such as the estimates for cold-related deaths, the toxicity level of particles derived from indoor sources and mental health, could result in a different balance of pollution impact depending on the assumptions made.
- While offering policymakers a support tool to include health as a criterion when developing and assessing home energy efficiency policy, the results presented here should be viewed with a clear understanding of the limitations associated with a modelling study.

INTRODUCTION

By 2030, the UK housing stock will undergo major changes to improve its energy performance,[1] motivated by the need to reduce emissions of greenhouse gases (GHGs), considerations of energy security/cost, and concern about fuel poverty with its presumed link to the UK's large burden of winter/cold-related mortality and morbidity.[2] Housing is responsible for one-quarter of total UK CO_2 emissions[3] and 52% of this is from space heating. Meeting the UK's ambitious energy efficiency targets will require investments to upgrade the energy performance of nearly all dwellings by 2030.[1] These changes to housing energy performance will comprise one of the largest natural experiments in the indoor environment in the coming decades and these are likely to have major impacts on the indoor environment and population health.[4 5] To date, health consequences have received limited examination,[6] though they are increasingly being recognised as an issue by the UK Government.[7]

Properly designed and implemented, actions to improve housing energy performance could have major co-benefits for public health,[4] although there are risks involved and the possibility of poorly designed interventions leading to unintended consequences (figure 1).[8–10] Energy efficiency retrofits that alter the fabric heat loss can also increase the air tightness of the dwelling,[11 12] increasing exposure to indoor-generated pollutants (eg, particulates, mould, radon). Living in cold or inefficient and poorly ventilated homes is linked to a range of health problems.[5 10 13] Retrofits that improve indoor temperatures may have positive impacts on mental health and cardiorespiratory disease,[5] but could have negative impacts on respiratory conditions due to the increased levels of indoor pollutants.[14 15] In the UK, most of our time is spent indoors and the majority of the health impact of more airtight buildings is likely to occur over the long term through low-dose exposure.[16]

While current English building regulations requires that adequate means of ventilation is provided to dwellings,[17] there is a lack of guidance for determining the level of ventilation required to protect health before or following an energy efficiency retrofit.[18] The only guidance that exists relates to the replacement of existing window trickle vents. Ultimately, additional ventilation following a retrofit is left to the discretion of the installer or household. The aim of this study is to illustrate the potential health impact of energy efficiency retrofits under different ventilation settings.

In this paper, we describe the results of a modelling study to quantify changes in exposures in the indoor environment and their associated health consequences attributable to housing energy efficiency retrofits. We do this to characterise possible health-related consequences in need of further scrutiny for the development of national policies and guidance on housing energy efficiency interventions. By doing so, we attempt to gain a better understanding of the trade-offs between risks and benefits for population health.

METHODS

We developed a household-level model to quantify the principal exposure and health pathways outlined in figure 1. The model comprised two parts:

1. A building physics model of English houses that quantifies indoor winter temperatures, exposures to particle pollution, secondhand tobacco smoke (STS), radon, mould growth and energy demand in relation to the energy performance of the dwelling; and
2. A model of the resulting health impacts based on a combination of life table methods and directly modelled changes in disease prevalence.

The two model components make up the Health Impact of Domestic Energy Efficiency Model (HIDEEM;

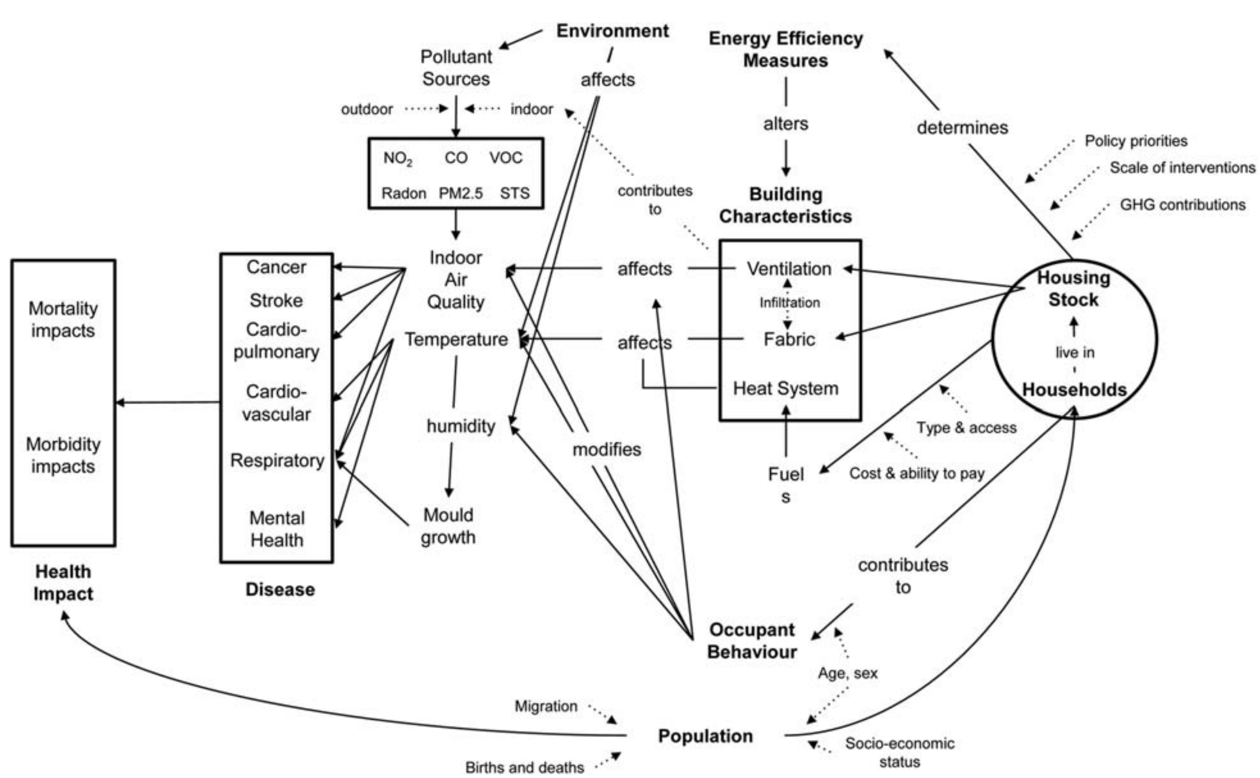

Figure 1 Connections between energy efficiency in housing and health (GHG, greenhouse gas; STS, secondhand tobacco smoke; VOC, volatile organic compound).

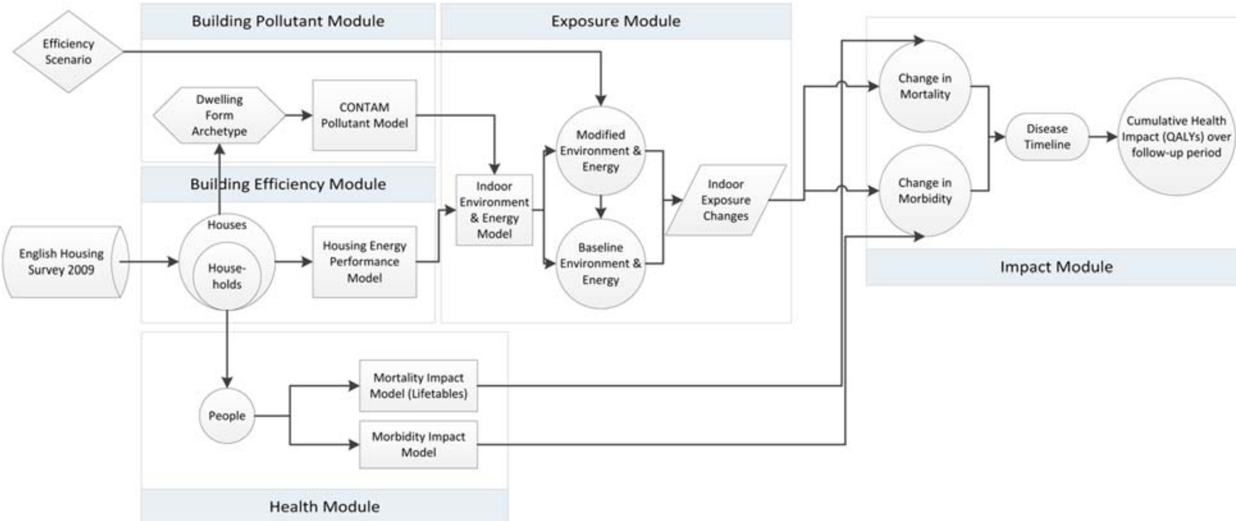

Figure 2 Health Impact of Domestic Energy Efficiency Model (HIDEEM) conceptual framework. The figure demonstrates the components of the model with solid lines representing input flows.

figure 2), an exposure-determinant and health impact model.

Other health outcomes that could be related to energy efficiency interventions but were not considered here include cold-related falls, changes in mental health impact (aside from temperature) and some forms of indoor pollutants (eg, volatile organic compounds, carbon monoxide poisoning, dust mites). However, such evidence can be sparse and the exposure–response uncertain. We have not modelled the impact of cold on respiratory disease (eg, chronic obstructive pulmonary disease) because the evidence required for robust quantification is still equivocal;[19] we hope to address this in future versions of the model. Also, we have not modelled the risk of overheating on energy efficiency, though this could have an important impact in the future. A difficulty with many empirical studies looking at the health effect of energy efficiency interventions is that the study designs and methods have not been sufficiently robust in their design or controlling for bias so as to draw strong conclusions.[5]

Part 1: Modelling the indoor environment

We developed a model that characterised the indoor environmental conditions of the 2010 English Housing Survey (EHS).[20] The indoor environmental conditions and changes in those conditions related to energy efficiency interventions were modelled using validated building physics and airflow models.[21–23] The modelling, described in detail elsewhere,[16 24 25] used representative archetype dwelling forms (informed by sampling from the EHS[26 27]) to represent the English dwelling stock. Each of these archetypes was modelled under different levels of air tightness and ventilation systems: window opening only, window trickle vents, extract fans, and combined use of trickle vents and extract fans. A total of 896 archetypes were modelled and matched to the EHS on the basis of dwelling type (eg, detached, semidetached, terraces and flats), floor area and notional permeability. The result was a model of indoor environmental conditions for a representative sample of English dwellings (see online supplementary appendix 1 for further details).

