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Environmental burden of disease associated with inadequate housing

A method guide to the quantification of health effects of selected housing risks in the WHO European Region

Edited by
Matthias Braubach
David E. Jacobs
David Ormandy

World Health Organization

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Methods for quantifying health impacts of selected housing risks in the WHO European Region

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The WHO European Centre for Environment and Health, Bonn Office, WHO Regional Office for Europe coordinated the development of this report.

ABSTRACT

This guide describes how to estimate the disease burden caused by inadequate housing conditions for the WHO European Region as well as for subregional and national levels. It contributes to the WHO series of guides that describe how to estimate the burden of disease caused by environmental and occupational risk factors. An introductory volume to the series outlines the general methodology.

In this context, the WHO Regional Office for Europe took up the challenge to quantify the health effects of inadequate housing and convened an international working group to quantify the health impacts of selected housing risk factors, applying the environmental burden of disease (EBD) approach.

The guide outlines, using European data, the evidence linking housing conditions to health, and the methods for assessing housing impacts on population health. This is done for twelve housing risk factors in a practical step-by-step approach that can be adapted to local circumstances and knowledge. This guide also summarizes the recent evidence on the health implications of housing renewal, and provides a national example on assessing the economic implications of inadequate housing.

The findings confirm that housing is a significant public health issue. However, to realize the large health potential associated with adequate, safe and healthy homes, joint action of health and non-health sectors is required.

Keywords

HOUSING – standards
RISK FACTORS
RISK ASSESSMENT – methods
HEALTH STATUS
ENVIRONMENTAL MONITORING
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Michael Baker (University of Otago, New Zealand) assisted with the development of the structure of the chapters and the summary tables found in each chapter. He also provided scientific editing for the chapters on damp and mould, traffic noise and formaldehyde.

Rokho Kim (WHO Regional Office for Europe) was involved in the project management during the first two years before taking over the Occupational Health Programme. He provided methodological guidance to the authors and reviewed various report chapters.

Finally, the editors would like to acknowledge the contribution and vision of Xavier Bonnefoy who worked in the WHO Regional Office for Europe as the Regional Advisor on Housing and Health until mid-2006 and initiated this project. Xavier Bonnefoy, who had established the WHO programme on housing and health in 2001, inspired all project meeting participants and authors in their work to provide a first European report on the Environmental Burden of Disease of inadequate housing. Xavier Bonnefoy died in November 2007.

Foreword

During the last century, large health improvements have been associated with increased quality of housing and urban settlements. Already in the 19th century, local governments in many European countries established housing improvement campaigns to respond to inadequate conditions of crowding, lack of hygiene and sanitation. However, the traditional risks are still prevalent in some areas, and modern risks have made their appearance. In some European countries, accidents in poorly designed homes kill more people than do road accidents, and indoor pollutants or mould cause asthma, allergies or respiratory diseases.

In more recent years, housing conditions have been demonstrated to be one of the major environmental and social determinants of population health and related health aspects have received increasing attention by the public health community. National reports, reviews and surveys as well as academic research and contributions from international agencies have added to the growing evidence base. Yet, we still face challenges in assessing the overall impact of housing on health, and in particular the health gains that could be associated with housing improvement schemes.

The WHO Regional Office for Europe has addressed the issue of healthy housing since the 1990s through its European Centre for Environment and Health in Bonn that took up the challenge to quantify the health effects of inadequate housing. Drawing from the recommendations of two international expert consultations, an international working group coordinated by WHO/Europe was tasked with the measurement of the health impacts of selected housing risk factors applying in particular the environmental burden of disease (EBD) approach.

The results of that work are presented in this report and show that – per 100 000 population – low indoor temperatures can cause 13 deaths, exposure to second-hand smoke 7 deaths, and exposure to radon 2-3 deaths per year. The use of solid fuels as a household energy source is associated with 17 deaths, and causes 577 Disability-Adjusted Life Years per year per 100 000 children under the age of five. Mould in homes leads to the loss of 40 Disability-Adjusted Life Years per 100 000 children each year, while traffic noise exposure and lack of home safety features cause an annual loss of 31 and 22 Disability-Adjusted Life Years per 100 000 population, respectively.

The findings confirm that housing is a significant public health issue and that policy-makers need to address it as a priority. Furthermore, they show the potential for primary prevention of a wide range of diseases and injuries through the improvement of housing conditions. However, public health workers cannot tackle the challenge alone. Healthy housing is a multisectoral responsibility, achievable only if all relevant players contribute to it, including not only public health, but also housing, engineering and construction, environment, social welfare, urban planning, and building management. The combination of actions from all these sectors shows the complexity of the subject as well as its great potential to increase the health status of our populations through providing adequate, safe and healthy homes.

Quantified health gains from improved housing conditions constitute an important component in decision-making on housing. We hope that this report will raise awareness of the housing-related health effects and support the application, adaptation and further development of the provided methodological examples by the scientific and policy community working on housing and health.

Guénaël R. Rodier, M.D.

Director, Division of Communicable Diseases, Health Security & Environment

Introduction

David Ormandy, Matthias Braubach

In 2003, the WHO published an introduction to the methodology for assessing the environmental burden of disease (EBD) (WHO 2003). This gave the background to, and a description of, the general method developed for quantifying the health impact (whether disease, injury or other health condition) attributable to a particular environmental risk at a population level.

The intention was to provide a means to help prioritize policies and actions directed at preventing or reducing the health impact of environmental risks, a means to identify high-risk groups in the population, and also a means to estimate health gains that interventions can bring.

Housing conditions are known to influence health, and there is a growing bank of evidence of the potential harmful effect that unsatisfactory housing can have on the health of occupiers. WHO recognizes that housing comprises four inter-related elements – the house (or dwelling), the home (the social, cultural and economic structure created by the household), the neighbourhood (or immediate housing environment), and the community (the population and services within the neighbourhood). Each of these individual elements has the potential to have a direct or indirect impact on physical, social and mental health, and two or more of them can have an even larger combined impact.

Housing is used by the whole population, but certain groups make greater use of it than others. These groups include young children, the elderly, the unemployed, those who are sick or for other physical or mental health reasons spend a greater proportion of time within the dwelling. The exposure to unsatisfactory housing conditions will be greater for these vulnerable groups than for the rest of the population.

In 2005, the WHO Regional Office for Europe (coordinated by the European Centre for Environment and Health, Bonn Office) organized the first of a series of workshops to examine the possibility of quantifying the negative impact of inadequate housing. The workshops brought together experts on a range of housing related subjects to investigate quantifying that impact using the EBD methodology. Two subsequent workshops were held to develop this approach, and the result was the commissioning of the work behind this report.

This report presents the results from using the EBD methodology to quantify the health impact of risks from particular unsatisfactory housing conditions. It does not cover all potential risks that could be attributed to inadequate housing, but it does demonstrate that this approach can be used effectively. The selection of the particular housing conditions covered by this report was based primarily on whether the relevant data existed and were available. However, limiting the report to those where the data were available would exclude some known high risk conditions (such as low indoor temperatures). Therefore, some chapters use alternative methods to quantify the risk from such conditions. In addition, where there exists EBD assessments of certain environmental risks (such as lead, environmental tobacco smoke, combustion of solid fuels, and radon), rather than duplicate the assessment, the report includes chapters that estimate the proportion of the burden that could be attributed to inadequate housing.

Each chapter in this report has been prepared by internationally recognized experts and subjected to peer review. That said, it is acknowledged that this work represents an important first step. It shows that the EBD methodology can be used to quantify the health impact of housing conditions where the appropriate data are available. And, by using that methodology, it

has provided a means to compare the quantifiable health impact of particular risks from housing conditions with the impacts from other environmental risks.

For those conditions where the EBD methodology could be used, the chapters provide an explanation of the topic and its health relevance, and summarize how the EBD was calculated and the sources for the data used. This is followed by an explanation of how the exposure-risk relationship was derived, and the EBD assessment results for the respective housing condition. The total EBD is given for Europe or the countries for which data are available, and where possible, the estimates are also provided for particular Member States. EBD results are provided in various forms: by the number of deaths attributable to the respective housing risk factor, by the number of Disability-Adjusted Life Years (DALYs)[1] attributable to the respective housing risk factor, or by the number of persons suffering from a given health outcome caused by the respective housing risk factor. Whenever possible, the EBD assessment is translated into the EBD per 100 000 population for the covered countries to provide a more consistent result. Several chapters also provide EBD results by the three epidemiological subregions (Euro A, B and C), which are used by WHO headquarters and cluster the 53 member States of the WHO European Region as shown below in Table 1. Any areas of uncertainty are set out and described, and suggestions are given for reducing that uncertainty. Finally, the policy implications are discussed.

Table 1: Epidemiological subregions of the WHO European Region

Subregion		**Member States covered**
Euro	A	Andorra, Austria, Belgium, Croatia, Cyprus, Czech Republic, Denmark, Finland, France, Germany, Greece, Iceland, Ireland, Israel, Italy, Luxembourg, Malta, Monaco, Netherlands, Norway, Portugal, San Marino, Slovenia, Spain, Sweden, Switzerland, United Kingdom
Euro	B	Albania, Armenia, Azerbaijan, Bosnia and Herzegovina, Bulgaria, Georgia, Kyrgyzstan, Montenegro, Poland, Romania, Serbia, Slovakia, Tajikistan, the former Yugoslav Republic of Macedonia, Turkey, Turkmenistan, Uzbekistan
Euro	C	Belarus, Estonia, Hungary, Kazakhstan, Latvia, Lithuania, Republic of Moldova, Russian Federation, Ukraine

Based on WHO, 2000

For conditions where alternative approaches were adopted because the necessary data did not exist or was not available, the respective chapters give an explanation of the approach adopted. Also included are chapters on the impact of housing interventions on health and on estimating the cost to the health sector attributable to unsatisfactory housing conditions.

In the first chapter, Maritta Jaakkola, Jeroen Douwes, Aino Nevalainen, and Ulla Haverinen-Shaughnessy estimate the burden of asthma among children in Europe that can be attributed to indoor exposure to dampness and mould. Michael D Keall, David Ormandy, and Michael G Baker then review the impact of housing conditions on the injuries and deaths from fires, drownings and falls related to housing conditions. Estimates of the EBD for tuberculosis that

[1] The WHO global burden of disease (GBD) measures burden of disease using the disability-adjusted life year (DALY). This time-based measure combines years of life lost due to premature mortality and years of life lost due to time lived in states of less than full health. The DALY metric was developed in the original GBD 1990 study to assess the burden of disease consistently across diseases, risk factors and regions. For further information, please see http://www.who.int/healthinfo/global_burden_disease/metrics_daly/en/index.html

can be ascribed to household crowding in Europe are provided by Michael Baker, Kamalesh Venugopal and Philippa Howden-Chapman.

The relationship between indoor cold and mortality is reviewed by Janet Rudge. While the EBD methodology could not be followed in this case, an estimate is given for the percentage of excess winter deaths related to cold housing using data from several studies. Wolfgang Babisch discusses the methods for quantifying ischaemic heart disease resulting from long term exposure to road traffic noise, and gives estimates of the EBD for Germany. Hajo Zeeb then discusses the relationship between indoor exposure to radon and lung cancer, but lacking country specific estimates, a summary of the evidence is given and some examples of studies in three European countries. Maritta Jaakkola reviews the evidence on the relationship between indoor environmental tobacco smoke and respiratory disease and provides estimates of the EBD for Europe.

The evidence on the link between health and lead in housing is discussed by David Jacobs, and an evidence summary is given as no detailed country specific estimates are available for Europe. Stefanos N. Kales, Tanzima Islam, and Min Kim review the relationship between indoor exposure to elevated levels of carbon monoxide, and provide an evidence summary. As indoor concentrations of formaldehyde are poorly characterized in Europe, Nicolas Gilbert and Mireille Guay, focusing on indoor exposure and an increased prevalence of lower respiratory symptoms in children, provide an evidence summary. As the disease burden from indoor smoke from solid fuel use has been fully assessed and reported, a further evidential summary is provided by Manish Desai, Eva Rehfuess, Sumi Mehta and Kirk Smith. Gary Evans discusses the relationship between housing quality and mental health, and reviews some of the studies that provide evidence.

Two chapters provide a different focus on the relationship between housing conditions and health. In the first, Hilary Thomson provides a synthesis of data on the health impact of energy efficiency improvements and the impact of neighbourhood renewal or regeneration. In the second, Simon Nicol, Mike Roys, Maggie Davidson, David Ormandy, and Peter Ambrose report on the development of a methodology to quantify the cost to the health sector attributable to unsatisfactory housing conditions.

The findings presented here should be of interest to a wide range of individuals and bodies involved in housing. They will be useful to those involved in the design and construction of housing, and those involved in the renovation and improvement of existing housing. The findings will inform policy-makers at local and national levels, and those responsible for setting health-based housing standards and requirements. For researchers and other academics, it is hoped that this report will encourage the collection of relevant data on other potential housing related health risks to help to give a greater understanding of the health impact burden that can be attributed to inadequate housing, and, it is also hoped that the work carried out to provide these findings can be further developed and refined.

References

WHO (2000). *The World Health Report 2000: Health systems: improving performance.* Geneva, World Health Organization (http://www.who.int/whr/2000/en/, accessed 12 June 2010).

WHO (2003). *Introduction and methods: Assessing the environmental burden of disease at national and local levels*. Geneva, World Health Organization (http://www.who.int/quantifying_ehimpacts/publications/9241546204/en/index.html, accessed 12 June 2010).

Indoor dampness and mould problems in homes and asthma onset in children

Maritta S. Jaakkola, Ulla Haverinen-Shaughnessy, Jeroen Douwes, Aino Nevalainen

1. Introduction

1.1 Background

Since the 1990s dampness, moisture and mould in indoor environments have been associated with adverse health effects in population studies in Europe, North America and elsewhere. Based on extensive reviews, the findings have been remarkably similar (IOM, 2004; WHO, 2009). Most commonly reported health effects are airways symptoms, such as cough and wheeze, but other respiratory effects, and skin and general symptoms have also been reported. Associations with both new-onset asthma and asthma exacerbations have been documented especially in children, and to some extent also in adults (Bornehag et al., 2001; Bornehag et al., 2004; Jaakkola, Jaakkola, 2004; Fisk et al., 2007). Asthma in childhood is the focus of this assessment, because it is the most common chronic disease in childhood and thus of major public health importance.

Asthma

The Global Initiative for Asthma (GINA, 2006) defines asthma as:

> ... a chronic inflammatory disorder of the airways in which many cells and cellular elements play a role. The chronic inflammation is associated with airway hyperresponsiveness that leads to recurrent episodes of wheezing, breathlessness, chest tightness, and coughing, particularly at night or in the early morning. These episodes are usually associated with widespread, but variable, airflow obstruction within the airways that is often reversible either spontaneously or with treatment.

Reversible airflow obstruction, enhanced bronchial reactivity and chronic airway inflammation form the basis for current definitions of asthma. They represent the major pathophysiological mechanisms leading to the symptoms of wheezing, breathlessness, chest tightness and cough by which physicians clinically identify this disorder, together with lung function measurements.

Dampness and mould problems in indoor environments

It is plausible that the causal exposures associated with the health effects typically observed in occupants of buildings with excess moisture, i.e. dampness or mould problems, can be both microbial and chemical in origin. At present, there are no comprehensive data on all exposures that can lead to relevant health effects, but useful surrogates for the exposures are observations of condensation, moisture or water damage and/or microbial growth in the indoor environment. Although different indicators and quantification systems of dampness and mould have been used, they generally appear to capture the extent of the problem well across different climates, cultures and building practices (see section 4).

Exposure agents in dampness and mould problem buildings

There is a relative lack of knowledge regarding the role of specific exposures in dampness and mould related health problems, largely due to their complex nature, the large variety of microbes that may play a role for the adverse health effects, and problems with quantitative exposure assessment methods for bioaerosols. Bioaerosols, i.e., particles of biological origin, may be found in elevated concentrations in the indoor air of damp and/or poorly ventilated buildings. Bioaerosols relevant to health in damp indoor environments include fungi (especially moulds

and yeasts), fungal spores, hyphae, as well as fungal fragments and allergens; bacteria and bacterial spores; microbial toxins and pro-inflammatory components (e.g. mycotoxins, (1→3)-β-D-glucans, endotoxin, exotoxins, peptidoglycans); arthropod allergens (e.g. from mites); algae; and amoebae (Jaakkola, Jaakkola, 2004; WHO, 2009). In addition to bioaerosols, indoor dampness may result in elevated concentrations of microbial volatile organic chemicals as well as increased chemical emissions of building materials, such as phthalates (Jaakkola, Jaakkola, 2004; Øie et al., 1999).

Selection of exposures and health outcome

A consistent association between dampness and mould problems in indoor environments and respiratory symptoms and asthma has been observed in a large number of studies conducted across many geographical regions (Bornehag et al., 2001; Bornehag et al., 2004; Zock et al., 2002; Fisk et al., 2007). Positive associations have been shown in infants (Øie et al., 1999), children (Brunekreef et al., 1989; Jaakkola et al., 1993; Andriessen et al., 1998; Zheng et al., 2002) and adults (Norbäck et al., 1999; Ruotsalainen et al., 1995; Kilpeläinen et al., 2001; Jaakkola et al., 2002; Park et al., 2008), and some evidence for dose-response relationships has also been demonstrated (Engvall et al., 2001).

For this burden of disease assessment from dampness and mould problems in indoor environments, we used questionnaire-based or inspector-reported indicators of dampness and mould growth in the home environment for assessing exposure, because these are applied in the epidemiological health effect studies we used to derive the risk estimates for exposure-response relationships.

Childhood asthma was chosen as the outcome for estimation of the burden of disease from indoor dampness and mould problems, because at present it has the strongest evidence base, and because asthma is the most common chronic disease in children, so its impact on the health burden at a population level is substantial. We conservatively excluded respiratory symptoms as separate outcomes, despite growing evidence on their relationship to indoor dampness and mould, because they are often related to asthma, and their separate inclusion would lead to double-counting of the burden. However, this conservative approach could also lead to an underestimation of the true burden.

2. Summary of the method

2.1 Methodological approaches

We calculated the population attributable fraction (PAF), which is the proportion of disease that can be ascribed to a specified exposure, using estimates of the exposure-response relation and prevalence of exposure based on a systematic search of previous studies. The PAF was subsequently applied to estimate the total burden of asthma in children in Europe in the form of deaths and disability adjusted life years (DALYs) that can be ascribed to indoor exposure to dampness and mould. Estimating the burden of disease therefore relied on the following three sources of information:

Exposure-risk relationship – Evidence for a significant exposure-risk relationship for asthma has been established in the 2007 meta-analysis (Fisk et al., 2007) and in other recent reviews (IOM, 2004; Bornehag et al., 2001; Bornehag et al., 2004; WHO, 2009). To select the best estimate for the risk ratio for onset of asthma in children, we conducted a structured review of publications on asthma onset in child populations.

Exposure assessment – Information was obtained from large population-based studies published in the last 10 years that reported the prevalence of household dampness and mould problems in Europe. We established three exposure risk categories (low, medium, and high).

Total burden of disease – Information was obtained on asthma occurrence based on the ISAAC phase III study (collected in 2002-2003) for the 6-7 year age group in 15 European countries, and from the WHO global burden of disease estimates for asthma among the 0-14 year age group (published in 2004) in the WHO Europe region (45 countries).

2.2 Literature search

A MEDLINE database search was performed with search terms 'dampness or moulds or microbial growth' AND 'asthma or respiratory tract disease'. Five studies with new-onset asthma as the outcome in a population of children were identified (Nafstad et al., 1998; Belanger et al., 2003; Wickman et al., 2003; Jaakkola et al., 2005; Pekkanen et al., 2007). These studies are summarized in Table 1. Since no meta-analysis including all of the studies on asthma onset is available to date, we chose the relative risk (RR) estimates for the burden of disease assessment based on these individual high quality studies. We focused on the induction of new disease (primary prevention) in order to distinguish it from the aggravation of a pre-existing disease, as asthma onset was considered the most important outcome in terms of public health. Therefore, we selected studies with a longitudinal design, including either cohort or incident case-control studies.

3. Exposure-risk relationship for dampness, mould and asthma

3.1 Evidence of exposure-risk relationship

Since the early 1990's an increasing body of evidence has accumulated on the relation between indoor dampness and mould, and respiratory infections, symptoms and asthma in both children and adults (Husman, 1996; Bornehag et al., 2001; Bornehag et al.,2004; Jaakkola, Jaakkola, 2004; IOM, 2004; WHO, 2009). The majority of the early childhood studies were cross-sectional or case-control studies. Their results were consistent with an effect of indoor dampness/mould exposure on asthma with estimated RR ranging between 1.4 and 2.2.

Fisk and colleagues (2007) recently published a meta-analysis of respiratory symptoms and asthma related to indoor dampness and mould problems. They reported an odds ratio (OR) of 1.37 (95% CI 1.23-1.53) for ever-diagnosed asthma and an OR of 1.56 (1.30-1.86) for current asthma for combined child and adult populations. No separate analysis of children and adults was conducted. An odds ratio for asthma development was also calculated for children and adults combined, but it was based on only four studies. When assessing the odds ratios for respiratory symptoms, they were usually somewhat higher in studies on children than in those on adults. For example, in Fisk et al.'s (2007) meta-analyses, the OR for cough was 1.75 (1.56-1.96) in children and 1.52 (1.18-1.96) in adults. For wheezing the ORs were 1.53 (1.39-1.68) and 1.39 (1.04-1.85) for children and adults, respectively. These estimates of odds ratio were based on studies with visible dampness and/or mould or mould odour as the exposure metric.

From the five studies identified in the systematic MEDline search, three studies investigated early signs of asthma up to the age of two years only (Nafstad et al., 1998; Belanger et al., 2003; Wickman et al., 2003). Because asthma diagnosis is less reliable in this age group the risk estimates should be interpreted with caution. A study from Finland investigated asthma in pre-schoolchildren 1-7 years old in an incident case-control study (Pekkanen et al., 2007) and one cohort study from Finland investigated asthma in children followed for over six years from the age of 1-7 years to the age of 7-13 years (Jaakkola et al., 2005). One study was hospital-based (Pekkanen et al., 2007), another included only infants with an asthmatic sibling (Belanger et al., 2003), while all the other studies were population-based. The exposure assessment was based on questionnaire-reported presence of signs of indoor dampness and moulds at home (Belanger et al., 2003; Wickman et al., 2003; Jaakkola et al., 2005) and/or such signs assessed in a home

inspection (Nafstad et al., 1998; Pekkanen et al., 2007). In addition, one study measured indoor fungi (Belanger et al., 2003).

The assessment of asthma varied among the studies, being based on reported wheezing in infancy (Belanger et al., 2003), reported episodes of wheezing and use of asthma medication (Wickman et al., 2003), reported doctor-diagnosed asthma that had started after baseline (Jaakkola et al., 2005) and a diagnosis by a paediatrician (Nafstad et al., 1998; Pekkanen et al., 2007). The OR for asthma was 1.7 – 2.6 in relation to questionnaire-based exposure assessment. The OR for an inspector-assessed exposure was 2.2 – 2.6, which was the same as or slightly higher than for questionnaire-based assessment. The risk related to measured fungi was lower (OR 1.1 – 1.2). The signs of dampness and mould problems included history of water damage; presence of moisture such as damp stains or windowpane condensation; presence of visible mould/mildew; and perceived mould odour. All of the five studies adjusted for an extensive set of confounders (Table 1) and the study by Nafstad et al. (1998) adjusted also for house dust mite allergens. The majority of studies had a response/follow-up rate of > 70% and were of high quality.

Nafstad et al. (1998) used an on-site home visit to confirm observations of water damage (i.e. damp stains or visible mould/mildew), and reported the highest OR of 2.6 (95% CI 1.6-4.2). In this study exposure assessment based on questionnaire report of home dampness gave exactly the same odds ratio for asthma. The case-control study of 1-7 year old children by Pekkanen et al. (2007) using on-site home visits to estimate exposure reported an OR of 2.24 (95% CI 1.25-4.01). The study by Jaakkola et al. (2005), which had the widest age range from 1 to 13 years and had the longest follow-up period (6 years), used mould odour as an exposure indicator and assessed exposure before the onset of asthma. It reported an incidence rate ratio (IRR) of 2.44 (95% CI 1.07-5.60). The birth cohort study by Wickman et al. (2003), which followed infants for a period of two years and used a questionnaire report of at least one of the following exposure indicators: smell and visible mould, water damage, persistent windowpane condensation, reported the lowest odds ratio of 1.74 (95%CI 1.28 –2.39). The two studies with a wider age range (Jaakkola et al., 2005; Pekkanen et al., 2007) had consistent risk estimates, suggesting that indoor dampness and mould-induced asthma continues to be important even after early childhood.

The study by Belanger et al. (2003) was based on parental reporting of wheezing and did not use any specific clinical markers for asthma (e.g. doctor diagnosis or use of asthma medication). Furthermore, it reported odds ratios separately for children whose mothers had asthma and for children whose mothers did not have asthma, which makes it difficult to make a direct comparison with the odds ratios reported in the other studies. However, it suggests that a genetic predisposition to asthma, measured as having a mother with asthma, modified the risk. Those with asthmatic mothers had a higher risk of developing asthma in relation to mould/mildew problems (OR 2.27, 95% CI 1.27-4.07) compared to those with no genetic predisposition, but the latter still showed a significantly increased risk (OR 1.83, 95% CI 1.04-3.22).

Table 1. Longitudinal and incident case-control studies on indoor dampness and mould and the onset of asthma in children

Reference	Study design	Study population	Exposure	Outcome	Adjustment for confounding	IRR or OR (95% CI) of asthma/other results	Comments
Nafstad et al., Norway, 1998*	Birth cohort followed for 2 years, nested incident case-control study within this cohort	Cohort of 3754 children born in Oslo 1992-93, 251 new cases (response rate 98%) and 251 controls (100%), 0-2 yrs	Questionnaire reported dampness problems + dampness problems confirmed by a trained home inspector. Presence of HDM in mattress of the child. Exposure assessed within 1 week of the dg	Clinical diagnosis of bronchial obstruction by paediatrician: - at least two episodes of symptoms and signs of airways obstruction or one episode lasting for more than 1 month	Matched for the time of birth; multivariate analysis: sex, birth weight, maternal age, siblings, pets, day care attendance, building type, parental atopy, breastfeeding, second-hand smoke exposure, socioeconomic conditions	Home dampness reported by parents OR 2.6 (1.7-4.0) Home dampness confirmed by a trained home inspector OR 2.6 (1.6-4.2) When controlling for dust mites: Dampness confirmed by a trained home inspector OR 3.8 (2.0-7.2) House dust mites > 2ug/g dust OR 3.7 (1.0-13.1)	Controls did not differ from the 2-yr cohort indicating no selection bias. Prevalence of house dust mites low: 4.5% among cases, 1.2% among controls.
Wickman et al., Sweden, 2003*	Birth cohort followed for 2 years	3692 children, 0-2 yrs; baseline response rate 75%, follow-up rate 90%; 312 new cases	Questionnaire report of at least one of the following indicators: smell and visible mould, persistent window condensation, water damage Exposure information collected before the onset of asthma	Questionnaire report of at least three episodes of wheezing after 3 months +treatment with inhaled steroids/signs of suspected hyperreactivity	Multivariate analysis: Sex, parental asthma or rhinitis, mother's age, socioeconomic status, breastfeeding, smoking mother, pets, year of building construction	Damp home environment OR 1.74 (1.28 –2.39)	Good follow-up rate. Cumulative incidence of asthma 8.5%.
Belanger et al., United States, 2003*	Birth cohort followed for one year	849 infants with an asthmatic sibling, 0-1 yr, response rate at baseline 69%; 380 cases with wheezing	Interview reported presence of persistent mould or mildew in the home living area during the previous year, assessed when child was 12 months old. Air sampling of fungal spores in main living area at 2-4 months	Interview report of wheeze: none, <30 days, $\geq$ 30 days during the first year of life	Multivariate analysis: sex, ethnicity, maternal education, smoking at home, exposure to indoor allergens, exposure to gas stove and wood stove	Children whose mothers had asthma: Reported mould/ mildew OR 2.27 (1.27-4.07); measured fungi per 20 colonies OR 1.23 (1.01-1.49) Children whose mother did not have asthma: Reported mould/ mildew OR 1.83 (1.04-3.22); measured fungi per 20 colonies OR 1.10 (0.99-1.23)	Reported mould/mildew exposure and measured fungi provided consistent results.

Reference	Study design	Study population	Exposure	Outcome	Adjustment for confounding	IRR or OR (95% CI) of asthma/other results	Comments
Jaakkola et al., Finland, 2005	Prospective population-based 6-year follow-up study	1916 children 1-7 yrs at baseline, 7-13 yrs at follow-up (follow-up rate 77%); 139 new cases	Questionnaire-report of 4 indicators at home at baseline: -history of water damage -presence of moisture -presence of visible mould -perceived mould odour Exposure information collected before the onset of asthma	Questionnaire-report of doctor-dg asthma that had started during the follow-up period, age at onset asked	Multivariate analysis: Age, sex, breast feeding, parental education, single parenting, type of child care, parental atopy, maternal smoking in pregnancy, exposure to second hand smoke, gas cooking, pets at home	Mould odour IRR 2.44 (1.07-5.60) Visible mould IRR 0.65 (0.24-1.72) Moisture in the surfaces IRR 0.92 (0.54-1.54) Water damage IRR 1.01 (0.45-2.26)	Good follow-up rate, no significant differences between baseline and 6-year cohort population, so no selection bias. Incidence rate of asthma was 125 per 10000 person-yrs (95% CI 104-146).
Pekkanen et al., Finland, 2007	Case-control study with new cases	121 new cases aged 1-7 yrs (response rate 70%) from the Kuopio University Hospital and 242 controls aged 1-8 yrs (response rate 62%)	Home inspection done after the diagnosis -excess moisture, moisture stains, visible mould, colour changes of materials, detached materials; location and severity	Clinical diagnosis of asthma by a paediatrician	Matching: birth year, sex, municipality; multivariate analysis: parental asthma, father's education, siblings, pets, day care attendance	Minor or major moisture damage in the main living area OR 2.24 (1.25-4.01) Visible mould in the main living area OR 2.59 (1.15-4.01) Mould odour in the main living area OR 2.96 (0.62-14.19) Damage in child's bedroom OR 1.97(1.00-3.90)	Moisture damage in general more common among controls, but homes of cases had more visible mould, mould odour, moisture damage in main living area and child's bedroom; OR increased with maximum severity of damage

* Study involved infants, at which age childhood asthma can not be diagnosed reliably. Risk estimates are therefore based on early signs of childhood asthma.

3.2 Synthesis of the evidence and selection of risk estimates

There were five studies with a longitudinal design showing similar associations between indoor dampness/mould problems and new-onset asthma in children. Their risk estimates and confidence intervals were comparable, but the definitions of exposure varied from general "dampness" indicators to more specific exposure indicators related to microbial growth (e.g. mould odour). The odds ratio estimates selected for burden of disease calculations were OR=2.2 (1.3-4.0) for a general indicator of dampness (Pekkanen et al., 2007) and OR=2.4 (1.1-5.6) for a specific indicator of mould growth (Jaakkola et al., 2005).

These estimates are close to those reported in numerous cross sectional studies, and the slightly higher risk estimates may be related to more specific health outcome (focusing on the onset asthma in children), and stronger study designs of the longitudinal studies. The majority of the studies came from Scandinavian countries and the United States. However, in the previous cross-sectional studies, the risk estimates have been in the same order of magnitude in other countries. Therefore, the selected estimates can be used for the European-level assessment of the burden of disease of onset asthma in children from exposures to indoor dampness and mould.

4. Exposure assessment for indoor dampness / mould

To date, the exact importance of exposure to any specific microbial agent emitted from micro-organisms (notably mould) growing in the indoor environment has not been conclusively identified. However, a recent longitudinal study of onset of asthma in adults indicated that hydrophilic fungi had the strongest association with asthma onset (Park et al. 2008). As such, there is not one specific microbial or chemical marker of exposure that could be recommended. Therefore, for the purpose of estimating the disease burden due to dampness and mould, we chose two indicators often used in the epidemiological health effect studies: i) a general indicator of dampness (referred to as "dampness" in the text), and ii) a more specific indicator of microbial growth based on visible mould and/or mould odour (referred to as "mould" in the text).

4.1 Measuring indicators of indoor dampness / mould

Different studies use different definitions of indoor dampness and mould, making comparison between studies somewhat difficult, but the terms describing dampness and mould appear to be applicable to various climates, cultures and building practices.

Occupants' perceptions have been the basis for assessing dampness/mould in most population studies. In these studies occupants were typically asked whether conditions such as leaks, floods, wet basements, window condensation, visible fungal growth, or mouldy odours were present currently and/or had been present in the past. Sometimes the extent of water damage and damp problems was also assessed. However, there was considerable variation in how these questions were stated, and prevalence estimates may vary, depending on the type of questions used, the level of detail requested, and the judgement of those filling in the questionnaires.

Reliance on self-reports, which are by definition subjective, may be a source of error in cross-sectional studies, as demonstrated by Dales and colleagues (1997), who reported that under some conditions allergy patients may be more likely than non-allergic people to report visible fungal growth. However, several studies have demonstrated that such bias is unlikely to occur (Verhoeff et al., 1995; Zock et al., 2002; Jaakkola, Jaakkola 2004). A study by Williamson et al. (1997) reported that occupants had a tendency to underestimate dampness. Nevalainen et al. (1998) concluded the same, suggesting that one explanation was the trained eye of the inspectors to rate their observations together with their knowledge of what represents critical problems. To overcome possible problems associated with reporters' bias some studies have used trained

inspectors who visit the house and provide an assessment of indoor dampness including the severity of the problem. However, in the study by Nafstad et al. (1998) both exposure assessment approaches led to exactly the same odds ratio for asthma.

Haverinen-Shaughnessy et al. (2005) studied moisture damage observations made by both occupants and independent inspectors and concluded that the inspectors observed more damage than did the occupants. The overall agreement between the inspector and the occupants was poor, whereas the agreement between the two inspectors was higher. Trained inspectors are more objective because they apply a standardized approach. On the other hand, trained inspectors lack the longer time perspective of the occupants. Hence, it is not quite evident which one of the two approaches (occupant reports or inspector observations) provides the most valid assessment of indoor dampness/mould problems.

4.2 Approaches for exposure assessment

Survey based prevalence estimates of dampness/mould in residential buildings have varied widely, from approximately 2 to 85%, depending on the study design, climate, and definition used (Bornehag et al., 2001). It is likely that the prevalence of dampness/mould in the housing stock has geographical variation and also changes over time depending on the economic situation and/or degree of housing deprivation. Also, increasing public awareness about the association between dampness/mould and poor health may prompt preventive and corrective actions. Environmental factors such as climate change and increasing demands for energy efficiency in buildings may also result in changes in the prevalence of dampness and mould problems. Therefore, the estimates of exposure for the purpose of assessing the asthma burden that is attributable to indoor dampness and mould problems should rely on relatively recent studies, taking into account climatic/regional differences, as well as differences in study design, methodology, and definitions.

There are also differences in exposure assessment based on different types of observations of dampness (e.g. high relative humidity, condensation on surfaces), moisture/water damage (e.g. signs of leaks, stained/discoloured building materials), or microbial growth (e.g. visible mould, mould odour). There are also differences between studies related to “current” or past exposures (e.g. occurred in the past 12 months, 5 years, etc.), as well as differences regarding the location of such observations within the building. Most of the studies do not differentiate between locations, but some studies emphasize dampness/mould in the child’s bedroom or other living areas. Some of the studies report the extent and/or severity of dampness/mould, but most are based on a dichotomous rating.

To cover the different exposure assessment approaches we chose to carry out separate burden of disease assessments for two types of indicators:

A general indicator for **“dampness”**, which includes observations of high relative humidity, condensation on surfaces, moisture/water damage, signs of leaks and stained/discoloured surface materials. This indicator reflects a larger spectrum of potential causal agents, including house dust mites and emission of chemicals, and comprises also milder problems. This indicator is widely used in many epidemiological studies.

A specific indicator for **“mould”** includes observations of visible microbial growth, especially visible mould, and mould odour. This indicator reflects more specific microbial origin and may reflect more extensive damage and higher exposure indoors. The fact that the signs are visible and/or can be smelled may also mean that this exposure could have more direct health relevance, as it is more likely to be accompanied by exposure agents in the breathing zone of humans.

4.3 Estimation of exposure in Europe

Exposure estimates were selected from a Medline search using the terms 'dampness OR mould OR microbial growth' and including studies published in the past 10 years. In addition, other large surveys providing data on indoor mould and dampness exposures were identified.

Large data sources are available from multinational studies that used the same definition of indoor dampness and mould problems throughout the study and therefore provided comparable estimates between countries and regions (Table 2). The LARES survey was undertaken in eight European cities in 2002 and 2003, consisting of data on approximately 400 dwellings from each city (WHO, 2007), and relying on on-site home visits. According to the dwelling inspections conducted by trained surveyors, visible mould growth was detected in at least one room of almost 25% of all visited dwellings. Country specific data were not reported in the preliminary overview of LARES findings. Findings related to other dampness/moisture related variables (including smell of dampness and signs of condensation) were not included in the report.

The European Community Respiratory Health Survey (ECRHS) investigated self-reported dampness and mould exposure in 38 study centres in 18 countries (Zock et al., 2000). Centres were located both in Europe (14 countries), and outside Europe (four countries). During the year prior to the interview water damage was observed in 12.4% (range 4-32%), water on basement floor in 2.2% (0-16%), and mould or mildew in 22.1% (5-56%) of the dwellings. Gunnbjörnsdottir et al. (2006) reported an overall prevalence of home dampness of 18% based on the ECRHS questionnaire conducted in the Nordic countries (Iceland, Norway, Sweden, Denmark) and Estonia eight years after the original survey.

Eurostat defines dampness as "rot in the house or damp or leaky roof" (2001) or "leaking roof, damp walls" (Eurostat, 2007) based on occupant reports (Lelkes, Zolyomi, 2010). Exposure to these types of problems varied among 13 countries from 4.2% (Finland) to 35.7% (Portugal) in 2001 and among 24 countries from 4.9% (Finland) to 37.5% (Poland) in 2007. In summary, LARES and ECRHS provided similar overall estimate for indoor mould problems of 25% and 22%, respectively. ECRHS and Eurostat also showed a similar range with respect to water damage (4-32% and 4-38%, respectively). However, it is not completely clear what type of water damage is referred to in these reports.