Dwelling energy performance was calculated as a notional heat loss value.[12] We used an empirical relationship between the dwelling heat loss value and standardised internal temperature (SIT)[i] to predict the bedroom and living room temperature, standardised at an external temperature of 5°C.[12 28] The SIT is a measure of the thermal condition of the dwelling ranked against all other dwellings, and is a function of the dwelling's energy and ventilation performance. The estimated average SIT (derived from an average temperature of the living room and bedroom) for each dwelling reflects the observed distribution shown in Oreszczyn *et al.*[11] The SIT to thermal performance relationship used in the model captures empirical rebound in temperature (eg, reduced heat flow, changes in occupant heating practices and temperature increases).[12] We used EHS data on dwelling fabric characteristics, heating system type and presence of ventilation systems to determine eligibility for energy efficiency upgrades (see online supplementary appendix 2).

Part 2: Quantification of health impact

We focused on a relatively restricted list of exposures that are supported by reasonably clear epidemiological evidence.[5] The health impact of changes in indoor air

[i]The standardised internal temperature (SIT) is derived from an empirical study of 1600 English dwellings with half-hourly temperature measurements for a period of 2–4 weeks over the winter period of 2001/2002 and 2002/2003. The SIT is derived from regression models of indoor on outdoor temperature for each dwelling. The models are used to derive a predicted indoor temperature at 5°C outdoor temperature.[12]

Table 1 Mortality and morbidity outcomes modelled and exposure–response relationships

Exposure	Health outcome	Exposure–response relationship	Reference
Mortality			
Standardised internal temperature	Winter excess cardiovascular (including excess cerebrovascular accident and myocardial infarction)	0.98 per °C	Derived from ref. 32
Secondhand tobacco smoke	Cerebrovascular accident	1.25 (if in same dwelling as smoker)	33
	Myocardial infarction	1.30 (if in same dwelling as smoker)	34
$PM_{2.5}$	Cardiopulmonary	1.082 per 10 μg/m^3	35 36
	Lung cancer	1.059 per 10 μg/m^3	As above
Radon	Lung cancer	1.16 per 100 Bq/m^3	37
Morbidity			
Standardised internal temperature (°C)	Mental health: Common mental disorders (GHQ-12 score 4+)	0.90 per °C	Based on Warm Front[38]
Mould (% MSI >1)	Asthma		
	Harm class II (hospital admission)	1.53 per 100%	Based on ref. [39] and used in HHSRS*
	Harm class III (GP consultation)	1.53 per 100%	As above
	Harm class IV (minor symptoms)	1.83 per 100%	As above

*Housing health and safety rating system.
GHQ, General Health Questionnaire; GP, general practitioner; HHSRS, housing health and safety rating system; MSI, mould severity index; $PM_{2.5}$, particulate matter with a diameter of 2.5 μm or less.

quality and temperature on (cause-specific) mortality was modelled using life table methods based on the IOMLIFET model[29] but applied to individuals in the EHS data based on their age, sex and specific exposure changes. Life tables were set up using 2010 age-specific population and (disease-specific and all-cause) mortality data for England and Wales from the Office for National Statistics (ONS), with separate life tables set up for males and females.[30] We modelled changes in five indoor exposures: SIT, STS, indoor and outdoor sources of particulate matter with a diameter of 2.5 μm or less ($PM_{2.5}$), radon and mould; the selected outcomes are listed in table 1. Impacts on morbidity for these same outcomes were estimated from the mortality estimates by applying age-specific and cause-specific ratios of years of healthy life lost due to disability (YLD) to the overall years of life lost (YLL) derived from WHO Global Burden of Disease data.[31]

Since some of the outcomes are subcategories of others, to avoid double counting we removed deaths in those subcategories from the larger categories. For outcomes affected by more than one exposure, we assumed the relative risks were multiplicative.

We assumed no time lags for cold-related deaths since these would likely to begin to occur within a year. For the other outcomes, a change in exposure would not necessarily lead to an immediate change in mortality in the population. Therefore, we incorporated disease-specific time functions to account for disease onset and cessation lags over time. The time lag functions were based on empirical evidence of the effect of smoking cessation on mortality over time,[40] and plausible assumptions about disease progression over time (see online supplementary appendix 3).

We separately estimated morbidity impacts on common mental disorders (CMDs) in adults and asthma in children using published estimates of the underlying disease prevalence in the population to which exposure-related relative risks were applied based on changes in SIT and mould growth, respectively (table 1). Mental health benefit is assumed to persist over 10 years (ie, exponential decay to zero over 10 years).

Model application: 2030 energy efficiency targets

The model was used to examine the effect of energy efficiency retrofits of the type and order proposed under 2030 GHG mitigation pathways for the English housing sector.[1] Where dwellings were eligible, the retrofits comprised installing double glazing, insulating cavity and solid walls, adding loft insulation, installing new condensing gas boilers, and adding draught proofing to improve dwelling air tightness in leaky dwellings (air leakage rate ≥7 m^3/m^2/h). In addition, non-operational extract fans in the kitchen and bathroom were repaired and window trickle ventilators[ii] were installed with glazing upgrades.

We examined three scenarios that addressed ventilation alongside the energy efficiency retrofits (table 2). They were:

[ii]A small purpose provided opening in a window or building envelope that facilitates ventilation in spaces when large openings (windows and doors) are closed and fans are turned off.

Table 2 Energy efficiency interventions modelled

Experiment energy efficiency retrofits	Ventilation scenarios Regulation	 Installer discretion	 No added ventilation
	Number of retrofits installed (1000s)		
Loft insulation	5320	5320	5320
Cavity wall insulation	6560	6560	6560
Solid wall insulation	5700	5700	5700
Double glazing installation	2430	2430	2430
Condensing boiler installation	10 730	10 730	10 730
Gas central heating installation	310	310	310
Draught proofing	3870	3870	3870
Trickle vent and extract fans	15 280	900	0
Extract fan installation only	350	350	0
Extract fan refurbishment	50	50	50
Trickle vent installation only	270	270	0

Note that trickle and extract fans include all new installations, extract fan only already have trickle vents, trickle only already have extract fans.

1. Purpose provided ventilation via extract fans and trickle vents (where not already present) was installed to ensure adequate indoor air quality in line with regulations (Regulation);
2. Purpose provided ventilation was installed (or repaired) only for dwellings that exhibit problems of mould or inadequate ventilation as reported in the EHS (~1.16 million dwellings—see online supplementary appendix 1; Installer Discretion); and
3. No purpose provided ventilation was added except for repairing broken extract fans and trickle vents for double glazing to reflect the lack of guidance surrounding energy efficiency retrofits (No Added Ventilation).

We assumed instantaneous installation for all retrofits in order to illustrate the effect of changes in exposures and associated health effect with all other unrelated conditions held constant. We also assumed that no changes occurred in the underlying health status of the population over time, an assumption which previous work has shown to have only a minor effect on life table calculations.[41]

Uncertainty analysis

We used Monte Carlo simulation to assess parametric uncertainty in the health impact estimates associated with the determinant of the exposure change (ie, the change in heat loss and air tightness due to each intervention), the exposure–response relationships and the utility weights for each health outcome. We report 95% credible interval estimates based on the 2.5th and 97.5th centiles of results generated from 500 model iterations.[42 43] See online supplementary appendix 4 for further details.

We also examined the uncertainty of the model due to two important structural assumptions: (1) the length of life lost in those dying of cold-related causes, and (2) the toxicity of particles derived from indoor sources. For cold, assessing chronic health impacts using exposure–response functions based on time series analyses implies that those who are vulnerable to cold-related risks have the same life expectancy as the population average. This is unlikely to be the case; instead it is likely that the people who die of cold-related events are people who have shorter than average life expectancy (see online supplementary appendix 5 for further discussion). To address this, we have examined the effect of assuming that those vulnerable to cold fall into a 'high-risk' subgroup of the population with elevated underlying cardiovascular risk. We then examined the shortening of remaining life expectancy in such a high-risk group as a function of (1) its size as a proportion of the total population (if overall cardiovascular deaths remain the same), and (2) the elevation of risk (relative risk) in the high-risk group compared with the remainder of the population. For particle toxicity, the epidemiology is dominated by studies of outdoor air pollution. However, it is unclear whether the same toxicity should be assumed for particles derived from indoor sources, whose concentration may rise if air tightness is increased. To account for this uncertainty, we performed calculations with and without the inclusion of the estimated effect of particles derived from indoor sources.

There is also uncertainty in the use of the mould severity index (MSI) used in the EHS that is derived from a visual inspection of the occurrence and extent of mould on windows, walls and ceilings. The potential uncertainty of the MSI measurement beyond the simple Monte Carlo treatment of the uncertainty in mould exposure is not examined here.

RESULTS

Indoor environmental exposure levels

The 2030 energy efficiency interventions resulted in improvements in energy performance, as well as appreciable increases in air tightness. The changes in indoor air pollutant concentrations reflected the ventilation

Table 3 Building performance and indoor environment conditions in the English stock for present day (baseline) and cumulative health effect after 50 years for selected exposure-specific diseases under the 2030 energy efficiency retrofit experiment with ventilation scenarios

	Baseline	Experiment ventilation scenarios		
	Intervention stock	Regulation	Installer discretion	No added ventilation
Sample		N		
Dwellings (1000s)		18 990	17 350	17 320
People (1000s)		44 740	41 130	41 060
Building characteristics		Mean (SD*)		
Fabric heat loss (W/K)	294 (167)	219 (120)	213 (115)	213 (116)
Ventilation heat loss (W/K)	75 (45)	70 (42)	51 (35)	50 (33)
Heat system efficiency (%)	76 (12)	88 (11)	89 (10)	89 (10)
Permeability ($m^3/m^2/h$)	16 (5)	11 (5)	11 (5)	11 (5)
Exposure†		Mean (95% credibility intervals)		
Standardised indoor temperature‡ (°C)	17.8 (0.7)	18.1 (18.1, 18)	18.1 (18.1, 18.1)	18.1 (18.1, 18.1)
STS§	0.5 (0.4)	0.5 (0.5, 0.4)	0.7 (0.7, 0.6)	0.7 (0.7, 0.7)
Indoor¶ $PM_{2.5}$ ($\mu g/m^3$)	9.4 (5.4)	4.6 (4.4, 4.2)	10.6 (10.1, 9.6)	11 (10.5, 9.9)
Outdoor $PM_{2.5}$ ($\mu g/m^3$)	6.2 (1.7)	6.8 (6.5, 6.2)	5.9 (5.6, 5.3)	5.8 (5.5, 5.2)
Radon (Bq/m^3)	22.9 (14.1)	22.4 (20.3, 20.1)	34.2 (30.7, 30)	35 (31.3, 30.7)
Mould (% with MSI >1)	14.9 (7.5)	12.3 (11.6, 11)	18.5 (17.8, 16.2)	18.8 (18.3, 16.5)
Heating energy (MWh/year)	22.9 (10.4)	16.6 (16.4, 16.3)	15.7 (15.6, 15.4)	15.6 (15.5, 15.4)
Health impact**		Total QALYs per 10 000 persons (95% credibility intervals)††		
Cardiovascular (winter)		119 (106, 131)	69 (57, 81)	65 (53, 77)
Heart attack		312 (287, 336)	−232 (−279, −185)	−271 (−319, −223)
Stroke		306 (282, 330)	−258 (−310, −206)	−296 (−349, −242)
Cardiopulmonary		1268 (1169, 1371)	−44 (−83, −6)	−130 (−166, −96)
Lung cancer		233 (209, 258)	−75 (−93, −57)	−97 (−115, −81)
Common mental disorder		2 (2, 4)	3 (3, 4)	3 (3, 4)
Asthma (children)		1 (4, 7)	−1 (−8, −4)	−1 (−9, −5)
Net impact		2241 (2085, 2397)	−539 (−678, −399)	−728 (−864, −592)

*Standard deviation is given for building characteristics as a measure of spread.
†Weighted average values of kitchen (10%), lounge (45%) and bedroom (45%).
‡Average between living room and bedroom temperature when 5°C outdoors.
§STS 1=average exposure level of smoking household.
¶Indoor sources of $PM_{2.5}$ relate to cooking only with an emission rate of 1.6 µg/min.
**Cardiovascular disease is modelled with equal risk across the population and toxicity of indoor and outdoor $PM_{2.5}$ is considered equal and as such the results are likely overestimating the impact—see uncertainty analysis for tests.
††Credibility intervals are derived from Monte Carlo analysis showing using the 5th and 95th centiles from 1000 model iteration results as limits.
MSI, mould severity index; $PM_{2.5}$, particulate matter with a diameter of 2.5 µm or less; STS, secondhand tobacco smoke; QALYs, quality adjusted life years.

strategy applied under the three different scenarios.[iii] Table 3 summarises the energy performance, indoor environmental conditions, changes in exposure levels and health impacts.

Scenario 1 (Regulation), where ventilation systems were added alongside all fabric and heating retrofits, resulted in a 30% reduction in annual heating energy demand, which is aligned with government objectives.[2] Wintertime temperatures increased by 0.3°C on average (with a SD of ±0.5), while added ventilation reduced indoor sources of pollutants (53% for $PM_{2.5}$, 11% for radon, 13% for STS, 23% for mould), but increased indoor exposure to outdoor-generated $PM_{2.5}$ (4.2%).

The 'Installer Discretion' scenario shows that mitigation measures applied due to perceptible conditions of inadequate ventilation or mould growth were insufficient to have wide benefit (in part due to the relatively small number of dwellings exhibiting these conditions, see online supplementary appendix 1). With the added ventilation, heat losses (33%) and heating energy (32%) were greater compared with the 'Regulation' scenario along with a modest increase in indoor temperatures. Outdoor sources of $PM_{2.5}$ reduced considerably (-10%), but indoor pollutants experienced sizable increases (8% for $PM_{2.5}$, 34% for radon, 33% for STS and 18% for mould).

Under the 'No Added Ventilation' scenario, there were still greater reductions in ventilation heat losses. The average indoor pollutant concentrations were

[iii]The modelled estimates for the baseline housing stock energy performance and indoor exposures were compared against observed national and sample stock distributions to check the accuracy of the outputs (see online supplementary appendix 1).

further elevated across the stock compared with scenario 2 (Installer Discretion).

Health impact of energy efficiency retrofits

The balance of the overall impact on mortality and morbidity is highly dependent on the assumptions made regarding the level of ventilation to mitigate reduced indoor air quality (table 3; figure 3). Over a follow-up period of 50 years, the net impact of the 2030 energy efficiency interventions under the 'Regulation' ventilation scenario resulted in 2241 quality adjusted life years (QALYs) gained per 10 000 persons for the 18.99 million affected dwellings. Selective targeting of ventilation system under the 'Installer Discretion' scenario resulted in −539 QALYs per 10 000 persons lost. While no added ventilation had an even greater overall negative impact of −728 QALYs per 10 000 persons lost among the intervention group.

If building regulations were met (scenario 1), the net impact on health is positive primarily because the reduction in exposure to particles of indoor origin is greater than the increase in outdoor-generated particles. Improved indoor temperatures have a net positive effect on cardiovascular disease, though this is dependent on assumptions of the remaining life expectancy of those vulnerable to the effects of cold (see Uncertainty analysis section).

Targeted extract fans and trickle vents in dwellings with a perceptive ventilation problem (scenario 2) offer only moderate modification on the long-term impact on health, a 30% improvement from no additional ventilation (scenario 3). However, despite these interventions, there remained a large number of dwellings that experienced an increase in fabric air tightness.

When no additional ventilation was provided alongside the dwelling energy efficiency retrofits, the increase in indoor sources of air pollutants resulted in a net negative impact on health, despite the reduced ingress of outdoor sources of particulates. Although sensitive to assumptions on the equal toxicity of indoor and outdoor $PM_{2.5}$ (see Uncertainty analysis section), reduced infiltration of outdoor air and increases in exposure to STS, radon and mould risk resulted in a net-negative impact on health.

Uncertainty analysis

Cold-related deaths risk group size

We use here scenario 2 to illustrate the sensitivity of the health impact estimates to changes in the concentration of cardiovascular risk within the population. Reducing the size of the 'high-risk' cardiovascular group in the population reduces the scale of the health benefit due to increased winter temperatures, though the overall impact is modest (see table 4). We illustrate this by concentrating the risk across increasingly smaller proportions of the population (from 100% to 0.1%), selected to represent the full range of plausible assumptions. An assumption of 100% of the excess winter cardiovascular deaths being in the high-risk group (ie, the whole population at risk) could result in a considerable overestimate of the change in the burden of winter time cardiovascular disease, while an estimate of 0.1% (ie, only 0.1% of the population are at risk) would effectively remove all of the potential benefit of increased temperatures for population health. Pending further research, it is difficult to estimate the correct level of adjustment. However, the impact is almost certain to be appreciably less than that implied by using time series coefficients applied without any correction.

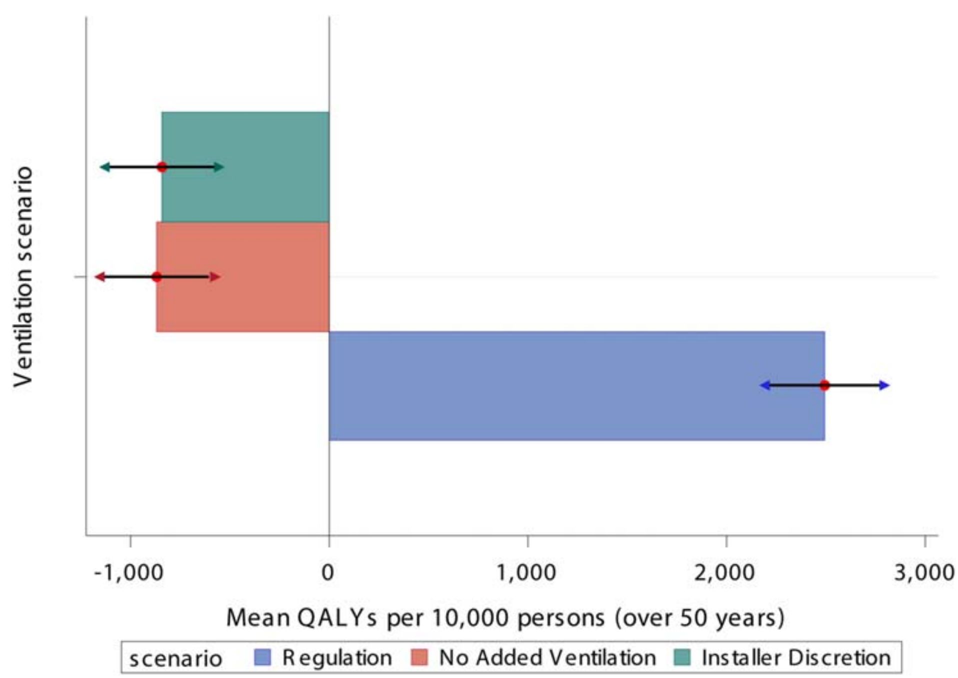

Figure 3 Net mortality and morbidity health effect (quality adjusted life years (QALYs) per 10 000 persons) for all selected exposure-specific diseases after 50 years for the 2030 energy efficiency experiment for different ventilation scenarios (arrows denote 95% credibility intervals). Note: cardiovascular disease is modelled with equal risk across the population and toxicity of indoor and outdoor $PM_{2.5}$ is considered equal and as such the results are likely overestimating the impact—see 'section, Uncertainty analysis' for tests ($PM_{2.5}$, particulate matter with a diameter of 2.5 μm or less).

Table 4 Cumulative health effect after 50 years for varying high-risk excess winter cardiovascular group size under the 2030 energy efficiency retrofit experiment for scenario 2 'installer discretion'

	Experiment ventilation scenario 2: 'Installer Discretion'			
	Size of 'high-risk' group*			
	100%†	10%	1%	0.1%
Net QALYs	Mean per 10 000 persons (95% credibility intervals)			
Cardiovascular (winter)	68.8 (56.8, 80.7)	34.1 (28.1, 40)	14.5 (12, 17)	4.8 (4, 5.7)
Heart attack	−232.1 (−279.1, −185.2)	−232.6 (−277.1, −188.1)	−232.7 (−276, −189.5)	−232.2 (−275.3, −189)
Stroke	−257.6 (−309.7, −205.5)	−257.2 (−307, −207.4)	−256.3 (−304.4, −208.2)	−257.3 (−305.5, −209.1)
Cardiopulmonary	−44.3 (−83.4, −5.6)	−46.6 (−85.6, −8.1)	−47.4 (−86.7, −8.8)	−44.2 (−83.4, −5.4)
Lung cancer	−74.9 (−92.9, −57.4)	−74.3 (−91.9, −57.2)	−75 (−92.9, −57.7)	−74.9 (−92.9, −57.5)
Common mental disorder	2.7 (2.8, 4.1)	2.7 (2.8, 4)	2.8 (2.8, 4.1)	2.7 (2.8, 4)
Asthma (children)	−1.3 (−8.4, −4.3)	−1.3 (−8.4, −4.4)	−1.3 (−8.4, −4.3)	−1.3 (−8.2, −4.2)
Net impact	−538.6 (−677.9, −399.3)	−575.2 (−706.5, −443.9)	−595.5 (−724.2, −466.7)	−602.2 (−729.6, −474.8)

*Proportion of the population in the group assumed to be at high risk for cardiovascular events.
†100% equivalent to whole population equally at risk.
QALYs, quality adjusted life years.