These exposure assessments are supported by country-specific studies. Three national studies were identified that relied on on-site home visits (Table 3). Brasche et al. (2003) reported signs of indoor dampness/mould in 21.9% of 5530 studied dwellings in Germany. Specifically, 9.3% of the dwellings had visible mould. Depending on dwelling type (single-family houses vs. apartment buildings), the overall prevalence of major or minor indoor mould or water damage ranged from 26-38% in Finland (Chelelgo et al., 2001) and the overall prevalence was 51% in the United States (Cho et al., 2006). A more recent Finnish study (Pekkanen et al., 2007) reported any or suspected damage in 86% and visible mould in 49% of the studied dwellings, and in the main living area a minor damage at 20% and a major damage at 10.5%. However, the study by Cho et al. was a prospective birth cohort study of atopic parents, and the study by Pekkanen et al. was a hospital-based case-control study of asthmatic (N=121) and non-asthmatic children (N=241), so the estimates may not be applicable for general population estimates.

Some 16 studies were identified that were based on occupant self-reporting. Six of these studies followed the ISAAC protocol and reported past or present dampness and/or mould in 3-36% of homes. However, the highest prevalence values reported by Tamay et al. (2007) and Bayram et al. (2004) were specific to children with allergic rhinitis symptoms and asthmatics, respectively. When excluding these two studies the range was 3-24% (Table 4). In the rest of the studies, the prevalence of self-reported dampness/mould varied from 1.5% to 29%. Specifically, the range was 5-27% for dampness/water damage, and 1.5-29% for mould.

Table 2. Prevalence of dampness/mould problems in homes from multinational studies

Reference	Target population	Method	Prevalence
WHO, 2006	Randomly selected households of eight European cities in 2002 and 2003, consisting of data for 300-500 dwellings from each city (3373 dwellings total, 8519 individuals total)	On-site home visits and questionnaire	25% mould growth in at least one room of all dwellings
Gunnbjörns dottir et al., 2006	16190 adults from Iceland, Norway, Sweden, Denmark and Estonia	Questionnaire (ECRHS)	18% living in damp housing
Zock et al., 2002	Random general population sample of 18 873 20-45 yrs old adults from 38 study centers in 18 countries	Interview-led questionnaire (ECRHS)	12.4% (4-32% per country) water damage in the last year 2.2% (0-16% per country) water on basement floor 22.1% (5-56% per country) mould or mildew in the last year
Eurostat, 2001	General population estimates in 13 countries	Questionnaire	4.2-35.7% with rot in the house or damp and leaky roof
Eurostat, 2007	General population estimates in 24 countries	Questionnaire (SILC survey)	4.9-37.5% with leaking roof or damp walls

Table 3. Prevalence of dampness/mould problems in homes from studies based on on-site home visits

Reference	Target population	Method	Prevalence
Cho et al., 2006	640 infants (8 mo) born in Cincinnati, OH, and northern Kentucky, United States in 2001-2003	On-site home visit (referring to Finland protocol)	51% minor mould or water damage 5% major mould or water damage with visible mould at 0.2m2 or more
Brasche et al., 2003	5530 randomly selected residences in Germany	On-site home visit	21.9% had signs of dampness (including mould) 9.3% had mould spots
Chelelgo et al., 2001	630 randomly selected Finnish residences	Home inspections (Finland protocol)	23% houses/11.5% apartments has notable moisture problems 15% houses/14.5% apartments has significant problems

Table 4. Prevalence of dampness/mould problems in homes from studies based on occupant self-reporting

Reference	Target population	Method	Prevalence
Turunen et al., 2008	Random population based sample of 1312 18-75 yrs old subjects in Finland	Questionnaire	5.3% had moisture/mould damage in the past 12 months 6.3% of those who had school age children (unpublished data) 8.8% of those who had children <7 yrs old (unpublished data) 1.2% reported mould odour inside the dwelling

Reference	Target population	Method	Prevalence
Simoni et al., 2007	Population sample of 20 016 children (mean age 7 yrs) and 13 266 adolescents (mean age 13 yrs) in northern and central Italy	Questionnaire (ISAAC protocol)	Mould/dampness (depending on the age group) 9.5-10.1% current 12.8-9.5% early 5.5-7.3% only current 8.8-6.7% only early 4.0-2.8% both
du Prel et al., 2005	25 864 children (6 yrs) in East Germany (all children entering elementary school living in the geographically defined area)	Questionnaire	6.1-10.4% living under damp housing condition in 2000 (depending on parental education level)
Bornehag et al., 2005	10 851 children (1-6 yrs) from the Swedish county of Värmland	Questionnaire (DBH)	17.8% water leakage 14.3% condensation 8.3% detached flooring materials 1.5% visible mould/damp spots
Jaakkola et al., 2005	Random sample of 1984 children drawn from the roster of Finland's Statistical Center (77.3% of the baseline study population)	Questionnaire	2.9% mould odour 4.6% visible mould 15.4% moisture in the surfaces 5.4% water damage 20% any of above
Zhang et al., 2005	996 children (4-12 yrs) from western Australia recruited from four standard school	Questionnaire	9% damp patches 28% condensation 19% moulds
Spengler et al., 2004	5951 children from 9 Russian cities	Questionnaire	22.4% water damage 10.4% water damage in the past 12 months 10% mould in the past 12 months
Salo et al., 2004	4185 ninth grade students from 22 randomly selected schools in Wuhan, China.	Questionnaire (ATS-DLD-78-C)	23.3% mould/water damage in the past 12 months
Lee et al., 2003	National survey of 35 036 children (6-15 yrs) from 22 elementary and 22 middle schools in Taiwan	Questionnaire (ISAAC protocol)	8.06/8.64% water damage 23.57/23.58% visible mould (boys/girls)
Wickman et al., 2003	4089 children at age 2 from southern Sweden (75% of all eligible children born in pre-defined areas fulfilling the inclusion criteria)	Questionnaire (BAMSE)	15.4% damp home environment
Rönmark et al., 1999	3525 children (7-8 yrs) enrolled from three municipalities in northern Sweden (Kiruna, Luleå, and Piteå)	Questionnaire (ISAAC protocol)	18% past or present dampness
Baker, Henderson, 1999	Random sample of 1954 representative of women with children <1 in Great Britain (stratified by age and tenure)	self-reporting (ALSPAC)	18.7-26.7% damp 21.2-28.6% mould (mortgaged – rented home)

Table 5. Additional studies (limited by pre-position of children or type of room)

Reference	Target population	Method	Prevalence
Tham et al., 2007	4759 children 1.5-6 yr in Singapore attending 120 randomly selected day-care centers	Questionnaire (ISAAC protocol)	5% dampness, 3% visible mould in child's bedroom
Tamay et al., 2007	2500 children aged 6-12 from randomly selected 6 primary schools in Istanbul	Questionnaire (ISAAC protocol)	35.8% of children with allergic rhinitis symptoms had dampness at home
Bayram et al., 2004	Cross-sectional population-based study carried out on 3164 schoolchildren aged between 6-18 from randomly chosen schools of Adana, Turkey	Questionnaire (ISAAC protocol)	34% of asthmatics had dampness at home
Freeman et al., 2003	4634 elementary schoolchildren living in a predominately Hispanic community in Passaic, New Jersey, United States	Questionnaire	12.3- 21.4% bathroom damp/mould depending on the ethnic group (black/non-Hispanic white/Mexican/Puerto Rican/Dominican

4.4 Summary of exposures in Europe

Different types of dampness/mould observations may reflect differences in climate and housing conditions. In European countries with cold climate (e.g. Finland, Sweden) the prevalence of self-reported indoor dampness ranged from 5 to 20% and visible mould or mould odour from 1.2 to 5%. In the Russian Federation, the prevalence of water damage in the previous 12 months was approximately 10%, and the prevalence of visible mould in the previous 12 months was also 10% (Table 6). In European countries with moderate/warm climate (e.g. United Kingdom, Germany and Italy) dampness and/or mould problems were reported in 6 to 29% of dwellings, and the estimates from the rest of the warm climate countries were in general within the same limits. Therefore, the level of exposure could be higher in warm climates, particularly with respect to visible mould. The occurrence of water damage ranged from 5-18%, and appeared to be less dependent on climate.

Table 6. Estimates of dampness/mould prevalence by type of exposure indicator and climate

	Cold climate	Moderate/warm climate
Water damage	18% (Bornehag et al., 2005) 5% (Jaakkola et al., 2005) 10% (Spengler et al., 2004)	9% (Zhang et al., 2005) 8-9% (Lee et al., 2003)
Condensation	14% (Bornehag et al., 2005) 15% (Jaakkola et al., 2005)	28% (Zhang et al., 2005)
Dampness	15% (Wickman et al., 2003) 18% (Rönmark et al., 1999)	6-10% (du Prel et al., 2005) 19-27% (Baker, Henderson, 1999)
Mould	1.2% (Turunen et al., 2008) 1.5% (Bornehag et al., 2005) 3-5% (Jaakkola et al., 2005) 10% (Spengler et al., 2004)	19% (Zhang et al., 2005) 24% (Lee et al., 2003 21-29% (Baker, Henderson, 1999)
Any combination of above	5% (Turunen et al., 2008) 20% (Jaakkola et al., 2005)	5.5-7% (Simoni et al., 2007) 23% (Salo et al., 2004)

In summary, we estimate that in cold climates 15% of dwellings have signs of dampness problems in general and 5% have signs of mould problems. In warm climates the corresponding estimates are 20% for dampness and 25% for mould. The estimate for water damage is 10%, regardless of climate. Thus, a conservative estimate for the range of exposure to "dampness" in Europe is 10-25% and for the assessment of burden of disease we used the central value of 15%. Similarly, a conservative estimate for the range of exposure to "mould" in Europe is 5-25% and for the assessment of burden of disease we used the central value of 10%.

5. Total burden of disease from asthma

5.1 Definition of asthma

Asthma diagnosis is based on a set of clinical symptoms and signs, including lung function changes, so there is no single test or 'gold standard' that could be used to define asthma. Furthermore, the variability over time of the condition typical for asthma, means that evidence of it may or may not be present on any one day, or at any one point in time, when someone is assessed. Thus, the clinical diagnosis of asthma is made on the basis of the clinical history, combined with physical examination and lung function tests over a period of time. The prevalence of physician-diagnosed asthma is substantially lower than the prevalence of asthma symptoms in the community (Asher et al., 1998). This is not surprising, since a clinical diagnosis of asthma can only be made if a person presents him or herself to a doctor. In addition, the symptoms related to asthma are non-specific and often occur with other respiratory diseases. Diagnosed asthma is also dependent on diagnostic practices and the access to health care.

Epidemiological studies have often defined asthma based on self-reported (or parental reported) symptoms related to asthma, including wheezing, chest tightness, breathlessness and coughing. However, a diagnosis based on reports of symptoms only may lead to considerable misclassification, as many other respiratory conditions also cause similar respiratory symptoms. Self-reports of doctor diagnosed asthma are a stronger definition of asthma than symptom report alone. Another approach used in some epidemiological studies has been to use lung function measurements such as bronchial responsiveness testing and spirometry and/or peak expiratory flow in combination with questionnaire-based information. Differences in asthma definition are likely to result in different estimates of asthma. However, bias in estimating the relative risk of asthma related to a specified exposure is only caused if asthma is assessed in a different way in the exposed and unexposed subjects. Usually the same asthma assessment method has been used in both of these comparison groups, so the misclassification in asthma assessment leads to underestimation of the risk ratio.

5.2 Estimating the burden of disease for indoor dampness and mould problems

For assessing the proportion of new-onset asthma in children attributable to mould in their home environment, we used a relative risk estimate of RR=2.4 (1.1-5.6) (from Jaakkola et al. 2005) and an exposure estimate of 10% (5-25%) for "mould". We used a risk estimate of RR=2.2 (1.3-4.0) (from Pekkanen et al. 2007) and exposure estimate of 15% (10-25%) for "dampness", as defined above. The population attributable fraction (PAF) was calculated using the formula:

$$PAF = \frac{p(RR-1)}{p(RR-1)+1}$$ where p = proportion of exposed; RR = relative risk for asthma onset.

For example, using the medium exposure estimates for calculating PAFs:

"Mould" and asthma onset (based on IRR of 2.4 for asthma)

$$PAF = \frac{0.10(2.4-1)}{0.10(2.4-1)+1} = 0.123$$

"Dampness" and asthma onset (based on OR of 2.2 for asthma):

$$PAF = \frac{0.15(2.2-1)}{0.15(2.2-1)+1} = 0.153$$

Results for the range of exposure estimates are shown in Tables 7 and 8.

Table 7. Proportion of asthma among children attributable to low, medium and high population exposure estimates to "mould"

Risk estimate (95% CI)	**PAF of new asthma related to population exposure to indoor "mould" (%)**		
	Low (5%)	**Medium (10%)**	**High (25%)**
2.4 (1.1-5.6)	6.5 (0.5-18.7)	12.3 (1.0-31.5)	25.9 (2.4-53.5)

Table 8. Proportion of asthma among children attributable to low, medium and high population exposure estimates to "dampness".

Risk estimate (95% CI)	**PAF of new asthma related to population exposure to indoor "dampness" (%)**		
	Low (10%)	**Medium (15%)**	**High (25%)**
2.2 (1.3-4.0)	10.7 (2.9-23.1)	15.3 (4.3-31.0)	23.1 (7.0-42.9)

The estimated percentage of asthma onset in children attributable to indoor "mould" in their living environment is approximately 12% and that attributable to "dampness" is approximately 15%. The range based on low to high exposure estimates reflects differences in occurrence of different types of exposures, i.e. direct observations of mould may be less prevalent than other types of dampness problems; and differences in occurrence of exposures due to differences in climate, regional differences in populations and housing stock, and other known and unknown factors. The range of PAF for different exposure estimates was from 6.5% to 26% for "mould" and 11% to 23% for "dampness".

6. Environmental burden of disease from dampness and mould in Europe

Table 9 presents calculations of the asthma burden from indoor mould and dampness problems in the home environment based on occurrence of asthma according to the ISAAC phase III data (collected in 2002-2003) for 6-7 years old children in selected European countries (Asher et al., 2006). ISAAC data based the diagnosis of asthma on asthma symptoms reported by parents and measurements of bronchial hyperresponsiveness.

Having estimated the PAF, we estimated DALYs and mortality attributable to mould and dampness problems in home environments by multiplying the asthma DALYs and mortality rates from WHO data by the calculated PAFs for children aged 0-14 years (Table 10). These data for all countries in Europe were produced as part of the WHO Global Burden of Disease Project update for 2004 (WHO, 2008). The BoD estimates are available for asthma DALYS and deaths in children aged 0-14 years.

Table 9. Prevalence of asthma related to mould and dampness problems in the home environment estimated based on occurrence of asthma in the ISAAC phase III data for children 6-7 years old

Country	Sample size	Response rate	Asthma prevalence	Mould-related asthma prevalence by different levels of exposure to indoor moulds			Dampness-related asthma prevalence by different levels of exposure to indoor dampness		
				Low PAF% 6.5 (0.5-18.7)	Medium PAF% 12.3 (1.0-31.5)	High PAF% 25.9 (2.4-53.5)	Low PAF% 10.7 (2.9-23.1)	Medium PAF% 15.3 (4.3-31.0)	High PAF% 23.1 (7.0-42.9)
		%	per 1000	per 1000	per 1000	per 1000	per 1000	per 1000	per 1000
Northern and eastern Europe									
Albania	2896	87.6	50	3 (0.2-9)	6 (0.5-16)	13 (1-27)	5 (1-12)	8 (2-16)	12 (3-21)
Estonia	2385	85.6	96	6 (0.5-18)	12 (1-30)	25 (2-51)	10 (3-22)	15 (4-30)	22 (7-41)
Georgia	2666	92.9	69	4 (0.3-13)	8 (0.7-22)	18 (2-37)	7 (2-16)	11 (3-21)	16 (5-30)
Lithuania	2772	92.0	66	4 (0.3-12)	8 (0.7-21)	17 (2-35)	6 (2-9)	10 (3-20)	15 (5-28)
Poland	4496	81.9	136	9 (1-25)	17 (1-43)	35 (3-72)	15 (4-31)	21 (6-42)	31 (9-58)
Russian Federation	2730	95.2	114	7 (1-21)	14 (1-36)	30 (3-60)	12 (3-26)	17 (5-35)	26 (8-49)
Sweden	2089	63.8	102	7 (1-19)	13 (1-32)	26 (2-55)	11 (03-24)	16 (4-32)	24 (7-44)
Ukraine	1950	99.1	125	8 (1-23)	15 (1-39)	32 (3-67)	13 (4-29)	19 (5-39)	29 (9-54)
Western Europe									
Austria	6876	87.8	74	5 (0.3-14)	9 (1-23)	19 (2-40)	8 (2-17)	11 (3-23)	17 (5-32)
Belgium	5645	77.8	75	5 (0.4-14)	9 (1-24)	19 (2-40)	8 (2-17)	11 (3-23)	17 (5-32)
Germany	3830	82.4	128	8 (1-24)	16 (1-40)	33 (3-68)	14 (4-30)	20 (6-40)	30 (9-55)
Italy	11 287	92.5	79	5 (0.4-15)	10 (1-25)	20 (2-42)	8 (2-18)	12 (3-25)	18 (6-34)
Portugal	5365	65.1	129	8 (1-24)	16 (1-41)	33 (3-69)	14 (4-30)	20 (6-40)	30 (9-55)
Spain	18 941	77.2	95	6 (0.5-18)	12 (1-30)	25 (2-51)	10 (3-22)	14 (4-29)	22 (7-41)
United Kingdom	1843	91.9	209	14 (1-39)	26 (2-66)	54 (5-112)	22 (6-48)	32 (9-65)	48 (15-90)

Source: modified from Asher et al., 2006, reprinted with permission from Elsevier.

The results show that indoor mould and dampness problems in the home environment attribute to a considerable burden of asthma in European children, accounting for 55 842 DALYs based on the exposure prevalence of 10% of indoor "mould" in home environment (the range based on the 95% CI of the risk estimate being from 4540 to 143 010) and 69 462 DALYs based on the exposure prevalence of 15% of indoor "dampness" in the home environment (the range being from 19 522 to 140 740).

It should be noted that these two environmental burden of disease assessments overlap, as damp indoor conditions are a prerequisite for microbial growth that has relevance for health. Thus, the environmental burden of disease (EBD) attributable to indoor "mould" is a more specific estimate for microbial growth indoors that is associated with asthma. This should be used when the aim of EBD assessment is to address the question of impact of indoor microbial growth specifically. The EBD attributable to indoor "dampness" is a more general indicator of moisture-related indoor problems. It probably includes also damage of less severity than those leading to mould growth, but on the other hand is more comprehensive, as it includes also other exposures that are associated with asthma (e.g. house dust mites and emission of phthalates). So for a more comprehensive EBD assessment due to dampness-related housing factors the EBD attributable to indoor dampness is recommended to be used.

Table 10. Asthma burden from indoor mould and dampness in children (age 0-14) measured as DALYs and deaths in 45 European countries[2] based on WHO, 2008 (BoD data for Europe, target population: 140 189 358 in 2004).

Burden of disease	**Burden of childhood asthma attributable to exposure to "mould" in the home according to different levels of exposure**			**Burden of childhood asthma attributable to exposure to "dampness" in the home according to different levels of exposure**		
	Low PAF 6.5% (0.5-18.7)	Medium PAF 12.3% (1.0-31.5)	High PAF 25.9% (2.4-53.5)	Low PAF 10.7 (2.9-23.1)	Medium PAF 15.3% (4.3-31.0)	High PAF 23.1% (7.0-42.9)
Deaths	43.9 (3.4-126.2)	83.0 (6.8-212.6)	174.8 (16.2-361.1)	72.2 (19.6-155.9)	103.3 (29.0-209.3)	155.9 (47.3-289.6)
DALYs	29 510 (2270-84 898)	55 842 (4540-143 010)	117 586 (10 896-242 890)	48 578 (13 166-104 874)	69 462 (19 522-140 740)	104 874 (31 780-194 766)

7. Uncertainties

Uncertainties in this assessment are related to the selected estimates of exposure-effect relations, selected estimates of exposures, and data used on asthma mortality and DALYs.

7.1 Assessment of exposure-effect relations

Outcome assessment in the studies from which the risk estimates were selected for this EBD assessment was based on doctor-diagnosis of asthma, which may be subject to some misclassification. However, the studies that provided the risk estimates were conducted in Finland, which has a national health care system, national guidelines for asthma diagnosis and

[2] Countries as listed in WHO BoD 2004 update (WHO, 2008): Albania, Andorra, Armenia, Austria, Belarus, Belgium, Bosnia and Herzegovina, Bulgaria, Croatia, Cyprus, Czech Republic, Denmark, Estonia, Finland, France, Georgia, Germany, Greece, Hungary, Iceland, Ireland, Italy, Latvia, Lithuania, Luxembourg, Malta, Monaco, Netherlands, Norway, Poland, Portugal, Republic of Moldova, Romania, Russian Federation, San Marino, Serbia and Montenegro, Slovakia, Slovenia, Spain, Sweden, Switzerland, The Former Yugoslav Republic of Macedonia, Turkey, Ukraine, United Kingdom

management and a national reimbursement system for asthma medications, so there is no reason to believe that the misclassification would vary by exposure status, as all social classes have adequate access to the health care system. As the asthma assessment was the same for the exposed and unexposed children, any misclassification in the asthma definition would lead to underestimating the true effect.

Avoidance behaviour could have resulted in exposure misclassification, i.e. those with asthma may have changed their living environment to reduce exposure levels. This is mostly a concern for cross-sectional studies and may lead to an underestimate of the true effect. It is not relevant to those studies where exposure was assessed before the onset of asthma, as was done in the Finnish 6-year follow-up study.

Several other factors are known as risk factors for childhood asthma, such as parental atopy, parental smoking indoors, short length of breastfeeding and having pets at home. These could be confounders for the studied effect, if they are also related to exposure status. For example, if children with lower socioeconomic status have more dampness/mould exposure at home and also have more exposure to second hand smoke, there could be a confounding influence. However, these as well as several other factors were adjusted for in the multivariate analysis, so confounding is not likely to explain the results.

Finally, there is the question whether the relative risks selected can be generalized into other populations. Four of the five longitudinal studies were from Nordic countries (Finland, Norway, Sweden) with a cold climate, while one was from the United States (Table 1). However, there is abundant evidence from previous cross-sectional studies that the risk estimates seem to be rather consistent throughout the world, including countries such as Taiwan with a subtropical climate (Bornehag et al., 2001). We limited our burden of disease assessment to children's asthma, because there were more longitudinal studies in child populations than in adults. In the future, if the number of studies on incident asthma in adults increases, an assessment including adulthood asthma as an outcome may be warranted.

7.2. Assessment of exposure prevalence

The estimates of the prevalence of indoor mould and dampness problems in home environments bear uncertainty because of several reasons: i) There is a relatively small number of studies that have assessed the prevalence of such exposures in different countries, ii) different studies have used different exposure definitions, and iii) with damp and moldy housing conditions there are probably several exposures that may induce asthma, and at the moment we do not know well the specific components that are the most relevant for health effects, such as asthma.

The studies on health effects assessed indoor mould and dampness exposures based on questionnaires, which could include some misclassification. Among the longitudinal studies reviewed in this chapter, odds ratios based on inspector- assessed exposure were the same or slightly higher when compared to parent-reported exposures (Nafstad et al., 1998; Pekkanen et al., 2007), which gives assurance that the selected risk estimates are valid. Based on the few studies conducted using on-site home visits, it appears that occupant self-reporting may give lower exposure estimates as compared to estimates based on on-site home visits conducted by trained inspectors, which would mean that our exposure prevalence values may underestimate the true exposures.

In the longitudinal studies on asthma, the same exposure assessment method was used for both the exposed and unexposed children, which reduces the concern for differential misclassification. In addition, the study by Jaakkola et al. (2005) assessed exposure before the onset of asthma, which should further diminish the concern of differential misclassification according to disease status.

Both occupant reporting and on-site visits typically rely on non-destructive (primarily visual) observation. Several factors cannot be taken into account in non-destructive investigation of buildings, such as the presence of hidden damage or microbial growth, and identification of specific types of microbes suspected to be more hazardous to health (e.g. toxin producing species). Neglecting these factors could lead to underestimation of exposures. Another problem that may be related to the use of surrogate measures for dampness/mould is that these measures may also be surrogates for other housing related factors that may associate with asthma. These factors may include, for example, general lack of hygiene, insufficient ventilation, high occupant density (inc. number of children in the household), and better survival of viruses and bacteria responsible for communicable diseases in humid indoor air, poverty, etc. Therefore, in order to assess the specific contribution of indoor dampness to the development of asthma, appropriate adjustment for social economic position, ventilation etc. may be required. Most of the studies summarized in this report (Table 1) adjusted for at least some of these parameters.

Occupant self-reporting may also involve reporting bias, e.g. symptomatic occupants may report more damp/mould than non-symptomatic occupants, but using exposure estimates from large population studies eliminate this concern. In addition, this is only a problem in cross-sectional and case-control studies and will not affect exposure estimates from longitudinal studies, which assessed exposure before the onset of asthma. The more relevant uncertainty related to exposure assessment is related to the fact that we applied exposure estimates from countries where such data was published to several countries from which such data is not available.

7.3. Assessment of asthma occurrence

Finally, the estimate for occurrence of asthma was based on the ISAAC project that defined asthma on the basis of asthma symptoms and bronchial hyperresponsiveness instead of clinical diagnosis (which was the primary outcome used for risk estimation). This may lead to some overestimation of burden, as the prevalence of asthma as defined by ISAAC may be higher than the prevalence of doctor diagnosed asthma.

8. Conclusions

Indoor mould and dampness problems in the home environment account for estimated 55 842 DALYs based on the exposure prevalence of 10% of indoor “mould” in home environment and 69 462 DALYs based on the exposure prevalence of 15% of indoor “dampness” in the home environment.

These two environmental burden of disease assessments overlap, as damp indoor conditions are a prerequisite for microbial growth that has relevance for health. Thus, the environmental burden of disease (EBD) attributable to indoor “mould” is a more specific estimate for microbial growth indoors that is associated with the respective health outcome. The EBD attributable to indoor “dampness” is a more general indicator of moisture-related indoor problems. It is more comprehensive, as it includes also other exposures that are associated with the respective health outcome (e.g. house dust mites and emission of phthalates). For a comprehensive EBD assessment due to dampness-related housing factors, therefore, the EBD attributable to indoor dampness should be considered.

Table 11. Summary of EBD of asthma onset in children caused by “mould” in home environment

Housing exposure	Indicators of “mould”			
Health outcome	Asthma deaths and DALYs in children			
Summary of EBD assessment	About 12% of new childhood asthma in Europe can be attributed to indoor mould exposure, which represents approximately 55 842 potentially avoidable DALYs and 83 potentially avoidable deaths per year.			
	Level	**Range**	**Geographic scope**	**Source of information**
(a) Exposure risk relationship	RR=2.4	1.1-5.6	Nordic countries, United States	Cohort studies with largely consistent findings (Jaakkola et al., 2005)
(b) Exposure assessment	10%	5-25%	Europe	Multiple cross-sectional and some cohort studies of indoor mould
(c) PAF	12.3%	1.0-31.5%	Europe	Derived from (a) and (b)
(d) Total burden of disease	Fatalities= 675 DALYs = 454 000	- -	Europe	WHO Global BOD Report (WHO, 2008)
(e) EBD from exposure	83 deaths (0.06/100 000) 55 842 DALYs (40/100 000)	7-213 deaths (0.005-0.15/100 000) 4540-143 010 DALYs (3-102/100 000)	Europe	Combined from (c) and (d) above (also see Table 10)
Main areas of uncertainty	Potential misclassification of exposure. Exposure-risk relationship derived from etiological studies in Nordic countries and the United States, may not be generalized to all countries in Europe. Household mould exposure varies considerably by housing conditions in different countries, hence wide range in EBD assessment			
Main implications	Reducing mould exposure in dwellings would contribute to significantly lower asthma DALYs.			

Table 12. Summary of EBD of asthma onset in children caused by “dampness” in the home environment

Housing exposure	Indicators of “dampness”			
Health outcome	Asthma deaths and DALYs in children			
Summary of EBD assessment	About 15% of new childhood asthma in Europe can be attributed to indoor dampness, which represents approximately 69 462 potentially avoidable DALYs and 103 potentially avoidable deaths per year.			
	Level	**Range**	**Geographic scope**	**Source of information**
(a) Exposure risk relationship	RR=2.2	1.3-4.0	Nordic countries, United States	Cohort and case-control studies with largely consistent findings (Pekkanen et al., 2007)
(b) Exposure assessment	15%	10-25%	Europe	Multiple cross-sectional and some cohortstudies of damp and mould
(c) PAF	15.3%	4.3-31.0%	Europe	Derived from (a) and (b)

(d) Total burden of disease	Fatalities= 675 DALYs = 454 000	- -	Europe	WHO Global BOD Report (WHO, 2008)
(e) EBD from exposure	103 deaths (0.07/100 000) 69 462 DALYs (50/100 000)	29-209 deaths (0.02-0.15/100 000) 19 522-140 740 (14-100/100 000)	Europe	Combined from (c) and (d) above (also see Table 10)
Main areas of uncertainty	Potential misclassification of exposure. Exposure-risk relationship derived from etiological studies in Nordic countries and the United States, may not be generalized to all countries in Europe. Household dampness exposure varies considerably by exposure measures used and housing conditions in different countries, hence wide range in EBD assessment.			
Main implications	Reducing dampness exposure in dwellings would contribute to significantly lower asthma DALYs.			

9. Policy implications and interventions

9.1 Interventions

The published intervention studies on remediation of indoor mould problems have mainly shown positive results on the occupants' respiratory health. Effects of remediation have decreased symptom rates among adults (Jarvis, Morey, 2001; Åhman et al., 2000; Sudakin et al., 1998) and decreased peak expiratory flow variability (a measure of asthma severity) (Ebbehoj et al., 2002). An insulation improvement of existing houses in New Zealand led to a drier and warmer indoor environment and improved self-rated health, decreased days off school or work, decreased visits to a general practitioner, and resulted in fewer hospital admissions for respiratory conditions (Howden-Chapman et al., 2007). Complete remediation of a mould-damaged school decreased the symptom prevalences of schoolchildren, while in another school that was only partially remediated the decrease of symptoms was not evident (Meklin et al., 2005). A review of case studies for both dampness and mould prevention and remediation approaches also identified that remediation measures are effective in reducing dampness and removing mould growth if adequate action, considering the root causes of the problem, is taken (WHO, 2010).

The main aim of remediation of mould problems in a building should be elimination of the exposure, i.e., removing the mould and replacing the contaminated materials. The cessation of mould exposure by removing the children to a non-mouldy environment has improved children's health, shown as a decrease in infections (Koskinen et al., 1995). However, it is not clear whether remediation measures can eliminate all exposing factors, as some of the schoolchildren continued to have symptoms even after comprehensive remediation measures of their schools (Haverinen-Shaughnessy et al., 2004; Rudblad et al., 2002). Do even very small amounts of remaining exposure have adverse effects on those who already had developed asthma or other respiratory conditions related to mould/dampness exposure? Are mould-induced asthma and other respiratory conditions irreversible once they have developed? From a public health perspective, these considerations place more emphasis on primary prevention, i.e. prevention of the development of disease.

There are relatively few data specifically on the effect of remediation of indoor mould problems on children's asthma. A recent intervention study on asthma exacerbation involving comprehensive removal of both dampness and mould in houses with symptomatic asthmatic children showed substantial improvement in asthma exacerbations and reduction in asthma symptoms (Kercsmar et al., 2006). A Swedish study observed the renovation of a day-care centre with indoor mould problems (Rylander, 1997). Renovation was shown to reduce

microbial exposure and this was accompanied by a slight improvement in bronchial hyperresponsiveness in the day-care nurses.

There are various methodological constraints related to intervention studies, originating from the large variation and quality in how the remediation is done, how the success of the remediation is evaluated, and how the process is handled as a whole (Haverinen-Shaughnessy et al., 2008). However, based on the literature reviewed it can be concluded that cessation of exposure to indoor moulds results in improvement of mould/dampness-induced asthma and that by remediation development of new cases of asthma can be prevented in both children and adults. More high-quality intervention studies are needed to fully demonstrate this.

9.2 Policy relevance

As shown in this EBD assessment, a significant portion of childhood asthma can be attributed to indoor mould and dampness. Asthma is in most developed countries the most significant chronic disease among children. Such health effects lead to increased morbidity, use of health services, increased absenteeism from school (and work), and decreased quality of life. A recent assessment from the United States estimated that their annual national cost of asthma that is attributable to dampness and mould exposure in the home is 3.5 billion US dollars (2.1-4.8 billion) (Mudarri, Fisk, 2007). Therefore, preventing and remediating such exposures would have great beneficial impact on public health and national economies, as well as prevent a considerable proportion of the suffering associated with asthma in children and probably also in adolescents and adults. Recently an expert review concluded that there is evidence that an intervention including combined elimination of moisture intrusion and leaks and removal of mould-contaminated items is effective for reducing symptoms from asthma (Krieger et al., 2010).

Exposure to dampness and mould always originates from problems in the building, its construction, use and functions, or maintenance. The causes of dampness or moisture damage may be traced down to failures in design, construction, use or maintenance of the building, or they may result from acute incidents such as storms, floods, or plumbing failures (Bornehag et al., 2001; Jaakkola, Jaakkola, 2004; IOM, 2004; Haverinen-Shaughnessy et al., 2006). Ventilation also plays a role in controlling the moisture conditions in the building. Most of the dampness, moisture and mould problems could be prevented by high-quality design and construction of buildings including their surroundings, and continuous maintenance and prompt remediation measures taken at any signs of indoor dampness or mould. Unfortunately, this connection between housing quality and health seems not yet be fully recognized by all the relevant stakeholders; thus, raising the awareness of the health effects related to indoor mould and dampness and potential preventive actions should be one of the housing-related priorities in Europe.

In addition to these relevant health effects, dampness and associated microbial growth are also harmful for the building. As an extreme situation, rot fungi, also resulting from prolonged dampness problems, may destroy the whole building. Therefore, in addition to the significant public health impact that indoor dampness and mould problems have, there are also economical and building-technical dimensions. It would therefore be beneficial from many points of view to prevent indoor dampness and mould problems from developing, and to repair any moisture damage or dampness at an early stage.

9.3 Recommendations for prevention of the harmful effects of indoor mould and dampness problems

Awareness of the harmful effects of indoor mould and dampness on occupants' respiratory health and the condition of the building should be raised by providing information to designers,

building and maintenance professionals, society and urban planning, environmental and health care professionals, as well as the general public.

Health care professionals should be educated about the link between home dampness and mould problems and asthma, and they should be encouraged to include questions on such exposures and give advise concerning remediation of such problems when investigating and treating asthma patients.

Risk of development of dampness and mould problems should be minimised by good design, construction and maintenance practices of buildings.

Importance of adequate insulation, ventilation, and moisture control for prevention of dampness and mould should be included in housing improvement programs.

10. References

Andersson MA et al. (1998). The mitochondrial toxin produced by Streptomyces griseus strains isolated from an indoor environment is valinomycin. *Applied and Environmental Biology*, 64:4767-4773.

Andriessen JW, Brunekreef B, Roemer W (1998). Home dampness and respiratory health status in European children. *Clinical & Experimental Allergy*, 28:1191-1200.

Arundel AV et al. (1986). Indirect health effects of relative humidity in indoor environments. *Environmental Health Perspectives*, 65:351-361.

Asher MI et al. (1998). Worldwide variations in the prevalence of asthma symptoms: International Study of Asthma and Allergies in Childhood (ISAAC). *European Respiratory Journal*, 12:315-335.

Asher MI et al. (2006). Worldwide time trends in the prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and eczema in childhood: ISAAC Phases One and Three repeat multicountry cross-sectional surveys. *Lancet*, 368:733-743.

Baker D, Henderson J (1999). Differences between infants and adults in the social aetiology of wheeze. The ALSPAC Study Team. Avon Longitudinal Study of Pregnancy and Childhood. *Journal of Epidemiology and Community Health*, 53:636-642.

Bayram I et al. (2004). The prevalence of asthma and allergic diseases in children of school age in Adana in southern Turkey. *The Turkish Journal of Pediatrics*, 46:221-225.

Belanger K et al. (2003). Symptoms of Wheeze and Persistent Cough in the First Year of Life: Associations with Indoor Allergens, Air Contaminants, and Maternal History of Asthma. *American Journal of Epidemiology*, 158:195-202.

Bible. *Leviticus Chapter 14*. Versus 34-45.

Black PN, Udy AA, Brodie SM (2000). Sensitivity to fungal allergens is a risk factor for life-threatening asthma. *Allergy*, 55:501-504.

Bloom E et al. (2007). Mass Spectrometry-Based Strategy for Direct Detection and Quantification of Some Mycotoxins Produced by Stachybotrys and Aspergillus spp. in Indoor Environments. *Applied and Environmental Biology*, 73:4211-4217.

Bouillard LA, Devleeschouwer MJ, Michel O (2006). Characteristics of the home bacterial contamination and endotoxin related release. *Journal de Pharmacie de Belgique*, 61:63-66.

Bornehag et al. (2001). Dampness in buildings and health. Nordic interdisciplinary review of the scientific evidence on associations between exposure to "dampness" in buildings and health effects (NORDDAMP). *Indoor Air*, 11:72-86.

Bornehag CG et al. (2004). Dampness in buildings as a risk factor for health effects, EUROEXPO: a multidisciplinary review of literature (1998-2000) on dampness and mite exposure in buildings and health effects. *Indoor Air*, 14:243-257.

Bornehag CG et al. (2005). 'Dampness' at home and its association with airway, nose, and skin symptoms among 10,851 preschoolchildren in Sweden: a cross-sectional study. *Indoor Air,* 10:48-55.

Brasche S et al. (2003). Prevalence, causes, and health aspects of dampness and mold in homes. Results of a population-based study in Germany. *Bundesgesundheitsblatt – Gesundheitsforschung – Gesundheitsschutz,* 46:683-693.

Brunekreef B et al. (1989). Home dampness and respiratory morbidity in children. *The American review of respiratory disease*, 140:1363-1367.

Centers for Disease Control and Prevention (1994). Acute pulmonary hemorrhage/hemosiderosis among infants – Cleveland, Ohio, 1993-1996. *Morbidity and Mortality Weekly Report*, 43:881-883.

Centers for Disease Control and Prevention (1997). Update: pulmonary hemorrhage/hemosiderosis among infants – Cleveland, Ohio, 1993-1996. *Morbidity and Mortality Weekly Report*, 46:33-35.

Centers for Disease Control and Prevention (2000). Update: pulmonary hemorrhage/hemosiderosis among infants – Cleveland, Ohio, 1993-1996. *Morbidity and Mortality Weekly Report*, 49:180-185.

Charpin-Kadouch C et al. (2006). Mycotoxin identification in moldy dwellings. *Journal of Applied Toxicology*, 26:475-479.

Chelelgo J et al. (2001). Analysis of moisture findings in the interior spaces of Finnish housing stock. *Air and Waste Management Association*, 51:69-77.

Chen CM et al. (2007). Social factors, allergen, endotoxin, and dust mass in mattress. *Indoor Air*, 17:384-393.

Chew GL et al. (2001). Fungal extracellular polysaccharides, beta (1–>3)-glucans and culturable fungi in repeated sampling of house dust. *Indoor Air*, 11:171-178.

Cho SH et al. (2005). Aerodynamic characteristics and respiratory deposition of fungal fragments. *Atmospheric Environment*, 39:5454-5465.

Cho SH et al. (2006). Mold damage in homes and wheezing in infants. *Annals of Allergy, Asthma & Immunology*, 97:539-545.

Claeson AS, Sandstrom M, Sunesson AL (2007). Volatile organic compounds (VOCs) emitted from materials collected from buildings affected by microorganisms. *Journal of Environmental Monitoring,* 9:240-245.

Dales RE, Miller D, McMullen E (1997). Indoor air quality and health: validity and determinants of reported home dampness and moulds. *International Journal of Epidemiology*, 26:120-125.

Douwes J (2005). (1→3)-ß-D-glucans and respiratory health: a review of the scientific evidence. *Indoor Air*, 15:160-169.

Douwes J, Pearce N, Heederik D (2002). Does environmental endotoxin exposure prevent asthma? *Thorax*, 57:86-90.

Douwes J et al. (1996). Measurement of ß(1→3)-glucans in the occupational and home environment with an inhibition enzyme immunoassay. *Applied and Environmental Biology*, 62:3176-3182.

Douwes J et al. (1998). Endotoxin and ß(1→3)-glucan in house dust and the relation with home characteristics: A pilot study in 25 german houses. *Indoor Air,* 8:255-263.

Douwes J et al. (1999). Fungal extracellular polysaccharides in house dust as a marker for exposure to fungi: relations with culturable fungi, reported home dampness, and respiratory symptoms. *Journal of Allergy and Clinical Immunology*, 103:494-500.

Douwes J et al. (2000). (1→3)-ß-D-glucan and endotoxin in house dust and peak flow variability in children. *American Journal of Critical Care Medicine*, 162:1348-1354.

Ebbehoj N et al. (2002). Building-related symptoms and mold: two step intervention study. *Indoor Air*, 12:273-277.

Eduard W (2006). Fungal spores. The Nordic Expert Group for Criteria Documentation of Health Risk from Chemicals. *Arbete och Hälsa*, 21:1-145.

Engvall K, Norrby C, Norback D (2001). Asthma symptoms in relation to building dampness and odour in older multifamily houses in Stockholm. *International Journal of Tuberculosis and Lung Disease*, 5:468-477.

Etzel R et al. (1998). Acute pulmonary hemorrhage in infants associated with exposure to Stachybotrys atra and other fungi. *Archives of Pediatrics & Adolescent Medicine*, 152:757-762.

Eurostat (2001). *Housing problems by socio-economic status*. Data from the European Community Household Panel (ECHP). (http://epp.eurostat.ec.europa.eu/portal/page/portal/microdata/echp, accessed 15 May 2009)

Eurostat (2007). *Housing problems by socio-economic status*. Data from the Statistics on income, social inclusion and living conditions survey (SILC). (http://www.euro.who.int/__data/assets/pdf_file/0005/97007/enhis_factsheet09_3_5.pdf, accessed 15 May 2009)

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*, 17:284-296.

Flannigan B, Samson RA, Miller JD (2001). *Microorganisms in home and indoor work environments*. New York, CRC Press.

Freeman NC, Schneider D, McGarvey P (2003). Household exposure factors, asthma, and school absenteeism in a predominantly Hispanic community. *Journal of Exposure Analysis and Environmental Epidemiology*, 13:169-176.

Gallup J et al. (1987). Indoor mold spore exposure: characteristics of 127 homes in southern California with endogenous mold problems. *Experientia Supplement*, 51:139-142.

Gehring, U (2001). ß(1→3)-glucan in house dust of German homes related to culturable mold spore counts, housing and occupant characteristics. *Environmental Health Perspectives*, 109:139-144.

GINA (2006). *Global Strategy for Asthma Management and Prevention. Global Initiative for Asthma*. (http://www.ginasthma.org, accessed 28.August 2009)).

Giovannangelo M et al. (2007). Determinants of house dust endotoxin in three European countries – the AIRALLERG study. *Indoor Air*, 17:70-79.

Godish T, Rouch J (1986): Mitigation of residential formaldehyde contamination by indoor climate control. *American Industrial Hygiene Association Journal*, 47:792-797.

Gorny RL (2004). Filamentous microorganisms and their fragments in indoor air–a review. *Annals Of Agricultural And Environmental Medicine*, 11:185-197.

Gorny RL, Dutkiewicz J, Krysinska-Traczyk E (1999). Size distribution of bacterial and fungal bioaerosols in indoor air. *Annals Of Agricultural And Environmental Medicine*, 6:105-113.

Gorny RL et al. (2002). Fungal fragments as indoor air biocontaminants. *Applied and Environmental Biology,* 68:3522-3531.

Green BJ (2006). Airborne fungal fragments and allergenicity. *Medical Mycology,* 44:245-255.

Gunnbjörnsdottir MI (2006). Prevalence and incidence of resporatory symptoms in relation to indoor dampness: the RHINE study. *Thorax*, 61:221-225.

Halonen M et al. (1997). Alternaria as a major allergen for asthma in children raised in a desert environment. *American Journal of Respiratory and Critical Care Medicine,* 155:1356-1361.

Haverinen-Shaughnessy U et al. (2004). Estimating effects of moisture damage repairs on students' health – a long-term intervention study. *Journal of Exposure Analysis and Environmental Epidemiology,* 14:58-64.

Haverinen-Shaughnessy U et al. (2005). Occurrence and Characteristics of Moisture Damage in Residential Buildings as a Function of Occupant and Engineer Observations. *Indoor and Built Environment,* 14:133–140.

Haverinen-Shaughnessy U et al. (2006). Children's homes – determinants of moisture damage and asthma in Finnish residences. *Indoor Air*, 1:248-255.

Haverinen-Shaughnessy U et al. (2008). Monitoring success of remediation: Seven case studies of moisture and mold damaged buildings. *Science of The Total Environment,* 399:19-27.

Hirvonen MR et al. (2001). Steptomyces anulatus induced inflammatory responses and cytotoxicity in macrophages are regulated by growth conditions of the microbe. *Inhalation Toxicology,* 13:55-68.

Howden-Chapman P et al. (2007). Effect of insulating existing houses on health inequality: cluster randomised study in the community. *British Medical Journal*, 334:434-435.

Husman T. (1996). Health effects of indoor-air micro organisms. *Scandinavian Journal of Work, Environment & Health,* 22:5-13.

Huttunen K et al. (2000). Inflammatory responses in RAW264.7 macrophages caused by mycobacteria isolated from moldy houses. *Environmental Toxicology and Pharmacology,* 8:237-244.

Huttunen K et al. (2001). Comparison of mycobacteria-induced cytotoxicity and inflammatory responses in human and mouse cell lines. *Inhalation Toxicology*, 13:977-991.

Hyvärinen A, Meklin T, Vepsäläinen A (2002). Nevalainen A. Fungi and actinobacteria in moisture-damaged building materials – concentrations and diversity. *International Biodeterioration & Biodegradation*, 49:27-37.

Institute of Medicine (IOM) (2004). *Damp Indoor Spaces and Health*. Washington D.C., The National Academies Press.

Jaakkola MS, Jaakkola JJK (2004). Indoor molds and asthma in adults. *Advances in Applied Microbiology*, 55:309-338.

Jaakkola JJK, Hwang B-F, Jaakkola N (2005). Home dampness and molds, parental atopy, and asthma in childhood: A six-year population-based cohort study. *Environmental Health Perspectives*, 113:357-361.

Jaakkola JJK, Jaakkola N, Ruotsalainen R (1993). Home dampness and molds as determinants of respiratory symptoms and asthma in pre-school children. *Journal of Exposure Analysis and Environmental Epidemiology*, 3*:*129-142.

Jaakkola MS et al. (2002). Indoor dampness and molds and development of adult-onset asthma: A population-based incident case-control study. *Environmental Health Perspectives*; 110:543-547.

Jarvis J, Morey P (2001). Allergic respiratory disease and fungal remediation in a building in a subtropical climate. *Applied Occupational and Environmental Hygiene*, 64:660-667.

Kercsmar CM et al. (2006). Reduction in asthma morbidity in children as a result of home remediation aimed at moisture sources. *Environmental Health Perspectives,* 114:1574-1580.

Kilpeläinen M et al. (2001). Home dampness, current allergic diseases, and respiratory infections among young adults. *Thorax*, 56:462-467.

Koskinen O et al. (1995). Respiratory symptoms and infections among children in a day care center with mold problems. *Indoor Air,* 5:3-9.

Krieger J et al. (2010). Housing interventions and control of asthma-related indoor biologic agents: A review of the evidence. *Journal of Public Health Management and Practice,* 16: S11-S20*.*

Lee YL et al. (2003). Indoor and outdoor environmental exposures, parental atopy, and physician-diagnosed asthma in Taiwanese schoolchildren. *Pediatrics*, 112:e389.

Meklin T et al. (2005). The effects of moisture damage repairs on microbial exposure and symptoms in schoolchildren. *Indoor Air,* 15:40-47.

Mudarri D, Fisk WJ (2007). Public health and economic impact of dampness and mold. *Indoor Air*, 17:226-235.

Murtoniemi T et al. (2001). Production of inflammatory mediators in RAW264.7 macrophages by microbes grown on six different plasterboards. *Inhalation Toxicology*, 13:233-247.

Murtoniemi T et al. (2003). The relation between growth of four microbes on six different plasterboards and biological activity of spores. *Indoor Air*, 13:65-73.

Nafstad P et al. (1998). Residential dampness problems and symptoms and signs of bronchial obstruction in young Norwegian children. *American Journal of Critical Care Medicine,* 157:410-414.

Nevalainen A et al. (1998). Prevalence of moisture problems in Finnish houses. *Indoor Air*, 4:45-49.

Norback D et al. (1999). Current asthma and biochemical signs of inflammation in relation to building dampness in dwellings*. International Journal of Tuberculosis and Lung Disease,* 3:368-376.

Norbäck D et al. (2000). Asthma symptoms in relation to measured building dampness in upper concrete floor construction, and 2-ethyl-1-hexanol in indoor air. *International Journal of Tuberculosis and Lung Disease,* 4:1016-1025.

Øie L et al. (1999). Ventilation in homes and bronchial obstruction in young children. *Epidemiology,* 10:294-299.

Park JH et al. (2000). Longitudinal study of dust and airborne endotoxin in the home. *Environmental Health Perspectives,* 108:1023-1028.

Park JH et al. (2008). Hydrophilic fungi and ergosterol associated with respiratory illness in a water-damaged building. *Environmental Health Perspectives*, 116:45-50.

Pearce N, Beasley R, Pekkanen J (2000). Role of bronchial responsiveness testing in asthma prevalence surveys. *Thorax,* 55:352-354.

Pearce N et al. (1998). *Asthma epidemiology: principles and methods*. New York, Oxford University Press.

Pearce N et al. (2000). Comparison of asthma prevalence in the ISAAC and the ECRHS. ISAAC Steering Committee and the European Community Respiratory Health Survey. International Study of Asthma and Allergies in Childhood. *European Respiratory Journal*, 16:420-426.

Peat JK, Dickerson J, Li J (1998). Effects of damp and mould in the home on respiratory health: a review of the literature. *Allergy*, 53:120-128.

Pekkanen J et al. (2007). Moisture damage and childhood asthma: a population-based incident case-control study. *European Respiratory Journal,* 29:509-515.

du Prel X, Krämer U, Ranft U (2005). Changes in social inequality with respect to health-related living conditions of 6-year-old children in East Germany after re-unification. *BMC Public Health*, 5:64.

Rintala H, Nevalainen A, Suutari M (2002). Diversity of streptomycetes in water-damaged building materials based on 16S rDNA sequences. *Letters in Applied Microbiology*, 34:439-443.

Rintala H et al. (2004). Detection of streptomycetes in house dust – comparison of culture and PCR methods. *Indoor Air*, 14:112-119.

Roponen M et al. (2001). Differences in inflammatory responses and cytotoxicity in RAW264.7 macrophages induced by Streptomyces anulatus grown on different building materials. *Indoor Air*, 11:179-184.

Rudblad S et al. (2002). Slowly decreasing mucosal hyperreactivity years after working in a school with moisture problems. *Indoor Air*, 12:138-144.

Ruotsalainen R, Jaakkola N, Jaakkola JJK (1995). Dampness and molds in day-care centers as an occupational health problem. *International Archives of Occupational and Environmental Health,* 66:369-374.

Rylander R (1997). Airborne (1->3)-ß-D-glucan and airway disease in a day-care center before and after renovation. *Archives of Environmental Health,* 52:281-285.

Rylander R (1999). Indoor air-related effects and airborne (1→3)-ß-D-glucan. *Environmental Health Perspectives,* 107:501-503.

Rönmark E et al. (1999). Different pattern of risk factors for atopic and nonatopic asthma among children–report from the Obstructive Lung Disease in Northern Sweden Study. *Allergy*, 54:926-935.

Salo PM et al. (2004). Indoor allergens, asthma, and asthma-related symptoms among adolescents in Wuhan, China. *Annals of Epidemiology,* 14:543-550.

Simoni M et al. (2005). SIDRIA-2 Collaborative Group.Mould/dampness exposure at home is associated with respiratory disorders in Italian children and adolescents: the SIDRIA-2 Study. *Occupational and Environmental Heath,* 62:616-622.

Solomon GM et al. (2006). Airborne mold and endotoxin concentrations in New Orleans, Louisiana, after flooding, October through November 2005. *Environmental Health Perspectives,* 114:1381-1386.

Spengler JD et al. (2004). Housing characteristics and children's respiratory health in the Russian Federation. *American Journal of Public Health,* 94:657-662.

Sudakin DL (1998). Toxigenic fungi in water damaged building: An intervention study. *American Journal of Industrial Medicine,* 34:183-190.

Tamay Z et al. (2007). Prevalence and risk factors for allergic rhinitis in primary school children. *International Journal of Pediatric Otorhinolaryngology,* 71:463-471.

Tham KW et al. (2007). Associations between home dampness and presence of molds with asthma and allergic symptoms among young children in the tropics. *Pediatric Allergy and Immunology,* 18:418-424.

Torvinen E et al. (2006). Mycobacteria and fungi in moisture-damaged building materials. *Applied and Environmental Biology,* 72:6822-6824.

Tuomainen A, Seuri M, Sieppi A (2004). Indoor air quality and health problems associated with damp floor coverings. *International Archives of Occupational and Environmental Health,* 77:222-226.

Turunen M et al. (2008). *Asuinympäristön laatu,terveys ja turvallisuus – internetpohjainen tiedonkeruu- ja palautejärjestelmä* (ALTTI). [Building environmental quality, health and safety – an internet based data collection and response system]. Publications of National Public Health Institute B8.

Verhoeff AP et al. (1994). Fungal propagules in house dust. II. Relation with residential characteristics and respiratory symptoms. *Allergy*, 49:540-547.

Verhoeff AP et al. (1995). Damp housing and childhood respiratory symptoms: the role of sensitization to dust mites and molds. *American Journal of Epidemiology*, 141:103-110.

Waegemaekers M et al. (1989). Respiratory symptoms in damp homes. A pilot study. *Allergy*, 44:192-198.

WHO (2007). *Large Analysis and Review of European housing and health Status. Preliminary overview*. Copenhagen, WHO Regional Office for Europe. (http://www.euro.who.int/__data/assets/pdf_file/0007/107476/lares_result.pdf, accessed 12 September 2009).

WHO (2008). *The Global Burden Of Diseases. 2004 Update*. Geneva, World Health Organization. (http://www.who.int/healthinfo/global_burden_disease/GBD_report_2004update_full.pdf, accessed 12 September 2009).

WHO (2009). *Guidelines for Indoor Air Quality: Dampness and Mould*. Copenhagen, WHO Regional Office for Europe. (http://www.euro.who.int/__data/assets/pdf_file/0017/43325/E92645.pdf, accessed 12 September 2009).

WHO (2010). *Technical and policy recommendations to reduce health risks due to dampness and mould*. Copenhagen, WHO Regional Office for Europe. (http://www.euro.who.int/__data/assets/pdf_file/0015/121425/E92998.pdf, accessed 12 September 2009).

Wickman M et al. (2003). Strategies for preventing wheezing and asthma in small children. *Allergy*, 58:742-747.

Zheng T et al. (2002). Childhood asthma in Beijing, China: a population-based case-control study. *American Journal of Epidemiology,* 156:977-983.

Housing conditions and home injury

Michael D. Keall, David Ormandy, Michael G. Baker

1. Introduction

Injuries have been defined (Baker et al., 1992) as:

> … acute exposure to physical agents such as mechanical energy, heat, electricity, chemicals, and ionizing radiation interacting with the body in amounts or at rates that exceed the threshold of human tolerance. In some cases (for example, drowning and frostbite), injuries result from the sudden lack of essential agents such as oxygen or heat.

Injuries therefore include burns, poisonings, ingestion of foreign objects, and fire-related injuries (including death from smoke inhalation), as well as drownings, falls, cuts and collisions with objects.

Injuries in the home present an important health burden worldwide. In Europe, almost 110 000 people die each year as a result of a home/leisure injury and an estimated 32 000 000 are hospitalised (Kuratorium für Verkehrssicherheit, 2007). The 2003-2005 home/leisure fatal injury rate is 22/100 000 over all Europe, which is more than twice the rate of road fatalities (10/100 000), and varies between a minimum of 12/100 000 in Ireland to a maximum of 72/100 000 in Latvia and Estonia (Kuratorium für Verkehrssicherheit, 2007). The injury burden is particularly important for children: in Europe, home injury deaths are highest in children under 5 years of age and then sharply decrease, in contrast to road traffic deaths, which increase with age (Sengoelge et al., 2010).

This chapter defines the housing-related burden of injury as related to those characteristics that can be improved through different building design, construction, or maintenance. Thus, the mere presence of a stairway in itself is not a housing-related injury source, because stairs are often necessitated by the space available for a house and may not be replaceable by lifts. However, a stairway that is excessively steep or that does not have safety features such as handrails does contribute to the injury burden of housing.

Over time, the application of building science has led to improvements in the design of housing features, such as ergonomic studies of stair design, with likely positive implications for safety that are difficult to quantify (Department for Communities and Local Government, 2008). Recent reviews of studies of the safety effects of housing improvements (Lyons et al., 2006; Kerr, 2007; Jacobs, Baeder, 2009) have identified a few discrete areas where sufficient evidence exists to estimate the burden of injury associated with the home. Studies that show associations between injury hazards (or lack of safety facilities) and the occurrence of injury are described below and their results are used to estimate the injury burden of housing.

This chapter uses data on the burden of home injury in the WHO European Region to estimate the impact of two modifiable features of housing on injury incidence, deaths, and disability adjusted life years (DALYs) from fire and falls. While this approach is likely to underestimate the true burden of home injury, the data sources are more robust, leading to a higher degree of certainty in the final estimate. The range of housing conditions considered is limited by the exposure data that are available and by gaps in the literature on the exposure-response relationship for many exposures. Consequently, this analysis has been restricted to two injury-hazard combinations: child (aged<15) deaths and DALYS from falls from second level or higher windows without window guards; deaths and DALYs due to domestic fires associated

with lack of smoke detectors. This paper must therefore be seen as only a first step in quantifying the injury burden associated with housing. This burden is likely to be considerably larger when limitations can be addressed in the quality of the evidence, the range of exposures measured, and the lack of relevant detail of injury circumstances currently recorded in surveillance systems.

2. Summary of the method

This analysis used the standard method for estimating the Environmental Burden of Disease (EBD), as described in the introductory chapter.

The population attributable fraction (PAF) is the proportion of disease that can be ascribed to a specified risk factor. In this context, PAF represents the proportion of injury in a population that would be prevented if exposure to remediable housing injury hazards were removed from the entire population. In this analysis we have used the following univariate formula for calculating PAF:

$$PAF = \frac{p(RR-1)}{p(RR-1)+1}$$

Where p = proportion of the population exposed, and RR is the relative risk for the condition in those exposed.

The PAF is then applied to the total burden of home injury in the WHO European Region, to estimate the proportion of cases, deaths and Disability Adjusted Life Years (DALYs) that is associated with specific inadequate housing conditions.

Estimating this EBD therefore relies on the following three sources of data:

Exposure-risk relationship – Obtained from a structured review of the literature on injury in the home and housing conditions

Exposure assessment – From existing estimates and scenarios. Where housing exposures cannot be estimated, we use a scenario-based approach, as outlined in the WHO report "Methodology for assessment of Environmental burden of disease" (Kay et al., 2000).

Total burden of disease – Obtained from previous WHO global burden of disease estimates based on injury reporting by states in the WHO European Region as well as the European Injury Database (European Home and Leisure Accident Surveillance System, 2009), which contains detailed injury data reported from a sample of hospitals.

3. Exposure-risk relationship for housing conditions and injury in the home

To produce an estimate of the relative risk of injury in the home and housing conditions, we reviewed studies that have investigated this risk and provided a quantified relative risk estimate. We used recent summaries of the literature as a means to identify studies and measure exposure-risk associations. One recent review (Jacobs, Baeder, 2009) evaluated evidence of those housing interventions that have sufficient evidence for implementation, require further field investigation, need further investigation, or have been shown to be ineffective (Table 1).

Table 1: Assessment of quality of evidence for links between housing exposures and injury

Sufficient Evidence	Needs More Field Evaluation	Needs Formative Research	No Evidence or Ineffective
• Installation of working smoke alarms • Isolation 4-sided pool fencing • Pre-set safe temperature hot water heaters	• Fall prevention by handrails, grab bars, stair-gates, window guards and improved lighting • Temperature-controlled water faucets • Safe ignition sources • Home modification to escape fires • Air conditioning during heat waves	• Ignition source controls • Escape exit signage • Improved smoke alarm and faucet design • Behaviour modification to escape fires • Automatic fire sprinkler systems for housing • Pool covers and alarms • Bathtub design to reduce falls • Stove and stove control design to prevent burns • Carbon monoxide exposure prevention through design and engineering • Improved enforcement of building and housing codes • Noise reduction	• 3-sided pool fencing

Source: Jacobs, Baeder, 2009

The main relevant studies that produced a quantitative relative risk or odds ratio are tabulated in Table 2.

There are other observational studies looking at the epidemiology of injuries that provide some insight into potentially effective measures. One such study of children under 15 years of age who were injured in falls from heights associated with buildings in Dallas, Texas, found that in more than two thirds of the balcony related falls, the child fell from between widely-spaced balcony rails (more than 10 cm apart). Clearly, more closely spaced balcony rails will enhance the safety of balconies, although such improvement in safety has not been quantified. A WHO expert meeting identified several priority features for reducing child injury: fire detectors, fencing of water, window catches and restrictors, adequate balustrades on balconies, restrictors of cupboard doors, handrails, thermostat mixers, socket protectors, fixing steps and stairs, carbon monoxide (CO) detectors, safe doorsteps/thresholds, automatic garage doors, and safe design of kitchens (World Health Organization, 2005).

There is also likely to be an injury burden attributable to many other home injury hazards, (such as inadequate handrails for steps, poor lighting, slippery surfaces, poor ergonomics), but statistically significant associations between such hazards and injury occurrence have only been shown in one observational study, to our knowledge (Keall et al., 2008).

Table 2: Summary of studies investigating the relationship between home injury hazard exposure and home injury

Reference	Study design, location, time period	Study population	Exposure measure	Outcome measure	Results (RR/OR, 95% CI)
Spiegel, Lindaman, 1977	Intervention study with subjects serving as their own controls	Children aged <15	Before and after programme (education, provision of free window guards)	Hospitalised fall injuries	50% reduction in falls, no confidence interval provided
Ahrens, 2008	Cross-sectional study, United States, 2000-2004	United States	Presence of smoke alarms	Deaths from home fires	Death rate twice as high in homes without working smoke alarms
Thompson, Rivara, 1998	Systematic review	Children 14 years of age or younger who are exposed to swimming pools	Provision of fencing on all sides of swimming pool**	Deaths or near deaths by drowning	0.27 (95% CI 0.16 to 0.47) for fenced vs unfenced swimming pool
Kendrick et al., 2008	Systematic review and meta-analysis	Children	Fall safety features in homes	Injury due to falls	No evidence of fall prevention effectiveness
Erdmann et al., 1991	Cross-sectional study, Washington, United States, 1969-1988	Children 14 years of age or younger	Legislation to preset new hot water heaters to 49 degrees. Rental homes hot water set to 49 degrees C and information package about dangers of hot water scalds provided to all homes.	Hospitalization for scalds from tap water	After 5 years, 11 degree reduction in mean temperature from 61 to 50 in homes with new hot water systems; drop in rate of hospitalization from 5.5 to 2.4 per annum.
Naughton et al., 2002	Case-control study, Chicago, United States, 1999	Population (typically affects elderly and children <1)	Presence of working air conditioner	Heat-related deaths during heat wave*	0.2 (0.1–0.7)

* See also Vandentorren et al. (2006) for analysis of risk factors in August 2003 heat wave in France

**Fencing on all sides is considerably more effective than fencing on three sides (Western Australia Health Department, 1988)

4. Exposure Assessment

The second component of the burden of disease calculation is an assessment of exposures to particular modifiable housing conditions that are implicated in home injury.

4.1 Method for measuring children's exposure to home fall hazards

Five European countries (Greece, Norway, Poland, Scotland and Sweden) are reported to have a national law requiring guards to prevent children from falling out of windows of height more than one storey/level (MacKay, Vincenten, 2007). Apart from the existence of laws, there is little or no basis on which to assess children's exposure to such fall hazards as no representative

surveys appear to be conducted at this time. Instead, we have used hospitalization and WHO data to determine exposures (see section 5).

4.2 Method for measuring exposure to excessively hot tap water

Five European countries reported that they have a national law requiring a maximum pre-set temperature for all water heaters or tap water in domestic settings. France's setting is the lowest, at 50°C (MacKay, Vincenten, 2007). In 2003, the French Consumer Safety Commission required that all new or renovated installations of domestic hot water produce water at a maximum temperature of 50°C for taps outside the kitchen (Consumer Safety Commission, 2003). For existing hot water systems, the Commission encouraged all households, but particularly those with young children, to install temperature-limiting devices, at least for hot water in the bathroom (WHO, 2008). There are some studies that have measured domestic hot water temperatures in the WHO European Region, but with a focus on identifying risk factors for the presence of legionella bacteria in the water. One recent study recorded domestic hot water temperatures in a sample of 450 German houses (Mathys et al., 2008). The temperatures measured had a median of 54°C for instantaneous hot water systems and 50.2°C for other systems. Another study of Italian houses found a mean operating temperature of 53°C (Borella et al., 2004). Recommended hot water temperature settings are often considered a balancing act between the risk of scalds due to excessively hot water on the one hand and on the other, the risk of having water temperatures low enough for legionella bacteria to survive, even though domestic hot water systems have rarely been implicated in cases of Legionnaire's Disease (Health Canada, 2007).

4.3 Methods for measuring exposure to lack of smoke detectors

England has information on the prevalence of domestic smoke detectors from its regular House Condition Survey (Department for Communities and Local Government, 2006). Some estimates for English housing are shown in Table 3.

Table 3: Prevalence of smoke detectors in English dwellings by type of dwelling

	2002		**2006**	
Type of dwelling	n (millions)	Proportion without smoke detectors	n (millions)	Proportion without smoke detectors
Non-bungalow*	19.3	23.6%	19.9	15.6%
Bungalow*	2.0	23.3%	2.0	17.3%
TOTAL	**21.3**	**23.5%**	**22.0**	**15.8%**

Data source: English House Condition Survey, unpublished data

*a bungalow is a single-storied house

LARES (Large Analysis and Review of European housing and health Status) was a WHO survey conducted in 2002/2003 in the eight cities as shown in Table 4. Data showing the presence or absence of smoke detectors were obtained from 3373 dwellings based on a physical inspection (Bonnefoy et al., 2003).

Table 4: Prevalence of homes lacking smoke detectors in some European cities

City (country)	% without smoke detectors*
Angers (France)	88.8
Bonn (Germany)	75.9
Bratislava (Slovakia)	91.2
Budapest (Hungary)	91.4
Ferreira do Alentejo (Portugal)	98.3
Forli (Italy)	95.8
Geneva (Switzerland)	85.4
Vilnius (Lithuania)	95.5
TOTAL	**90.9**

Data source: WHO LARES Survey Database, 2002/03, unpublished data

* excludes "don't know" responses

There are also some estimates available from a German nongovernmental organization web site[3] on domestic smoke detector prevalence in other countries. Although the sources of the estimates are not always specified, the countries listed (with proportion of households without smoke detectors) include: Germany (69%), Sweden (30%), Norway (2%) and the Netherlands (32%). A population weighted average of all these estimates, using the estimates in the previous sentence plus the 2006 United Kingdom estimate shown in Table 3 and the LARES city estimates to represent other respective countries, is 66%. This will be an optimistic estimate of European prevalence of homes without smoke detectors as the larger countries represented here are relatively wealthy and are likely to have higher smoke detector fitment rates than most of the smaller European countries.

4.4 Methods for measuring exposure to pools without pool fencing

Exposure measurements should ideally identify the number of children who live in houses with unfenced and fenced domestic swimming pools. Although such exposure measures are not obtainable for any European country, we do know something about swimming pool fencing legislation in the WHO European Region (only currently existent in France and Sweden) and about the number of swimming pools in France (Table 5). Norway has a law that requires open water on private property to be secured to prevent childhood drowning, but the law does not specify swimming pools (MacKay, Vincenten, 2007). Regulations were introduced in France in January 2004 requiring all new swimming pools to have protective devices. Two years later, all private existing pools were required to have protective devices (Thélot et al., 2008). These devices consist of either:

- A barrier at least 1.10 m high, provided with a gate, preferably with automatic closing;
- An audible alarm;
- A cover.

Note that only the first option has evidence supporting its effectiveness (see Table 1). Table 5 shows data for France on the number of child drownings in private swimming pools together

[3] See http://www.rauchmelder-lebensretter.de/internationaler-vergleich.html

with the number of private swimming pools (Ermanel, Thélot, 2005). In 2005, there were 25 730 000 households in France (Institut National d'Etudes Démographiques, 2007), so about 4% of households had a private swimming pool in 2004. Note that these data are mainly for the period before the 2004 legislation came into effect. If we apply the exposure-risk relationship from Table 2 (Thompson, Rivara, 1998) under the assumption that no pool had fencing, then we would expect that (100%-27%)=73% of the child deaths in private swimming pools would have been saved.

This example, however, highlights some of the uncertainties in attributing burden of disease. First, we do not know the proportion of French swimming pools to which children were exposed that were fenced. Second, the exposure-risk relationship applies to children aged 14 and under (Thompson, Rivara, 1998). Clearly, fencing will be considerably more effective in the case of young children (such as those aged 5 and under. Table 5 shows annual counts of drownings for this age group in French private swimming pools).

Table 5: French data on number of children aged 0-5 drowning in private swimming pools together with number of private swimming pools

Year	Number of deaths of children aged <6 in private swimming pools	Number of private swimming pools
2000	32	708 000
2001	23	773 000
2002	14	854 000
2003	25	928 000
2004	17	1 056 000

Source: Ermanel, Thélot, 2005

4.5 Limitations for examining other injury hazards in housing

England is one of the few countries in the world that carries out a regular survey of private housing quality/conditions, the English House Condition Survey (Department for Communities and Local Government, 2006). The survey carried out in 2005/6 attempted to capture data on particular serious hazards, defined under a framework developed for the English Housing Health and Safety Rating System, which categorises housing conditions that may increase the likelihood of the occurrence of an adverse health event (such as injury) together with the likelihood of health consequences of various levels of severity (Office of the Deputy Prime Minister, 2006). In the future, it may be possible to use these data to estimate population attributable fractions for housing injury hazards in England. The data are not available at this stage, however. The United States also carries out a regular representative survey of housing quality (known as the American Housing Survey), but the data are unlikely to reflect European conditions and are not considered further here.

Poisoning is another important injury class that could potentially be reduced by home features such as safe storage cabinets, but no exposure data (prevalence of safe storage in houses), nor evaluations of the efficacy of safe storage exist currently to inform any estimates of poisoning events potentially prevented. The burden of unintentional poisoning is largely imposed on children, as is well summarized in the following extract from the WHO Europe report on child injury (WHO, 2008):

> Poisoning remains the third leading cause of unintentional injury death; 3000 children and adolescents aged 0–19 years died from acute poisoning in 2004 in the Region. Inequality is substantial in the Region both between countries and within countries. The countries with

> the highest and lowest rates differ by thirtyfold, and 90% of poisoning deaths occur in the low- and middle-income countries in the Region. The home is the most common setting for childhood poisoning, and children are particularly at risk when harmful substances are stored in non-child-proof containers or within easy reach. … If all countries in the Region had the same rate as the country with the lowest rate, then 93% of deaths could be averted. Evidence from countries with low rates shows that modifying the environment, such as child-resistant closures, safe storage, reducing the availability of toxic substances, dispensing medicines in nonlethal quantities and establishing poison control centres, is a good investment in prevention.

A recent survey of parents of young children in 14 European countries found that safe storage of poisons/medicines etc was a commonly cited safety practice amongst these parents (Vincenten et al., 2005).

5. Total burden of disease from certain types of injury in the home

The final component of the burden of disease calculation is data on the burden of disease, obtained from counts of injuries. This section describes the data sources and presents the data in the form of tables within subsections referring to particular injury causes.

5.1 Methods for measuring burden of disease from home injury

For this project, two main data sources were used to estimate injury rates and total numbers:

1) The European Injury Database (IDB) is hosted by the European Commission and reports summarized data from a sample of hospitals in certain European countries (European Home and Leisure Accident Surveillance System, 2009). The injury counts combine routine causes of death statistics, hospital discharge registers and data sources specific to injury areas, including road accidents and accidents at work. An estimate of population rates is also extrapolated in the web-based tables on the assumption that the sampled hospitals represent all hospitals in the country. The number of reporting hospitals is quite small and only a few countries are represented at this stage. Given these limitations, we use the IDB data only to estimate proportions of injuries of particular types in particular settings. These estimated proportions are then applied to the Global Burden of Disease data described below.

2) DALYs for injury and counts of injury deaths have also been calculated for all countries of the WHO European Region as part of 2004 update for the WHO Global Burden of Disease (GBD) project[4]. The burden of disease due to injury is presented according to three classes of injury: child drownings; fires; child falls. These data are combined with estimates of proportions of injuries per setting derived from the IDB, described above, to estimate injury burden associated with the home setting.

[4] See http://www.who.int/healthinfo/bod/en/index.html

5.2 Summary of burden of disease from fires

The burden data for fires in the WHO European Region are shown in Table 6.

Table 6: DALYs, deaths, population and rates for fire-related injuries in European states

Country*	DALYs	Deaths	Population	DALY rate per million	Death rate per million
Albania	1509	15.4	3 134 402	481.3	4.9
Andorra	6	0.2	72 297	78.3	3.2
Armenia	1763	45.3	3 026 880	582.5	14.9
Austria	740	47.1	8 253 381	89.6	5.7
Azerbaijan	10 615	333.3	8 305 938	1278.0	40.1
Belarus	16 573	729.9	9 847 821	1682.9	74.1
Belgium	1855	100.3	10 359 676	179.1	9.7
Bosnia and Herzegovina	910	22.1	3 905 325	233.0	5.7
Bulgaria	2990	109.9	7 794 839	383.6	14.1
Croatia	597	46.2	4 539 880	131.4	10.2
Cyprus	219	7.4	826 813	265.1	9.0
Czech Republic	1544	74.4	10 194 511	151.5	7.3
Denmark	985	65.9	5 402 910	182.3	12.2
Estonia	2546	148.8	1 348 345	1888.2	110.4
Finland	1656	93.4	5 231 166	316.7	17.9
France	10 248	504.1	60 591 139	169.1	8.3
Georgia	1286	61.2	4 516 983	284.7	13.6
Germany	8439	484.0	82 642 615	102.1	5.9
Greece	1313	103.4	11 079 234	118.5	9.3
Hungary	4020	178.4	10 113 272	397.5	17.6
Iceland	55	2.3	292 903	188.2	8.0
Ireland	627	37.2	4 067 737	154.1	9.2
Israel	548	19.1	6 573 979	83.3	2.9
Italy	4682	284.3	58 433 920	80.1	4.9
Kazakhstan	19 095	537.8	15 106 857	1264.0	35.6
Kyrgyzstan	5955	83.8	5 152 508	1155.8	16.3
Latvia	3741	203.6	2 315 283	1615.9	87.9
Lithuania	3352	145.7	3 440 158	974.4	42.4
Luxembourg	52	1.7	452 421	114.1	3.8
Malta	37	2.1	400 124	91.4	5.3
Monaco	1	–	32 394	31.2	–
Netherlands	982	43.3	16 263 535	60.4	2.7
Norway	1139	57.9	4 608 551	247.1	12.6
Poland	18 625	499.7	38 245 488	487.0	13.1
Portugal	1659	111.4	10 471 588	158.5	10.6
Republic of Moldova	2940	133.4	3 925 170	748.9	34.0
Romania	11 679	366.8	21 725 785	537.6	16.9
Russian Federation	301 976	12 245.6	144 691 716	2087.0	84.6
San Marino	1	–	29 641	23.2	–
Serbia and Montenegro*	2576	84.1	10 516 739	245.0	8.0
Slovakia	2271	55.8	5 386 700	421.6	10.4
Slovenia	194	7.5	1 997 206	97.0	3.8

Country*	DALYs	Deaths	Population	DALY rate per million	Death rate per million
Spain	4094	220.7	42 778 189	95.7	5.2
Sweden	1565	113.6	8 997 689	174.0	12.6
Switzerland	664	36.0	7 392 067	89.8	4.9
Tajikistan	4940	102.1	6 467 378	763.8	15.8
The former Yugoslav Republic of Macedonia	670	13.1	2 030 308	330.0	6.5
Turkey	32 304	445.0	72 020 502	448.5	6.2
Turkmenistan	13 782	365.9	4 766 006	2891.7	76.8
Ukraine	60 044	2736.8	47 247 660	1270.8	57.9
United Kingdom	7877	454.0	60 050 590	131.2	7.6
Uzbekistan	26 844	520.4	26 208 820	1024.2	19.9
European Region	**604 785**	**23 101.6**	**883 277 039**	**684.7**	**26.15**

Data source for population, deaths, DALYs: WHO Global Burden of Disease Project, 2004 update http://www.who.int/healthinfo/bod/en/index.html

*Countries are defined as they were in 2004 at the time that these data were collated

The figures for deaths from domestic fires were found for Germany (310 out of 346 fire deaths in 2007 were in the home[5]) and the United Kingdom (374 deaths in 2004[6] out of a total of 454 deaths shown in Table 6). Thus deaths from domestic fires constituted 90% and 82% of all fire deaths for Germany and the United Kingdom respectively. The lower proportion in the United Kingdom could be related to the higher prevalence of domestic fire detectors in the United Kingdom. The GBD data in Table 6 only lists total deaths and DALYs without classifying by the setting. We used 82% as the multiplier to estimate domestic fire deaths and DALYs as a conservative proportion of all deaths and DALYs. This is likely to be conservative on average as the United Kingdom has one of the highest smoke detector fitment rates.