Toxicity of indoor particulate matter

There is uncertainty about the relative toxicity of particles generated from indoor sources compared with those from outdoor sources. Some evidence suggests these might be as toxic or perhaps even more toxic as particulate matter (PM) derived from outdoor sources.[35 36] Analysis in which indoor-generated $PM_{2.5}$ was assumed to have no adverse effect on health had a significant impact on the results (see table 5), reducing the overall net health impact by around 78% compared with the base case results (which assumed equal toxicity to outdoor particulates). Though the effect may be uncertain, there is very likely to be some impact from indoor sources and we would stress the need for more empirical studies that measure and assess the toxicity of indoor $PM_{2.5}$, and the balance of indoor and outdoor particles on health.

DISCUSSION

This modelling work shows that predicted changes in indoor environmental exposures following housing energy efficiency interventions of the type being proposed by the UK Government may have an appreciable impact on health. This approach can be applied to different country settings but with regard to existing conditions, and information on the housing stock and households therein.

There is an expectation that retrofits that seek to reduce space heating energy demand will increase indoor temperatures,[12] but such interventions will also affect the dwelling air tightness and its ventilation. Although indicative, our modelling suggests that reducing fabric heat loss and increasing air tightness may reduce exposure to outdoor pollutants and raise indoor temperatures. However, without added ventilation, indoor concentrations are increased with associated adverse health impacts which are greater than those associated with indoor temperatures, leading to an overall negative impact on health. As demonstrated, this conclusion is sensitive to assumptions made about the toxicity of particles from indoor sources, an area where further research is urgently needed.

In the various scenarios, for purposes of illustration, we assumed an instantaneous installation and a lagged health impact associated with step changes in some exposures. However, the reality will be that these interventions and potential impacts will be realised over a longer period of time. Under the UK's mitigation targets, virtually all English dwellings will need retrofitting by 2030 (ie, 20 million over 15 years or 3650 per day). Putting in place effective measures to address ventilation now can have long-term health effects for both existing and future households.

Although associations between indoor temperatures and mental well-being have been reported,[38] it is unclear how long the benefit to mental well-being would persist following improved temperatures. Given the high

Table 5 Cumulative health effect after 50 years for indoor $PM_{2.5}$ toxicity equal to outdoor sources and with no effect of indoor $PM_{2.5}$ under the 2030 energy efficiency retrofit experiment for scenario 2 'installer discretion'

	Experiment ventilation scenario 2	
	Indoor particulate matter toxicity	
	Equal to outdoor	No effect
Net QALYs	Mean per 10 000 persons (95% credibility intervals)	
Cardiovascular (winter)	68.8 (56.8, 80.7)	81.6 (69.8, 93.4)
Heart attack	−232.1 (−279.1, −185.2)	−186 (−225, −147)
Stroke	−257.6 (−309.7, −205.5)	−212.1 (−255.1, −169)
Cardiopulmonary	−44.3 (−83.4, −5.6)	200.8 (170.5, 233.5)
Lung cancer	−74.9 (−92.9, −57.4)	−47 (−59.8, −34.5)
Common mental disorder	2.7 (2.8, 4.1)	2.8 (2.9, 4.1)
Asthma (children)	−1.3 (−8.4, −4.3)	−1.3 (−8.1, −4.2)
Net impact	−538.6 (−677.9, −399.3)	−161.2 (−240.3, −82)

$PM_{2.5}$, particulate matter with a diameter of 2.5 μm or less; QALYs, quality adjusted life years.

prevalence of CMD in the population, any small shift can be highly influential on the results. While there is very likely to be benefit that accrues beyond a single year and maybe a seasonal effect for a period afterwards, the long-term benefit will likely be affected by the risk of reoccurring episodes of mental health driven by factors other than thermal environment.

The underlying assumptions regarding housing air tightness and occupant ventilation practices (eg, window opening behaviour) are both extremely important. The EHS shows that 71% of homes have no extract fans (or working extract fans); in other words, these homes are naturally ventilated and thus, the exposure to indoor-generated pollutants will be highly determined by the air tightness of the dwelling and the practices of the occupants. Our model has examined the uncertainty of these practices on our estimates and therefore, provides a reasonable spread on the likely true impact.[43] From our scenarios, we found that added ventilation accompanying efficiency retrofits mitigated the health risk associated with increased air tightness (scenario 1), but that this mitigation must be applied beyond 'problem homes' (scenario 2), only the widespread installation of ventilation systems results in a net benefit to health (scenario 1), and providing no additional ventilation poses a potential risk to health (scenario 3).

The provision of added ventilation to offset potential increases in indoor concentrations of pollutants following fabric energy retrofits is an important issue for public health. While the spirit of the building regulations suggests that adequate ventilation should be provided following changes to a dwelling, there is no explicit guidance for installers on what and when to install such systems. The Housing Health and Safety Rating System provides an 'after-the-fact' route through which remediation of poor indoor air quality could be addressed, but it is both unlikely and undesirable to rely on this system to address issues that could otherwise be avoided. Clearly assumptions on how a household ventilates their dwelling will have an important impact on creating a healthy indoor environment. Dwellings with higher ventilation rates have been shown to have reduced health burdens,[10 44] though the association with air change rates and specific diseases can be equivocal.[45] Occupant ventilation practices have also been shown to be counter-productive to creating a healthy indoor environment. A study of Dutch households showed that many neglect the annual maintenance required to ensure that ventilation system operation is not compromised.[46] Education around ventilation will be essential to minimise exposure to indoor pollutants following retrofits. Our work highlights that the potential health impacts following efficiency retrofits are not necessarily positive and that there may be risk trade-offs that will depend on the retrofit installation regulatory framework. Having stronger regulation around energy efficiency retrofits and ventilation will help to realise multiple benefits (eg, energy savings and health).

STRENGTHS AND LIMITATIONS

Modelling studies provide a method of examining complex problems by drawing together data from a range of sources in order to explore the potential impact of interventions on population health. While quantifying the potential health impact of policy options is preferable over qualitative assessment, doing so is subject to several difficulties, primarily the availability of evidence[47] and the potential to add scientific credibility to uncertain predictions.[48] The modelling also involves many uncertainties. For instance, the limited set of observed data on how such retrofits affect indoor air quality remains an impediment, with only a few studies looking at the determinants of indoor air quality (eg, infiltration).[5] There is a paucity of evidence relating to some of the most important health outcomes—especially in relation to cold.[49] In the overall balance of health calculations, morbidity impacts are potentially larger than those of mortality, for example, the effect of improved temperatures on CMD,[5] but the evidence is still uncertain, and this gap in the research evidence should be addressed.

The modelling results are presented as QALYs; however, it is clear that these changes in disease outcomes would have an impact on health and social care services beyond these utility estimates. As the average age of the UK population increases so too does the demand on health services. Preventative actions, such as improving energy and ventilation performance, may help to mitigate some of this demand.

The exposure modelling in this experiment concentrated on indoor conditions. The experiment did not alter outdoor pollutant concentrations related to proposed energy supply decarbonisation,[1] which may reduce outdoor levels of particulate matter in the future.[50] This would further tip the balance towards installing mitigating ventilation systems so as to dilute 'stale' indoor air. Refining the model to include assumptions on energy systems and transport could further improve the estimates of the potential health impact associated with UK's GHG abatement measures.

CONCLUSIONS AND POLICY IMPLICATIONS

On balance, if properly implemented, actions to mitigate climate change through energy efficiency in housing can have benefits to health by reducing exposure to cold and outdoor air pollutants. They will also offer indirect health benefits by providing more resilience to protect indoor thermal conditions during extreme cold and heat events. Modelling studies of the type presented here are needed to ensure housing policies are developed in ways that capitalise on this potential for improving health. Such studies, however, should be used with acknowledgment of their uncertainty and limitations, and do not supplant the need for well-designed empirical studies that can validate models and offer policymakers more evidence, and provide greater confidence around policy impact.

We have shown that, unless specific remediation is used, reducing the ventilation of dwellings will improve energy efficiency at the expense of increased exposure to indoor air pollutants and risk to health. However, an important conclusion of this work is that, with careful attention to retrofit installation and ventilation practices, these potential negative impacts can be removed.

The policy agenda and evidence base on the health impact of home energy efficiency is still evolving. Guidance for installers regarding adequate levels of ventilation to protect health is now needed before the large-scale introduction of energy efficiency measures into the housing stock.

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Contributors IH developed the integrated health and exposure model, and was responsible for developing the experiment and crafting the text. JM and ZC developed the health model and contributed to the text. PD, BJ and CS all developed portions of the exposure models and contributed to the text. MD and PW were project leads, guided the study design and contributed to the text.

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Correction

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Health effects of home energy efficiency interventions in England: a modelling study

Supplementary materials

Appendix 1 – Energy performance and exposure modelling

Indoor exposure modelling details

The pollutant exposure modelling used CONTAMv2.4c, which is a validated multi-zone airflow and pollutant transport simulation tool[1,2]. Five contaminants are modelled: second-hand tobacco smoke (STS), PM2.5 from internal sources, PM2.5 from external sources, radon, and moisture (as a precursor for mould). A series of pollutant sources and sinks were placed within appropriate building zones. External concentrations of pollutants were specified where relevant. The CONTAM models then predict the concentrations of the pollutants within each building zone every 15 minutes for a year. Models were created only for the ground-floor flats and the radon concentrations for first-floor flats were assumed to be half this, and for second-floor or higher flats were assumed to be zero. In modelling the indoor environment, we assumed that occupants opened windows during summer daytime hours (i.e. 9 AM to 5 PM) and whenever using the kitchen and bathroom, trickle vents were open at all times and extract fans were used whenever present in the kitchen or bathroom.