[5] Data source: Deutscher Feuerwehrverband (2009). "Feuerwehr-Jahrbuch (Fire Brigade Yearbook)" from http://www.feuerwehrverband.de/statistik.html

[6] Data source: http://www.kiddefyrnetics.co.uk/utcfs/Templates/Pages/Template-66/0,8070,pageId%3D34942%26siteId%3D405,00.html

5.3 Summary of burden of disease from child falls

The burden data for child falls in the WHO European Region are shown in Table 7.

Table 7: Children aged 0-14: DALYs, deaths, population and rates for child fall-related injuries in European states

Country*	DALYs	Deaths	Population	DALY rate per million	Death rate per million
Albania	3068	8.4	854 134	3592.1	9.8
Andorra	10	0.0	10 409	944.2	1.9
Armenia	1754	2.7	658 648	2663.0	4.2
Austria	1323	4.2	1 318 381	1003.9	3.2
Azerbaijan	3392	5.7	2 191 075	1547.9	2.6
Belarus	5540	21.2	1 593 829	3475.7	13.3
Belgium	1957	7.2	1 776 506	1101.4	4.1
Bosnia and Herzegovina	1363	0.0	691 711	1970.8	0.0
Bulgaria	2590	8.2	1 096 761	2361.3	7.5
Croatia	1070	4.0	716 778	1493.2	5.6
Cyprus	181	0.2	168 532	1071.8	0.9
Czech Republic	2398	3.6	1 534 603	1562.8	2.3
Denmark	1156	0.6	1 016 404	1137.0	0.6
Estonia	490	0.0	210 623	2326.0	0.0
Finland	1644	3.0	917 119	1792.9	3.3
France	15 028	35.3	11 162 245	1346.3	3.2
Georgia	1216	6.5	877 797	1384.8	7.5
Germany	9433	34.6	12 066 607	781.7	2.9
Greece	1538	3.2	1 594 950	964.4	2.0
Hungary	2404	4.5	1 618 720	1485.2	2.8
Iceland	69	0.3	65 492	1055.1	5.0
Ireland	681	2.5	846 087	805.4	3.0
Israel	1282	2.6	1 839 175	697.1	1.4
Italy	8184	21.8	8 202 421	997.8	2.7
Kazakhstan	13 559	75.8	3 742 069	3623.4	20.3
Kyrgyzstan	8251	41.4	1 635 822	5043.8	25.3
Latvia	1068	6.6	346 358	3083.0	19.1
Lithuania	1498	3.3	597 872	2506.2	5.5
Luxembourg	102	0.0	84 283	1214.8	0.0
Malta	72	0.3	71 580	999.4	4.8
Monaco	6	0.0	5965	1046.3	4.3
Netherlands	2242	9.6	3 005 000	746.1	3.2
Norway	1059	0.0	910 048	1163.4	0.0
Poland	20 044	20.5	6 434 374	3115.2	3.2
Portugal	2091	8.5	1 647 181	1269.4	5.2
Republic of Moldova	1955	9.4	816 217	2394.6	11.5
Romania	11 702	31.7	3 496 087	3347.1	9.1
Russian Federation	102 321	279.4	22 447 125	4558.3	12.4
San Marino	3	0.0	4157	611.9	0.0
Serbia and Montenegro*	3173	4.4	1 976 497	1605.3	2.2
Slovakia	2562	1.5	930 230	2754.2	1.6

Country*	DALYs	Deaths	Population	DALY rate per million	Death rate per million
Slovenia	416	0.8	287 619	1445.3	2.7
Spain	5588	22.0	6 161 400	907.0	3.6
Sweden	1334	0.0	1 587 979	839.9	0.0
Switzerland	1159	4.0	1 252 228	925.2	3.2
Tajikistan	5270	36.2	2 588 783	2035.8	14.0
The former Yugoslav Republic of Macedonia	880	0.0	410 140	2146.2	0.0
Turkey	54 479	162.0	20 668 660	2635.8	7.8
Turkmenistan	5805	26.0	1 556 220	3729.9	16.7
Ukraine	21 386	77.4	7 155 694	2988.6	10.8
United Kingdom	7776	14.3	10 893 907	713.8	1.3
Uzbekistan	26 648	176.3	8 909 144	2991.1	19.8
European Region	**370 219**	**1191.9**	**162 651 646**	**2276.1**	**7.3**

Data source for population, deaths, DALYs: WHO Global Burden of Disease Project, 2004 update http://www.who.int/healthinfo/bod/en/index.html

*Countries are defined as they were in 2004 at the time that these data were collated

Table 8 uses data from the IDB project, showing the proportion of all home falls for children that were classified as "fall/jump from greater height" and excluded falls on the level and falls on stairs and falls of less than one metre (counts of which were all specified elsewhere in the database). These were falls occurring at the residence, but excluded falls in the yard (i.e., excluding those settings specified as: playground in residential area, garden, private driveway, parking area, garage, carport, path, walking area). By excluding falls from trees, etc, it is likely that mainly falls from windows were therefore counted.

Table 8: 2002-2005 average annual number of home injuries classified as "Fall/jump from greater height" (greater than 1 metre) for children aged under 15: count of cases and proportion of all home fall injuries in the database

	Cases (sample)	Incidence Rate per thousand	Rate as proportion of all fall rate
Austria	284	3.25	5.9%
Denmark	591	1	1.5%
France	2528	7	6.5%
Netherlands	<5	-	-
Portugal	<5	-	-
Sweden	278	1	2.2%
United Kingdom*	<5	-	-

Data source: EU Injury Database – EU Commission/DG Sanco.

*2002 only. When there are fewer than five cases in the given country per year, no figures are reported.

Falls from a greater height will include some falls from windows and some from balconies. A study of children aged under 15 injured in falls from heights associated with buildings in Dallas, Texas, found that 52% had fallen from windows and 45% from balconies (Istre et al., 2003). Falls from bunk beds are unlikely to constitute a substantial portion of the injury burden of falls (Lyons, Oates, 1993) and can obviously be prevented by not having such beds in the first place

(Mayr et al., 2000). Although such studies indicate that housing features such as bunk beds and widely-spaced balcony rails could be associated with preventable injury (and hence part of the burden of injury associated with housing conditions), we will conservatively restrict the estimation of the burden only to those features that have been the focus of an intervention study. Of the various housing features related to falls, only the installation of window guards have been found to be effective to our knowledge (Spiegel, Lindaman, 1977).

These IDB data show that hospitalised falls in the home setting as described above classified as "fall/jump from greater height" were likely to constitute between 1.5% and 6.5% of all hospitalised falls in all settings for children. The rate of such injuries as a proportion of all fall injuries will vary according to factors such as children's exposure to unlatched/unguarded windows in each country concerned. The fraction of such falls as a proportion of all falls for children varies considerably between the countries shown in Table 8. It is therefore appropriate to estimate burden of disease due to children's falls from unguarded windows using a range of rates to give a realistic EBD range. The range of home fall rates applied to all the European countries is 1.5% to 6.5%. These rates are then halved to conservatively estimate rates of falls from windows at home as a proportion of all falls on the basis of the single study we could find that analysed such injuries (Istre et al., 2003).

5.4 Summary of burden of disease from child drownings

The burden data for drownings of children aged 0-14 in the WHO European Region are shown in Table 9.

Table 9: Children aged 0-14: DALYs, deaths, population and rates for child drownings in European states

Country*	DALYs	Deaths	Population	DALY rate per million	Death rate per million
Albania	1162	31.1	854 134	1361	36.5
Andorra	1	0.0	10 409	76	1.8
Armenia	437	11.9	658 648	664	18.1
Austria	157	4.0	1 318 381	119	3.0
Azerbaijan	1745	47.3	2 191 075	796	21.6
Belarus	2186	59.3	1 593 829	1371	37.2
Belgium	547	14.9	1 776 506	308	8.4
Bosnia and Herzegovina	3	0.0	691 711	5	0.0
Bulgaria	608	16.4	1 096 761	554	14.9
Croatia	271	7.2	716 778	377	10.0
Cyprus	60	1.6	168 532	354	9.5
Czech Republic	598	15.7	1 534 603	390	10.2
Denmark	203	5.3	1 016 404	200	5.2
Estonia	174	4.7	210 623	825	22.2
Finland	397	10.5	917 119	433	11.4
France	3077	82.0	11 162 245	276	7.4
Georgia	240	6.4	877 797	273	7.3
Germany	1953	52.2	12 066 607	162	4.3
Greece	275	7.1	1 594 950	172	4.5
Hungary	644	17.3	1 618 720	398	10.7
Iceland	1	0.0	65 492	10	0.0
Ireland	67	1.6	846 087	79	1.9
Israel	180	4.6	1 839 175	98	2.5
Italy	1340	34.3	8 202 421	163	4.2

Country*	DALYs	Deaths	Population	DALY rate per million	Death rate per million
Kazakhstan	14 571	402.9	3 742 069	3894	107.7
Kyrgyzstan	6603	185.1	1 635 822	4036	113.2
Latvia	974	26.4	346 358	2813	76.2
Lithuania	848	23.5	597 872	1419	39.2
Luxembourg	19	0.5	84 283	223	6.0
Malta	9	0.2	71 580	128	3.4
Monaco	0	0.0	5 965	10	0.0
Netherlands	863	23.6	3 005 000	287	7.9
Norway	313	8.4	910 048	344	9.2
Poland	1973	52.4	6 434 374	307	8.1
Portugal	646	17.2	1 647 181	392	10.5
Republic of Moldova	1938	53.4	816 217	2374	65.5
Romania	4717	128.3	3 496 087	1349	36.7
Russian Federation	47 471	1287.1	22 447 125	2115	57.3
San Marino	0	0.0	4157	6	0.0
Serbia and Montenegro*	1153	30.8	1 976 497	583	15.6
Slovakia	358	9.6	930 230	385	10.3
Slovenia	58	1.5	287 619	203	5.4
Spain	1539	41.4	6 161 400	250	6.7
Sweden	328	8.6	1 587 979	206	5.4
Switzerland	305	8.0	1 252 228	244	6.4
Tajikistan	8510	238.8	2 588 783	3287	92.3
The former Yugoslav Republic of Macedonia	242	6.5	410 140	589	15.9
Turkey	6440	170.0	20 668 660	312	8.2
Turkmenistan	14 261	400.7	1 556 220	9164	257.5
Ukraine	13 328	363.9	7 155 694	1863	50.9
United Kingdom	878	22.0	10 893 907	81	2.0
Uzbekistan	34 127	954.9	8 909 144	3831	107.2
European Region	**178 795**	**4901.3**	**162 651 646**	**1099**	**30.1**

Data source for population, deaths, DALYs: WHO Global Burden of Disease Project, 2004 update http://www.who.int/healthinfo/bod/en/index.html

*Countries are defined as they were in 2004 at the time that these data were collated

Although data for all drownings or near-drownings were obtained, the setting of the accident was not available (in particular, whether the event occurred at private housing or not). The IDB database was queried for "Mechanism of injury: Drowning and near-drowning" by children (see Table 10), but the data were scarce for most countries. Only France and the Netherlands reported 5 or more cases of home drownings or near-drownings per year from the sample of hospitals reporting to the IDB project. Note that different numbers of hospitals of different sizes may report data each year, so the figures cannot be interpreted as rates that can be compared between countries. However, by comparing the numbers of cases reported for drownings in all settings, we get an approximate proportion of all child drownings that can be estimated to occur at home. This was estimated to be approximately 18% in the Netherlands and 32% in France.

Table 10: 2002-2005 average annual number and percent of “drowning or near-drowning” children aged under 5 at home

	Cases (sample – home)	Cases (sample – all)	% at home**
France*	12	37	32%
Netherlands	21	118	18%
Sweden	<5	6	
United Kingdom	<5	11	

Data source: EU Injury Database – EU Commission/DG Sanco

*2003-2006 data

**These are approximate figures only as different hospitals may have reported cases for the IDB in different years.

We estimated drownings potentially prevented by pool fencing using the following approach.

Table 5, above, shows annual counts of private swimming pool drownings of children aged 0-5. Data from the WHO GBD project 2004 update show that 59 children aged 0-4 died from drowning in France in 2004. Inferring an average annual rate of child drownings in French private swimming pools of 17.8 per year (by averaging over Table 5 and somewhat crudely – and conservatively – multiplying by 5/6 to estimate the rate for 0-4-year-olds from this rate for 0-5-year-olds), we can then approximate the French private pool drownings as 30.2% of all French child drownings for those aged 0-4. Using this proportion for the rest of the WHO European Region, 31.4% of 2 468 total drowning deaths (according to the WHO GBD project) results in 775 avoidable deaths of children aged under 5 estimated to occur in home swimming pools in the WHO European Region. The prevalence of swimming pools is likely to be much higher in southern WHO European Region countries than in the north, and higher in wealthier countries, so this extrapolation may be most appropriate when restricted to: Andorra, Austria, Croatia, Cyprus, France, Gibraltar, Greece, Hungary, Israel, Italy, Malta, Monaco, Portugal, Slovenia, Spain, where there were 120 deaths due to drowning of children aged under 5 according to the WHO GBD (2004 update) project. These are countries with a GDP level per capita of at least $18 000 according to mainly 2008 data (CIA, 2009). For these countries, the assumption that the same proportion of drowning deaths occurs in private swimming pools as in France leads to an estimate of 38 deaths annually.

5.5 Summary of burden of disease from child scalds from domestic hot water

Table 11 shows data from IDB data for injuries with “Type of injury: Burns, scalds (thermal)” and “Mechanism of injury: Hot liquid” for children aged under 15 in bathroom or kitchen of home. Many of these will be scalds from domestic hot water, as well as from cooking liquids and hot drinks. “Cases” represents the number of accidents recorded in the sample of hospitals participating to the IDB project. “Incidence Rate” represents the number of accidents per 1000 inhabitants, not standardised by age.

Table 11: 2002-2006 annual case numbers and extrapolated incidence rates (per 1000 persons) of burns and scalds by water for children aged under 15 in bathroom or kitchen of home

Year	Austria		Denmark		France		Netherlands		Portugal		Sweden	
	Cases	Incidence Rate	Cases	Incidence Rate	Cases	Incidence Rate	Cases	Incidence Rate	Cases	Incidence Rate	Cases	Incidence Rate
2002	28	1	64	0	N/A	N/A	17	0	45	1	32	0
2003	36	2	57	0	77	1	30	0	92	1	35	0
2004	35	2	60	0	47	0	26	0	79	1	27	0
2005	35	2	76	0	40	0	104	0	52	1	25	0
2006	22	2	65	0	160	1	N/A	N/A	N/A	N/A	37	1

Data source: EU Injury Database – EU Commission/DG Sanco

6. Environmental burden of disease from injury associated with two aspects of housing conditions

6.1 EBD estimate for the WHO European Region

Lack of smoke detectors

The exposure values for different countries are shown above. For those without exposure estimates, the population weighted average for those countries with proportions of homes without smoke detectors estimates supplied was used, a value of 66%. The population attributable fraction (PAF) for lack of smoke detectors on fire-related deaths for these countries is:

$$PAF = \frac{p(RR-1)}{p(RR-1)+1} = \frac{0.66 \times (2.0-1.0)}{0.66 \times (2.0-1.0)+1.0} = 0.40 = 40\%$$

Where p = proportion of the population exposed, and RR is the relative risk for the condition in those exposed.

The EBD assessment for the contribution of housing conditions to fire injury deaths in the WHO European Region for those countries without exposure estimates is therefore:

Fire-related deaths attributable to lack of smoke detectors (assuming 44% fitment)

= PAF × death rate

= PAF × rate of fire-related deaths × proportion of deaths estimated to be in the home setting

=0.40 × 37.2 × 0.82 = 12.2 fire-related deaths/100 000

These estimates are shown in Table 12 which also includes estimates for deaths and DALYs. As discussed above, 82% of all fire deaths are conservatively estimated to occur in the home.

Table 12: EBD of fire-related injury from housing conditions

Country	% houses without detectors	PAF	DALYs	Deaths	Population	DALY rate per million	Death rate per million
France*	88.8%	0.47	3971	195.30	60 591 139	65.53	3.22
Germany**	69.0%	0.41	2838	162.77	82 642 615	34.35	1.97
Hungary*	91.4%	0.48	1581	70.18	10 113 272	156.36	6.94
Italy*	95.8%	0.49	1887	114.60	58 433 920	32.29	1.96
Lithuania*	95.5%	0.49	1349	58.63	3 440 158	392.10	17.04
Netherlands**	32.0%	0.24	196	8.65	16 263 535	12.06	0.53
Norway**	2.0%	0.02	18	0.94	4 608 551	3.99	0.20
Portugal*	98.3%	0.50	678	45.48	10 471 588	64.71	4.34
Slovakia*	91.2%	0.48	892	21.92	5 386 700	165.64	4.07
Sweden**	30.0%	0.23	298	21.60	8 997 689	33.07	2.40
Switzerland*	85.4%	0.46	252	13.66	7 392 067	34.09	1.85
United Kingdom***	15.8%	0.14	885	51.03	60 050 590	14.74	0.85
Countries without data****	66.0%*	0.40	182 720	6757.80	554 885 215	329.29	12.18
Total			**197 565**	**7522.55**	**883 277 039**	**223.67**	**8.52**

* See Table 4 (estimates from LARES survey, which only surveyed one city within the respective country)

** See http://www.rauchmelder-lebensretter.de/internationaler-vergleich.html

*** See Table 3 (English House Condition Survey, unpublished data)

**** Weighted European average (see section 6.1 above)

Based on estimates shown in the second column of Table 12 of exposure to housing without smoke detectors and reported fire-related DALYs and deaths, this EBD represents 7500 additional deaths and almost 200 000 DALYs across the WHO European Region that can be attributed to lack of smoke detectors (Table 12).

Lack of window guards

Table 13 shows two contrasting scenarios of children's exposure to housing without window guards on windows higher than ground floor level and reported fall-related DALYs and deaths. These estimates are combined with high and low scenarios of the proportion of child-hospitalised falls that were from higher windows (from Table 8). This EBD represents between 200-1300 additional deaths across the WHO European Region that can be attributed to lack of window guards.

Table 13: EBD of child fall injury from lack of window guards.

High or low impact scenario	Proportion of falls that are from home windows	Scenario: % with guards	PAF	DALYs	Deaths	Population	DALY rate per million	Death rate per million
High	3.25%	10%	47.4%	5699	18.3	162 651 646	35	0.11
Low	0.75%	50%	33.3%	926	3.0	162 651 646	6	0.02

Table 13 uses two exposure scenarios: high impact scenario (6.5% of all child fall hospitalizations are from high windows; only 10% of such windows have window guards across the WHO European Region) and low impact (1.5% of all child fall hospitalizations are from

high windows; 50% of such windows have window guards across the WHO European Region). Population figures are specific to children aged 0-14 (2004 data).

Lack of pool fencing

We have not included an EBD related to this exposure in our final estimate due to data gaps. However, a general estimate of a potential EBD associated with lack of domestic pool fencing can be derived. Table 14 shows two contrasting scenarios of children's exposure to this hazard, suggesting that between 21.7 and 25.2 additional deaths across the wealthier (with 2008 GDP level per capita of at least $18 000) southern states of the WHO European Region can be reasonably attributed to lack of pool fencing.

Table 14: EBD of child (aged <5) drownings in southern European countries from lack of pool fencing for two exposure scenarios

High or low impact scenario	Scenario: % of pools with fencing	PAF	Deaths	Population (millions)	Death rate per million
High	25%	66.9%	25.2	27.3	0.9
Low	50%	57.4%	21.7	27.3	0.8

Table 14 uses a high impact scenario (25% of private swimming pools have fencing) and low impact (50% of private swimming pools have fencing). Population figures are specific to children aged 0-4 in the southern WHO European Region (2004 data) with higher 2008 GDP per capita levels (Andorra, Austria, Croatia, Cyprus, France, Gibraltar, Greece, Hungary, Israel, Italy, Malta, Monaco, Portugal, Slovenia, Spain).

Burns to children

There are other aspects of the home environment associated with the child scald injury burden. The WHO European Report on Child Injury Prevention mentions other environmental measures in the home (WHO, 2008):

> A number of environment modifications hold promise to reduce thermal injuries: modification of building codes and standards and construction materials, improved heating and lighting equipment in homes, modification of cooking facilities and separation of cooking from living areas. Such prevention strategies have not been well evaluated, however.

6.2 EBD estimates for the WHO European Region: summary by exposure type

Table 15 combines the high and low scenarios proposed above for lack of window guards with the estimate of the burden of injury associated with lack of smoke detectors to provide ranges of overall death and DALY EBD estimates.

Table 15: EBD estimates summary of injury due to housing conditions in the WHO European Region.

High or low impact scenario	Window guards		Smoke detectors			
	Deaths, children aged <15	DALYs, children aged <15	Deaths, all age groups	DALYs, all age groups	TOTAL deaths	TOTAL DALYs
High	18.3	5699	7523	197 565	7541	203 265
Low	3.0	926			7526	198 491

In Table 15, injury categories include: child (aged<15) deaths and DALYs from falls from second level or higher windows without window guards; deaths and DALYs due to domestic fires associated with lack of smoke detectors. The estimates are based on WHO Global Burden of Disease (2004 update) and European IDB data.

7. Uncertainty

7.1 Areas of uncertainty

The analytical approach used in this report has a number of limitations.

Limited evidence base for exposure-response relationship: The evidence base for quantifying the exposure-response relationship between housing quality and home injury is small and of poor quality, without control of potential confounders, and based in single countries where country-specific factors may have influenced the outcomes.

Exposure assessment: Exposure estimates were rarely available as there are no national surveys of housing quality in Europe, outside of England. Some estimates that were not necessarily representative were used. Specifically, to estimate prevalence of housing without smoke detectors, the LARES survey provided single city estimates, which we have used to represent the respective country. For housing characteristics without exposure estimates, we have used a sensitivity analysis derived from using a range of plausible exposures as potential scenarios, tending to be conservative in these choices (by choosing values that were likely to be lower levels or intensities of exposure).

Lack of stratification of exposure levels and housing hazards: Housing hazards have strongest effect on injury risk for particular age groups. For example, the lack of fencing of swimming pools presents a higher risk for households with young children. Unfortunately, even for the one country for which we have data on the prevalence of swimming pools, we do not know the ages of those exposed, nor do we know the proportions of pools without safety features.

Injury incidence: Surveillance of injury data needs to include information on the setting of the accident to inform injury prevention initiatives and to motivate initiatives via analyses of injury burden such as that in the current paper. However, injury data will always be incomplete. These limitations are likely to be greater for derived measures such as DALYs, which also rely on accurate mortality data.

As the range of housing conditions considered is limited by the exposure data that are available and there are gaps in the literature on the exposure-response relationship for many exposures, this analysis has been restricted to just two injury-hazard combinations. The injury burden associated with housing will be estimated to be considerably larger when limitations can be addressed in the quality of the evidence, the range of exposures measured, and the lack of relevant detail of injury circumstances currently recorded in surveillance systems.

7.2 Reducing uncertainty

The estimates contained in this report could be improved in a number of ways.

Data on housing quality should ideally be available for more countries in the WHO European Region than just England. Such data could inform a considerably more precise estimate of EBD of housing from home injury.

Data for coding the location of the injury need to be improved. The International Classification of Diseases code ICD-10 has a fourth digit assigned to code the location, which includes

"home" as one category. Recent analysis has shown that this coding is poorly completed for most European countries (Suarez-Garcia et al., 2009).

Similarly, intentional injuries should be analysed separately to unintentional ones. Intentional injuries constitute a large proportion of home injuries (Sethi, personal communication, 2009). Generally, unintentional injuries are likely to be most amenable to home environmental injury prevention initiatives. The analysis in the current paper is relatively unaffected by the intentionality of the injury.

Researchers should be encouraged to conduct more high quality studies of the relationship between housing quality, home hazards, safety features and home injury.

Future EBD analyses could attempt to carry out a more sophisticated analysis that considered the distribution of exposures to housing conditions and home injury across different segments of the population and the fact that vulnerability to these exposures varies according to age and other factors. Such analyses should include more injury types and more types of housing conditions.

8. Conclusions

The EBD of home injury from two aspects of housing conditions is summarized in Table 16. This is likely to be an underestimate, because only a small number of housing exposures and injury types were included. The true EBD of injury associated with housing is large.

Improving housing quality and the prevalence of safety features such as smoke detectors would reduce home injury levels in Europe. The importance of this strategy is dependent on the prevalence of housing hazards. This strategy is likely to be most important in the least developed countries, and the most deprived subpopulations within developed countries, which are likely to suffer from the poorest housing quality.

Table 16. Summary of EBD of injury from household conditions in the WHO European Region

Housing exposure	Lack of window guards at second level and higher; lack of smoke detectors			
Health outcome	Injury deaths and DALYs			
Summary of EBD assessment	The lack of safety features causes about 7500 deaths and about 200 000 DALYs per year; mostly due to the lack of smoke detectors			
	Level and outcome measure	**Range**	**Demographic / geographic scope**	**Source of information**
(a) Exposure risk relationship				
Lack of window guards	RR 2.0, hospitalised falls	No CI provided	Children <15	Single intervention study
Lack of smoke detectors	RR 2.0	No CI provided	All age groups	Cross-sectional study
(b) Exposure assessment				
Lack of window guards		10%-50% of windows above ground level	Western Europe	Table 13
Lack of smoke detectors		2%-98% dwellings depending on country	Western Europe	Table 12

(c) PAF				
Lack of window guards		33.3%-47.4% child deaths from falls from windows	WHO European Region, 0-14 years	Derived from (a) and (b)
Lack of smoke detectors		2%-50% fire deaths depending on country	WHO European Region, all ages	Derived from (a) and (b)
(d) Total burden of disease	789 000 deaths 19 973 000 DALYs		WHO European Region (all injuries in all settings)*	Country reports to European Office of WHO 2004
(e) EBD from unsafe housing conditions				
Lack of window guards	Ca. 10 deaths (0.007/100 000) Ca. 3310 DALYs (2.0/100 000)	3-18 deaths (0.002 – 0.011/100 000) 926-5699 DALYs (0.6 – 3.5/100 000)	WHO European Region, 0-14 years	See Table 15
Lack of smoke detectors	7523 deaths (0.9/100 000) 197 565 DALYs (22.4/100 000)		WHO European Region, all ages	See Table 15
Main areas of uncertainty	Few etiological studies in developed countries to establish exposure-risk relationship; limited scope of exposure data			
Main implications	Considerable reductions in injury burden in the home are achievable via mainly low-cost safety features.			

* from http://www.who.int/healthinfo/global_burden_disease/estimates_regional/en/index.html

9. Policy implications

Injury prevention programs in most settings rely strongly on initiatives to address environmental injury hazards. Hazards in the home setting are difficult to quantify as homes tend to be private spaces, for which regulators are reluctant to impose standards or laws, and few surveys are conducted to gather data on home injury hazards. This situation leads to a paucity of reliable research to support home injury prevention initiatives and little exposure data on which to build EBD estimates, which are potentially an important motivator for policy development and orientation.

Nevertheless, the current analysis has identified the fitment of window guards on second floor and higher windows, and the widespread installation of smoke detectors as interventions with some supporting evidence of their effectiveness and a basis on which to estimate exposure levels or exposure scenarios. The instigation of these relatively low-cost measures in the WHO European Region would yield potential savings of about 7500 deaths and about 200 000 DALYs. These measures should be strongly supported by policy initiatives, including regulation where possible.

Other potentially effective measures include the provision of fencing for private swimming pools and reducing excessively high domestic hot water temperatures.

10. References

Ahrens M (2008). Home Smoke Alarms: The Data as Context for Decision. *Fire Technology*, 44:313-327.

Baker SP et al. (1992). *The injury fact book*. 2nd ed. New York, Oxford University Press.

Bonnefoy X et al. (2003). Housing and Health in Europe: Preliminary Results of a Pan-European Study. *American Journal of Public Health*, 93:1559-1563.

Borella P et al. (2004). Legionella infection risk from domestic hot water. *Emerging Infectious Diseases*, 10:457-464.

CIA (2009). *The World Factbook 2009*. Washington, DC, Central Intelligence Agency.

Consumer Safety Commission (2003). *Recommendation on the danger of burns presented by domestic hot water* 10/03. Paris, Consumer Safety Commission.

Department for Communities and Local Government (2008). *Review of Health and Safety Risk Drivers*. London, Department for Communities and Local Government.

Department for Communities and Local Government (2006). *English House Condition Survey – Technical Report* (2004 Edition). London, Department for Communities and Local Government.

Deutscher Feuerwehrverband (2009). *Feuerwehr-Jahrbuch* [Yearbook of Fire Brigades]. Bonn, Deutscher Feuerwehrverband [German Fire Fighter Association].

Ermanel C, Thélot B (2005). *Surveillance épidémiologique des noyades*. Enquête noyades 2004. Saint-Maurice, Institut de Veille Sanitaire.

European Home and Leisure Accident Surveillance System (2009). *European Injury Database (IDB).* (https://webgate.ec.europa.eu/idb, accessed 15 May 2010).

Health Canada (2007). *Bacterial Waterborne Pathogens – Current and Emerging Organisms of Concern*. (http://hc-sc.gc.ca/ewh-semt/pubs/water-eau/pathogens-pathogenes/legionella-eng.php, accessed 19 January 2010).

Institut National d'Etudes Démographiques (2007). *Households by number of persons in 1999 and 2005*. (http://www.ined.fr/en/pop_figures/france/couples_households_families/households_number_people/, accessed 12 April 2010).

Istre GR et al. (2003). Childhood injuries due to falls from apartment balconies and windows. *Injury Prevention*, 9:349-352.

Jacobs DE, Baeder A (2009). *Housing interventions and health: a review of the evidence*. Columbia, National Center for Healthy Housing.

Kay D, Prüss A, Corvalan C (2000). *Methodology for assessment of Environmental burden of disease*. Geneva, World Health Organization. (http://www.who.int/quantifying_ehimpacts/methods/en/wsh0007.pdf, accessed 12 August 2010).

Keall MD et al. (2008). Association between the number of home injury hazards and home injury. A*ccident Analysis & Prevention*, 40:887–893.

Kendrick D et al. (2008). Preventing Childhood Falls at Home: Meta-Analysis and Meta-Regression. *American Journal of Preventive Medicine*, 35:370-379.

Kerr K (2007). Prevention of injuries at home. *Injury Prevention*, 13:141.

Kuratorium für Verkehrssicherheit (2007). *Injuries in the European Union: a statistics summary 2003-2005*. Vienna, Kuratorium für Verkehrssicherheit.

Lyons TJ, Oates RK (1993). Falling out of Bed: A Relatively Benign Occurrence. *Pediatrics,* 92:125-127.

Lyons RA et al. (2006). Modification of the home environment for the reduction of injuries. *Cochrane database of systematic reviews 4*.

MacKay M., Vincenten J (2007). *Child Safety Summary Report Card for 18 Countries – 2007*. Amsterdam, European Child Safety Alliance, Eurosafe.

Mathys W et al. (2008). Occurrence of Legionella in hot water systems of single-family residences in suburbs of two German cities with special reference to solar and district heating. *International Journal of Hygiene and Environmental Health,* 211:179-185.

Mayr JM et al. (2000). Bunk beds – a still underestimated risk for accidents in childhood? *European Journal of Pediatrics, 1*59:440-443.

Naughton MP et al. (2002). Heat-related mortality during a 1999 heat wave in Chicago. *American Journal of Preventive Medicine*, 22:221-227.

Office of the Deputy Prime Minister (2006). *Housing Health and Safety Rating System: Operating Guidance.* London, Office of the Deputy Prime Minister.

Sengoelge M, Hasselberg M, Laflamme L (2010). Child home injury mortality in Europe: a 16-country analysis. *European Journal of Public Health.* Published online 29 April 2010 doi:10.1093/eurpub/ckq047.

Sethi D (2009). Personal communication on the ratio between intentional and unintentional injuries. 25 May 2009.

Spiegel CN, Lindaman FC (1977). Children Can't Fly: A Program to Prevent Childhood Morbidity and Mortality from Window Falls. *American Journal of Public Health*, 67:1143-1147.

Suarez-Garcia I et al. (2009). Mortality due to injuries by place of occurrence in the European Region: analysis of data quality in the WHO mortality database. *Injury Prevention, 1*5:275-277.

Thélot B et al. (2008). *Surveillance épidémiologique des noyades.* Enquête noyades 2006. Saint-Maurice, Institut de Veille Sanitaire.

Thompson DC, Rivara F (1998). Pool fencing for preventing drowning in children. *Cochrane Database of Systematic Reviews Reviews 1.*

Vandentorren S et al. (2006). August 2003 Heat Wave in France: Risk Factors for Death of Elderly People Living at Home. *European Journal of Public Health*, 16:583-591.

Vincenten JA et al. (2005). Parents' perceptions, attitudes and behaviours towards child safety: a study in 14 European countries. *International Journal of Injury Control and Safety Promotion*, 12:183-189.

Western Australia Health Department (1988). *Preschool Drowning in Private Swimming Pools*. Perth, Western Australia, Inter-Government Working Party on Swimming Pool Safety.

World Health Organization (2005). *Preventing children accidents and improving home safety in the European Region. Identifying means to make dwellings safer*. Report of a WHO expert meeting. Copenhagen, WHO Regional Office for Europe. (http://www.euro.who.int/__data/assets/pdf_file/ 0008/98666/ Bonn_accident_rep.pdf, accessed 5 January 2010)

World Health Organization (2008). European report on child injury prevention. Copenhagen, WHO Regional Office for Europe. (http://www.who.int/violence_injury_prevention/child/injury/world_report/ European_ report.pdf, accessed 12 February 2010)

Household crowding and tuberculosis

Michael G Baker, Kamalesh Venugopal, Philippa Howden-Chapman

1. Introduction

Tuberculosis: Tuberculosis (TB) is a disease caused by infection with mycobacteria, mainly *Mycobacterium tuberculosis*. Most infection is transmitted from people with active pulmonary or respiratory tract TB via airborne droplets generated through respiratory effort (coughing or sneezing). If these droplets are inhaled by a susceptible person, that person may develop latent tuberculosis infection (LTBI). About 10% of people with LTBI (also called latent TB) subsequently progress to active TB themselves. The risk of progression to active disease is highest in the first two years after infection, but persists for life unless treated (Heyman, 2004).

TB remains one of the most important causes of ill health and premature mortality worldwide. About 9 million new cases of TB occur each year (WHO 2002). In eastern Europe and Africa, TB deaths are increasing after almost 40 years of decline. Including people who are also infected with HIV/AIDS, approximately 2 million patients die from TB annually (WHO, 2002). The rising global caseload is almost certainly driven upwards in subSaharan Africa by the spread of HIV/AIDS and in eastern Europe by the deterioration of health in general and of TB control in particular. There is also a large reservoir of cases in Asia.

The World Health Organization (WHO) declared TB a global emergency in 1993 because of concern about the huge scale of the modern TB epidemic (The National Foundation for Infectious Diseases, 1999). This disease disproportionately affects the poor, who are more vulnerable to infection and suffer more from the consequences. Indeed, there is growing recognition that controlling TB has the potential to reduce poverty. The Millennium Development Goals link achievement of health outcomes with TB control as an indicator for progress in the elimination of poverty (Department for International Development, 2004).

There were 422 830 TB cases reported in Europe in 2006, which was 7% of cases reported to WHO that year (Institut de Veille Sanitaire, 2008). These cases were reported by 51 of the 53 countries of the WHO European Region (no cases were reported from Monaco and San Marino). There was a marked East-West gradient in incidence, with 73% cases reported from the East (Institut de Veille Sanitaire, 2008). In 2006 the average rate of TB ranged from 10.4/100 000 in the old EU states of western Europe (EU-15) to 110.3/100 000 in the 12 countries classified as eastern Europe. By contrast with western Europe, the incidence of TB is increasing in eastern Europe. Anti-tuberculous drug resistance is also rising in eastern Europe where multidrug resistance is becoming a serious problem (Aziz et al., 2006).

Household crowding and TB: TB is generally associated with poverty and deprivation (Spence et al., 1993; Barr et al., 2001). Household crowding is one manifestation of poverty and could be a mechanism that mediates the link between deprivation and TB. Several studies have explored the relationship between household crowding and TB incidence (Hill et al., 2006; Saiman et al., 2003). The importance of this risk factor remains unresolved because of a lack of agreement about the most appropriate definition of crowding, contradictory findings due in large part to the lack of a common definition of crowding, a reliance on ecological design, and the failure to control for potential confounders in many studies.

Household exposure to a known TB case is probably the most important mode of TB transmission. This conclusion is supported by both epidemiological and microbiological data.

For example, in a study of active TB among Iraqi schoolchildren and their household contacts, the authors concluded that 77.2% of new TB cases were attributable to household contacts (Al Kubaisy et al., 2003). It is therefore not surprising that household crowding, which increases the frequency and duration of contact between infectious cases and household members, has often been shown to be a risk factor for TB. Increased use of laboratory typing methods has also helped to clarify the important role of contacts outside the household, particularly in areas with high prevalence of disease (Warren, 1999).

The finding that TB rates are associated with crowded living conditions is hardly surprising from what we know about the effective transmission of this bacteria in households via small droplet nuclei (Musher, 2003). Several modelling approaches have also been used to show how transmission of TB is increased by contact with infectious cases in confined spaces (Beggs et al., 2003). These modelling methods have been applied particularly to health care settings where TB patients are being cared for and undergoing procedures that may increase the release of infectious material into the air.

Aims of this chapter: This chapter aims to estimate the environmental burden of disease (EBD) for TB that can be attributed to household crowding in Europe. It follows the method developed by the World Health Organization for such studies (WHO, 2003). It reviews the evidence on the relationship between household crowding and TB to assess the strength of the evidence and the size of the effect. It then uses this exposure-response knowledge along with data on the level of exposure to household crowding in Europe to estimate the population attributable fraction (PAF) for TB from household crowding. It then uses data on TB burden of illness in Europe to estimate the impact of eliminating household crowding on TB incidence, deaths, and DALYs.

2. Summary of the method

This analysis used the standard method for estimating the Environmental Burden of Disease (EBD) proposed by the WHO (WHO, 2003).

In brief, this method relies on calculating the PAF which is the proportion of disease that can be ascribed to a specified risk factor. In this context, PAF represents the proportion of TB in a population that would be prevented if exposure to household crowding were removed from the entire population. In this analysis we have used the following univariate formula for calculating PAF:

$$PAF = \frac{p(RR-1)}{p(RR-1)+1}$$

where p = proportion of the population exposed, and RR is the relative risk for the condition in those exposed.

The PAF is then applied to the total burden of TB, in this case in Europe, to estimate the proportion of cases, deaths and disability adjusted life years (DALYs) that can be ascribed to household crowding.

Estimating this EBD therefore relies on the following three sources of data:

- Exposure-risk relationship – Obtained from a structured review of the literature on TB and household crowding
- Exposure assessment – Obtained from census data for European countries that reported household crowding levels
- Total burden of disease – Obtained from previous WHO global burden of disease estimates based on TB case and mortality reporting by states in WHO Europe region

The remainder of this chapter describes the key steps in obtaining this essential information and using it to estimate the EBD of TB from exposure to household crowding.

3. Exposure-risk relationship for household crowding and TB

3.1 Method for establishing exposure-risk relationship

To produce an estimate of the relative risk of TB associated with household crowding we reviewed epidemiological studies that have investigated this risk. This literature search used Medline 1960-2008 and Embase 1988-2008, applying the following two search strategies:

1. Tuberculosis (disease and death) and household crowding:

- Tuberculosis (mesh heading and key word)
- AND Case-control studies (mesh heading and key word), Cohort studies (mesh heading and key word), Ecological studies (key word), Geographic information system (mesh heading and key word), Risk factors (mesh heading and key word)
- AND Crowding (mesh heading and key word), Overcrowding (key word)

2. Latent tuberculosis infection (LTBI) and household crowding

- Latent tuberculosis (mesh heading and key word), Tuberculosis infection (key word), Tuberculin test (mesh heading and key word)
- AND Survey (key word), Cross-sectional studies (mesh heading and key word), Risk factors (mesh heading and key word)
- AND Crowding (mesh heading and key word), Overcrowding (key word)

Studies were selected based on the following criteria:

- English language – Main paper written in English allowing full review of its design and methods.
- Household crowding – Was measured in a meaningful way and clearly reported (for example, as persons per room (ppr)).
- Control for socioeconomic status – One or more measure of socioeconomic status (such as income, education, unemployment, home ownership) was used to produce an estimate of the impact of household crowding adjusted for the effects of poverty and deprivation.
- Control for migration (where important) – For studies carried out in developed countries, where a high proportion of cases are related to migration from high-TB-incidence countries, then an indicator of migration was used in the analysis.

Selected studies were appraised based on their study design. We placed greater weight on studies that used more robust designs, such as case-control and cohort studies, which generally allow for greater control of important confounders, compared with ecological studies.

3.2 Summary of exposure-risk relationship

This search identified 41 studies that investigated the risk of TB in relation to household crowding. These studies include 13 case-control studies, seven cross-sectional studies and 21 ecological studies. Most (35) of these studies were of TB cases, but two used TB mortality and four used LTBI. A meta-analysis would be difficult because of the heterogeneity of these studies.

Case-control studies: Of the 13 case-control studies identified that considered household crowding as a potential risk factor for TB, six were excluded from this review. One was carried out in a low incidence country by Tocque et al. (2001) in Liverpool (United Kingdom) and reported no effect from household crowding. However, it included few details on how household crowding was considered in the analysis. It also used a very high level of matching between cases and controls (age, sex, ethnicity, postcode) to control for social deprivation which would probably have resulted in 'overmatching' of housing conditions between cases and controls. A second study, carried out in Mexico, specifically investigated the role of biomass stoves (Perez-Padilla et al., 2004) and controlled for household crowding in the analysis, but did not report its independent effect. Another study aimed to investigate the effectiveness of the Bacillus Calmette-Guérin (BCG) vaccine (Arbelaez et al., 2000). Although it did find a modest effect for household crowding (which was simply recorded as yes or no), it did not report an adjusted effect size for household crowding. A fourth case-control study aimed to investigate the effects of passive smoking on the risk of LTBI progressing to TB. While it did record household crowding, cases and controls were effectively highly matched for this exposure. Another case-control study was excluded because of its small sample size and lack of adjustment for confounders (Coetzee et al., 1998). A further case-control study investigated risk factors for LTBI in children ≤5 years rather than TB disease (Besser et al., 2001). It recorded the number of people in the house, but did not include a measure of household crowding.

Of the seven selected case-control studies, six investigated the role of household crowding as a risk factor for TB and one used LTBI as the outcome. These case-control studies are listed below and tabulated in Table 1:

- A case-control study by Hill et al., 2006 (Hill et al., 2008) in the Gambia found a strong effect from household crowding with an adjusted odds ratio (aOR) of 5.12 (95% CI 1.75-14.62) for TB for households in the most crowded category (≥4 persons and ≥2 ppr) compared to those in the least crowded (<4 persons and <2 ppr).
- A case-control study by Coker et al. (2006) in Samara (Russian Federation) found a strong effect from household crowding with an aOR of 3.77 (95% CI 2.06-6.88) for TB for households with the least quartile of space per person compared to the quartile with the most space.
- A case-control study by Shetty et al. (2006) carried out in India found no effect from household crowding, with an aOR of 1.03 (95% CI 0.89-1.19) for households where ppr was >2 compared with those ≤2 ppr.
- A case-control study by Lienhardt et al. (2005) carried out in three countries in West Africa found a significantly increased risk of TB for households which included larger numbers of adults with an aOR of 2.80 (95% CI 1.71-4.57) for households with high occupancy (>10 adults) compared to those with 1 to 5 adults. Household density (ppr) was not a significant risk factor in the multivariate analysis.
- A case-control study by Tipayamongkholgul et al. (2005) carried out in Thailand found a significantly increased risk of TB for households which included the highest level of crowding compared with the least crowded with an aOR of 11.18 (95% CI 2.35-53.20) for

households with ≥5 ppr compared with those that had ≤1 ppr in the multivariate logistic regression.

- A case-control study by Tekkel et al. (2002) carried out in Estonia found a non-significantly increased risk of TB with household crowding, with an aOR 1.49 (95% CI 0.93-2.39) for those living in household with >1ppr.
- A case-control study by Saiman (2001) investigated risk factors for LTBI, rather than TB, in New York children aged 1-5 years. It found a non-significant elevated risk associated with household crowding in the univariate analysis. In this population other risk factors were overwhelmingly important, notably contact with an adult with active TB, foreign birth, foreign travel, and a relative with a positive tuberculin skin test (TST).

Cross-sectional studies: Seven cross-section studies investigated the prevalence of TB and its relationship to household crowding and other risk factors. Two of these studies were excluded as they did not measure or report the effects of household crowding (Rathi et al., 2002; Lockman et al., 2002). Of the remaining five studies, three looked at cases of TB (Gustafson et al., 2004; Yu et al., 1988), one focused on LTBI in household contacts of cases (Tornee et al., 2004), and one reported results of a LTBI/tuberculin survey (Plant et al., 2002). All of these studies were carried out in high TB incidence developing countries. These cross-sectional studies are listed below and tabulated in Table 1.

- One of the cross-sectional TB studies carried out in Guinea-Bissau (Gustafson et al., 2004) found that adult crowding (>2 adults per household) was a significant risk factor (OR 1.68, 95% CI 1.18-2.39) for TB. By contrast, the other two TB studies did not find such an association. The main focus of one was on investigating the role of biomass cooking fuels as a risk factor for TB in India and it relied on self-reported active TB status (Mishra et al., 1999). The other study with a negative finding used a large TB screening programme carried out in Shanghai (Yu et al., 1988).
- The cross-sectional study of LTBI among household contacts of TB cases found that the risk of being infected was significantly associated with household crowding, as measured by ppr (Tornee et al., 2004).
- The one LTBI/tuberculin survey did not find a relationship between LTBI and crowding as measured by sharing a bedroom with one or more other people (Plant et al., 2002).

Ecological studies: A total of 21 identified ecological studies investigated the association between TB incidence and living in a geographic area with higher levels of crowding. All assigned exposure to crowding and other factors based on the domicile of the case. Of these studies three measured household crowding in a composite deprivation score so the independent effect of household crowding could not be measured and these were excluded (Spence et al., 1993; Tocque et al., 1998; Bhatti et al., 1995). Three studies used only univariate analysis so were unable to distinguish the independent contribution of household crowding (Barr et al., 2001; van Rie et al., 1999; Siddiqi et al., 2001). Two studies did not include measures of crowding at the household level (Munch et al., 2003; Ponticiello et al., 2005) and one was restricted to migrant cases (Davidow et al., 2003). One study did not include measures of socioeconomic position in the multivariate analysis (Drucker et al., 1994). A further study used an analytic method that could not produce an effect size for exposure to household crowding (Acevedo-Garcia, 2001).

Table 1. Summary of studies investigating the relationship between exposure to household crowding and risk of TB

Reference	Study design, location, time	Study population	Exposure measure	Outcome measure	Adjusted covariates	Results (RR/OR, 95% CI)
Case-control studies						
Hill et al., 2006	Case-control, Gambia, 2002-04	100 TB cases and 200 age sex matched clinic controls, all ≥15 years	Crowding index with 3 categories (category 1= HH<4 persons and <2 ppr to category 3=HH≥4 persons and ≥2 ppr)	Sputum smear positive TB	TB case in household; Occupation; Employment; House construction; Smoking; Ethnic group	For category 2 crowding vs. reference (category 1) OR=2.58 (0.69-9.68), for category 3 vs. reference OR=5.05 (1.75-14.62). Other significant risk factors were past household exposure to a known TB case.
Coker et al., 2006	Case-control, Samara, Russian Federation, 2003	334 TB cases and 334 age sex matched controls from population registry, all adults	Crowding measures as living space/ person (4 categories)	Culture confirmed TB cases	Assets; Employment; TB contact; History of prison; History of illicit drug use; Drinking raw milk; Diabetes	For third least living space vs. reference (quartile with most space) aOR=1.74 (95% CI=0.92-3.20), for second least aOR=1.89 (95% CI=1.03-3.47), for least aOR=3.77 (95% CI=2.06-6.88). Other significant risk factors were low assets, diabetes, history of imprisonment, unemployment, history of illicit drug use, relative with TB, drinking raw milk.
Shetty et al., 2006	Case-control, India, 2001-03	189 cases and 189 age-sex matched controls recruited from relatives of non-TB in-patients, all ≥15 years	Crowding measured as ppr (≤2, >2)	TB cases	Education; Household income; Smoking; Cooking (biomass fuels); Separate kitchen; Alcohol; Chronic disease	For >2 ppr aOR=1.03 (95% CI=0.89-1.19). Significant risk factors were low education level, not having a separate kitchen, and chronic disease (mainly diabetes)
Lienhardt et al., 2005	Case-control, three countries in West Africa, 1999-2001	687 TB cases and age-matched controls (one household and one neighbourhood control each), all >15 years	Crowding measured as number of people in household and ppr (<1,1-2 and >2)	Smear positive TB cases	Sex; HIV infection; Smoking; Marital status; Family history of TB; Home ownership	For 6-10 adults in household vs. reference (1-5 adults) aOR=1.37 (95% CI=1.03-1.82), for >10 adults vs. reference aOR=2.80 (95% CI=1.71-4.57). Weak positive association between TB and ppr. Other significant risk factors were male sex, HIV infection, smoking, history of asthma, family history of TB, marital status and renting the house.
Tipayamongkholgul et al., 2005	Case-control, Thailand, 2002-03	130 TB cases, 130 age-sex matched hospital controls, all with BCG immunisation, all < 15 years	Crowding measured as average ppr (<1.1, 1.1-2.9, 3.0-4.9, 5.0+)	TB	Age; Frequency of illness; Passive smoking	In group with no history of TB patient contact (n=192), for 1.1-2.9ppr vs. reference (≤1ppr) aOR=1.04 (95% CI=0.34-3.22), for 3.0-4.99ppr aOR=1.44 (95% CI-0.46-4.57), for 5+ persons aOR=11.18 (95% CI=2.35-53.20).

Reference	Study design, location, time	Study population	Exposure measure	Outcome measure	Adjusted covariates	Results (RR/OR, 95% CI)
Tekkel et al., 2002	Case-control, Estonia, 1999-2000	248 cases and 248 controls matched by age, sex and county, all >15 years	Crowding measured as ppr (≤1, >1)	TB	Place of birth; Marital status; Education	For ppr>1 OR=1.49 (95% CI=0.93-2.39). Significant risk factors: non-married marital status, low education level, low income, previous prison, not having own home, unemployment, smoking, alcohol consumption, shortage of food, contact with TB patients.
Saiman et al., 2001 (Saiman et al., 2003)	Case-control study, New York, 1996-98	92 cases and 192 matched controls, aged 1-5 years	Crowding defined as ppr ≥0.71. Also measured sharing bathroom or kitchen with other tenants	LTBI	Logistic regression adjusting for: contact with TB case or TST positive person; foreign birth; foreign travel; single parent household	Non-significant elevated risk associated with household crowding in univariate analysis (OR=2.71, 95% CI=0.85-10.60). Significant risk factors were contact with an adult with active TB, foreign birth, foreign travel, and a relative with a positive TST.
Cross sectional studies						
Gustafson et al., 2004	Prospective community study, Bissau, Guinea-Bissau, 1996-98	247 adult cases of TB compared with 25 189 adults living in the area in May 1997. (≥15 years)	Crowding measured as >2 adults per household	Intra-thoracic TB	Sex; Age; Ethnicity; Living area; Type of household; Child crowding; House quality	Adult crowding (>2 adults per household) aOR 1.68 (95% CI 1.18-2.39) and poor quality of housing (aOR 1.66, 95% CI 1.24, 2.22). Other significant risk factors were increasing age, male sex, ethnic group other than the largest group.
Tornee et al., 2004	Cross-sectional study, Bangkok, Thailand, 2002-03	500 household contacts <15 years of 342 index cases with sputum smear positive pulmonary TB	Crowding measured as average number of ppr.	LTBI	Contact with index case; Duration of contact; Relationship to contact; Illness in TB contact (cavitation, sputum smear positive, cough, treatment delay); House type.	For 2.1-3 ppr vs. reference (≤2 ppr) aOR=1.31 (95% CI=0.60-2.88), for having >3 ppr vs. reference aOR=2.63 (96% CI=1.18-5.85). Other significant risk factors: close contact to female index case, exposure to mother/father with TB, exposure to index case with cavitation or with 3+ sputum smear grade.
Plant et al., 2002	Cross-sectional study, Vietnam, 1997-99	1395 prospective migrants aged >15 years, assessed with TST	Crowding measured as sharing bedroom with none, one, or two or more others	TST positive	Age; smoking	No increased risk of TST positive result associated with sharing bedroom with others. Significant risk factors were age, gender and smoking
Mishra et al., 1999	Cross-sectional study on biomass fuels as risk factor, India, 1992-93	260 162 persons aged 20+ in India's 1992-93 National Family Health Survey self-reported active TB	Crowding measured as ≥3 ppr	Self-reported active TB	Age; Sex; Ethnicity; Education level; House type; Cooking fuel type; Separate kitchen	No relationship between TB and crowding. Persons living in households that primarily use biomass for cooking fuel had higher prevalence of TB than persons living in households that used cleaner fuels (aOR = 2.58; 95% CI = 1.98-3.37).
Yu et al., 1988	Cross sectional study, Shang-hai, China	TB survey of 30 287 employees using chest x-ray	Area of housing, split into >4 m^2 or ≤4 m^2	TB	Age; Sex; Smoking; Contact history; Occupational class	No relationship between TB rate and housing area. Significant risk factors were history of contact with case and smoking

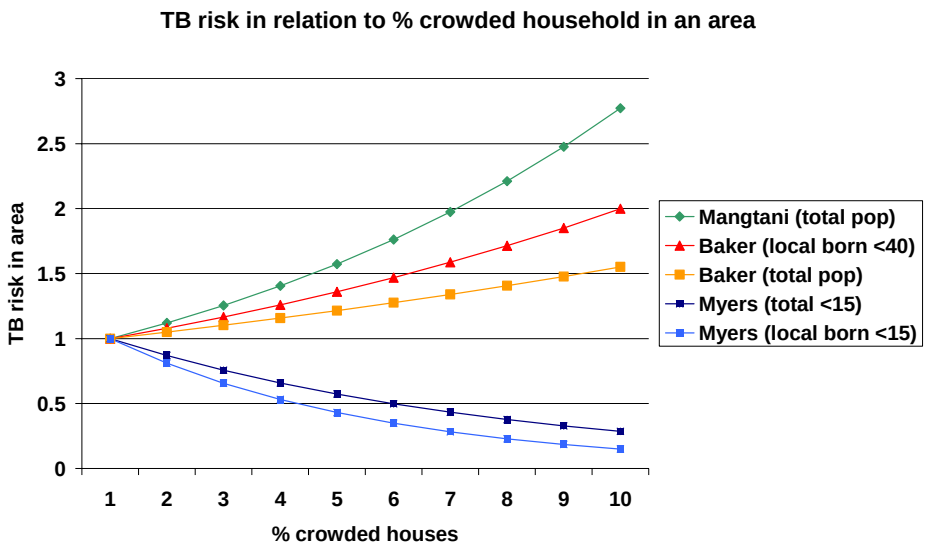
This left 10 ecological studies in the review (Mangtani et al., 1995; Elender et al., 1998; Baker et al., 2008; Cantwell et al., 1998). All but one (Antunes et al., 2001) were carried out in developed countries, notably the United Kingdom (Mangtani et al., 1995; Elender et al., 1998), the United States (Myers et al., 2006; Cantwell et al., 1998), Canada (Wanyeki et al., 2006; Clark et al., 2002), Germany (Kistemann et al., 2002) and New Zealand (Baker et al., 2008). These studies mainly focused on TB disease, but two used deaths from TB as the main outcome (Elender et al., 1998; Antunes et al., 2001).

Nine of these ecological studies found a significant positive association between household crowding and TB risk. One found that household crowding became protective after adjusting for multiple socio-demographic factors including income, ethnicity, and migration (Myers et al., 2006).

These studies suggest that people living in neighbourhoods that contain more crowded households have a small, but significantly, raised risk of TB after adjusting for socioeconomic factors and the level of migration. Since migration from high incidence countries is a very strong risk factor for TB, and is highly associated with lower socioeconomic position, poor housing and ethnicity, it may be difficult to produce robust estimates of the independent effect of these risk factors.

Three of these studies calculated incident rate ratios (IRR) for the increase in TB risk in relation to the increase in percent of crowded households. Two used percent of households with >1 ppr (Mangtani et al., 1995; Myers et al., 2006). One used percentage of household with a bedroom deficit of one or more (Baker et al., 2008). Two of these studies also provided estimates for subpopulations which are more likely to be representative of the risk attributed to the effects of household crowding (younger, local-born populations). These relationships are shown graphically below (Fig. 1).

Fig. 1. Summary of exposure-response relationship between household crowding and risk of TB from three ecological studies, adjusted for socioeconomic status and other factors (2 with subgroup analyses)



Note: the Mangtani and Myers papers used >1ppr as a marker of crowding, whereas the Baker paper used ≥1 bedroom deficit.

Synthesis of evidence on household crowding and TB exposure-risk relationship: The evidence base for establishing the exposure-risk relationship between household crowding and TB is relatively small and incomplete and contains some inconsistent findings.

For ***low incidence countries***, such as western Europe, North America and Australasia, there is an absence of high quality case-control studies that have investigated the effect of household crowding. The one case-control study carried out in this setting did not report how household crowding was measured and cases and controls appear to have been effectively matched for housing conditions (Tocque et al., 1999). The one case-control study of LTBI (Saiman et al., 2003) investigated risk factors in New York children aged 1-5 years. It found a non-significant elevated risk associated with household crowding, but only in the univariate analysis. In this population other risk factors were overwhelmingly important. Studies of TB in low-incident-TB countries have mostly been ecological. Nine out of ten of these ecological studies that used reasonably robust methods to control for important confounders (socioeconomic position, migration) found that the incidence of TB was higher in neighbourhoods with higher average levels of household crowding.

For ***medium incidence countries***, such as central and eastern Europe (Euro B+C), this review identified two case-control studies. A case-control study by Coker et al. (2006) in Samara (Russian Federation) found a strong effect from household crowding with an aOR of 3.77 (95% CI 2.06-6.88) for TB, but did not report a result using an exposure measure that would be easy to apply (such as ppr). A case-control study by Tekkel et al. (2002) carried out in Estonia found a non-significantly increased risk of TB with household crowding, with an aOR 1.49 (95% CI 0.93-2.39) for those living in household with >1ppr.

Most of the studies using more robust methods (case-control and cross-sectional) were in ***high incidence countries***. Of the four TB case-control studies carried out in high incidence countries (Gambia, West Africa, India, Thailand), three found a significantly elevated risk of TB associated with household crowding and household size (Hill et al., 2006; Lienhardt et al., 2005; Tipayamongkholgul et al., 2005), and one found no risk (Shetty et al., 2006). The five reported cross-sectional studies of TB and LTBI that met selection criteria were also carried out in relatively high incidence countries (Guinea-Bissau, Thailand, Vietnam, India, China). Two found a significant increased risk of TB/LTBI associated with measures of household crowding and three found no association.

On the basis of this evidence, we propose a RR of 1.5 for the effect of household crowding (>1 ppr) on the risk of TB, with a plausible range of 1.2 to 2.0. We consider that the evidence supports application of this exposure-risk relationship to medium and high TB incidence countries.

4. Exposure assessment for household crowding

4.1 Methods for measuring exposure to household crowding

Crowding in households relates to situations where the number of people residing in a household exceeds the capacity of the household to provide adequate shelter, space, and facilities for its occupants. The simplest measures of crowding simply report occupancy, being the number of people or families in a dwelling. Most measures of crowding also consider the size of the dwelling and report the numbers of people per room or per bedroom. There is no consensus, however, in defining the point at which a dwelling may be considered ‘crowded’. Crowding is subjective and likely to vary according to culture and context. Thus definitions vary between surveys and between international organizations.

The two most commonly used definitions of crowding are *persons per room* and *households per dwelling unit*. Each of these definitions was included among data collected during the first phase of the Housing Indicators Programme (UNCHS, World Bank, 1992). Surveys have also shown that *floor area per person* is a more precise and policy-sensitive measure than the other two indicators, but is less commonly collected. UN Habitat, the United Nations Centre for Human Settlements (UNCHS) has developed and tested a series of crowding indicators in low-income settlements. These indicators include: the percentage of housing units with more than one household, in-house living area per person, percentage of housing units with more than three persons per room, number of households per building and per housing unit and number of persons per building (UNCHS, World Bank, 1992).

The World Development Indicators report the percentage of people living in crowded dwellings. Crowding refers to the number of households living in dwellings with ≥2 ppr as a percentage of all households in the country and in urban areas (World Bank, 2005).

The American Crowding Index is derived by dividing the number of usual household residents by the number of rooms (excluding bathrooms, porches, balconies, foyers, hallways or half-rooms)(US Census Bureau, 2003). These standards can also be used to convert household crowding density from a continuous exposure variable to a discrete variable for ease of handling (Statistics New Zealand, 2002).

The official Eurostat definition of crowding is >1ppr (Eurostat, 2002) but there is no European national norm to define overcrowding. Alternative definitions and standards in different European countries have been summarized in a report by Edgar and Meert (2005). The Eurostat definition of crowding combines Eurostat data on households and residents with the statistical information on room number and floor area, identifying the number of households with < 1 room per person and the number of households with < 14 square meters per person.

Some crowding measures take into account social as well as physical aspects of crowding. According to these definitions a crowded household is one, where depending on the age, sex and relationship of the household members, one or more additional bedrooms are required to meet the sleeping needs of the members. This measure has been developed by the Canada Mortgage and Housing Corporation and is called the Canadian National Occupancy Standard. Statistics New Zealand has used this standard in its reports on household crowding (Canadian Ministry of Housing Corporation, 1991).

4.2 Summary of exposure levels to household crowding

Household crowding levels for 15 European countries are shown in Table 2 (Eurostat, 2002). These Eurostat data are based on crowding being defined as situations where the number of occupants is higher than the number of rooms. Crowding is markedly higher in the countries of southern Europe: Greece had a proportion of 25.2%, Portugal 20.6%, Italy 21.3% and Spain 14.2%. In the Netherlands and the United Kingdom, in contrast, the proportions were only 0.8% and 3.4% respectively. The World Development Indicators Report provides household crowding data for some additional European countries (Table 2). These data use a higher crowding threshold of >2ppr.

To calculate the PAR we need to estimate the proportion of the population exposed to household crowding. Since most housing data provide only the proportion of households that are crowded, we need to adjust these estimates. Crowded households generally contain more people than the average household. In the United Kingdom the proportion of people exposed to crowding was almost exactly twice the proportion of households classified as overcrowded (with a multiplier ranging from 1.92 to 2.05 over the period from 1998 to 2008, based on the annual General Lifestyle Survey results (Fig. 2). A similar relationship has been seen in New Zealand where the ratio of those exposed to household crowding in relation to the proportion of houses that are

crowded had ranged from 1.86 to 2.00 over the Census periods from 1991 to 2006 (Baker et al., 2011). We have therefore used a multiplier of two in subsequent calculations for converting from the proportion of reported crowded households to the proportion of the population exposed to household crowding.

Table 2. Exposure to household crowding in Europe

Countries	Households living in crowded conditions (%) (2001)[a]	Households living in crowded conditions (%) (2001)[b]	Average household size (2001)[a b]	Total population in 2001 ('000)[c]	Estimated percentage of people exposed to crowding [d]
Subregion Euro A*					
Austria	7.5		2.4	8020.9	15
Belgium	3.9		2.4	10 263.4	7.8
Denmark	3.9		2.2	5349.2	7.8
Finland	8.2		2.1	5181.1	16.4
France	5.7		2.4	60 853.1	11.4
Germany	6.1		2.2	82 259.5	12.2
Greece	25.2		2.6	10 931.2	50.4
Ireland	11.8		3.0	3833.0	23.6
Italy	21.3		2.6	56 967.7	42.6
Luxembourg	6.4		2.5	439.0	12.8
Netherlands	0.8		2.3	15 987.1	1.6
Norway		1.0	2.2	4503.4	2.0
Portugal	20.6		3.0	10 256.7	41.2
Spain	14.2		2.9	40 476.7	28.4
Sweden	4.0		2.9	8882.8	8.0
United Kingdom	3.4		2.3	58 999.8	6.8
Subregions Euro B+C*					
Armenia		4.0	4.3	3801.2	8.0
Estonia		3.0	2.4	1367	6.0
Latvia		4.0	2.7	2364.3	8.0
Lithuania		7.0	2.6	3487	14.0
Russian Federation		7.0	2.8	14 4819.1	14.0
The former Yugoslav Republic of Macedonia		8.0	3.9	2031.1	16.0

a Crowding refers to the number of households living in dwellings with ≥1ppr as a percentage of the total number of households in the country (Data source: Eurostat, 2002)

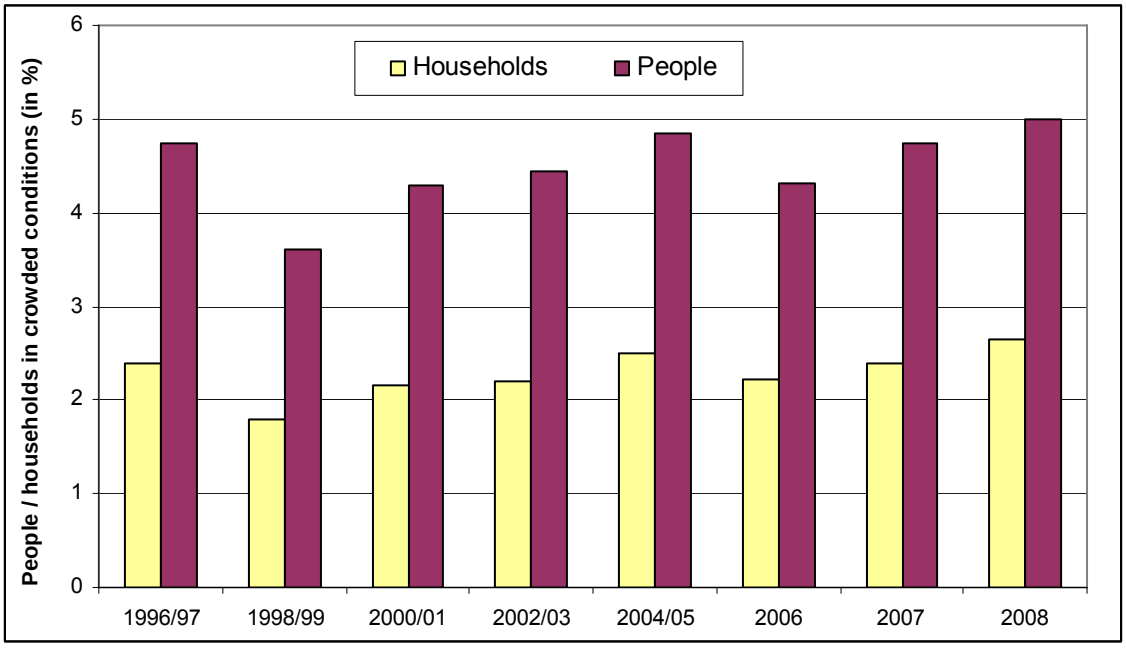
b Crowding refers to the number of households living in dwellings with ≥2ppr as a percentage of the total number of households in the country and in urban areas (Data source: World Development Indicators Report, 2006)

c Data source: WHO Health For All database

d Calculated as double the proportion of households classified as crowded

* The list of countries for the European subregions is provided by Table 1 of the Introduction chapter

Fig. 2. Relationship between households that are crowded and people exposed to household crowding in the United Kingdom, 1996/97-2008.



Data source: General Lifestyle Survey, ONS: Great Britain; reported at: http://www.poverty.org.uk/82/index.shtml

Synthesis of the evidence on exposure to household crowding in Europe

The main focus of this EBD assessment is on Euro B+C. We only have crowding data from the World Development Indicators Report based on the higher crowding level of ≥2ppr and then only for 6 countries (Table 2). These data show a range of crowding levels from 6.0% of the population exposed in Estonia to 16.0% in the former Yugoslav Republic of Macedonia. For this analysis, we will assume an even level of household crowding for the Euro B+C region of 10% of the population exposed to ≥1ppr with a plausible range from 6.0 to 14.0% (which is highly conservative given that the data are based on ≥2ppr).

5. Total burden of disease from TB

5.1 Method for measuring burden of disease from TB

TB data for Europe were obtained from the European Health For All Database operated by the World Health Organization Regional Office for Europe (http://data.euro.who.int/hfadb/). These data are based on newly diagnosed TB cases, all forms (ICD-9:010-018; ICD-10: A15-A19) during the given calendar year. Data are reported annually to WHO by national health agencies. For the purpose of this report, we calculated average annual case counts for a 5-year period centred on 2004 (i.e. 2002-2006 inclusive).

DALYs for TB have also been calculated for all countries of Europe as part of the WHO Global Burden of Disease Project[7]. These data are reported for 2004 (WHO, 2008). More recently, DALYs for TB have been calculated for selected countries in Europe (van Lier et al., 2007) which are broadly similar to WHO estimates. However, because they are only available for selected countries in western Europe, the earlier but more comprehensive Global Burden of Disease Project estimates for 2004 will be used instead.

[7] See http://www.who.int/healthinfo/bod/en/index.html

5.2 Summary of burden of disease from TB

TB burden data for Europe are shown in Table 3.

Table 3. TB incidence, deaths and DALYs for European WHO Member States and grouped into subregions Euro A and Euro B+C

Country	Population[a]	Cases[b]	Deaths[a]	DALYs[a]	Case rate per 100 000	Death rate per 100 000	DALY rate per 100 000
Andorra	72 297	9	1	9	12.17	1.16	13.10
Austria	8 253 381	934	45	634	11.31	0.55	7.68
Belgium	10 359 676	1098	119	1069	10.59	1.15	10.32
Croatia	4 539 880	1210	171	2492	26.64	3.76	54.89
Cyprus	826 813	31	4	69	3.75	0.45	8.29
Czech Republic	10 194 511	1040	69	987	10.20	0.68	9.68
Denmark	5 402 910	375	38	389	6.93	0.70	7.19
Finland	5 231 166	356	68	491	6.80	1.30	9.39
France	60 623 894	5231	946	6348	8.63	1.56	10.47
Germany	82 627 588	6005	455	5229	7.27	0.55	6.33
Greece	11 079 234	603	81	1112	5.44	0.73	10.04
Iceland	292 903	9	4	16	3.21	1.40	5.59
Ireland	4 067 737	382	39	382	9.40	0.96	9.38
Israel	6 573 979	455	46	492	6.92	0.70	7.48
Italy	58 474 754	4020	462	3691	6.87	0.79	6.31
Luxembourg	452 421	37	2	31	8.22	0.39	6.88
Malta	400 124	20	2	13	4.95	0.43	3.25
Monaco	32 394	0	0	2	0.00	0.94	5.84
Netherlands	16 263 535	1216	82	916	7.48	0.51	5.63
Norway	4 608 551	277	46	233	6.01	0.99	5.06
Portugal	10 471 588	3673	378	4920	35.07	3.61	46.98
San Marino	29 641	0	0	0	1.35	0.00	1.63
Slovenia	1 997 206	268	13	273	13.40	0.65	13.67
Spain	42 795 448	7147	462	6473	16.70	1.08	15.12
Sweden	8 997 689	441	91	482	4.90	1.01	5.36
Switzerland	7 392 067	528	28	276	7.15	0.38	3.74
United Kingdom	59 964 948	7332	463	5995	12.23	0.77	10.00
Total Euro A*	**422 026 335**	**42 696**	**4115**	**43 024**	**10.12**	**0.98**	**10.19**
Albania	3 134 402	532	120	1770	16.97	3.83	56.46
Armenia	3 026 880	1721	308	7441	56.85	10.16	245.83
Azerbaijan	8 305 938	5225	844	20 910	62.91	10.16	251.75
Belarus	9 847 821	5228	1120	24 177	53.08	11.37	245.50
Bosnia and Herzegovina	3 905 325	1935	266	3879	49.54	6.80	99.34
Bulgaria	7 794 839	3158	282	4631	40.51	3.62	59.40
Estonia	1 348 345	523	108	2023	38.79	7.99	150.06
Georgia	4 516 983	4354	589	12 463	96.38	13.05	275.91
Hungary	10 113 272	2195	298	4852	21.70	2.94	47.98
Kazakhstan	15 106 857	26 054	2867	68 926	172.47	18.98	456.26
Kyrgyzstan	5 152 508	6278	934	22 927	121.85	18.13	444.97

Country	Population[a]	Cases[b]	Deaths[a]	DALYs[a]	Case rate per 100 000	Death rate per 100 000	DALY rate per 100 000
Latvia	2 315 283	1553	191	3810	67.09	8.25	164.58
Lithuania	3 440 158	2303	322	5974	66.94	9.35	173.65
Poland	38 246 730	8933	932	14 772	23.36	2.44	38.62
Republic of Moldova	3 925 170	4465	676	17 313	113.75	17.23	441.08
Romania	21 725 785	27 411	2103	47 876	126.17	9.68	220.36
Russian Federation	144 695 560	12 5392	39 173	93 5596	86.66	27.07	646.60
Serbia and Montenegro	10 516 739	3681	366	5622	35.00	3.48	53.46
Slovakia	5 386 700	785	54	1028	14.58	1.00	19.08
Tajikistan	6 467 378	4733	2252	45 686	73.18	34.82	706.41
The former Yugoslav Republic of Macedonia	2 030 308	628	96	1455	30.95	4.73	71.65
Turkey	72 024 776	18 576	3867	68 931	25.79	5.37	95.71
Turkmenistan	4 766 006	3448	522	12 844	72.34	10.95	269.50
Ukraine	47 281 800	39 299	10 892	252 755	83.12	23.04	534.57
Uzbekistan	26 208 820	21 398	4120	104 212	81.64	15.72	397.62
Total Euro B+C*	**461 284 383**	**319 807**	**73 302**	**1 691 873**	**69.33**	**15.89**	**366.77**
European Region	**883 310 718**	**362 503**	**77 417**	**1 734 897**	**41.04**	**8.76**	**196.41**

Data sources:

a Population, deaths, DALYs: 2004 data from WHO Global Burden of Disease Project (WHO, 2008), http://www.who.int/healthinfo/bod/en/index.html

b Case data: Average of 2002-06 from European Health for All Database, World Health Organization Regional Office for Europe, http://data.euro.who.int/hfadb/

* The list of countries for the European subregions is provided by Table 1 of the Introduction chapter

6. Environmental burden of TB from household crowding

6.1 EBD estimate for Europe

This analysis made the following simplifying assumptions:

- To restrict the analysis to subregion Euro B+C because the evidence base for the exposure-response relationship is stronger for medium and high TB incidence countries. This estimate uses a RR of 1.5 for those exposed to household crowding ≥1ppr with a plausible range of estimates from 1.2 to 2.0.
- To assume an even level of household crowding for the region but produce a range of plausible estimates. This analysis assumes 10.0% exposed to ≥1ppr with a range from 6.0 to 14.0%.
- To apply this estimate to the total TB rate for subregion Euro B+C. This is an average rate of 69.3/100 000 with a reported range from 14.6 – 172.5 cases/100 000.

The population attributable fraction (PAF) for housing crowding on TB in subregion Euro B+C is:

$$PAF = \frac{p(RR-1)}{p(RR-1)+1} = \frac{0.1 \times (1.50-1)}{0.1 \times (1.50-1)+1} = 0.048$$

Where p = proportion of the population exposed, and RR is the relative risk for the condition in those exposed.

The EBD assessment for the contribution of housing crowding to TB incidence in subregion Euro B+C is therefore:

TB attributable to household crowding = PAF * TB incidence

= 0.048 * 69.3 = 3.3 cases TB/100 000 for subregion Euro B+C

These estimates are shown in Table 4.1 which also includes estimates for deaths and DALYs.

Table 4.1 EBD of TB from household crowding for European subregions

Region	**PAF**	**Cases**	**Deaths**	**DALYS**	**EBD Cases**	**EBD Deaths**	**EBD DALYS**
		per 100 000	**per 100 000**	**per 100 000**	**per 100 000**	**per 100 000**	**per 100 000**
Euro A*	NC	10.12	0.98	10.19	NC	NC	NC
Euro B+C*	0.048	69.33	15.89	366.77	3.33	0.76	17.61
European Region	**NC**	**41.04**	**8.76**	**196.41**	**NC**	**NC**	**NC**

NC = not calculatable

* The list of countries for the European subregions is provided by Table 1 of the Introduction chapter

Based on the incidence of TB (319 807 cases per year), reported TB deaths (73 302), and estimated DALYs (1 691 873) this EBD represents 15 351 TB cases, 3 518 additional deaths, and 81 210 DALYs each year across the Euro B+C subregions that can be attributed to household crowding.