From the CONTAM model outputs, annual average indoors pollutant concentrations (weighted to reflect exposure levels experienced around the home – with 45% of the occupant time in the living room, 45% in the bedroom and 10% in the kitchen) within the archetype dwellings for a range of permeability's (i.e. 0.5 to 30 m3m-2hr-1). Interpolation using a 4th-order polynomial was used to estimate concentrations for permeability's not directly modelled in CONTAM. The permeability for each EHS dwelling was calculated using a physics-based method that uses details relevant to air infiltration, i.e. openings and cracks in the fabric (walls, windows, flues, vents, etc…) and normalises this by a fabric to volume ratio. Each EHS dwelling was matched into one of the 16x4 archetype and ventilation categories and the exposure concentrations were calculated using the pollutant models. The heat loss due to overall permeability for each dwelling is calculated by using the infiltration level, building volume and heat capacity of air.[3]

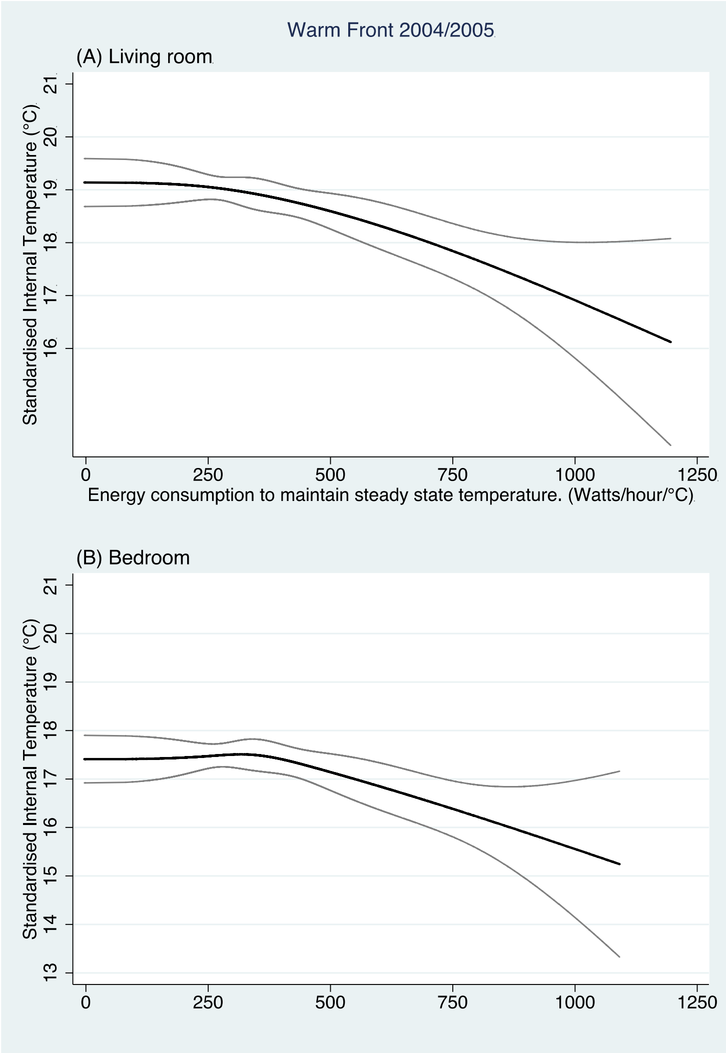
For the purposes of this paper, to determine the dwellings that exhibit problems of mould or inadequate ventilation we used information from the EHS under the ‘Interior’ and ‘Damp’ data tables. Ventilation types and problems are presented in Appendix Table 1.

Appendix Table 1 - Type of ventilation in English dwellings and dwellings experiencing ventilation-related problems, as defined in the EHS

Ventilation type	**N Dwellings**
Window opening only	13,502,140
Window trickle vents	675,490
Extract fans	3,078,700
Trickle vents and extract fans	2,175,050
Perceptible ventilation problem	
Broken extract fan in kitchen or bathroom(s)	48,830
Higher than average risk of damp and mould	899,920
Condensation in bathroom or kitchen	278,520
Inadequate ventilation in bathroom or kitchen	91,260
Inadequate ventilation in living room or bedroom	107,390
Condensation in living room or bedroom	164,710
All dwellings with an above problem	1,165,380

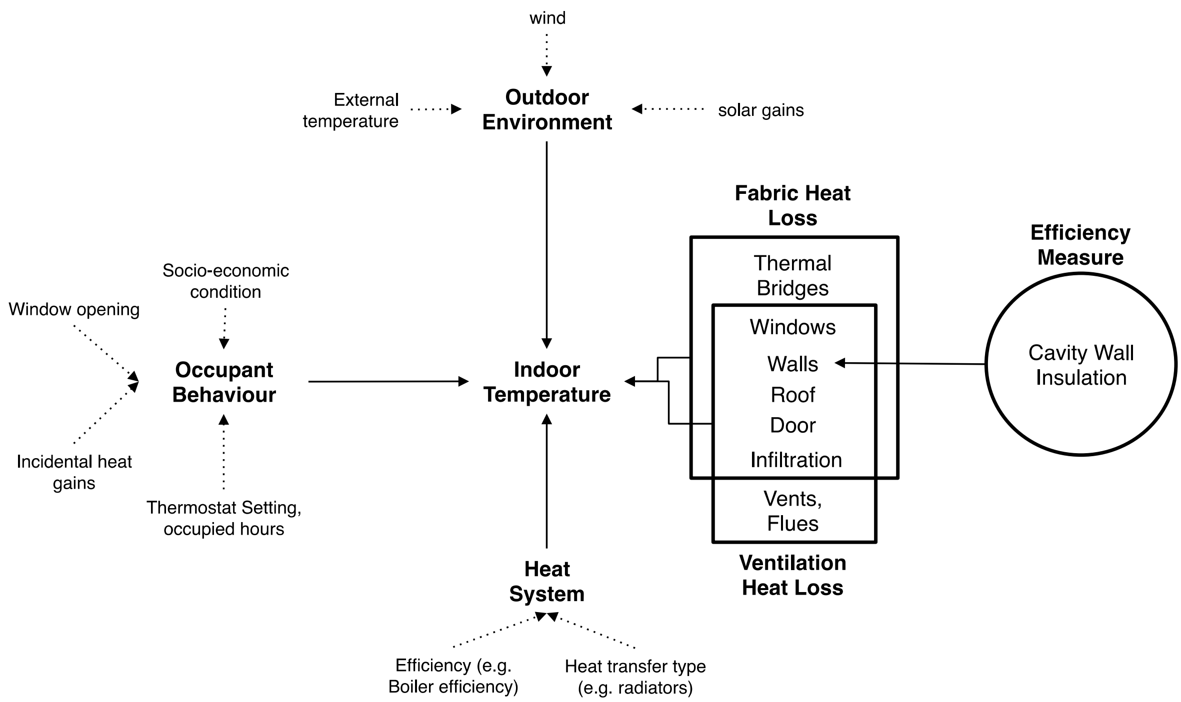
An estimate of the thermal performance of the EHS dwellings was also made using a buildings-physics based approach to determine the overall dwelling heat loss through the fabric along with its airtightness (i.e. infiltration).[3] The fabric heat loss[1] (W/K) for each EHS dwelling is calculated using U-values ($Wm^{-2}K^{-1}$) for each building fabric feature (i.e. walls, windows, doors, floor) inferred from the dwelling age, wall construction and location along with the area (m^2).[4] We used the calculated dwelling heat loss along with the overall heat system efficiency to estimate the standardised indoor temperature for each dwelling[5,6]. The method estimates the internal living room and bedroom temperature standardised for an external temperature of 5 °C to reflect winter conditions; the living room and bedroom temperature are averaged to estimate the whole house average. The method accounted for the likely occupant behaviour response to heating older leakier homes, i.e. older less efficient dwellings are harder to heat and have lower internal temperatures at 5 °C outdoors, even accounting for incomes,[5]. Dwellings with high E-values (the least energy efficient homes) have the lowest indoor temperatures, and temperatures increase approximately linearly as E-values fall, i.e. with improving energy efficiency (Appendix Figure 1). The SIT reaches a plateau of around 18.2°C at E-values to the left of the inflexion point at around 250 W/K, suggesting that this is a temperature which the average householder living in a reasonably energy efficient home considers sufficient for comfort. Risk of mould growth for each EHS dwelling was calculated using the standardized internal temperature estimate and moisture concentration modelling in CONTAM.

[1] Heat loss is defined as the thermal energy lost for every degree difference between the indoor and outdoor temperature measured in W/K.



Appendix Figure 1 - Standardized daytime living room temperature and standardized night time bedroom temperature against required rate of energy consumption to maintain steady state temperature (A and B) and. Graphs show predicted values and 95% confidence intervals. From Oreszczyn et al. (2006)

The change in exposure concentrations (Δ) for each EHS dwelling was determined by modelling a base-case scenario and then with the subsequent energy efficiency retrofits in all eligible houses for the specified scenarios. The efficiency retrofits were modelled by making an adjustment to the relevant building component (see Appendix Table 4 below). This building-physics approach accounted for the determinants of indoor environmental exposures; see Appendix Figure 2 for example.



Appendix Figure 2 - Pathways of fabric efficiency retrofits to indoor temperature

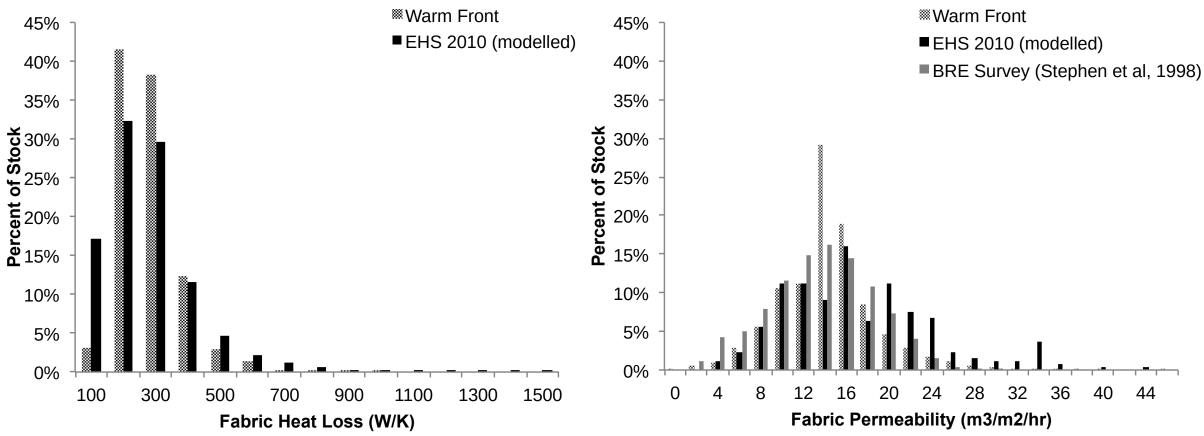
Energy Performance

The modelled estimates for the base case energy performance were compared against observed national and sample stock distributions to check the accuracy of the model outputs, see Appendix Table 2 and Appendix Figure 3.[5,7–10] The modelled average dwelling fabric heat loss is 274 W/K and is greater than both Warm Front and national modelled estimates.[8,11] The modelled average heat system efficiency is 76% compared to national estimates of 74%.[11] The modelled mean English dwelling permeability is 14 $m^3m^{-2}hr^{-1}$ compared to 17 $m^3m^{-2}hr^{-1}$ in Warm Front and 14 $m^3m^{-2}hr^{-1}$ from an observed national survey.[7,9] The modelled English dwelling exposure concentrations (STS, PM2.5, radon, temperature and mould) were compared with relevant observed surveys and found to be very close or within a range in all cases but mould, see Appendix Table 3.[5,12–17] These comparisons provide some confidence that the building energy and ventilation performance in our model represents the observed distribution of performance among the dwelling stock. Also, the model is also able to estimate the observed range of indoor temperatures and pollutant levels.

Appendix Table 2 – Comparison of modelled English housing stock building performance and values from Warm Front and national estimates (DECC) and surveys (Stephen)

	Modelled	Warm Front[a]		National	
Building Performance	**Mean**	**Mean**	**Source**	**Mean**	**Source**
Fabric heat loss (W/K)	274	224	Oreszczyn et al. 2006	203.8	DECC, 2012
Heat system efficiency (%)	76%	67%	Hong et al. 2009	74%	DECC, 2012
Permeability ($m^3m^{-2}hr^{-1}$)	13.8	17.2	Hong et al. 2006	13.9	Stephen, 1998

Note: [a]Warm Front Study



Appendix Figure 3 – Comparison of modelled English housing stock fabric heat loss and fabric permeability compared to Warm Front and BRE survey

Appendix Table 3 – Comparison of modelled English housing stock exposure concentrations and observed survey or estimates of concentrations in houses

Exposures	**Modelled**	**Comparison**	**Source**
Temperature - living room (°C)	18.6	17.9 - 19.1	Oreszczyn et al. 2006, Hong et al. 2006, OPDM 1998
Temperature - bedroom (°C)	17.1	15.9 - 18.5	Oreszczyn et al. 2006, Hong et al. 2006, OPDM 1998
Indoor[a] $PM_{2.5}$ ($\mu g/m^2$)	17	17 - 25	Hanninan et al. 2004, Dimitroupolou et al. 2006
Indoor $PM_{2.5}$[b]	10.9	9.3*	Shrubsole et al. 2012
Outdoor $PM_{2.5}$	6.1	6.1*	Shrubsole et al. 2012
Radon (Bq/m^3)	26.2	21	Gray et al. 2009
Mould (% with MSI >1)	11.5	14.6 - 21.2	OPDM 1998, Oreszczyn et al. 2006
% of homes with smoker	21.2	21	ONS 2008

Note: a) Weighted average values of kitchen (10%), lounge (45%) and bedroom (45%); b) Indoor sources of $PM_{2.5}$ relate to cooking only with an emission rate of 1.6 μg/min; * Indicates modelled estimate.

Appendix Table 4 – Energy efficiency improvement values used in building physics modelling of indoor environmental condition changes

Intervention	Type	Component	Value	Unit	Source
Lofts to 250mm	Insulation	Roof u-value	0.22	$W/m^2 K$	RdSAP v9.83 2005
	Infiltration	Direct adjustment	0.10	N_{ach}*	Hong et al, 2004
Wall Insulation (Solid External)	Insulation	External wall u-value	0.58	$W/m^2 K$	RdSAP v9.83 2005
	Infiltration	Direct adjustment	0.2	N_{ach}	Hong et al, 2004
Wall Insulation (Cavity fill)	Insulation	External wall u-value	0.33	$W/m^2 K$	RdSAP v9.83 2005
	Infiltration	Direct adjustment	0.20	N_{ach}	Hong et al, 2004
Double Glazing	Insulation	Glazing u-value	2.00	$W/m^2 K$	RdSAP v9.83 2005
	Infiltration	Draught stripping percentage	0.98	N_{ach}	Hong et al, 2004
Install Condensing Boilers	Efficiency	Main system efficiency	93	%	RdSAP v9.83 2005
Draught Proofing	Infiltration	Floor infiltration	0.10	N_{ach}	RdSAP v9.83 2005
	Infiltration	Glazing draught stripping percentage	0.98	N_{ach}	RdSAP v9.83 2005
	Infiltration	Direct adjustment	0.20	N_{ach}	Hong et al, 2004

Notes: * Nach = Number of air changes per hour

Appendix 2 – English Housing Energy Efficiency

Housing stock

The model uses the house and household stock from the 2010 English Housing Survey (EHS), conducted over the years 2010 and 2011, as the basis for the modelling. The EHS provides a statistically random representative sample of the English stock on which the health impact of energy efficiency interventions can be modelled.

The EHS survey collects information on the overall condition of English homes and the households living in them. The survey provides data on key housing stock characteristics (including age, type and size) and households (age, tenure, number of occupants, income, vulnerability) based on physical surveys and interviews. The surveyed 'dwelling sample' of properties where physical inspections were carried out contains 16,150 occupied or vacant dwellings, or 0.7% of the housing stock of 22.2 million dwellings in England [18]. The EHS provide a factor with which to weight variables in order to represent houses or households in England. For the purpose of the modelling, the houses weighting was used as it represents the occupants of the dwellings that will be affected by energy efficiency improvements.

The EHS includes details on the household occupants of the surveyed houses. The occupant details include their age, sex, employment status, smoking practices, income and a number of other features. The occupant variables used in the modelling relate only to age, sex and whether an active smoker lives in a house.

Converting the EHS for building efficiency modelling input

In order to use the EHS housing stock data in the modelling, the EHS data must undergo a conversion process in order to create a set of key input variables required for calculating the ventilation characteristics and thermal performance [4]. The building physics component of the model uses the Standard Assessment Procedure (SAP) as the core calculation method to predict the ventilation and fabric heat loss and heat system efficiency.

The conversion process uses variables collected in the EHS in order to infer features that are necessary to run a SAP-like estimation of the building efficiency. These include details such as: dwelling and household information, geometry, ventilation, fabric heat loss, and space heating systems, see Appendix Table 5.

Appendix Table 5 - Building characteristics and components from EHS conversion

Characteristic	Component
Geometry	Gross floor area (GFA), volume, number of storeys, storey height, façade area, fabric component area (glazing, doors, party walls, roof, ground floor)
Glazing	Type, draught proofing
U-values	Glazing, roof, external walls, party walls, doors, thermal bridges, thermal mass parameter
Walls	Wall type, thickness,
Infiltration	Floor, fabric, draught lobby, additional infiltration, chimneys, flues, fans and passive vents
Heat system	Type, efficiency

Energy efficiency retrofits

Changes in exposures are made through the introduction of energy efficiency retrofits to those dwelling variants not already having had such an intervention as determined by the EHS.

Retrofits are applied by altering key parameters within the building efficiency modelling. Sources for the changes to fabric heat loss are draw from RdSAP version 9.83 [3], which provides several tables relating to u-values of dwelling components with varying levels of energy efficiency. For airtightness adjustments, research from Warm Front that assessed the impact of retrofits on airtightness is used to determine the adjustment to dwelling infiltration rates post-intervention [7].

Dwellings are deemed eligible based on rules that relate to each component or where an EHS variable exists. The rules for each retrofit are:

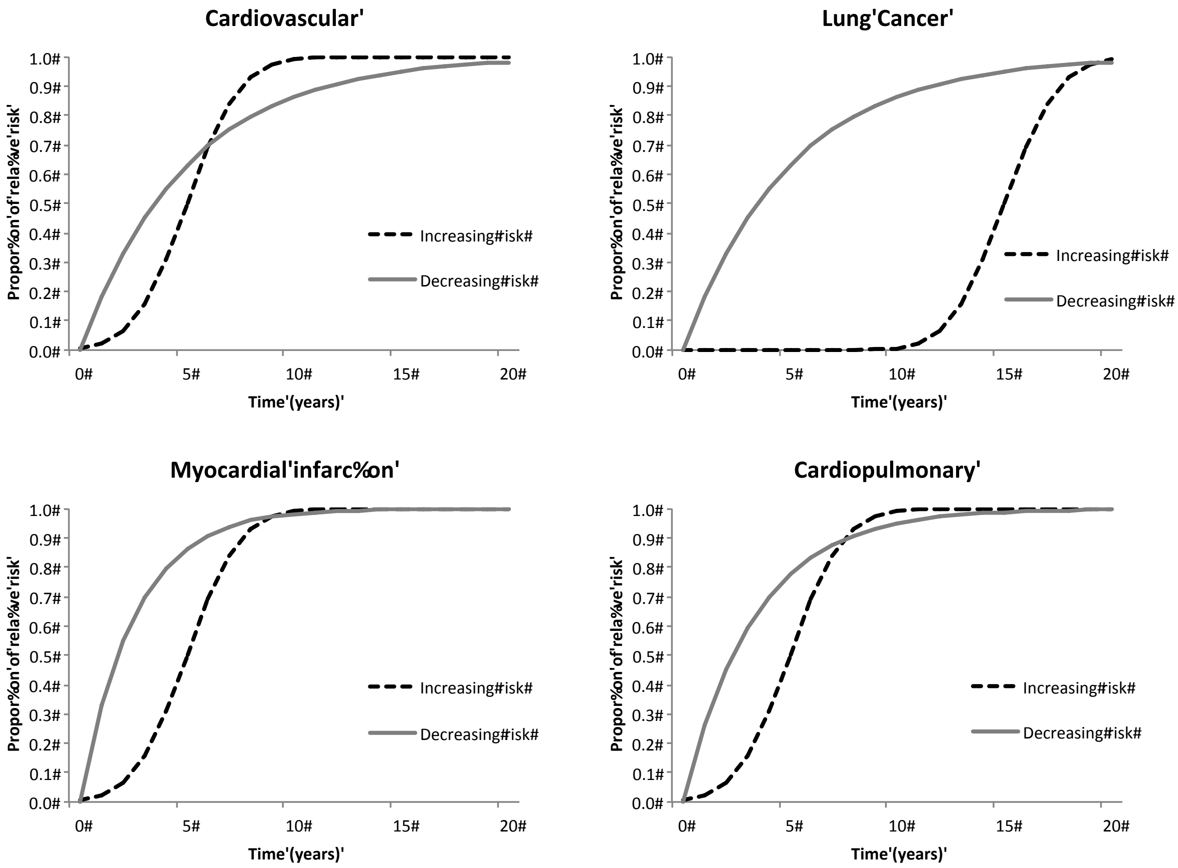
- Lofts to 250mm: EHS variable (EPulin05e) 'Energy upgrade loft insulation' recorded as 'Yes'
- Solid Wall Insulation: EHS variable (wallinsx) 'type of wall and insulation' recorded as 'other'
- Cavity Wall: EHS variable (wallinsx) 'type of wall and insulation' recorded as 'cavity uninsulated'
- New Double Glazing: Modified EHS variable (typewin) 'Predominant type of window' combined to form three groups, single, double, mixed, recorded as 'single'
- Install Condensing Boilers: EHS variable (EPublr5e) 'Energy upgrade boiler' recorded as 'Yes'
- Draught Stripping: All dwelling with infiltration >7 $m^3m^{-2}hr^{-1}$ are eligible for draught stripping
- Trickle vents: All dwellings with no trickle vents
- Extract fans: All dwellings with no extract fan systems or trickle vents only