6.2 EBD estimate for specific states in Europe

Table 4.2 presents EBD estimates for all states in subregion Euro B+C. These estimates have been calculated in the same way as for subregion Euro B+C as a whole. They assume a consistent PAF for all states which is then applied to the different TB burden in each state (cases, deaths, DALYs).

Table 4.2 Estimated EBD of TB from household crowding for Euro B+C countries

Country	Cases[a] per 100 000	Deaths[b] per 100 000	DALYS[b] per 100 000	EBD Cases per 100 000	EBD Deaths per 100 000	EBD DALYS per 100 000
Albania	16.97	3.83	56.46	0.81	0.18	2.71
Armenia	56.85	10.16	245.83	2.73	0.49	11.80
Azerbaijan	62.91	10.16	251.75	3.02	0.49	12.08
Belarus	53.08	11.37	245.50	2.55	0.55	11.78
Bosnia and Herzegovina	49.54	6.80	99.34	2.38	0.33	4.77
Bulgaria	40.51	3.62	59.40	1.94	0.17	2.85
Estonia	38.79	7.99	150.06	1.86	0.38	7.20
Georgia	96.38	13.05	275.91	4.63	0.63	13.24
Hungary	21.70	2.94	47.98	1.04	0.14	2.30
Kazakhstan	172.47	18.98	456.26	8.28	0.91	21.90
Kyrgyzstan	121.85	18.13	444.97	5.85	0.87	21.36
Latvia	67.09	8.25	164.58	3.22	0.40	7.90
Lithuania	66.94	9.35	173.65	3.21	0.45	8.34
Poland	23.36	2.44	38.62	1.12	0.12	1.85
Republic of Moldova	113.75	17.23	441.08	5.46	0.83	21.17
Romania	126.17	9.68	220.36	6.06	0.46	10.58
Russian Federation	86.66	27.07	646.60	4.16	1.30	31.04
Serbia and Montenegro	35.00	3.48	53.46	1.68	0.17	2.57
Slovakia	14.58	1.00	19.08	0.70	0.05	0.92
Tajikistan	73.18	34.82	706.41	3.51	1.67	33.91
The former Yugoslav Republic of Macedonia	30.95	4.73	71.65	1.49	0.23	3.44
Turkey	25.79	5.37	95.71	1.24	0.26	4.59
Turkmenistan	72.34	10.95	269.50	3.47	0.53	12.94
Ukraine	83.12	23.04	534.57	3.99	1.11	25.66
Uzbekistan	81.64	15.72	397.62	3.92	0.75	19.09
Euro B+C	**69.33**	**15.89**	**366.77**	**3.33**	**0.76**	**17.61**

Data sources:

a Case data: Average of 2002-06 from European Health for All Database, World Health Organization Regional Office for Europe, http://data.euro.who.int/hfadb/

b Population, deaths, DALYs: 2004 data from WHO Global Burden of Disease Project (WHO, 2008), http://www.who.int/healthinfo/bod/en/index.html

6.3 Sensitivity analysis of EBD estimate

A sensitivity analysis is shown in the summary table. This analysis was based on using

- The RR estimate of 1.5 with range of estimates from 1.2 to 2.0.
- An even level of household crowding for the region of 10.0% with a range from 6.0% to 14.0%. Given that the World Development Indicators use a higher crowding threshold of ≥2ppr this is very conservative as an estimate of those exposed to ≥1ppr.
- To apply this estimate to the total TB rate for subregion Euro B+C. This is an average rate of 69.3/100 000 with a reported range from 14.6 – 172.5 cases/100 000.

7. Uncertainty

7.1 Areas of uncertainty

The analytical approach used in this report has a number of limitations.

Exposure assessment: There was no uniform definition of household crowding used in these epidemiological studies to estimate the exposure-response relationship. The individual studies reviewed here used several different measures of household crowding which in some cases differed from the way household crowding was measured in the exposure data. Single indicators of household crowding, such as person per room (ppr), can only ever provide a limited measure of the intensity of exposure to other people in the home environment. Household crowding is measured in quite different ways in specific studies and routine population surveys. Common measures are having ≥1ppr or ≥2ppr. Some studies simply split the population into quartiles or quintiles and compare the most with the least crowded. We have used a sensitivity analysis derived from using a range of plausible relative risk estimates.

Incomplete exposure data: High quality data on levels of household crowding is not available for all countries. Eurostat data covered most countries in western Europe but did not provide data for central and eastern Europe. We therefore used crowding data from the World Bank (World Development Indicators, 2006). However, these data were only available for six countries in subregion Euro B+C. In addition, these data used the exposure measure of ≥2ppr so are likely to markedly underestimate exposure to ≥1ppr. We also used the simplifying assumption that crowded households contain on average twice as many people as uncrowded households.

Limited evidence base for exposure-response relationship: As noted earlier in this report, the evidence base for quantifying the exposure-response relationship between household crowding and TB is still quite small. For that reason we have used a range of RR (1.2, 1.5, 2.0) to give some indication of the uncertainly that exists. Most of the studies using more robust designs (case-control) have been carried out in developing countries where levels of household crowding are very much higher than in developed countries. It is not certain that these findings can be extrapolated to populations experiencing very low levels of household crowding. Because of this limitation, we have only carried out EBD estimates for subregion Euro B+C.

Lack of stratification of exposure levels and TB risk: Household crowding on its own is not a risk factor for TB. It is only a risk factor in combination with the presence of cases of active TB. In other words, exposure to people with active TB is a ‘necessary cause’. Household crowding can be thought of as interacting with the prevalence of infectious TB in the population to increase the risk of such transmission. Consequently, the effects of household crowding appear relatively strong in populations where there is a moderate to high prevalence of active TB. In many developed countries TB is becoming uncommon in the local-born population so the effects of household crowding are becoming hard to measure at a population level. However, their effects will be important for subpopulations where active TB is still occurring, notably in migrants from high TB prevalent countries. Ideally, we would want to carry out a more sophisticated analysis that considered the distribution of crowding and TB across different segments of the population and the fact that vulnerability to this exposure is likely to vary according to age and other factors.

Confounding: We acknowledge the potential for a number of household exposures and demographic factors to confound the relationship between household crowding and TB. Two of the most important are socioeconomic status and migration from developing countries with high TB rates. Adjustment for these factors was part of the selection criteria for studies included in

our analysis of the exposure-response relationship. Even after considering these factors, there is still considerable potential for unmeasured confounders to operate.

Potential confounders that were not considered by all studies include exposure to active smoking and exposure to indoor air pollution from combustion processes (passive smoking, solid fuel use). There is good evidence that the risk of TB is increased by smoking. A meta-analysis of this association concluded that smoking is a risk factor for both LTBI and TB disease, with a RR of 1.4 to 1.6 for smokers in an infected population (Bates et al., 2007). Exposure to passive smoking is also likely to increase the risk of TB (Lin et al., 2007). An assessment of the EBD of disease from household exposures to indoor smoke from solid fuels concluded that they also contribute to an increased risk of TB. The increased risk for TB in adults over 15 years of age has been estimated at RR 1.5 (95% CI 1.0-2.4)(Desai et al., 2004). Other potential exposures include geographic and cultural differences in the way people occupy their homes and the amount of time they spend indoors. There are likely to be large differences in levels of ventilation between cold and hot climates.

Disease incidence and burden of disease (BoD) data: TB is under intensive surveillance in most countries because of the need to respond to each case. However, disease data will always be incomplete. These limitations are likely to be greater for derived measures such as DALYs which also rely on accurate mortality data.

7.2 Reducing uncertainty

The estimates contained in this report could be improved in a number of ways.

- Data on levels of household crowding should be available for most countries, particularly those that conduct a periodic census. In addition, it should be possible to analyse these data to produce exposure estimates in a more comparable form, notably the percentage of population exposed to ≥1ppr and ≥2ppr.
- Work in this area would be supported by establishing a strong consensus around a definition of household crowding and how it can be measured and quantified in a meaningful way. Similarly, it would be useful to encourage more comprehensive collection and analysis of housing data to allow measurement of exposure to household crowding in comparable ways across countries and subpopulations.
- Researchers should be encouraged to conduct more high quality studies of the relationship between TB and household crowding in a range of settings. It would be particularly useful to have the results of case-control studies carried out in a range of developed low-prevalence countries. Such studies would ideally take advantage of highly discriminatory molecular methods to distinguish recent infections which would allow more accurate assessment of the contribution of household crowding to disease risk.
- Future EBD analyses could attempt to carry out a more sophisticated analysis that considered the distribution of crowding and TB across different segments of the population and the fact that vulnerability to this exposure is likely to vary according to age and other factors.

8. Conclusions

The EBD of TB from household crowding is summarized in Table 5. Reducing levels of household crowding would reduce levels of TB in Europe. The importance of this strategy is dependent on the prevalence of crowding. Its overall contribution to TB reduction is also affected by the rate of TB. In general, high levels of household crowding are associated with high rates of TB. This strategy is therefore likely to be most important in the least developed countries, and the most deprived subpopulations within developed countries.

Table 5. Summary of EBD of TB from household crowding

Housing exposure	Household crowding, people per room (ppr)			
Health outcome	Tuberculosis (TB) cases, deaths and DALYs			
Summary of EBD assessment	About 5% of TB in Europe (B+C sub regions) can be attributed to household crowding which represents 15 351 cases, 3518 deaths, and 81 210 DALYs per year.			
	Level	**Range**	**Geographic scope**	**Source of information**
(a) Exposure risk relationship	RR 1.5	1.2-2.0	Euro B + C*, Developing countries	Case-control and cross-sectional studies with largely consistent findings
(b) Exposure assessment	10.0%	6.0-14.0% people exposed to > 1 ppr	Euro B + C*	World Development Indicators Report
(c) PAF	4.8%	1.2-12.3% TB cases, deaths and DALYs	Euro B + C*	Derived from (a) and (b)
(d) Total burden of disease	319 807 TB cases per year 73 302 deaths per year 1 691 873 DALYs per year	14.6 – 172.5 cases/100 000 1.0-34.8 deaths/100 000 19.1-706.4 DALYs/100 000	Euro B + C*	Country reports to European Office of WHO (see Table 3)
(e) EBD from household crowding	15 351 TB cases (3.3/100 000) 3518 deaths (0.8/100 000) 81 210 DALYs (17.6/100 000)	0.8-8.5 cases per 100 000# 0.2-2.0 deaths per 100 000# 4.4-45.1 DALYs per 100 000#	Euro B + C*	Combined from (c) and (d) above
Main areas of uncertainty	Few etiological studies in developed countries to establish exposure risk relationship Limited geographic scope of household crowding data and some variability in definition of household crowding so specific population attributable fractions could not be calculated for each country Household crowding and TB rates vary considerably between countries, hence wide range in EBD assessment			
Main implications	Reducing household crowding would contribute to reduced transmission of TB Given the importance of household transmission for many infections disease this strategy could help reduce population burden of many other infectious diseases.			

* The list of countries for the European subregions is provided by Table 1 of the Introduction chapter.
# Calculated from range in estimates of PAF, which are in turn based on the range of values for exposure risk relationship and exposure assessment.

9. Policy implications

TB control programs have a strong emphasis on swiftly identifying and treating cases of active disease. This strategy is worthwhile as it has the potential to remove the 'necessary cause' of disease which is exposure to an infectious case. We argue that there is also good evidence to support an additional focus on reducing household crowding as a population health strategy to combat TB.

Reducing levels of household crowding is likely to be most important for those populations with both high rates of TB and high rates of household crowding. This situation applies to some countries in Europe and to specific, usually socio-economically deprived, subpopulations across

that region. These subpopulations are likely to particularly include migrants from high-TB-incidence countries. These findings therefore provide a further argument for housing policies that seek to promote an adequate supply of affordable, and suitable sized, houses to minimise pressure on deprived populations to live in crowded conditions.

Housing policies to reduce household crowding are also likely to contribute to reduced transmission of all infectious diseases that are transmitted from person to person. Such diseases are known to include a range of respiratory infections in children (such as meningococcal disease (Baker et al., 2000; Pereiro et al., 2004), *Haemophilus influenzae* type b (Jafari et al., 1999; Arnold et al., 1993), pneumonia (Victora et al., 1994; Fonseca et al., 1996), bronchiolitis (Bulkow et al., 2002; Cardoso et al., 2004)), enteric infections (such as hepatitis A (Barros et al., 1999; Letaief et al., 2005), *Helicobacter pylori* (Malaty et al., 2001; Broutet et al., 2001)) and infections transmitted from direct skin contact (bacterial skin infections (Cardoso et al., 2004) and hepatitis B (Milne et al., 1987)).

Reducing levels of household crowding may also reduce population vulnerability to pandemic infections, notably influenza. Modelling work has estimated that the home environment is the setting where about half of influenza transmission occurs. Longini et al. (2005) estimate that family members are the source of 28% of transmission and a further 20% occur as part of household clusters. Other evidence about the importance of household transmission of influenza comes from the observation that unvaccinated household contacts of vaccinated children have 42% lower rates of influenza than control households where the children have not been vaccinated (Hurwitz et al., 2000).

10. References

Acevedo-Garcia D (2001). Zip code-level risk factors for tuberculosis: neighborhood environment and residential segregation in New Jersey, 1985-1992. *American Journal of Public Health,* 91:734-741.

Al Kubaisy W, Al Dulayme A, Hashim DS (2003). Active tuberculosis among Iraqi schoolchildren with positive skin tests and their household contacts. *Eastern Mediterranean Health Journal*, 9:675-688.

Antunes JLF, Waldman EA (2001). The impact of AIDS, immigration and housing overcrowding on tuberculosis deaths in Sao Paulo, Brazil, 1994-1998. *Social Science & Medicine,* 52:1071-1080.

Arbelaez MP, Nelson KE, Munoz A (2000). BCG vaccine effectiveness in preventing tuberculosis and its interaction with human immunodeficiency virus infection. *International Journal of Epidemiology,* 29:1085-1091.

Arnold C, Makintube S, Istre GR (1993). Day care attendance and other risk factors for invasive Haemophilus influenzae type b disease. *American Journal of Epidemiology,* 138:333-340.

Aziz MA et al. (2006). Epidemiology of antituberculosis drug resistance (the Global Project on Anti-tuberculosis Drug Resistance Surveillance): an updated analysis. *Lancet,* 368:2142-2154.

Baker M et al. (2000). Household crowding a major risk factor for epidemic meningococcal disease in Auckland children. *Pediatric Infectious Disease Journal,* 19:983-990.

Baker M et al. (2008). Tuberculosis associated with household crowding in a developed country. *Journal of Epidemiology and Community Health*, 62:715-721.

Baker M et al. (2011). *The distribution of household crowding in New Zealand: Census results from 1991 to 2006.* Wellington, He Kainga Oranga/Housing and Health Research Programme.

Barr RG et al. (2001). Neighborhood poverty and the resurgence of tuberculosis in New York City, 1984-1992. *American Journal of Public Health,* 91:1487-1493.

Barros H, Oliveira F, Miranda H. (1999). A survey on hepatitis A in Portuguese children and adolescents. *Journal of Viral Hepatitis,* 6:249-253.

Bates MN et al. (2007). Risk of tuberculosis from exposure to tobacco smoke: a systematic review and meta-analysis. *Archives of Internal Medicine,* 167:335-342.

Beggs CB et al. (2003). The transmission of tuberculosis in confined spaces: an analytical review of alternative epidemiological models. *International Journal of Tuberculosis & Lung Disease,* 7:1015-1026.

Besser RE et al. (2001). Risk factors for positive mantoux tuberculin skin tests in children in San Diego, California: evidence for boosting and possible foodborne transmission. *Pediatrics*, 108:305-310.

Bhatti N et al. (1995). Increasing incidence of tuberculosis in England and Wales: a study of the likely causes. *British Medical Journal*, 310:967-969.

Broutet N et al. (2001). Helicobacter pylori infection in patients consulting gastroenterologists in France: prevalence is linked to gender and region of residence. *European Journal of Gastroenterology & Hepatology,* 13:677-684.

Bulkow LR et al. (2002). Risk factors for severe respiratory syncytial virus infection among Alaska Native children. *Pediatrics*, 109:210-216.

Canadian Ministry of Housing Corporation (1991). *Core housing need in Canada*. Ottawa, Canadian Government Print.

Cantwell MF et al. (1998). Tuberculosis and race/ethnicity in the United States: impact of socioeconomic status. *American Journal of Respiratory and Critical Care Medicine,* 157:1016-1020.

Cardoso MRA et al. (2004). Crowding: risk factor or protective factor for lower respiratory disease in young children? *BMC Public Health,* 4:19.

Clark M, Riben P, Nowgesic E (2002). The association of housing density, isolation and tuberculosis in Canadian First Nations communities. *International Journal of Epidemiology*, 31:940-945.

Classen CN et al. (1999). Impact of social interactions in the community on the transmission of tuberculosis in a high incidence area. *Thorax*, 54:136-140.

Coetzee N, Yach D, Joubert G (1988). Crowding and alcohol abuse as risk factors for tuberculosis in the Mamre population. Results of a case-control study. *South African Medical Journal*, 74:352-354.

Coker R et al. (2006). Risk factors for pulmonary tuberculosis in Russia: case-control study. *British Medical Journal,* 332:85-87.

Davidow AL et al. (2003). Rethinking the socioeconomics and geography of tuberculosis among foreign-born residents of New Jersey, 1994-1999. *American Journal of Public Health*, 93:1007-1012.

Department for International Development (2004). *Tuberculosis Factsheet.* London, Department for International Development. (http://webarchive.nationalarchives.gov.uk/+/http://www.dfid.gov.uk/pubs/files/tuberculosis-factsheet.pdf, accessed 28 June 2008).

Desai MA, Mehta S, Smith KR (2004). *Indoor smoke from solid fuels: Assessing the environmental burden of disease at national and local levels.* WHO Environmental Burden of Disease Series, No.4. Geneva, World Health Organization. (http://www.who.int/quantifying_ehimpacts/publications/9241591358/en/, accessed 2 September 2003).

Deutch S et al. (2004). Crowding as a risk factor of meningococcal disease in Danish preschool children: a nationwide population-based case-control study. *Scandinavian Journal of Infectious Diseases,* 36:20-23.

Drucker E et al. (1994). Childhood tuberculosis in the Bronx, New York. *Lancet,* 343:1482-1485.

Edgar B, Meert H (2005). *Fourth review on statistics of homelessness in Europe: The ETHOS definition of homelessness*. Brussels, European Federation of National Organisations Working with the Homeless.

Elender F, Bentham G, Langford I. (1998). Tuberculosis mortality in England and Wales during 1982-1992: its association with poverty, ethnicity and AIDS. *Social Science & Medicine,* 46:673-681.

Eurostat (2002). Eurostat yearbook 2002: *The statistical guide to Europe data 1990-2000*. Luxembourg, Eurostat.

Fonseca W et al. (1996). Risk factors for childhood pneumonia among the urban poor in Fortaleza, Brazil: a case-control study. *Bulletin of the World Health Organization*, 74:199-208.

Gustafson P et al. (2004). Tuberculosis in Bissau: Incidence and risk factors in an urban community in subSaharan Africa. *International Journal of Epidemiology*, 33:163-172.

Hawker JI et al. (1999). Ecological analysis of ethnic differences in relation between tuberculosis and poverty. *British Medical Journal*, 319:1031-1034.

Heyman DL (2004). *Control of communicable diseases manual*. Washington, American Public Health Association.

Hill PC et al. (2006). Risk factors for pulmonary tuberculosis: a clinic-based case control study in The Gambia. *BMC Public Health,* 6:156.

Hurwitz ES et al. (2000). Effectiveness of influenza vaccination of day care children in reducing influenza-related morbidity among household contacts. *Journal of the American Medical Association,* 284:1677-1682.

Institut de Veille Sanitaire (2008). *EuroTB and the national coordinators for tuberculosis surveillance in the WHO European Region. Surveillance of tuberculosis in Europe. Report on tuberculosis cases notified in 2006.* Saint-Maurice, Institut de Veille Sanitaire.

Jafari HS et al. (1999). Efficiency of Haemophilus influenzae type b conjugate vaccines and persistence of disease in disadvantaged populations. *American Journal of Public Health,* 89:364-368.

Kistemann T, Munzinger A, Dangendorf F (2002). Spatial patterns of tuberculosis incidence in Cologne (Germany). *Social Science & Medicine,* 55:7-19.

Letaief A et al. (2005). Age-specific seroprevalence of hepatitis A among school children in Central Tunisia. *American Journal of Tropical Medicine & Hygiene,* 73:40-43.

Lienhardt C et al. (2005). Investigation of the risk factors for tuberculosis: A case-control study in three countries in West Africa. *International Journal of Epidemiology,* 34:914-923.

Lin HH, Ezzati M, Murray M (2007). Tobacco smoke, indoor air pollution and tuberculosis: a systematic review and meta-analysis. *PLoS Medicine*, 4:e20.

Lockman S et al. (1999). Tuberculin reactivity in a pediatric population with high BCG vaccination coverage. *International Journal of Tuberculosis & Lung Disease,* 3:23-30.

Longini IM et al. (2005). Containing pandemic influenza at the source. *Science,* 309:1083-1087.

Malaty HM et al. (2001). Helicobacter pylori infection in preschool and school-aged minority children: Effect of socioeconomic indicators and breast-feeding practices. *Clinical Infectious Diseases,* 32:1387-1392.

Mangtani P et al. (1995). Socioeconomic deprivation and notification rates for tuberculosis in London during 1982-91. *British Medical Journal,* 310:963-966.

Milne A et al. (1987). A seroepidemiological study of the prevalence of hepatitis B infections in a hyperendemic New Zealand community. *International Journal of Epidemiology,* 16:84-90.

Mishra VK, Retherford RD, Smith KR (1999). Biomass cooking fuels and prevalence of tuberculosis in India. *International Journal of Infectious Diseases*, 3:119-129.

Munch Z et al. (2003). Tuberculosis transmission patterns in a high-incidence area: a spatial analysis. *International Journal of Tuberculosis & Lung Disease,* 7:271-277.

Musher DM (2003). How contagious are common respiratory tract infections? *The New England Journal of Medicine,* 348:1256-1266.

Myers WP et al. (2006). An ecological study of tuberculosis transmission in California. *American Journal of Public Health,* 96:685-690.

The National Foundation for Infectious Diseases (1999). *Factsheet on tuberculosis: A global emergency*. Bethesda, The National Foundation for Infectious Diseases. (http://www.nfid.org/factsheets/tb.shtml, accessed 28 June 2008).

Pereiro I et al. (2004). Risk factors for invasive disease among children in Spain. *Journal of Infection,* 48:320-329.

Perez-Padilla R et al. (2001). Cooking with biomass stoves and tuberculosis: A case control study. *International Journal of Tuberculosis & Lung Disease*, 5:441-447.

Plant AJ et al. (2002). Predictors of tuberculin reactivity among prospective Vietnamese migrants: the effect of smoking. *Epidemiology & Infection*, 128:37-45.

Plant AJ et al. (2005). Results of tuberculosis screening in applicants for migration in Vietnam and Cambodia. *International Journal of Tuberculosis & Lung Disease,* 9:157-163.

Ponticiello A et al. (2005). Deprivation, immigration and tuberculosis incidence in Naples, 1996-2000. *European Journal of Epidemiology,* 20:729-734.

Rathi SK et al. (2002). Prevalence and risk factors associated with tuberculin skin test positivity among household contacts of smear-positive pulmonary tuberculosis cases in Umerkot, Pakistan. *International Journal of Tuberculosis & Lung Disease,* 6:851-857.

Saiman L et al. (2001). Risk factors for latent tuberculosis infection among children in New York City. *Pediatrics*, 107:999-1003.

Shetty N et al. (2006). An epidemiological evaluation of risk factors for tuberculosis in South India: a matched case control study. *International Journal of Tuberculosis and Lung Disease,* 10:80-86.

Siddiqi K, Barnes H, Williams R (2001). Tuberculosis and poverty in the ethnic minority population of West Yorkshire: an ecological study. *Communicable Disease and Public Health,* 4:242-246.

Spence DP et al. (1993). Tuberculosis and poverty. *British Medical Journal,* 307:759-761.

Statistics New Zealand (2002). *Indicator 2c: American Crowding Index: People Per Room*. Auckland, Statistics New Zealand.

Tekkel M et al. (2002). Risk factors for pulmonary tuberculosis in Estonia. *International Journal of Tuberculosis and Lung Disease*, 6:887-894.

Tocque K et al. (1998). Tuberculosis notifications in England: the relative effects of deprivation and immigration. *International Journal of Tuberculosis and Lung Disease*, 2:213-218.

Tocque K et al. (1999). Social factors associated with increases in tuberculosis notifications. *European Respiratory Journal,* 13:541-545.

Tocque K et al. (2001). A case-control study of lifestyle risk factors associated with tuberculosis in Liverpool, North-West England. *European Respiratory Journal*, 18:959-964.

Tornee S et al. (2004). Risk factors for tuberculosis infection among household contacts in Bangkok, Thailand. *Southeast Asian Journal of Tropical Medicine & Public Health,* 35:375-383.

Tipayamongkholgul M et al. (2005). Factors associated with the development of tuberculosis in BCG immunized children. *Southeast Asian Journal of Tropical Medicine & Public Health,* 36:145-150.

United Nations Centre for Human Settlements (1993). *The housing indicators programme: Report of the executive director (Volume I)*. Nairobi, United Nations Centre for Human Settlements.

US Census Bureau (2003). *Structural and occupancy characteristics of housing*. Washington, US Census Bureau.

van Lier EA, Havelaar AH (2007). *Disease burden of infectious diseases in Europe: a pilot study*. RIVM report 215011001/2007. Bilthoven, The National Institute for Public Health and the Environment.

van Rie A et al. (1999). Childhood tuberculosis in an urban population in South Africa: burden and risk factor. *Archives of Disease in Childhood,* 80:433-437.

Victora CG et al. (1994). Risk factors for pneumonia among children in a Brazilian metropolitan area. *Pediatrics,* 93:977-985.

Wanyeki I et al. (2006). Dwellings, crowding, and tuberculosis in Montreal. *Social Science & Medicine,* 63:501-511.

WHO (2002). *The world health report 2002: Reducing risks, promoting healthy life*. Geneva, World Health Organization. (http://www.who.int/whr/2002/en/, accessed 8 November 2004).

WHO (2003). *Introduction and methods: assessing the environmental burden of disease at national and local levels.* WHO Environmental Burden of Disease Series, No. 1. Geneva, World Health Organization. (http://www.who.int/quantifying_ehimpacts/publications/9241546204/en/index.html, accessed 24 August 2006).

WHO (2008). *The Global burden of disease: Update 2004*. Geneva, World Health Organization. (http://www.who.int/healthinfo/global_burden_disease/GBD_report_2004update_full.pdf, accessed 25 September, 2009).

World Bank (2005). *World development indicators*. Washington, The World Bank.

World Bank (2007). *World development indicators report 2006*. Washington, The World Bank.

Yu GP et al. (1988). Risk factors associated with the prevalence of pulmonary tuberculosis among sanitary workers in Shanghai. *Tubercle,* 69:105-112.

Indoor cold and mortality

Janet Rudge

1. Introduction

In Europe alone, there are about one-quarter of a million excess winter deaths each year (Mercer, 2003). Excess winter deaths are conventionally defined (according to Curwen, 1990) as the number of deaths in winter (Dec-March) above the average for the previous and subsequent 4-month seasons (Aug-Nov; Apr-July). A relationship with temperature is evident since, within any one country, numbers of excess winter deaths increase as outdoor winter temperatures fall. However, the proportional excess, defined as the ratio of observed deaths minus expected deaths, divided by the number of expected deaths ((observed – expected)/expected) varies between countries. Those with temperate climates exhibit greater excess than those with extremely cold winters. For example, relative excess winter mortality is approximately twice as high in the United Kingdom compared with the Scandinavian countries (Laake, Sverre, 1996). Therefore, the implication is that outdoor temperature does not account for all the seasonal variation. Indoor temperatures could also play a part, because of corresponding differences in building characteristics and their variable effectiveness in maintaining warm indoor environments in winter. This chapter addresses the evidence for contributions made by excess winter mortality due to indoor winter temperatures to the burden of housing-related disease in Europe.

Previously, the influence of influenza epidemics on numbers of winter deaths has in part confounded the excess due to temperature. However, in recent years influenza-related deaths are known to be a very small percentage of overall deaths in England (Donaldson, Keatinge, 2002) and Scotland (Bowie, Jackson, 2002), while temperature-related excess winter mortality remains strongly evident. A growing body of epidemiological evidence now exists to show links between indoor temperatures and excess winter mortality and morbidity in various European regions, notwithstanding the difficulties of demonstrating direct causality (Eurowinter Group, 1997; Aylin et al., 2001; Wilkinson et al., 2001). The most reliable evidence is currently available for mortality, while morbidity prevalence in relation to indoor temperatures still needs further research.

Cold indoor temperatures are caused by a combination of factors. Firstly, energy inefficient building design and/or heating systems can make homes difficult to heat. In conjunction with poor building characteristics, low household income and high fuel prices both further exacerbate heating affordability. Energy inefficient housing and difficulties with paying heating bills vary widely in Europe (Whyley, Callender, 1997; Healy, 2003).

To date, studies that relate cold homes and health effects have been largely based in the United Kingdom, Ireland and New Zealand, where the fuel poverty issue has a higher profile. Increasingly, epidemiological research is showing that the problem of cold indoor temperatures is nevertheless replicated in other countries. Where buildings are designed primarily for coping with extreme summer temperatures, in Mediterranean climates for example, houses may not effectively protect against low temperatures during the relatively brief, but cold, winter season. Meanwhile, cold-related mortality is consistently far greater than that associated with high summer temperatures (Keatinge et al., 2000), despite the increasing frequency of extreme hot weather events driven by climate change.

2. Summary of the method

The evidence concerning excess winter deaths and housing with low indoor temperatures suggests there is a relationship between excess winter deaths and cold housing. While the evidence is not strong enough to determine a robust quantitative relationship, a preliminary estimate can be made to produce an approximate number of excess deaths that could be attributable to low temperature housing. The main steps required for estimating the disease burden include:

- Compile the number of excess winter deaths for countries and/or regions, defined as the numbers of all-cause deaths in winter (Dec-March) in excess of the average for the previous and subsequent 4-month seasons (Aug-Nov; Apr-July).
- Multiply this number by 30% to derive the number of excess winter deaths that – using the best estimate based on evidence – can be considered attributable to the cold housing conditions.

3. Exposure-risk relationship between low temperature and mortality

3.1 Relationship between outdoor temperature and mortality

About one third of cold-related mortality is explained by indirect effects of influenza, air pollution and season. However, the relationship between cold weather and mortality is largely attributable to the direct effect of exposure to cold temperatures, partially by means of increased stress on the circulatory system. Cold effects become apparent over a relatively short time (within a week), which confirms the direct effect of cold exposure (Kunst et al., 1993).

Up to 70% of excess winter deaths are due to cardiovascular disease (CVD), and about half of the remaining are due to respiratory disease (RD) (Mercer, 2003). In England, half of the total is due to cardiovascular and one third to respiratory disease (Press, 2003). Although greater absolute numbers of excess winter deaths are due to cardiovascular disease, winter has the greatest proportional effect on respiratory disease (Collins, 2000; Kunst et al., 1993). This is also the cause of most excess winter hospital admissions in England and winter pressures on hospital beds (Damiani et al., 2001). However, the relationships between respiratory and cardiovascular disease can confound the numbers of deaths attributed to each (Stewart et al., 2002; Crombie, 1995). In fact, Wilkinson et al. (2004) found that pre-existing respiratory disease was the single strongest predictor of excess winter death among people aged 75 years and over in Britain, but was most clearly associated with death from cardiovascular disease. Deaths directly attributed to influenza and hypothermia represent only a small proportion of excess winter mortality (Bowie, Jackson, 2002).

There is normally a ‘U-shape’ relationship observed between mortality and mean daily (outdoor) temperature, with numbers of deaths increasing as temperatures either fall below or rise above a certain threshold. The shape of the relationship is found to vary with latitude (Curreiro et al., 2002). The optimum or threshold (external) temperature band is described by Kunst et al. (1993) as 20-25°C as the daily maximum, or 15-25°C average temperature (Ballester et al., 2003) but it varies according to climate. However, within any one country in the northern hemisphere, excess winter mortality generally increases in areas furthest north. This may appear to contradict the findings that there is a relatively large impact in temperate climates. Nevertheless, it is consistent with effects of increasing latitude on temperature whilst influential conditions other than temperature remain similar throughout any one country. Relative temperature change, rather than absolute low temperature levels, may be most

important (Rudge, 1996). Increased temperature variability showed more direct effect on respiratory mortality than extreme hot or cold days (Braga et al., 2002). Cold effects are more delayed than those of heat (Kunst et al., 1993).

The Eurowinter study (1997) used a threshold of 18°C for comparisons across eight regions of widely varying climates in Europe. Mortality rates in each region for ischaemic heart disease (IHD), cardiovascular disease (CVD), respiratory disease (RD) and all causes were at or near their minimum value when the mean daily temperature was 18°C. Other studies report temperatures for lowest mortality varying from 10°C in Oslo, or 14°C in Finland, to 20°C in England. This makes it difficult to select a common threshold below which to compare the excess winter effect. Table 1 illustrates the variation of measures and criteria used in some of these studies.

Table 1: Comparison of measures used in European studies of excess winter mortality

Location	**Numbers /% increase of deaths per °C reduction below threshold**			**Threshold**	**Age group**	**Reference**
	All cause	**Respiratory Disease (RD)**	**Cardiovascular Disease (CVD)**			
Finland	2000-3000 extra deaths in 'cold season'	Relative excess daily mortality: 90%	Coronary heart disease: 30%; cerebrovascular: 40%	14°C	(80% are 65+)	Nayha, 2005
United Kingdom (England and Wales)	3500 approx (i.e. 2 per 10 000)			'winter'	45 +	Laake, Sverre, 1995
United Kingdom (Scotland)	2.9%	4.8%	3.4%	11°C (daytime mean)		Carder et al., 2005
Netherlands		5.15%	1.69%	16.5°C		Huynen et al., 2001
London, United Kingdom	4.2%			5.25°C		Pattenden et al., 2003
Sofia, Bulgaria	1.8%			~0.46°C		Pattenden et al., 2003
United Kingdom (England)			2%	19°C		Wilkinson et al., 2001
Oslo, Norway	1.4%	2.1%	1.7%	10°C		Nafstad et al., 2001
8 regions incl:						
South Finland	0.27%			18°C	50-59 and 65-74	Eurowinter Group, 1997
London	1.37%					
Athens	2.15%					

Researchers for the project 'Assessment and prevention of acute health effects of weather conditions in Europe' (PHEWE) considered weather-related mortality variations in 16 cities throughout Europe. The PHEWE project defines winter: Oct – Mar; summer: Apr- Sept, whereas the conventional definition is winter: Dec-Mar; summer: previous Aug-Nov and

following Apr-Jul. The selected exposure indicators were the maximum apparent temperature, for different lag periods in different seasons, which is an index of thermal discomfort dependent on air temperature and dewpoint temperature (Michelozzi et al., 2007). Results of this study published so far have confirmed that increases in emergency winter hospital admissions were particularly noticeable for respiratory disease in all 16 cities studied.

The Eurowinter study (1997) considered climate, home temperature and some aspects of individual behaviour in relation to seasonal mortality. It found greater increases in all-cause mortality with a given fall of temperature in regions with warmer winters, in populations with cooler homes, and among people who exhibited less protective behaviour against the cold. This illustrates some of the complexity of identifying causal effects of indoor cold on health, because of the inter-relationships between climate, buildings, expectations and behaviour. Another study compared the number of energy efficiency measures present and affordability of heating bills with national seasonal mortality data (Healy, 2003). Those countries with the poorest housing, judged by certain indicators within the available data, had the highest excess winter mortality, and this also coincided with countries that have milder climates.

3.2 Relationship between outdoor temperature and morbidity

To date, there have been few studies of cold-related outcomes other than deaths. A London-wide study showed that respiratory general practitioner consultations increased by 10% per degree Celsius (°C) decrease below 5°C (Hajat, Haines, 2002). Some studies have shown winter peaks of hospital admissions for heart failure in Spain (Martinez-Selles et al., 2002) and in Scotland (Stewart et al., 2002). Maheswarana et al. (2004) found that only respiratory disease showed a winter excess for hospital admissions in South Yorkshire, England. An index related to risk of cold homes is a predictor of excess winter emergency hospital episodes for respiratory disease (Rudge, Gilchrist, 2007).

Mortality statistics do not fully reflect the levels of morbidity due to cold-related disease, but numbers of deaths are more available than hospital admissions. Excess winter deaths are therefore the outcome selected here for consideration

3.3 Relationship between indoor temperature and mortality

The link between excess winter deaths and cold temperatures is well established. Considering that people spend much of their time indoors, it has been argued that there is a theoretical basis for suggesting that home heating is a modifier of some of the risk posed by low outdoor temperatures (University College London et al., 2006). This appears to be borne out by the various studies showing associations between poor housing or colder homes and excess winter mortality. It is further supported by the fact that countries with more extreme winter climates, which generally have more energy efficient housing, exhibit lower excesses of winter deaths.

Most excess winter deaths are attributed to cardiovascular and respiratory diseases (Wilkinson et al., 2001; Aylin et al., 2001; Khaw, 1995). According to Khaw (1995), the seasonal variation in blood pressure is more strongly related to indoor than to outdoor temperature. Cardiovascular conditions include ischaemic heart disease and stroke; respiratory conditions affected or exacerbated by the cold include influenza-like disease, asthma, Chronic Obstructive Pulmonary Disease (COPD), and respiratory viruses. The biological mechanisms for the effect of cold on these groups of diseases are explained in a WHO Environmental Health Series Report (WHO, 1987). This report concluded that there is no demonstrable risk to the health of ‘healthy sedentary people living in temperatures of between 18 and 24°C’, assuming appropriate clothing, insulation, humidity, radiant temperature, air movement and stable physiology. However, for certain vulnerable groups, including the very old, a minimum of 20°C was recommended, while temperatures below 12°C were thought to be a health risk for similar groups. According to Collins (1986), below 16°C there is increased risk from respiratory

infection, while below 12°C there is increased strain on the cardiovascular system. After 2 hours or more at less than 6°C, deep body temperature falls and there is risk of hypothermia. Temperature variations within a building can also cause thermal stress on the respiratory and circulatory systems (Lloyd, 1990; Hunt, 1997; Goodwin, 2000). In this respect, it should be noted that measured average temperatures disguise the extremes that can be experienced within the home. For example, homes without central heating tend to display a wider range of temperatures between rooms than homes with central heating, although they may present as having very similar average whole house temperatures (Rudge, Winder, 2002).

Indoor cold is known to exacerbate the respiratory condition known as chronic obstructive pulmonary disease (COPD) (Collins, 2000), which is also characterized by repeat hospital admissions. For example, this diagnosis accounted for more than 40% of emergency respiratory hospital episodes in one London Borough over a 4-year period (Rudge, Gilchrist, 2007), where there was found to be a noticeable winter excess for emergency respiratory episodes in general.

People appear to be better protected going out from a warm house into cold outdoor conditions than from a cold house (Goodwin, 2000), indicating the importance of the link between effects of indoor and outdoor conditions. The proportion of excess winter mortality associated with respiratory and cardiovascular diseases is widely described as the proportion that is cold-related (Wilkinson et al., 2001), without disaggregating the causes as indoor or outdoor cold.

3.4 Relationship between indoor temperature and morbidity

A decrease in living room temperature is associated with increased blood pressure, which increases cardiovascular risk (Khaw, 1995). Increased indoor temperatures as a result of housing interventions (heating systems and thermal insulation) have a significant impact on health conditions, improving both the mental health of the affected residents as well as the physical health conditions (cardiovascular and respiratory) (Green, Gilbertson, 2008; Howden-Chapman et al., 2007). Walker et al. (2006) showed that increased heating use and higher temperatures are associated with reduced levels of environmental problems such as mould and condensation, which are predictive for general health outcomes and specifically adult wheezing, similar to a study of thermal insulation improvements coordinated by WHO (2008).

3.5 Population at risk

According to Laake and Sverre (1996), age is the most important risk factor for a winter death. Older people are at greatest risk of indoor cold-related health effects because they generally spend more time indoors and are less mobile, while their thermo-regulatory system may also be impaired. In the United Kingdom, older people are the most likely to be living in least energy efficient housing and unable to afford sufficient heating for comfort (DEFRA, 2006). For England and Wales about 93% of excess winter deaths are among those over 64 years old (Hajat et al., 2007). Similarly, data from 20 western European countries showed a highly significant positive correlation between total mortality rates for those aged 65 years and over and relative excess winter mortality (Laake, Sverre, 1996). This is therefore the population group considered most relevant for the purposes of estimating the burden of disease due to cold homes.

4. Exposure assessment

4.1 Evidence from population surveys

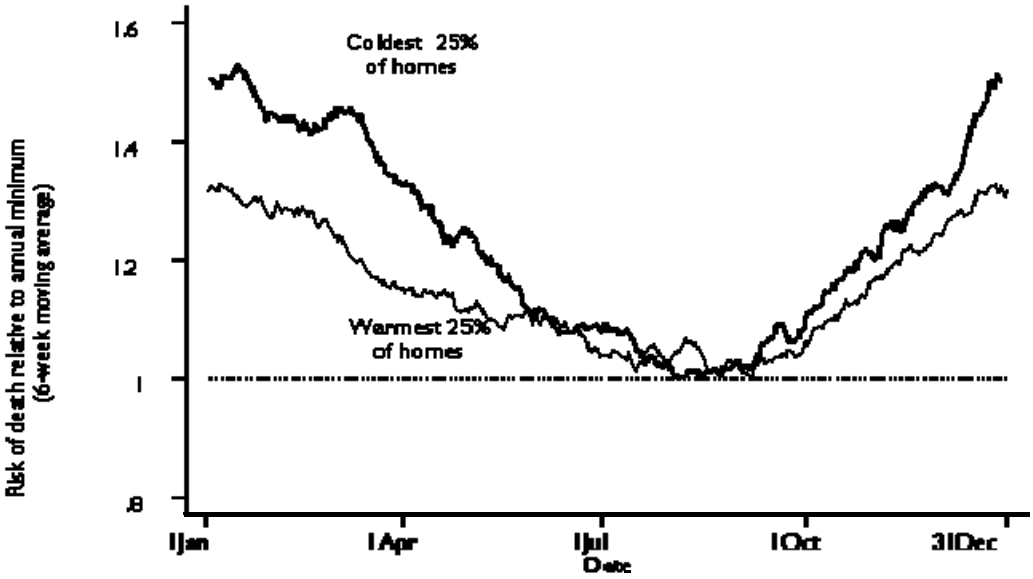
Few research papers have attempted to determine the fraction of excess winter deaths attributable to housing, due to the complex nature of establishing direct causality and difficulties in separately distinguishing the effects of indoor and outdoor cold.

The Eurowinter study (1997) used regional mortality for two age groups, 50-59 and 65-74 years, in Finland, the Netherlands, Germany, England, Italy and Greece and concluded that "striking differences indoors were higher living room temperatures and more frequent bedroom heating in the colder countries, all at a given level of outdoor cold". This indicates the differences found between temperate and more extreme climates and the effect of indoor as opposed to outdoor temperatures. Percentage increases in deaths were calculated per 1°C fall in temperature below 18°C, by region, over a period of up to 4 years. Evidence showed links between mortality and home heating independent of outdoor cold stress, and outdoor cold stress independently of home heating, despite correlations between cold exposure factors. Outdoor cold stress was indicated by the proportion of people who became cold enough to shiver at 7°C, controlling for age and gender. Various mortality indices were significantly related to bedroom heating hours or to living room temperature, independent of outdoor stress, and to outdoor stress independent of indoor factors. Keatinge and Donaldson (2000) suggest that half of excess winter deaths are attributable to indoor cold and half to outdoor cold. In Siberia, warm clothing and warm housing prevented any increase in CVD mortality as outdoor temperatures fell to extremes of -48.2°C (Donaldson et al., 1998).

Clinch and Healy (2000) compared excess winter mortality in Ireland with Norway over 1986-1995. Ireland has notably poor energy efficiency standards and a mean dwelling temperature of 15°C, while Norway has high thermal efficiency standards and indoor home temperatures (21°C on average). After controlling for multiple confounding variables, over 40% of excess winter mortality in Ireland attributable to cardiovascular and respiratory diseases might be associated with poor thermal housing standards. These diseases accounted for 85% of the total. Indoor attributed deaths were disaggregated from outdoor attributed deaths by comparing the mean excess winter death rates for both diseases in Norway and Ireland over the ten year period.

Aylin et al. (2001) found a significant association between winter mortality and temperature, with a 1.5% higher odds of dying in winter with every 1°C reduction in 24 hour mean winter temperature. Respiratory disease showed the strongest associations with temperature. Lack of central heating was associated with a higher risk of dying in winter (OR 1.016 (95% CI: 1.009-1.022) for all causes). Wilkinson et al. (2001) reported greater excess cold-related deaths were associated with low indoor temperatures, older buildings and thermal efficiency. Notably, low socioeconomic status was not strongly related to winter death unless considered in combination with the cost of home heating. This research found a 20% greater risk of excess winter death in the predicted 25% coldest homes than in the predicted 25% warmest homes (see Fig 1). On average, the effect of cold weather on cardiovascular mortality decreased by 0.15% for each increase in indoor temperature of one degree (95% CI 0.03%, 0.28%) (University College London et al., 2006). Of all-cause excess winter deaths (including flu), 50-60% in England and Wales are specifically cold-related, being attributable to cardiovascular or respiratory diseases (Wilkinson et al., 2001).

Fig. 1: Seasonal fluctuation in mortality in cold and warm homes



Based on Wilkinson et al., 2001. Curves represent top and bottom quarters of the distribution of predicted indoor (hall) temperatures at 5°C outside temperature.

4.2 Evidence from intervention studies

The Watcombe Housing Study, in southwest England, included 480 participants of 119 local authority owned houses that received a range of upgrades, including central heating and insulation (Barton et al., 2007). The interventions improved energy efficiency, producing warmer, drier houses. For those living in the intervention houses, some respiratory conditions improved significantly compared with the control group. One of the general benefits was increased whole-house comfort, which contributed to improved self-reported well-being. The Warm Front study, which evaluated the English government energy efficiency programme, also found psychosocial benefits from improved thermal comfort and expanded use of space (Wilkinson et al., 2007). Howden-Chapman et al. (2007) demonstrated that installing insulation led to significantly warmer and drier homes, significantly improved self-reported health and fewer general practitioners' visits and hospital admissions for respiratory conditions. The research team suggest that health benefits may not have been due to average temperature and humidity changes, which were relatively small, but rather to larger changes in exposure to very low temperatures and high humidity.

5. Total burden of deaths related to cold

About 60% of the variation in excess winter deaths is due to cold (Wilkinson et al., 2001). The upper limit on the burden of cold-related deaths is determined by the Excess Winter Death Index, which can be calculated from national mortality statistics. The lower limit, even if it were a small proportion of the total, is likely to imply a substantial figure. For example, in the United Kingdom a middle estimate could represent an annual figure of between 5000 and 20 000 excess winter deaths (Wilkinson, personal communication, 2006). Kunst et al. (1993) concluded that the relation between cold weather and mortality "is largely attributable to the direct effects of exposure to cold temperatures", and state that approximately one third of cold-related mortality can be explained by the indirect effects of influenza, air pollution and season. However, the remaining two-thirds could not be fully attributed to direct effects because of other potential confounders that were not accounted for in their study. The conclusion from this work would

therefore be that an upper limit of about 65% of winter excess deaths is directly related to (both indoor and outdoor) cold temperatures. Keatinge and Donaldson (2000) estimate 50% apportionment of excess winter deaths to indoor temperatures. The study from Ireland states that 40% of respiratory and cardiovascular deaths are related to indoor temperature and that these diseases account for 85% of the total. Therefore indoor cold-related deaths are 34% (40% of 85%) of total excess winter deaths (Clinch, Healy, 2000). In short, the literature shows that between 30% and 50% of excess winter mortality is attributable to housing.

Table 2 offers a summary of studies and expert opinions on the extent of the indoor effect of cold on excess winter deaths. Some of these studies have not included further potential confounders of the health relationship with low temperatures, such as socioeconomic status or poverty, although others have. However, various studies in England have found little or no link between deprivation and excess winter mortality (Shah, Peacock, 1999; Wilkinson et al., 2001; Aylin et al., 2001). This is probably because the deprivation measures conventionally used depend on housing tenure, which is not necessarily a good indication of low indoor temperatures. For example, social housing is an indicator of low income, but housing in this category is generally more energy efficient than the private rented or owner-occupied sectors.

Based on these estimates from different studies and sources, a conservative estimate of about 30% of total excess winter deaths is related to cold housing. This estimate is highly unlikely to over-estimate the burden of disease calculation.

6. Environmental burden of deaths from exposure to cold housing

The housing-related burden of deaths from exposure to cold housing is half of the total burden from direct effects of cold, which in section 5 was estimated to be 60%. Thus, the housing-related burden of death from exposure to cold housing is 30% of the total (Wilkinson, personal communication, 2006). As Mercer (2003) points out, while many countries clearly regulate for indoor climatic conditions in public buildings (or workplaces) there is little or no regulation (as opposed to recommendations) for private homes. Published data on indoor climatic conditions and thermoregulatory behaviour patterns in private homes are also scarce. Since outdoor temperature data are readily available, while data on indoor conditions is not, the assessment of exposure to low indoor temperatures must rest with some threshold of outdoor temperature, based on available evidence. The Eurowinter Group (1997) considered a wide range of climates in their study, and concluded that, since mortality rates were least in all regions surveyed at, or near, the mean daily temperature of 18°C, this should be the threshold below which excess mortality was calculated.

Some of the studies described earlier provide evidence to indicate that many households in Europe and elsewhere experience indoor temperatures below 16°C. This temperature is the threshold suggested by Collins (1986) below which there is increased risk of respiratory infections. It is evidently below the 18°C described as comfortable for normal sedentary activity in living rooms.

Wilkinson et al. (2001) found that hall temperatures below 16°C at an outside temperature of 5°C ranged from 39% of the oldest to 15% of the most recently built properties. The Warm Front study found standardized daytime living-room and night-time bedroom temperatures to be less than 16°C in 21% and 46% of dwellings respectively (Hutchinson et al., 2006). Moreover, 20% of standardized living-room temperatures were still below 14.9°C in homes that received heating and insulation interventions (University College London et al., 2006). According to Clinch and Healy (2000), Ireland has a mean dwelling temperature of 15°C, while Norway has indoor home temperatures of 21°C on average. This serves to illustrate further that countries with climates that may be regarded as mild tend to have indoor temperatures lower than those with more extreme winters.

Table 2: Summary of assessments of indoor cold effect on excess winter deaths.

Author/year	Location/years	Study design/population	Exposure assessment	Outcome assessment	Adjusted co-variates	EWDs attributable to direct cold effects	EWDs attributable to 'cold' housing
Eurowinter Group, 1997	Regions in Finland, Italy, Netherlands, Germany + London, (1988-92) Palermo and Athens (1992)	• Regional mortality vs mean outdoor winter temp (Oct-March) for 2 age groups: 50-59 and 65-74 years, male/female; • Behavioural, heating and temperature survey of c.1000 persons per region	No. days per year colder than 18°C	% increased daily mortality (all cause) per °C fall from <18°C (range: 0.27% in S Finland to 2.15% in Athens)	Adjusted for outdoor temp of 7°C: • Living room temps • Bedroom heating hours • No. outdoor excursions • Outdoor clothing levels and activity/shivering		Mortality indices related to indoor temperature factors independently of outdoor cold stress and vice versa
Keatinge, Donaldson, 2000; Keatinge, 2007	Based on evidence from Eurowinter study (above) Personal communication						50%
Wilkinson et al., 2001	England, 1986-96	Linked datasets by postcode: • CVD mortality statistics • Housing data for 21 000 dwellings covered by EHCS, matched by postcode to regional	% households with predicted hall temp. < 16°C at 5°C outdoor temp, at 3pm, after 4 hours of central heating	20% greater risk of excess winter CVD deaths in predicted 25% coldest homes than in 25% warmest	• Age of dwellings • Lack of/dissatisfaction with heating system • Cost of heating the dwelling • Low household income • Household size	60%	
Wilkinson, 2006	Personal communication based on accumulated research experience in England					50-60%	ca. 30% (half of total cold-related EWDs)
Clinch and Healy, 2000	Ireland 1986-95	Cross country comparison with Norway as 'control group', with high levels of energy efficiency housing standards + mean indoor temp of 21°C	Poor thermal housing standards + mean dwelling temp of 15°C in Ireland	50% CVD and 57% RD excess winter deaths in Irish residents	Adjusted for RD/CVD risk factors: • Demography • Smoking prevalence • Diet • Obesity rates • Level of air pollution		40% of combined CVD and RD deaths; i.e. 34% of total (CVD and RD form 85% of total)
Healy, 2006	United Kingdom (England, Wales), Ireland Personal communication based on own research						ca. 33%

The evidence on indoor temperatures is summarized in Table 3.

Table 3: Summary of exposure from indoor temperature data in reference studies

Country/year	Average indoor °C			Number of dwellings	Standardized condition	Building type/occupants	Reference
	Hall*	Living-room	Bed-room				
England 2001-2002 (Warm Front Study)		21% <16°C (daytime)	46% <16°C (night-time)	470 (pre-intervention)	Outdoor 5°C	Privately owned/rented households qualifying for receipt of Warm Front grant	Hutchinson et al., 2006
England 1991	<16°C: 29% all dwellings; almost 20% post-1980 dwellings			21 173 (EHCS data)	Predicted for outdoor 5°C, at 3pm, after 4 hours central heating	Nationally representative sample	Wilkinson et al., 2001
Athens 1994-1995		19.2°C			Outdoor temp 7°C		Eurowinter, 1997
S Finland 1994-1995		21.7°C			Outdoor temp 7°C		Eurowinter, 1997
Frankfurt, Germany 2006	Av house temp: 8.5% homes <18°C, 50% of time			351			WHO, 2007b
New Zealand 2001-2002			13.2	679		Uninsulated dwellings/ households including respiratory patient	Howden-Chapman et al., 2007
Ireland	Mean dwelling temp: 15°C						Clinch, Healy, 2000
Norway	Average indoor home temp: 21°C						Clinch, Healy, 2000

* note that the hall temperature is sometimes used as representative of the mean whole house temperature

Table 4 shows the estimated annual excess winter deaths related to indoor cold temperatures for certain countries of the WHO European Region, calculated as 30% of the average total excess, according to available figures.

Table 4: Estimated excess winter deaths (EWD) related to cold housing per year by country

Country	Source of EWD data	Year range	EWD per year	Estimated EWD due to cold housing per year
England and Wales	National statistics site. Available at: http://www.statistics.gov.uk/StatBase/ssdataset.asp?vlnk=7089&Pos=&ColRank=2&Rank=272	1991-2005*	31 772	9532
Germany	WHO (2007a): Housing, energy and thermal comfort. A review of 10 countries within the WHO European Region. WHO Regional Office for Europe, Copenhagen. Available at: http://www.euro.who.int/document/e89887.pdf	1992-2003	32 119	9636
Poland		1991-2002	14 680	4404
Portugal		1991-2003	9047	2714
Romania		1991-2004	17 538	5261
Turkey		2001-2003	8622	2587
The former Yugoslav Republic of Macedonia		1995-2004	884	265
Armenia	Bonnefoy, Kim, Green, Monolbaev (unpublished data): Excess Winter Deaths in central and eastern Europe. Data available at http://www.apug.nrw.de/pdf/vortrag-perspektive-who.pdf	1990-1999	2149	645
Bulgaria		1990-2000	7367	2210
Kyrgyzstan		1990-1999	1585	476
Lithuania		1991-2000	1578	473
Total estimated EWD/year for the 11 countries			**127 341**	**38 203**

* provisional figures for 2005/2006

7. Uncertainty

Seasonal mortality studies are all subject to the ecological fallacy, where the characteristics of areas are assumed to apply to individuals in those areas. This is recognized by Clinch and Healy (2000), who did not claim to have demonstrated causality. Similarly, the Eurowinter study used building, heating and behaviour characteristics of individuals in their samples as representative of the regions studied and related these to previous years' regional mortality data. This necessarily gives rise to uncertainty with regard to apportioning the effect of indoor temperatures on health.

Differing measures used in different studies, such as excess death definitions, consideration of different lag effects, comparison of building characteristics, or indicators of energy efficiency, or measures for fuel poverty or cause of death also limit the ability to compare studies The definition of excess winter deaths as used by Curwen (1990) depends on 'winter' being December to March. The actual excess due to cold weather in that case may be underestimated when, for example, the months of November or April include cold spells (Donaldson et al., 2001).

8. Conclusion

At present, mortality data, as opposed to morbidity, are the only routinely available national data. The annual burden of disease due to cold homes can be conservatively estimated as a

proportion of 30% excess winter deaths, according to available evidence and expert opinion, and equals 38 200 EWD for the eleven countries covered (Table 5). This is related to a temperature threshold of 18°C (Eurowinter, 1997). However, a different threshold for different parts of Europe may be advisable to account for differences between those with cold and those with mild winters.

Although cold-related deaths are regarded as those attributed to cardiovascular and respiratory disease, excess winter mortality statistics are not routinely published according to cause. If they were available in the future, the proportion attributable to RD and CVD could then be the basis for calculating the burden attributable to cold housing, as this proportion may vary from country to country. This is likely to increase the 30% estimate. The population group most at risk is 65 years or older.

Table 5. Summary of EBD of cold housing

Housing exposure	Indoor winter temperature from inadequate housing design and energy inefficiency		
Health outcome	Excess winter mortality primarily from cardiovascular and respiratory disease		
Summary of EBD evidence	Excess winter deaths (EWD) due to cold homes account for 30% of all EWD and cause an estimated 38 203 deaths per year for 11 countries with available mortality data.		
	Level	**Geographic scope**	**Source of information**
(a) Exposure risk relationship	60% of excess winter deaths due to cold temperatures both indoor and outdoor and 30% of excess winter deaths due to low indoor temperatures	England	Wilkinson et al., 2001
(b) Exposure assessment	Effect of cold weather on cardiovascular mortality decreased by 0.15% for each increase in indoor temperature by one °C	England	University College London et al., 2006
(c) PAF	30% of excess winter deaths are related to cold housing	Europe	Eurowinter, 1997; Clinch, Healy, 2000 (see Table 2)
(d) Total burden of disease	127 341 excess winter deaths per year	Eleven selected European countries	See Table 4
(d) EBD from cold temperatures in housing	38 203 excess winter deaths per year (12.8/100 000)	Eleven selected European countries	See Table 4 (based on the average population size of the eleven countries for the reported years)
Main areas of uncertainty	Ecologic fallacy. Building, heating and behaviour characteristics create uncertainty in apportioning the effect of indoor temperatures on health. Differing definitions of excess deaths, winter, different lag effects, indicators of energy efficiency, or measures for fuel poverty or cause of death.		
Main implications	The burden of disease (excess winter deaths) from cold can be reduced by measures that result in warmer indoor temperatures.		

9. Policy implications

Howden-Chapman et al. (2007) showed the beneficial health impact of improving domestic insulation, specifically, which further supports the hypothesis that the burden of disease from cold-related disease can be reduced by measures that result in warmer indoor temperatures. Their study also shows that it is possible to carry out randomized studies of housing

interventions related to cold housing, using appropriate study design and consultation with the sample population, so that further evidence may be forthcoming from future research.

10. References

Aylin P et al. (2001). Temperature, housing, deprivation and their relationship to excess winter mortality in Great Britain, 1986-1996. *International Journal of Epidemiology*, 30:1100-1108.

Ballester F, Michelozzi P, Iniguez C (2003). Weather, climate and public health. *Journal of Epidemiology and Community Health*, 57:759-760.

Barton A et al. (2007). The Watcombe Housing Study: the short term effect of improving housing conditions on the health of residents. *Journal of Epidemiology and Community Health*, 61:771-777.

Bonnefoy X et al. *Excess Winter Deaths in central and eastern Europe.* Copenhagen, WHO Regional Office for Europe. (http://www.apug.nrw.de/pdf/vortrag-perspektive-who.pdf, accessed 19 November 2009)

Bowie N, Jackson G (2002). *The Raised Incidence of Winter Deaths*. Occasional Paper No.7. (http://www.gro-scotland.gov.uk/statistics/library/occpapers/the-raised-incidence-of-winter-deaths.html, accessed 17 October 2007)

Braga ALF, Zanobetti A, Schwartz J (2002). The effect of weather on respiratory and cardiovascular deaths in 12 US cities. *Environmental Health Perspectives*, 110:859-863.

Carder M et al. (2005). The lagged effect of cold temperature and wind chill on cardiorespiratory mortality in Scotland. *Occupational and Environmental Medicine,* 62:702–710.

Clinch JP, Healy JD (2000). Housing standards and excess winter mortality. *Journal of Epidemiology and Community Health*, 54:719-720.

Collins KJ (1986). Low indoor temperatures and morbidity in the elderly. *Age and Ageing*, 15:212-220.

Collins KJ (1993). Cold and heat-related illnesses in the indoor environment. In: Burridge R, Ormandy D, eds. *Unhealthy Housing: Research, Remedies and Reform*. London, E&FN Spon Ltd.:117-140.

Collins KJ (2000). Cold, cold housing and respiratory illnesses. In: Rudge J, Nicol F, eds. *Cutting the cost of cold: Affordable Warmth for Healthier Homes.* London, E&FN Spon Ltd.:36-47.

Crombie DL et al. (1995). Concurrence of monthly variations of mortality related to underlying cause in Europe. *Journal of Epidemiology and Community Health*, 49:373-378.

Curriero FC et al. (2002). Temperature and Mortality in 11 Cities of the Eastern United States. *American Journal of Epidemiology*, 155:80-87.

Curwen M (1990). Excess winter mortality: a British phenomenon? *Health Trends,* 22:169–175.

Damiani M, Dixon J (2001). *Managing the pressure*. London, King's Fund.

DEFRA (2006). *The United Kingdom Fuel Poverty Strategy – 4th Annual Progress Report*. London, Department for Environment, Food and Rural Affairs. (http://www.dti.gov.uk/files/file29688.pdf, accessed 01 December 2007).

Donaldson GC, Keatinge WR (2002). Excess winter mortality: influenza or cold stress? Observational study. *British Medical Journal,* 324:89-90.

Donaldson GC et al. (1998). Cold related mortalities and protection against cold in Yakutsk, eastern Siberia: observation and interview study. British Medical Journal, 317:978-982.

Donaldson G et al. (2001). Heat- and cold-related mortality and morbidity and climate change. In: Department of Health *Health effects of climate change in the United Kingdom*. Expert Group on Climate Change and Health in the United Kingdom. London, Department of Health.

Eurowinter Group (1997). Cold exposure and winter mortality from ischaemic heart disease, cerebrovascular disease, respiratory disease, and all causes in warm and cold regions of Europe. *Lancet,* 349:1341-1346.

Goodwin J (2000). Cold stress, circulatory illness and the elderly. In: Rudge J, Nicol F, eds. *Cutting the cost of cold: Affordable Warmth for Healthier Homes*. London: E&FN Spon Ltd.:48-61.

Green G, Gilbertson J (2008). *Warm Front Better Health*. Health Impact Evaluation of the Warm Front Scheme. Published by the Centre for Regional, Economic and Social Research – Sheffield Hallam University, United Kingdom.

Hajat S, Haines A (2002). Associations of cold temperatures with GP consultations for respiratory and cardiovascular disease amongst the elderly in London. *International Journal of Epidemiology*, 31:825-830.

Hajat S, Kovats RS, Lachowycz K (2007). Heat-related and cold-related deaths in England and Wales: who is at risk? *Occupational and Environmental Medicine*, 64:93–100

Healy JD (2003). Excess winter mortality in Europe: a cross country analysis identifying key risk factors. *Journal of Epidemiology and Community Health*, 57:784-789.

Healy JD (2006). Personal communication. 26 October 2006.

Howden-Chapman P et al. (2007). Effect of insulating existing houses on health inequality: cluster randomized study in the community. *British Medical Journal*, 334:460-464.

Hunt S (1997). Housing-related disorders. In: Charlton J, Murphy M, eds. *The Health of Adult Britain: 1841-1994*. London. The Stationery Office.

Hutchinson EJ et al. (2006). Can we improve the identification of cold homes for targeted home energy efficiency improvements? *Applied Energy*, 83:1198-1209.

Huynen MMTE et al. (2001). The Impact of Heat Waves and Cold Spells on Mortality Rates in the Dutch Population. *Environmental Health Perspectives,* 109:463-470.

Keatinge WR (2007). Personal communication. 14 May 2007.

Keatinge WR, Donaldson GC (2000). Cold weather, cold homes and winter mortality. In: Rudge J, Nicol F, eds. *Cutting the cost of cold: Affordable Warmth for Healthier Homes*. London: E&FN Spon Ltd.:17-24.

Keatinge WR et al. (2000). Heat related mortality in warm and cold regions of Europe: observational study. *British Medical Journal*, 321:670–673.

Khaw KT (1995). Temperature and cardiovascular mortality. *Lancet,* 345:337-338.

Kunst AE, Looman CWN, Mackenbach JP (1993). Outdoor air temperature and mortality in the Netherlands: a time-series analysis. *American Journal of Epidemiology*, 137:331-341.

Laake K, Sverre JM (1996). Winter Excess Mortality: A Comparison between Norway and England plus Wales. *Age and Ageing*, 25:343-348.

Lloyd EL (1990). Hypothesis: Temperature Recommendations for Elderly People: Are We Wrong? *Age and Ageing*, 19:264-267.

Martınez-Selles M et al. (2002). Annual rates of admission and seasonal variations in hospitalizations for heart failure. *The European Journal of Heart Failure*, 4:779–786.

Mercer JB (2003). Cold – an underrated risk factor for health. *Environmental Research,* 92:8-13.

Michelozzi P et al. (2007). Assessment and prevention of acute health effects of weather conditions in Europe: The PHEWE project – background, objectives, design. *Environmental Health*, 6:doi:10.1186/1476-069X-6-12.

Nafstad P, Skrondal A, Bjertness E (2001). Mortality and temperature in Oslo, Norway, 1990–1995. *European Journal of Epidemiology*, 17:621-627.

Nayha S (2005). Environmental temperature and mortality. *International Journal of Circumpolar Health,* 64:451-458.

Office for National Statistics (2007). *Excess winter deaths by age group, Government Office Region and country of usual residence, England and Wales, 1991/1992-2004/2005 and 2005/2006.* Newport, Office for National Statistics. (http://www.statistics.gov.uk/statbase/Expodata/ Spreadsheets/D7089.csv, accessed 01 May 2007).

Pattenden S, Nikiforov B, Armstrong BG (2003). Mortality and temperature in Sofia and London. *Journal of Epidemiology and Community Health*, 57:628-633.

Press V (2003). *Fuel Poverty and Health. A guide for primary care organizations and public health and primary care professionals*. London, National Heart Forum.

Rudge J (1996). British weather: conversation topic or serious health risk? *International Journal of Biometeorology*, 39:151-155.

Rudge J, Gilchrist R (2007). Measuring the health impact of temperatures in dwellings: investigating excess winter morbidity and cold homes in the London Borough of Newham. *Energy and Buildings*, 39:847–858.

Rudge J, Winder R (2002). Central heating installation for older, low income households: What difference does it make? In: Levin H, ed. *Indoor Air 2002: Proceedings of the 9th International Conference on Indoor Air Quality and Climate, Vol. 4.* Santa Cruz, California:1078-1083.

Shah S, Peacock J (1999). Deprivation and excess winter mortality. *Journal of Epidemiology and Community Health*, 53:499–502.

Stewart S et al. (2002). Heart Failure in a Cold Climate: Seasonal Variation in Heart Failure-Related Morbidity and Mortality. *Journal of the American College of Cardiology*, 39:760-766.

Thomson H, Petticrew M, Morrison D (2001). Health effects of housing improvement: systematic review of intervention studies. *British Medical Journal*, 323:187-190.

University College London, Sheffield Hallam University, London School of Hygiene and Tropical Medicine (2006). *Health impact evaluation of England's home energy efficiency scheme (Warm Fron*t). Headline Results. Report to the Department of the Environment Food and Rural Affairs. London.

Walker J et al. (2006). Does usage of domestic heating influence internal environmental conditions and health? *European Journal of Public Health, 1*6:463-469.

Warm Front Study Group (2006). *Health impact evaluation of Warm Front: Summary results.* (http://www.warmerhealthyhomes.org.uk/media/PDF/warm_front_summary%20results.pdf, accessed 15 May 2007)

WHO (1987). *Health impact of low indoor temperatures*. WHO Environmental Health Series No 16. Copenhagen, WHO Regional Office for Europe. (http://whqlibdoc.who.int/euro/ehs/EURO_EHS_16.pdf, accessed 22 May 2008).

WHO (2007a). *Housing, energy and thermal comfort. A review of 10 countries within the WHO European Region*. Copenhagen, WHO Regional Office for Europe. (http://www.euro.who.int/document/e89887.pdf, accessed 22 May 2008).

WHO (2007b). *WHO case study on housing, energy and thermal insulation*. Copenhagen, WHO Regional Office for Europe. (http://www.euro.who.int/Housing/Activities/20060519_1, accessed 19 March 2009).

WHO (2008). *Preliminary results of the WHO Frankfurt housing intervention project.* Copenhagen, WHO Regional Office for Europe. (http://www.euro.who.int/__data/assets/pdf_file/0011/ 98696/E91699.pdf, accessed 19 March 2009).

Whyley C, Callender C (1997). *Fuel poverty in Europe: Evidence from the European Household Panel Survey*. Report by Policy Studies Institute. Newcastle upon Tyne: National Energy Action (NEA).

Wilkinson P (2006). Personal communication. 15 Dec 2006.

Wilkinson P et al. (2001). *Cold comfort. The social and environmental determinants of excess winter death in England, 1986-96*. Bristol: The Policy Press.

Wilkinson P et al. (2004). Vulnerability to winter mortality in elderly people in Britain: population based study. British Medical Journal, 329:647-652.

Wilkinson P et al. (2007). Energy, energy efficiency, and the built environment. *Lancet*, 370:1175-1187.

Traffic noise exposure and ischaemic heart disease

Wolfgang Babisch, Rokho Kim

1. Introduction

It is well known that high noise levels can cause hearing loss and tinnitus (Official Journal of the European Union, 2003). The exposure limit of the EU Directive on the minimum health and safety requirements regarding the exposure of workers to noise refer to a time-weighted weekly average noise exposure level of 85 decibels (dB(A)) of five eight-hour working days. Such auditory effects are not likely with respect to traffic noise. However, non-auditory physiological effects of noise have been studied in humans for a couple of decades using laboratory and empirical methods (Passchier-Vermeer et al., 2000). Biological reaction models have been derived, which are based on the general stress concept (Lercher, 1996; Ising et al., 1980). Stress hormones and classical biological risk factors including blood pressure, blood lipids, glucose level, blood clotting factors and cardiac output have been shown to be elevated in subjects that were exposed to high levels of noise in the laboratory and the field (Babisch, 2003; Berglund et al., 1995; WHO, 1999).

An organisms' ability to effectively cope with stimuli that threaten homeostasis is, in part, determined by their ability to initiate a cascade of neurochemical and behavioural responses; a sequelae of events which together are regarded as the prototypical stress response. This normally transient response enhances cognitive alertness, suppresses non-essential behaviours (like feeding or mating), augments immune response (via cell translocation) and triggers an activation of the hypothalamic pituitary adrenal (HPA) axis. The activation of the HPA axis includes the immediate release of corticotropin-releasing hormone (CRH) from the hypothalamus into the portal blood system where CRH binds to receptors located on the anterior pituitary gland. The activation of these receptors by CRH causes the release of adrenocorticotropin-releasing hormone which travels through the general circulation to activate receptors located on the adrenal glands. This evokes the release of glucocorticoids (cortisol in humans, corticosterone in rodents) which eventually acts to inhibit the HPA axis (negative feedback mechanism), returning the chemical changes to pre-stress levels. In addition to the activation of the HPA axis, stressors evoke an increase in the activity of the sympathetic nervous system resulting in elevated plasma catecholamine concentrations. The activation of the sympathetic nervous system, like that of the HPA axis, is beneficial in the short term insofar as it mobilizes energy resources and prepares the organism for the *fight or flight* response. When chronically activated, however, these neurochemical changes can promote the onset of stress-related disorders, including anxiety, panic, depression and increase one's risk of developing cardiovascular disease. The nature of the stress response (i.e. magnitude and duration of activation) plays a large role in the etiology of stress-related disorders. Several factors interact to influence the stress response, including the type of stressor, stressor regimen (acute, intermittent, chronic), as well as organismic factors such as past experience, genetic make-up, in addition to behavioural and psychological coping variables, such as perceived control/predictability. It is important to note that one's ability to effectively cope with any given stressor requires efficient behavioural, psychological and physiological change.

Noise is considered as being an unspecific stressor that contains both physical and psychological attributes, and arouses the autonomous nervous system and the endocrine system. From this, the hypothesis emerged that persistent noise stress increases the risk of stress-related diseases, including immunosuppressive, gastrointestinal and cardiovascular disorders. Hypertension and

ischaemic heart diseases have been most frequently investigated with respect to noise because of the high prevalence in developed countries.

This chapter addresses methods to quantify impacts on ischaemic heart disease (IHD) from long-term exposure to road traffic noise. It then produces an estimate of the likely burden of ischaemic heart disease that can be attributed to road traffic noise for Germany.

2. Summary of the method

The main steps required for estimating burden of disease from road traffic noise include:

- Establishing exposure-response relationships between noise exposure and IHD risk using results of published meta analyses.
- Estimating the percentage of the study population that is exposed to road traffic noise exceeding harmful levels (in this case 60 dB(A) during the day (16 hours, from 7.00 to 23.00) and/or 50 dB(A) during the night (8 hours, from 23.00 to 7.00).
- Compiling the health statistics for ischaemic heart disease (in cases, deaths and/or DALYs) from Global Burden of Disease estimates.
- Calculating the population attributable fraction (PAF), for each age group and exposure level. The PAF is estimated using the following formula:

$$PAF = \frac{\sum(P_i \times RR_i) - 1}{\sum(P_i \times RR_i)}$$

where: P_i =Proportion of the population in exposure category i

RR_i = relative risk at exposure category i compared to the reference level

- The population attributable fraction is multiplied by the total annual burden of ischaemic heart disease (in cases, deaths and/or DALYs) for the population of concern to estimate the burden attributable to road traffic noise. This is an exposure-based approach.

3. Exposure-risk relationship for noise exposure and ischaemic heart disease

3.1 Method for establishing exposure-risk relationship

Classical, systematic and quantitative reviews have been published in the past, summarizing the results of studies that were carried out towards the end of the last century (Babisch, 2000; Babisch, 2006). Experts have assessed the evidence of the relationship between community noise and cardiovascular disease outcomes such as hypertension and ischaemic heart diseases (IHD), including myocardial infarction (MI) (Berglund et al., 1995; Babisch, 2006; Morrell et al., 1997; WHO, 2009).

3.2 Summary of exposure-risk relationship

Although individual studies are often lacking in statistical significance, the evidence of the association is high, particularly with respect to ischaemic heart diseases. Findings consistently showed higher risks of subjects who live in dwellings exposed to higher road or aircraft noise (for review of studies see Babisch, 2006). It was concluded that ambient noise may be detrimental to health if the daytime (16 hours) noise level outdoors at the facade of the dwellings exceeds 65 dB(A) (European Environment Agency, 2001).

Recently, a meta-analyses was carried out to derive a continuous exposure-response curve for the association between the noise level and the relative risk of MI (Babisch, 2006). It was developed within the WHO projects on Night Noise Guidelines (WHO, 2009) and Environmental Noise Burden of Disease (WHO, 2011). The curve can be used for a quantitative risk assessment as suggested by the WHO working group on the Environmental Noise Burden of Disease, using the noise mapping data which are more and more available according to the European Environmental Noise Directive. Sixty-one epidemiological studies on the association between transportation noise and cardiovascular outcomes were evaluated for their feasibility in a meta-analysis.

Five studies fulfilled the strict inclusion criteria: (1) peer-reviewed in the international literature, (2) reasonable control of possible confounding, (3) objective assessment of exposure (sound level), (4) objective assessment of outcome (clinical data), (5) type of study (analytic), and (6) multilevel dose-response assessment. All studies referred to the road traffic noise level during the 16 hour daytime[8] and the incidence of myocardial infarction as the outcome. For the reference category "≤60 dB(A)" was used. Study subjects were men for reasons of statistical power (higher incidence in middle-aged subjects). The orientation of rooms was considered for the exposure assessment (at least one bedroom or living room facing the street). The characteristic of the studies considered for the meta-analysis regarding IHD are shown in Table 1. It also gives information regarding some major studies of the association between road traffic noise and high blood pressure (hypertension). Hypertension is a major risk factor for IHD. Full information about the review can be taken from the original report (Babisch, 2006).