Appendix 3 – Disease onset time functions

In reality, following an intervention, a change in exposure may not lead to an immediate change in a health outcome in the population. There would likely be a delay that differs by disease and whether there was a beneficial effect on the disease risk (i.e. positive health impact) or an increase in the disease risk (i.e. negative health impact). For example, an increase in radon exposure would lead to almost no increase in lung cancer risk in the population for several years due to the latency period of the disease.[19] To account for this, disease-specific time functions were incorporated to account for disease onset and cessation lags over time (see Appendix Figure 4). The time lag functions were based on empirical evidence of the effect of exposure changes on mortality over time, where available, as in the work on smoking cessation and $PM_{2.5}$.[20,21] However, where such evidence was not yet available, the shapes of the time lags were based on plausible assumptions regarding disease progression over time.

In this study, time lags were applied to the initial cohort over a period of 50 years into the future, for which the output from the life tables were age-specific changes in life years (LYs) accumulated over the period. Independent multiple health impact assessments were performed for each exposure-health outcome pathway over the full range of expected exposure changes (increases and decreases) for which age- and sex-specific relationships between changes in exposure and cumulative changes in life years over 50 years were determined.

Using this approach requires several assumptions regarding the life tables and mortality and morbidity impact, for example: mortality rates vary only with age and sex; changes in exposure affect mortality risk at all ages; the age- and cause- specific baseline mortality rates do not change over time; and, the time lags in risk follow an appropriate time profile. For morbidity these include: the baseline prevalence is not age- or sex- dependant (for direct estimates); morbidity does not depend on e.g. socio-economic factors, underlying health status, etc.; baseline population disease prevalence is assumed to represent an individual's probability of having the disease (direct estimates); and a fixed ratio exists between mortality and morbidity impacts at the population level (indirect estimates). There may also be other disease- exposure specific assumptions, for example that mould is associated with respiratory illness in children only (up to age 14) and temperature is associated with mental health in adults only (age 16 and over) during winter months.



Appendix Figure 4 – Lag functions used for modelling the impact of cardiovascular, cardiopulmonary, myocardial infarction and lung cancer mortality following changes in exposure

Appendix 4: Probabilistic sampling of exposure determinants

Sampling of exposure-determinants (intervention impact)

Uncertainty in the exposure-determinants (i.e. interventions) was captured by sampling from a distribution around the mean change in the physical building component associated with an intervention. The mean values were derived from the RdSAP estimates. Where no estimate of the standard error was known, a standard approach of using 10% of the parameter mean for the standard error was used.[22] Normal distributions were used to specify the uncertainty in the exposure-determinants. For heating and insulation interventions, the means were desired target levels and therefore likely to be normally distributed. For ventilation changes, there is limited available evidence and therefore normal distributions were also specified.

Sampling of exposure-response functions

Using a similar approach to the interventions, shape parameters were defined for each exposure-outcome pathway using estimates of 95% credibility intervals (CI) from the original source references, where available. Normal distributions based on the CI of the central estimates were used for the relative risks; however, where the uncertainty was great or the evidence was limited, uniform distributions (i.e. uninformative prior) over an appropriate range were used. Normal distributions were applied to the relative risks associated with cardiovascular disease, common mental disorder, and asthma.

Sampling of utility weights

Since there is variation in the utilities within each disease category, utility weights for morbidity estimates were sampled using uniform distributions with +/- 10% as the upper and lower level ranges. These were applied to CVD, stroke, heart attack, CMD, and asthma.

Appendix 5: Loss of life expectancy with cold death

Modelling health impact related to changes in indoor temperature draws on analysis by Wilkinson *et al.* 2001 on the change in excess winter death (as a ratio of non-winter death) due to cardio-vascular disease (CVD). The relationship is from a time-series analysis of mortality data and indoor temperatures, standardised to 5 °C during the winter daytime [23]. The analysis provided a trend estimate of 2% reduction in winter: non-winter ratio of CVD, adjusted for deprivation and variation in excess winter death (EWD) by region, per increase in indoor hall temperature. In this modelling, the impact of changes in standardised temperature is used to determine the change in EWD [5].

Among the multiple uncertainties relating to the quantification of the impact of cold-related deaths is the loss of life expectancy associated with each cold death. Cold does not induce new disease or events, but rather accelerates events (especially cardiovascular events) in people with pre-existing sub-clinical or clinical disease. For example, the additional people dying from a heart attack or stroke on cold days will be people with already established atherosclerosis in whom the effect of cold is sufficient to precipitate (early) the thrombotic obstruction of an already narrowed coronary or cerebral artery. Such a thrombotic obstruction would have been likely to occur eventually anyway, but the patho-physiological effects of cold bring about the obstruction at a point earlier than it would otherwise have occurred – with consequential clinical sequelae including death in some cases.

In consequence, it is likely that the people who die of cold-related events are people who have shorter than average life expectancy. The difficulty for modelling of cold-related QALYs is that the risks of cold-related death are determined from time-series studies from which it is impossible to determine the degree of life-shortening (i.e. loss of life expectancy).

Applying relative risks for cold death derived from time-series studies to life tables makes the implicit assumption that those who die of cold are representative of the population as a whole and therefore have average age-specific life expectancy. This is almost certainly untrue given that in nearly all cases they must have pre-existing underlying disease.

To address this, we have examined the effect of assuming that those vulnerable to cold fall into a high risk sub-group of the population with elevated underlying risk of cardiovascular death. This was done through life tables set up to include all-cause and cardiovascular mortality in England and Wales and considered remaining life expectancy at age 70 (to represent older populations at higher risk of cold-related mortality). We divided the population into two groups: a “high risk” group (of cold-related mortality) and a “low risk” group (i.e. the rest of the population). We then performed life table simulations to examine the effect on remaining life expectancy in the high risk group (relative to the low risk group) as a function of (i) its size as a proportion of the total population, and (ii) the elevation of risk (relative risk) in the high risk group compared to the remainder of the population (i.e. concentrating the level of cardiovascular risk in the high risk group). Results are show in Appendix Table 6.

Appendix Table 6 – Relationship between cardiovascular high-risk group size and life expectancy

Proportion of the population in the group assumed to be at high-risk for cardiovascular events	Approx. remaining life expectancy at age 70 in high risk group* (years)	Approx. life expectancy in high risk group relative to that calculated using population average mortality rates
100% (i.e. whole population equally at risk = default of applying time-series cold relative risk to life table)	14.5	100%
10%	7.5	50%
5%	5.5	38%
1%	<3	21%
0.1%	~1	7%

*For a given size of the high risk group (as a proportion of the total population), the life expectancy declined with the increasing relative risk for cardiovascular death in that group. However, the decline showed considerable flattening after a relative risk of around 20 or so. The results shown here are the 'effective asymptote' of life expectancy for the high risk group at high relative risk.

From this it can be seen, for example, that if the vulnerable population at risk of cold death can be assumed to be around 10% of the population, then their life expectancy will be only around half that of the population as a whole. Likewise, if the vulnerable high risk group is assumed to be 1% of the population, life expectancy would be little more than a fifth of that in the population as a whole.

Using these figures, we calculate several alternative estimates for the loss of life expectancy associated with cold-related death, using correction factors to the original life-table estimates as suggested by the figures in the last column of the table above. Specifically, the modelling output gives three additional estimates to the (uncorrected) life-table calculations, with 'global' correction factors of 0.38, 0.21 and 0.07 to the total of loss of life expectancy (and hence of QALYs) corresponding to assumptions that the high risk group vulnerable to cold death is confined to 5%, 1% and 0.1% of the population respectively.

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Checklist for reporting modelling studies

Dimension of Quality	**Reporting item**	**YES/NO**	**Notes**
STRUCTURE			
Decision problem/objective	Is there a clear statement of the decision problem?	YES	The decision problem is stated in the third paragraph of the Introduction (paragraph beginning “While current English building regulations…”
	Is the objective of the evaluation specified and consistent with the stated decision problem?	YES	Though not a strict evaluation the objective of the study is set out in the last paragraphs of the introduction, with the line beginning “We do this to characterize…”
	Is the primary decision-maker specified?	YES	The paper is examining a point of principle relevant to many bodies and decision-makers, including individual householders. Guidance notes on retrofitting of properties are issued by various bodies including independent agencies such as the Institute for Sustainability. Some, but not all, aspects are covered by building regulation. We suggest the UK Government as the key target as it is responsible for many aspects of guidance and regulation.
Scope/perspective	Is the perspective of the model clearly stated?	YES	The perspective is stated in the first paragraph of the Methods section.
	Are the model inputs consistent with the stated perspective?	YES	The model inputs are described (and referenced) in the Methods section (pages 5 to 7). They relate to the characteristics of the housing stock, and in particular to changes in the distribution of dwelling air permeability, which are described and justified (see, in particular, page 5). The basis for assumptions about thermal and indoor sources of pollutant emission rates is referenced (see references 11,18-20 & 23). Life-table data are based on

			published sources as described (see page 6).
	Are definitions of the variables in the model justified?	YES	See above.
	Has the scope of the model been stated and justified?	YES	Described in the Methods.
	Are the outcomes of the model consistent with the perspective, scope and overall objective of the model?	YES	The main outputs of the model are distributions of indoor temperature and air pollutant exposures (i.e. radon, mould risk, PM2.5 from indoor and outdoor sources, and second-hand tobacco smoke) changes in the population and population-level health impacts (see for example tables 1 and 3).
Rationale for structure	Is the structure of the model consistent with a coherent theory of the health condition under evaluation?	YES	It is established that reducing home ventilation may increase indoor air pollutants and that these pollutants pose a risk to health through a number of established health outcomes (page 5). Therefore, to evaluate the intervention it is necessary to combine a building stock model, an exposure model and a health impact model.
	Are the sources of data used to develop the structure of the model specified?	YES	References are provided for all data sources used to inform the development of the scenarios (see pages 5 and 6).
	Are the causal relationships described by the model structure justified appropriately?	YES	See above.
Structural assumptions	Are the structural assumptions clearly stated and justified?	YES	There are many structural assumptions. These are justified (and referenced) throughout the Methods section. In particular, the broad structure of the model is described at the start of the section. We further test structural uncertainty in the work.
	Are the structural assumptions reasonable given the overall objective, perspective and scope of the model?	YES	The structure used (the combination of three sub-models: a building stock model, a model of exposure and a population-level health impact model) is required to make

			predictions of the type presented in the paper.
Strategies/comparators	Is there a clear definition of the options under evaluation?	YES	The options under evaluation are the three scenarios described in the section titled "Modelling Application".
	Have all feasible and practical options been evaluated?	N/A	The paper presents three potential future scenarios covering a range of plausible strategies. Innumerable variants are possible, but the scenarios specified illustrate the main options.
	Is there justification for the exclusion of feasible options?	N/A	No major categories of alternative options have been excluded.
Model type	Is the chosen model type appropriate given the decision problem and specified causal relationships within the model?	YES	See above.
Time horizon	Is the time horizon of the model sufficient to reflect all important differences between options?	YES	See page 5 and table 3. We show evidence of health impacts over a time course that allows for the evolution of selected disease risk with appropriate onset lag.
	Are the time horizon of the model, the duration of treatment and the duration of treatment effect described and justified?	YES	The interventions are assumed to occur instantaneously. The time horizon of the modelled health impact is 50 years, in order to cover the lifetime of the interventions and allow for sufficiently long disease development.
Disease states/pathways	Do the disease states (state transition model) or the pathways (decision tree model) reflect the underlying biological process of the disease in question and the impact of the interventions?	YES	We used a standard life table. The relationships between indoor air pollutant exposure and cardio-respiratory and lung cancer mortality are well established and described in Table 1.
Cycle length	Is the cycle length justified?	N/A	
Parsimony	Is there indication that the structure of the model is as simple as possible and that any simplifications are justified?	NO	More simple (or more complex) exposure models can be used to study radon. However, these would not be appropriate to model the distribution of exposures at the population level. Simpler health models would not have captured the time-varying nature of the

			health impacts.
DATA			
Data identification	Are the data identification methods transparent and appropriate given the objectives of the model?	YES	All data sources are listed and referenced in the Methods section (pages 5 to 7).
	Are results reported in a way that allows the assessment of the appropriateness of each parameter input and each assumption in the target settings?	N/A	
	Where choices have been made between data sources, are these justified appropriately?	N/A	The most recent version of the housing survey at the time of the model development was used to specify the building stock model. Other data, including the baseline mortality data from ONS (see page 5), were chosen to match the year of this survey as closely as possible.
	Where data from different sources are pooled, is this done in a way that the uncertainty relating to their precision and possible heterogeneity is adequately reflected?	N/A	
	Are the data used to populate the model relevant to the target audiences (i.e., decision-makers) and settings?	YES	All data sources are listed and referenced in the Methods section. These data are commonly used in assessments by, e.g., the Department of Energy and Climate Change.
	Has particular attention been paid to identifying data for the important parameters in the model?	YES	The basis of the building stock model was the English Housing Survey 2010 (5). The baseline health data were obtained from ONS (page 5).
	Has the quality of the data been assessed appropriately?	YES	All data are from reliable sources (see references in the Methods section) and used commonly in the field of research.
	Where expert opinion has been used, are the methods described and justified?	N/A	
Data modelling	Is the data modelling methodology based on justifiable statistical and epidemiological techniques?	YES	Although it does not fit the category of data modelling, the life table (see page 5/6) is a well-established technique and is used widely to model health

			impacts in epidemiological studies.
Baseline data	Is the choice of baseline data described and justified?	YES	All baseline data were chosen to match the year of the housing stock survey. Baseline data for the exposure model are described at the bottom of page 7. Baseline data for the health model are described on page 5/6
	Are transition probabilities calculated appropriately?	YES	We used a standard life table. The relationship between temperature and air pollutant exposure and mortality and morbidity are well established and described in table 1. The time lag functions used are shown in appendix figure 3.
Treatment effects	If relative treatment effects have been derived from trial data, have they been synthesized using appropriate techniques?	YES	The treatment in this case is the energy efficiency intervention. The relationship between temperature and air pollutants exposure and selected cardio-respiratory and cancer mortality and morbidity is well established and described in Table 1.
	Have the methods and assumptions used to extrapolate short-term results to final outcomes been documented and justified? Have alternative assumptions been explored through sensitivity analysis?	YES	See above.
	Have assumptions regarding the continuing effect of treatment once treatment is complete been documented and justified? Have alternative assumptions been explored through sensitivity analysis?	N/A	The interventions would result in a permanent change to the housing stock.
Risk factors	Has evidence supporting the modeling of risk factors as having an additive or multiplicative effect on baseline probabilities or rates of disease incidence or mortality been presented?	YES	Described on Page 5, to avoid double counting we removed deaths in those sub-categories from the larger categories. For outcomes affected by more than one exposure, we assumed the relative risks were multiplicative.
Data incorporation	Have all data incorporated into the model been described and referenced in	YES	All data incorporated into the model is described and referenced in the Methods

	sufficient detail?		section and in further detail in the Appendices.
	Has the use of mutually inconsistent data been justified (i.e., are assumptions and choices appropriate)?	N/A	
	Is the process of data incorporation transparent?	YES	See above.
	If data have been incorporated as distributions, has the choice of distribution for each parameter been described and justified?	N/A	
	If data have been incorporated as distributions, is it clear that second order uncertainty is reflected?	N/A	
Assessment of uncertainty	Have the four principal types of uncertainty been addressed?	YES	Both parametric uncertainty and structural uncertainty analyses were used to determine sensitivity of the model results on key areas of uncertainty. Parametric uncertainty was carried out for (1) the intervention effect on exposures and (2) exposure-response relationships, and (3) utility weights for outcomes. Structural uncertainty tests were carried out for 1) toxicity of indoor PM2.5 and 2) cold-related death group size. Distributions of modelled indoor pollutant concentrations were compared with empirical measurements in Appendix 1.
	If not, has the omission of particular forms of uncertainty been justified?	NO	See below.
Methodological	Have methodological uncertainties been addressed by running alternative versions of the model with different methodological assumptions?	YES	The three scenarios are based on different methodological assumptions (see pages 6 and 7).
Structural	Is there evidence that structural uncertainties have	YES	See above.

	been addressed via sensitivity analysis?		
Heterogeneity	Has heterogeneity been dealt with by running the model separately for different subgroups?	YES	The exposure model was run for different housing types, and health impacts calculated by age, sex and for smokers and non-smokers.
Parameter	Are the methods of assessment of parameter uncertainty appropriate?	YES	The selected assumptions used in the uncertainty analyses reflect the range of plausible alternatives.
	If data are incorporated as point estimates, are the ranges used for sensitivity analysis stated clearly and justified?	N/A	
	Which sensitivity analyses were carried out?	YES	See above. Uncertainty analyses that varied assumptions about intervention effect on exposures, exposure-response relationships, and utility weights for outcomes. Structural sensitivity analysis that examined differences in pollutant toxicity and cold-related disease risk.
CONSISTENCY			
Internal consistency	Is there evidence that the mathematical logic of the model has been tested thoroughly before use?	N/A	The building stock, exposure and health models are based on established and tested mathematical principles (see references 20, 21, 23 and 37).
External consistency	Are any counterintuitive results from the model explained and justified?	N/A	All results are in line with expected patterns and distributional effects.
	If the model has been calibrated against independent data, have any differences been explained and justified?	N/A	
	How was the model calibrated?	YES	Model outputs have been compared against known exposure distributions. The exposure model was calibrated against published observed data. This is described at the start of the Results section and a comparison is shown in appendix 1.
	Calibration - description of source data	YES	See references in Table 1.

	Calibration - description of search algorithm	N/A	
	Calibration - description of goodness-of-fit metric	YES	
	Calibration - description of acceptance criteria	N/A	
	Calibration - description of stopping rule	YES	See reference 37.
	Have the results of the model been compared with those of previous models and any differences in results explained?	YES	Magnitudes of exposure changes and health impacts compared with references 21, 27, 35.
VALIDITY			
Output plausibility	Has evidence of face validity - evaluation by experts in the subject matter area for a wide range of input conditions and output variables, over varying time horizons – been presented?	N/A	
Predictive validity	Was the validity of the model tested?	NO	Both the exposure model (see reference 20) and the health model (see reference 24) are well established and validated methods.
	Is there a description of how the validity of the model was checked?	NO	See above.
	How was the validity quantified? (e.g., % explained)	NO	See above.
COMPUTER IMPLEMENTATION	Is the software used in the study listed and its choice justified?	YES	Exposure model software (CONTAM) listed on page 5. Health model software (IOMLIFET) listed on page 5.
TRANSPARENCY	Is the model available to the reader?	YES	The individual components of the model are freely available.
	Is a detailed document describing the calibration methods available?	N/A	
	Do the authors provide relevant appendices?	YES	Additional supporting figures are provided in an appendix.
SPONSORSHIP	Is disclosure of relationship between study sponsor and performer of the study provided?	YES	See statements on page 13.