Fig. 1 and Table 2 show the pooled effect estimates (odds ratios and 95% confidence intervals) derived from the meta-analyses. The confidence intervals include the RR of 1.0. However, when the upper two noise categories are taken together, the effect estimate is OR = 1.25 (p = 0.068, 2-sided test) in the total sample (Babisch, 2008). In the subsample of subjects that had lived for more than 10 or 15 years in their dwellings (dependent on the data given in the references) the odds ratio is significant (OR = 1.44, p = 0.020) (Babisch, 2008). The linear trend of the association was nearly significant in the subsample (p = 0.067). Fig. 2 and Table 2 show the polynomial fit of the curve for the total sample (OR = 1.629657 – 0.000613 x $(L_{day})^2$ + 0.000007357 x $(L_{day})^3$, R^2 = 0.96). Alternatively to the polynomial fit, a regression coefficient was calculated to show the increase in risk per 10 dB(A) increase of the noise level (Babisch, 2008): OR = 1.17 (95% CI = 0.87-1.57), noise level range 57.5-77.5 dB(A).

[8] Different studies apply different definitions of day and night noise related to time zones and biological rhythms. The 16-hour period considered "daytime noise" (L_{day}) can thus range from 6.00 to 22.00, or from 7.00 to 23.00. "Night noise" (L_{night}) correspondingly takes place from 22.00 to 6.00 or 23.00 to 7.00.

Table 1. Selected overview of studies investigating the relationship between exposure to road traffic noise and cardiovascular diseases

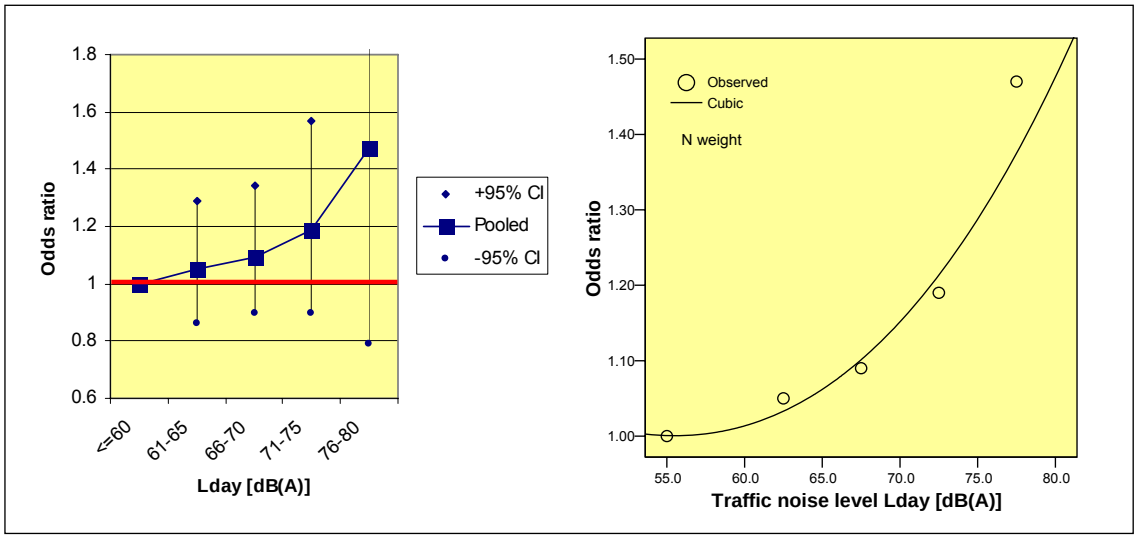
Reference	Study design, location	Study population	Exposure measure	Outcome measure	Adjusted covariates	Results (RR/OR, 95% CI)
Babisch et al., 1988; Babisch, 2006	Cross-sectional, Caerphilly (United Kingdom)	2512 males, 45-59 yrs	Road traffic noise, outdoors, $L_{Aeq,16h}$	Prevalence of MI	Age, social class, BMI, employment status, marital status, smoking, physical activity, family history of IHD, pre-existing diseases	51-55 dB(A): 1 56-60 dB(A): 1.00 (0.58-1.71) 61-65 dB(A): 0.90 (0.56-1.44) 66-70 dB(A): 1.22 (0.63-2.35)
Babisch et al., 1993a; Babisch, 2006	Cross-sectional, Speedwell (United Kingdom)	2348 males, 45-63 yrs	Road traffic noise, outdoors, $L_{Aeq,16h}$	Prevalence of MI	Age, social class, BMI, smoking, employment status, physical activity, family history of IHD, pre-existing diseases	51-55 dB(A): 1 56-60 dB(A): 1.02 (0.57-1.83) 61-65 dB(A): 1.22 (0.70-2.12) 66-70 dB(A): 1.07 (0.59-1.94)
Babisch et al., 1994	Case-control (hospital-based), Berlin (Germany)	243 males, 41-70 yrs	Road traffic noise, outdoors, $L_{Aeq,16h}$	Incidence of MI	Age, social class, education, BMI, smoking	≤ 60 dB(A): 1 61-65 dB(A): 1.48 (0.57-3.85) 66-70 dB(A): 1.19 (0.49-2.87) 71-75 dB(A): 1.25 (0.41-3.81) 76-80 dB(A): 1.76 (0.11-28.5)
Babisch et al., 1994	Case-control (population-based), Berlin (Germany)	4035 males, 31-70 yrs	Road traffic noise, outdoors, $L_{Aeq,16h}$	Incidence of MI	Age, social class, education, BMI, smoking, shift work, marital status, urban area	≤ 60 dB(A): 1 61-65 dB(A): 1.16 (0.82-1.65) 66-70 dB(A): 0.94 (0.62-1.42) 71-75 dB(A): 1.07 (0.68-1.68) 76-80 dB(A): 1.46 (0.77-2.78)
Babisch et al., 1999	Cohort, Pooled Caerphilly and Speedwell sample (United Kingdom)	3950 males, 45-63 yrs	Road traffic noise, outdoors, $L_{Aeq,16h}$	Incidence of major IHD (MI)	Age, social class, BMI, smoking, physical activity, family history of IHD, prevalence of IHD, pre-existing diseases,	≤ 60 dB(A): 1 61-65 dB(A): 0.65 (0.27-1.57) 66-70 dB(A): 1.18 (0.74-1-89)
Babisch et al., 2005	Case-control (population-based), Berlin (Germany)	2857 males, (1258 females), 31-70 yrs	Road traffic noise, outdoors, $L_{Aeq,16h}$	Incidence of MI	Age, education, employment status, BMI, smoking, shift work, marital status, family history of MI, noise sensitivity, diabetes mellitus, high blood pressure	≤ 60 dB(A): 1 61-65 dB(A): 1.01 (0.77-1.32) 66-70 dB(A): 1.13 (0.86-1.49) 71-75 dB(A): 1.27 (0.88-1.84)
Björk et al., 2006	Cross-sectional, Region Skane (Sweden)	13 557 males and females, 18-80 yrs	Road traffic noise outdoors, $L_{Aeq,24h}$	Prevalence of high blood pressure	Age, sex, BMI	<50 dB(A): 1 50-54 dB(A): 1.01 (n.s.) ≥55 dB(A): 1.05 (n.s.)

Reference	Study design, location	Study population	Exposure measure	Outcome measure	Adjusted covariates	Results (RR/OR, 95% CI)
Maschke et al., 2003	Cross-sectional, Berlin (Germany)	1718 males and females, 16-90 yrs	Road traffic noise outdoors, $L_{Aeq,16h}$, L_{night}	Prevalence of high blood pressure	Age, sex, socioeconomic index, BMI, smoking, alcohol consumption, physical activity, subjective noise sensitivity	Day, living room: <60 dB(A): 1 60-65 dB(A): 0.96 (0.65-1.41) >65 dB(A): 1.28 (0.82-2.01) Night, bedroom: <50 dB(A): 1 50-55 dB(A): 1.50 (0.88-2.53) >55 dB(A): 1.89 (1.09-3.21)
Bluhm et al., 2007	Cross-sectional, Sollentuna (Sweden)	310 males and 357 females, 19-80 yrs	Road traffic noise (and aircraft noise) outdoors, $L_{Aeq,24h}$	Prevalence of high blood pressure	Age, sex, smoking, occupational status, type of housing, years of residence, room orientation	Per 5 dB(A), range 45-70 dB(A): 1.38 (1.06-1.80) ≤45 dB(A): 1 45-50 dB(A): 1.74 (0.60-5.01) 50-55 dB(A): 2.07 (0.82-5.43) >55 dB(A): 3.47 (1.27-9.43)
Jarup et al., 2008	Cross-sectional, 6 large European cities	2404 males and 2457 females, 45-70 yrs	Road traffic noise (and aircraft noise) outdoors, $L_{Aeq,24h}$	Prevalence of high blood pressure	Country, age, sex, education, alcohol intake, BMI, smoking, physical activity	Road traffic noise: Per 10 dB(A), range 45-70 dB(A): 1.097 (1.003-1.201)
Belojevic et al., 2008	Cross-sectional, Belgrade (Serbia)	995 males and 1508 females, adults	Road traffic noise outdoors, L_{night}	Prevalence of high blood pressure	Age, sex, education, BMI, smoking, family history of high blood pressure, physical activity, alcohol consumption, subjective noise sensitivity, years of residence	Males: ≤45 dB(A): 1 >45 dB(A): 1.58 (1.03-2.42) Females: ≤45 dB(A): 1 >45 dB(A): 0.90 (0.59-1.38)
Barregard et al., 2009	Cross-sectional, Lerum (Sweden)	1857 males and females, adults	Road traffic noise outdoors, $L_{Aeq,24h}$	Prevalence of high blood pressure: prevalence of antihypertensive drug medication	Age, sex, BMI, family history of hypertension, smoking, education, subjective occupational noise exposure	Males (hypertension): 45-50 dB(A): 1 51-55 dB(A): 1.1 (0.6-1.8) 56-70 dB(A): 1.2 (0.7-2.1) Females (hypertension: 45-50 dB(A): 1 51-55 dB(A): 1.1 (0.6-1.8) 56-70 dB(A): 1.1 (0.6-1.7)

MI: Myocardial infarction; IHD: Ischaemic heart disease; BMI: Body Mass Index

It should be noted that the purpose of the meta-analysis was not primarily significance testing. The evidence of the association as such is based on the consistency of findings from individual studies. Rather, it was anticipated to derive a 'best guess' pooled exposure-response relationship that can be used for quantitative risk assessment, taking into account that information about the length of residence and room orientation is not available on a population level. Most noise maps refer to the noise level at the most exposed façade, for which the exposure-response curve can be applied.

Figures 1 and 2: Exposure-response curve for road traffic noise level (Lday) and incidence of myocardial infarction



Source: Babisch, 2008

Table 2: Relative risks of myocardial infarction by categories of road traffic noise levels

Noise level (Lday), dB(A)	Odds ratio (95% CI) (categorical)	Relative risk (polynomial fit)
>60-65	1.05 (0.86-1.29)	1.031
>65-70	1.09 (0.90-1.34)	1.099
>70-75	1.19 (0.90-1.57)	1.211
>75-80	1.47 (0.79-2.76)	1.372

Data source: Babisch, 2008

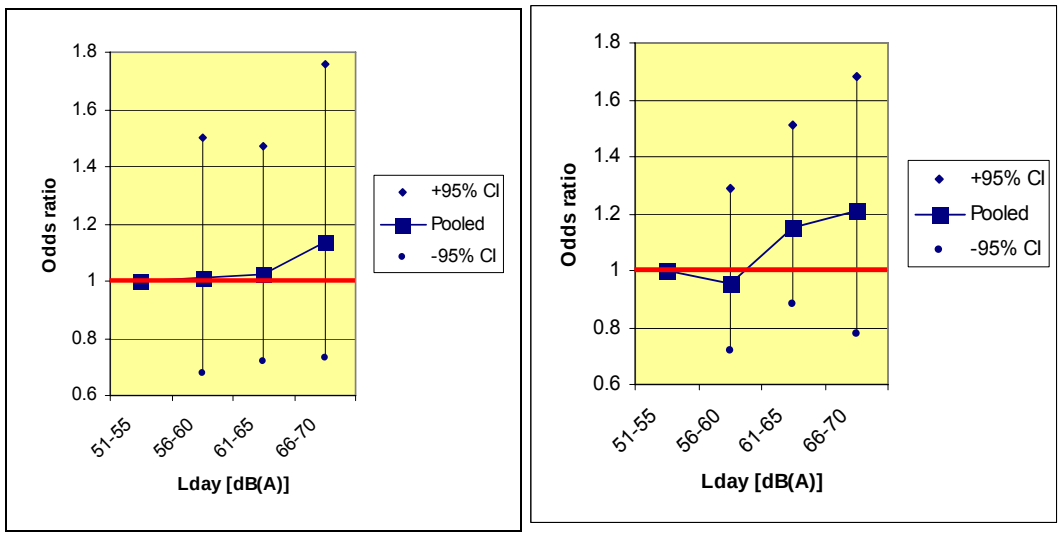
Note: Odds ratios refer to Figures 1 and 2

Myocardial infarction was considered for the meta-analysis because it was commonly assessed in the respective studies. The noise impact on myocardial infarction may have been easier to detect by epidemiological studies because the misclassification in the diagnosis of myocardial infarction is less likely than for all ischaemic heart diseases (coded as 410-414 in ICD 9, and I20-I25 in ICD 10), including other acute and subacute forms of ischaemic heart disease and angina pectoris.

Because there is no exclusive causal mechanism postulated specifically to myocardial infarction, it has been suggested that the population attributable fraction of traffic noise could be applied to all ischaemic heart disease. This is supported by Fig. 3 and 4. It shows the association between road traffic noise level during the day (L_{day}) and the prevalences of myocardial infarction and

ischaemic heart diseases based on 2 respective studies, where all information was collected within each study (Babisch, 2006; Babisch et al., 1993b). Ischaemic heart disease comprises: acute myocardial infarction, other acute and subacute forms of ischaemic heart disease, old myocardial infarction, ischaemic signs in the electrocardiogram, angina pectoris, coronary atherosclerosis and chronic ischaemic heart disease. It can be seen that the associations with the noise level look quite similar.

Figures 3 and 4: Exposure-response curve for road traffic noise level (Lday) and prevalence of myocardial infarction (left) or all ischaemic heart diseases (right)



Source: Babisch et al., 1993b

4. Exposure assessment for road traffic noise

4.1 Methods for measuring exposure to road traffic noise

Energy-based indicators of exposure (L_{eq}) are adequate and sufficient for the assessment of the relationship between long-term exposure to community noise and chronic diseases, e.g. cardiovascular disorders. Such indicators should measure noise during various periods of the day. Examples include L_{day} (e.g. day-noise indicator 7:00 to 23:00), $L_{day,12h}$ + $L_{evening,4h}$ (day-noise indicator 7:00 to 19:00 and evening-noise indicator 19:00 to 23:00), and L_{night} (night-noise indicator 23:00 to 7:00). For estimating health impacts according to the method proposed here, L_{day} is a useful indicator.

When information for noise for the various periods of the day, i.e. day/evening/night, is available, weighted and non-weighted indicators can easily be calculated for use in health studies and related quantitative risk assessment. This includes the indicators L_{den} (weighted day-evening-night noise indicator) and L_{night} according to the European Environmental Noise Directive (Official Journal of the European Communities, 2002), which are considered for noise mapping.

4.2 Summary of exposure levels in Europe

The exposure-response curve shown in Fig. 2 and Table 1 can be applied to the "noise mapping" data – which refer to L_{den} in accordance to the Directive – when conversions between L_{den} and L_{day} are made: $L_{den} \approx L_{day} - 2 \times \ln[(L_{day}-L_{night})/22.4)]$ (Bite et al., 2004). In Europe, for example, systematically assessed exposure data of most European countries are lacking. EU member

States have recently started to systematically assess the environmental noise due to traffic and commercial activities.

With respect to sleep disturbance, indicators describing the noise of an individual event (L_{max}: maximum sound level; L_{AE}: sound exposure level; number of events) may additionally be useful indicators (e. g. with respect to aircraft noise over-flights), especially since L_{night} alone would not capture the hours that delineate a child's sleep period.

5. Environmental burden of ischaemic heart disease from traffic noise

5.1 EBD estimate for Europe

According to the WHO Global Burden of Disease 2000 study which was updated in 2004, ischaemic heart disease is the leading cause of death in developed and developing countries (22.8% and 9.4% of total deaths, respectively) (WHO, 2002; Mathers et al., 2003). EU Member States are currently assessing the noise exposure in their countries according to the Environmental Noise Directive (Official Journal of the European Communities, 2002). However, during the development of this chapter, no reliable exposure data for the whole of Europe are available.

However, crude approximations of the disease burden (in DALYs) for ischaemic heart disease attributable to transport noise may be estimated, assuming the same exposure patterns across the countries and subregions with an population attributable fraction 3% in Euro A, i.e. approximately the same as in Germany. This assumption would be conservative for some European countries, as the noise exposure may be higher in these countries than in Germany. According to older data approximately 80 million people (EU15 population status: approximately 360 Mio) were exposed to noise levels during the day (L_{day}) of more than 65 dB(A) (European Commission, 1996; CALM, 2004). This accounts for approximately 22% of the EU15 population (the comparative estimate for Germany would be approximately 16%).

5.2 EBD estimate for specific states of Europe

Case study: Estimation of the disease burden for Germany

According to the German noise exposure model ("Lärmbelastungsmodell") it is estimated (reference year 1999) that approximately 16% of the German population are exposed to road traffic noise levels exceeding 65 dB(A) during the day (6-22 h) at the façade of their houses (approximately 15% exposed to noise levels >60 to 65 dB(A) and approx. 18% to noise levels >55 to 60 dB(A)) (Federal Environmental Agency, 2005). During the night the noise levels tend to be 7-10 dB(A) lower.

The risk ratios used for the exposure categories ($L_{day)}$ of >60 to 65, >65 to 70, >70 to 75, and >75 to 80 dB(A) are 1.031, 1.099, 1.211, and 1.372, respectively, as specified in Table 1. 15.3%, 9.0%, 5.1% and 1.5% of the population are in those categories, respectively, and the remaining 69.1% are exposed to an L_{day} lower than 60 dB(A) (i.e. which have a RR of 1.0).

The country-specific population attributable fraction and the attributable cases can be calculated based on the distribution of population in different exposure categories and the disease incidence rates. Using the formula of the population attributable fraction provides the following results:

$$PAF = \frac{(1.031 x\, 0.153 + 1.099 x\, 0.090 + 1.211 x\ \ 0.051 + 1.372 x\, 0.015 + 1 x\, 0.691) - 1}{(1.031 x\, 0.153 + 1.099 x\, 0.090 + 1.211 x\ \ 0.051 + 1.372 x\, 0.015 + 1 x\, 0.691)} = 0.0291$$

The resulting population attributable fraction of myocardial infarction due to road traffic noise for the German population in the year 1999 is therefore 2.9%.

According to the national health statistics, 849 557 cases of ischaemic heart diseases (ICD 9, No. 410-414) including 133 115 cases of acute myocardial infarction (ICD 9, No. 410) were detected in the year 1999 (Statistisches Bundesamt, Robert Koch-Institut, 2005). 2.9% of this number results in approximately 3900 cases per year (rounded) of myocardial infarctions in Germany being attributable to traffic noise exposure (Table 3). When generalizing the exposure-response curve for myocardial infarction to all ischaemic heart diseases, the cases attributable to road traffic noise would account for approximately 24 700 IHD-cases (or 25 300 DALYs) in Germany per year (World Health Organization, 2007). Additional research is needed to strengthen this hypothesis. While considering only the cases of myocardial infarctions as attributable to noise exposure may strongly underestimate the real burden, the evidence may not yet be solid enough to support the application to all ischaemic heart diseases, although this may currently be a likely interpretation of the evidence.

5.3 Sensitivity analysis

A categorical approach was used to pool the effect estimates of different noise studies for different noise categories. The confidence intervals given in Table 1 and shown in Fig. 1 can be used to assess the range of uncertainty by applying the formula of the population attributable fraction (PAF). Since all lower confidence intervals include the RR of 1, the lower EBD estimate of the population attributable fraction is 0; the upper EBD estimate is the PAF = 0.1153 (11.5%). This range appears large, but one has to bear in mind that the point estimate of PAF = 0.0291 has the highest statistical probability. Since it is generally accepted that environmental noise affects the cardiovascular system, exposure-response relationship shown in Fig. 2 is a "best guess" curve that can be used for the moment. It is based on the available studies and data. The data base must continuously be updated to enlarge the database and to narrow the confidence intervals. When the analyses of studies were restricted to subjects with long years of residence larger and often significant effect estimates were found (Babisch et al., 1999; Bluhm et al., 2007; Maschke et al., 2008; Belojevic et al., 2008; Barregard et al., 2009).

6. Uncertainty

6.1 Areas of uncertainty

The main uncertainty currently stems from the exposure-response relationship. Uncertainty of the exposure assessment will depend upon the specific assessment.

Noise indicator

The exposure-response curve refers to the noise indicator $L_{day,16h}$. It is often argued that people may not be at home during the day and the indicator may not reflect the true exposure. Such argument is based on a toxicological way of thinking (e. g. accumulation of exposure over time, e.g. '$\mu g/m^3$'). However, this concept does not apply to the non-auditory effects of noise. Community noise indicators are a determinants of the noise load to which people are exposed whenever they are exposed in the respective environment (e.g. their dwellings), and which may then cause stress reactions (e.g. during the evening when people want to relax, concentrate or communicate). Road traffic noise levels during the day and the night are usually highly correlated ($r > 0.90$), which means that the day noise indicator serves also as a surrogate for relative effects of the night noise exposure of road traffic. An averaged 24-hour noise level based on personal dosimetry would not be an appropriate determinant for noise-induced health effects. The adverse effects of noise are highly dependent on the activities that are disturbed. In

this respect, for example, 85 dB(A) during work may cause less of an effect than 60 dB(A) at leisure at home or 45 dB(A) during sleep.

With respect to the history of exposure it is important to note that doubling of the traffic volume corresponds with an increase of the noise exposure of only approximately 3 dB(A) due to the logarithmic nature of the decibel. The range of exposures, however, is approximately 30 dB(A) from side streets to very busy streets. Doubling or halving of traffic volume is very unlikely to have occurred to a larger extent in studies where retrospective years of exposure were considered. Study subjects were selected at random from streets all over the city. Any non-differential exposure misclassification tends to dilute (underestimate) the true noise effects.

According to the European Directive relating to the Assessment and Management of Environmental Noise (Official Journal of the European Communities, 2002), European Member States are currently assessing noise maps. These maps can be used for the assessment of EBD due to noise. However, one has to bear in mind that the national estimates of noise exposure underestimate the true exposure, because only large agglomerations and major roads are considered at present.

Meta-analysis

The confidence intervals of the pooled exposure-response curve are still large. However, in epidemiological noise studies, higher and significant risk estimates were found, when the lengths of exposure (years of residence), room orientation and window opening habits were considered (Babisch et al., 1999; Bluhm et al., 2007). The exposure-response curve was derived from male study subjects due to considerations of statistical power in individual noise studies (ischaemic heart disease is more frequent in middle-aged male subjects). Although there are differences in the absolute risk between males and females, it seems to be reasonable to assume that in relative terms, females may be just as affected by noise stress as males. Road traffic noise studies sometimes showed stronger effects in males (Babisch et al., 2005; Herbold et al., 1989; Jarup et al., 2008), sometimes in females (Bluhm et al., 2007). However, in future noise studies, potential gender differences should be addressed.

Potential confounding or aggravating by other traffic exposures

Air pollutants have also been shown to be associated with cardiovascular endpoints. Individuals exposed to road noise are also likely to be exposed to air pollution arising from road traffic. It is not yet entirely clear whether the impact of noise on ischaemic heart disease is independent, additive or synergistic to the impact of outdoor air pollution. Air pollution studies have not controlled for noise and vice versa (Jarup et al., 2007). However, very few new studies are available where both factors were considered showing that both factors contribute individually to the increase in cardiovascular risk (de Kluizenaar et al., 2007).

The biological plausibility of the noise effects' hypothesis, however, is well documented based on laboratory and animal experiments. The general noise reaction model was well established before noise effects' research moved from the laboratory into the field to test epidemiological noise hypotheses with respect to long-term effects of noise. The noise exposure of the subjects was assessed for each house individually and controlled for individual confounding factors. Air pollution studies often referred to spatial exposures; particularly, those that have been used for quantitative risk assessments (Pope III et al., 2002; WHO, 2006). For the time-being, the curve (Fig. 2) may well be a good guess exposure-response relationship which will be updated with new incoming data from research. Noise studies from aircraft noise, where subjects experience, if at all, only moderate increase of air pollution levels, also seem to confirm the relation between exposure to noise and effects on cardiovascular diseases (mainly high blood pressure).

In summary, although the effect of noise on myocardial infarction or ischaemic heart disease seem plausible, and the currently most likely interpretation of the available evidence, additional

studies investigating the impact of noise separately, as well as the combined effects, are needed to confirm the relationship (Jarup et al., 2007; Schwela et al., 2005).

6.2 Approaches for reducing uncertainty

More studies are needed to confirm the effect estimates or to alter them accordingly. With larger numbers of subjects considered in the meta-analysis the confidence intervals may narrow. With respect to causal inference, exposure misclassification should be reduced by accounting for room orientation and other exposure modifiers, e. g. window opening habits. However, such information can only be obtained from scientific studies to test the noise hypothesis as such. On a population level individual information regarding personal attitudes and living conditions are not available. For EBD calculations, therefore, the weaker (and maybe non-significant) effect estimates must be used that include a certain degree of exposure misclassification. These estimates, however, refer to larger numbers of the entire population and not only specific groups with certain exposure conditions, which balances the estimates of the population attributable risk.

7. Conclusions

It appears that road traffic noise is a significant risk factor for ischaemic heart diseases, causing ca. 3% of all myocardial infarction in Germany, according to older exposure data from the year 1999. The proportional attributable fraction, however, may be different when using updated exposure data which are currently assessed within the framework of the European Noise Directive. The risk increases when the average noise levels is greater than 60 dB(A) during the day, which corresponds with noise levels during the night of approximately 50 dB(A). To support the extension of the effect from myocardial infarction to all ischaemic heart diseases would benefit from additional evidence. Equally, disentangling the various health impacts caused by traffic, including noise, air pollution in terms of particulate matter but maybe also other components such as solvents, would benefit from additional studies where the potential confounders are adequately controlled for, but also possible aggravating effects further investigated.

Together with air pollution, noise is an environmental risk factor to health that may be largely underestimated.

Table 3. Summary of EBD of ischaemic heart disease from road traffic noise

Exposure	Road traffic noise during day (16 hours)			
Health outcome	Ischaemic heart diseases (IHD cases) including myocardial infarction (MI)			
Summary of EBD assessment	About 3% of myocardial infarction in Germany can be attributed to road traffic noise outside the dwellings which represents 3900 potentially avoidable cases of myocardial infarction (MI) or 24 700 avoidable cases of ischaemic heart disease (IHD) or 25 300 DALYs per year.			
	Level	**Range**	**Geographic scope**	**Source of information**
(a) Exposure risk relationship	RR 1.17 per 10 dB(A)	0.87-1.57	Germany	Cohort and case-control studies with largely consistent findings
(b) Exposure assessment	31% people exposed to road traffic noise levels > 60 dB(A)	>60 to 80 dB(A)	Germany	Taken from national report

(c) PAF	2.9%	0.0-11.5% IHD cases	Germany	Derived from this report, section 5.
(d) Total burden of disease	133 115 MI 849 557 IHD cases 872 400 DALYs		Germany	Derived from national health statistics.
(e) EBD from traffic noise exposure	3900 MI (4.8/100 000) 24 700 IHD cases (30.1/100 000) 25 300 DALYs (30.8/100 000)	0 – 97 946 IHD cases	Germany	Derived from this report, section 5.
Main areas of uncertainty	Large confidence intervals of exposure-response relationship, missing or incomplete exposure data for many countries, unknown impact (confounding) of other traffic related exposures, extrapolation from MI to all IHD for the calculation of DALYs. In addition, the assessment is based on 1999 data possibly not representative for today's exposure conditions.			
Main implications	Reduce traffic noise exposure of the population			

8. Policy and practice implications

Measures to reduce exposure to road traffic noise can be taken at various levels and imply different types of actions, as for example the following:

- Modification of transport policies or regulations
  - Reduction of transit in city centres, residential centres, around schools and other noise-sensitive areas
  - Speed reductions
  - Promotion of public transportation
  - Limitation of heavy vehicles, e. g. during night-time
  - More rigorous regulations pertaining to noise from motorcycles and cars
  - Time limitation, e.g. of heavy vehicles in city centres
  - Traffic zoning
  - Incentive instruments to motivate car producers to develop less noisy cars
  - Economic instruments to reduce external costs of noise exposure
- Introduction of technical measures
  - Use of quieter road surfaces
  - Use of less noisy tires
  - Technical control of vehicles
- Modification of housing
  - Noise-insulation of certain housing elements or materials (e.g. windows)
  - Sound barriers along traffic arteries
- Land use measures (for longer term results)
  - Improved planning to move traffic away from noise-sensitive areas

For the future assessment of the environmental burden of disease associated with road traffic noise (for example WHO, 2011), the European data collected for large urban areas under the Environmental Noise Directive could provide a more appropriate and reliable international data source.

In addition to traffic noise, the quantification of the health impacts of neighbour noise from housing surroundings and indoor noise sources would be desirable but is currently not feasible due to lack of adequate data.

9. References

Abbey DE et al. (1999). Long-term inhalable particles and other air pollutants related to mortality in nonsmokers. *American Journal of Respiratory and Critical Care Medicine,* 159:373-382.

Babisch W (2000). Traffic noise and cardiovascular disease: epidemiological review and synthesis. *Noise and Health*, 2:9-32.

Babisch W (2003). Stress hormones in the research on cardiovascular effects of noise. *Noise and Health*, 5:1-11.

Babisch W (2006). Transportation noise and cardiovascular risk: Updated review and synthesis of epidemiological studies indicate that the evidence has increased. *Noise and Health*, 8:1-29.

Babisch W (2008). Road traffic noise and cardiovascular risk. *Noise and Health*, 10:27-33.

Babisch W et al. (1988). Traffic noise and cardiovascular risk. The Caerphilly Study, first phase. Outdoor noise levels and risk factors. *Archives of Environmental Health*, 43:407-414.

Babisch W et al. (1993a). Traffic noise and cardiovascular risk: the Speedwell study, first phase. Outdoor noise level and risk factors. *Archives of Environmental Health*, 48:401-405.

Babisch W et al. (1993b). Traffic noise and cardiovascular risk: the Caerphilly and Speedwell studies, second phase. Risk estimation, prevalence, and incidence of ischemic heart disease. *Archives of Environmental Health*, 48:406-413.

Babisch W et al. (1994). The incidence of myocardial infarction and its relation to road traffic noise – the Berlin case-control studies. *Environment International*, 20:469-474.

Babisch W et al. (1999). Traffic noise and cardiovascular risk: The Caerphilly and Speedwell studies, third phase – 10 years follow-up. *Archives of Environmental Health*, 54:210-216.

Babisch W et al. (2005). Traffic noise and risk of myocardial infarction. *Epidemiology*, 16:33-40.

Babisch W (2006). *Transportation noise and cardiovascular risk. Review and synthesis of epidemiological studies. Dose-effect curve and risk estimation*. WaBoLu-Hefte 01/06. Dessau, Federal Environmental Agency.

Barregard L, Bonde E, Öhrström E (2009). Risk of hypertension from exposure to road traffic noise in a population-based sample. *Occupational and Environmental Medicine*, 66:410-415.

Belojevic GA et al. (2008). Nighttime road-traffic noise and arterial hypertension in an urban population. *Hypertension Research*, 31:775-781.

Berglund B, Lindvall T (1995). *Community noise*. Archives of the Center for Sensory Research 2(1). Stockholm, Center for Sensory Research.

Bite M, Bite PZ (2004). Zusammenhang zwischen den Straßenverkehrslärmindizes LAeq(06-22) und LAeq(22-06) sowie Lden. *Zeitschrift für Lärmbekämpfung*, 51:27-28.

Björk J (2006). Road traffic noise in southern Sweden and its relation to annoyance, disturbance of daily activities and health. *Scandinavian Journal of Work, Environment & Health*, 32:392-401.

European Commission (1996). *Commission Green Paper on future noise policy. COM (96)540*. Brussels, European Commission.

CALM (2004). *Research for a quieter Europe in 2020. An updated strategy paper of the CALM Network – Oct. 2004*. Brussels, European Commission Research Directorate-General.

Bluhm GL et al. (2007). Road traffic noise and hypertension. *Occupational and Environmental Medicine*, 64:122-126.

De Kluizenaar Y et al. (2007). Hypertension and road traffic noise exposure. *Journal of Occupational and Environmental Medicine,* 49:484-492.

Directive 2002/49/EC (2002). Directive of the European Parliament and of the Council of 25 June 2002 relating to the assessment and management of environmental noise. *Official Journal of the European Communities*, L189:12-25.

Directive 2003/10/EC (2003). Directive of the European Parliament and of the Council of 6 February 2003 on the minimum health and safety requirements regarding the exposure of workers to the risks arising from physical agents (noise). *Official Journal of the European Communities,* L42:38-44.

Dockery DW et al. (1993). An association between air pollution and mortality in six U. S. cities. *The New England Journal of Medicine*, 329:1753-1759.

European Environment Agency (2001). *Traffic noise: exposure and annoyance*. Copenhagen, European Environmental Agency. (http://themes.eea.eu.int/Sectors_and_activities/transport/indicators/consequences/noise_exposure/Noise_TERM_2001.doc.pdf (accessed June 9, 2008).

Federal Environmental Agency (2005). *Data on the environment. The state of the environment in Germany, 2005 edition*. Dessau, Federal Environmental Agency.

Health Council of the Netherlands (2004). *The influence of night-time noise on sleep and health*. Report No. 2004/14E. The Hague, Gezondheidsraad.

Herbold M, Hense HW, Keil U (1989). Effects of road traffic noise on prevalence of hypertension in men: results of the Luebeck Blood Pressure Study. *Social and Preventive Medicine*, 34:19-23.

Ising H et al. (1980). Health effects of traffic noise. *International Archives of Occupational and Environmental Health*, 47:179-190.

Jarup L, Babisch W, Schwela D (2007). Cardiovascular noise & outdoor pollutants. In: Kephalopoulos S et al., eds. *Proceedings of the International Workshop on "Combined Environmental Exposure: Noise, Air Pollutants and Chemicals", 15-16 January 2007, Ispra*. Luxembourg, Office for Official Publications of the European Communities:17-24. (http://web.jrc.ec.europa.eu/pce/documentation/eur_reports/JRC_Workshop_Combined_Environmental_Exposure_Proceedings_FINAL.pdf, accessed 04 May 2008).

Jarup L et al. (2008). Hypertension and exposure to noise near airports – the HYENA study. *Environmental Health Perspectives*, 116:329-333.

Lercher P (1996). Environmental noise and health: an integrated research perspective. *Environment International*, 22:117-128.

Maschke C, Wolf U, Leitmann T (2003). Epidemiological examinations of the influence of noise stress on the immune system and the emergence of arteriosclerosis. Report 298 62 515. *WaBoLu-Hefte* 01/03.

Mathers CD et al. (2003). *Global burden of disease in 2002: data sources, methods and results*. Global programme on evidence for health policy discussion paper no. 54. Geneva, World Health Organization. (http://www.who.int/healthinfo/paper54.pdf, accessed August 28, 2006).

Morrell S, Taylor R, Lyle D (1997). A review of health effects of aircraft noise. *Australian and New Zealand Journal of Public Health*, 21:221-236.

Passchier-Vermeer W, Passchier WF (2000). Noise exposure and public health. *Environmental Health Perspectives*, 108 (suppl. 1):123-131.

Pope III CA et al. (1995). Particulate air pollution as a predictor of mortality in a prospective study of U. S. adults. *American Journal of Respiratory and Critical Care Medicine*, 151:669-674.

Pope III CA et al. (2002). Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *Journal of the American Medical Association*, 287:1132-1141.

Porter ND, Flindell IH, Berry BF (1998). *Health effect-based noise assessment methods: a review and feasibility study*. NPL Report CMAM 16. Teddington, National Physical Laboratory.

Schwela D, Kephaloupoulos S, Prasher D (2005). Confounding or aggravating factors in noise-induced health effects: air pollutants and other stressors. *Noise and Health*, 7:41-50.

Stansfeld S, Haines M, Brown B (2000). Noise and health in the urban environment. *Reviews on Environmental Health*, 15:43-82.

Statistisches Bundesamt, Robert Koch-Institut (2005). Gesundheitsdaten online, Gesundheitsberichterstattung des Bundes. (http://www.gbe-bund.de, accessed 20 June 2007).

Van Kempen EEMM et al. (2002). The association between noise exposure and blood pressure and ischaemic heart disease: A meta-analysis. *Environmental Health Perspectives*, 110:307-317.

WHO (1999). *Guidelines for Community Noise.* Geneva, World Health Organization. (http://whqlibdoc.who.int/hq/1999/a68672.pdf, accessed 13 June 2009).

WHO (2000). *Air Quality Guidelines for Europe, 2nd edition.* Copenhagen, WHO Regional Office for Europe. (http://www.euro.who.int/__data/assets/pdf_file/0005/74732/E71922.pdf, accessed 13 June 2009).

WHO (2002). *The world health report 2002*. Geneva, World Health Organization. (http://www.who.int/whr/2002/en/, accessed 13 June 2009).

WHO (2006). *Air Quality Guidelines Global Update 2005.* Copenhagen, WHO Regional Office for Europe. (http://www.euro.who.int/__data/assets/pdf_file/0005/78638/E90038.pdf, accessed 13 June 2009).

WHO (2007). *WHOSIS (World Health Organization Statistical Information System)*. Geneva, World Health Organization. (http://www.who.int/whosis/en/, accessed 15 October 2008).

WHO (2009). *Night Noise Guidelines.* Copenhagen, World Health Organization (http://www.euro.who.int/_data/assets/pdf_file/0017/43316/E92845.pdf; accessed 10 May 2010).

WHO (2011). *Burden of disease from environmental noise.* Copenhagen, WHO Regional Office for Europe. To be published in April 2011.

Indoor radon and lung cancer

Hajo Zeeb

1. Introduction

Radon (^{222}Rn) is a noble gas formed from radium (^{226}Ra), which is a decay product of Uranium (^{238}U). Uranium and radium occur naturally in soils and rocks from where it emanates and concentrates in enclosed spaces like underground mines or houses. Radon gas is an important source of ionizing radiation of natural origin and a major contributor to the ionizing radiation dose received by the general population. The International Agency for Research on Cancer classified radon gas as a Group 1 human carcinogen in 1988 (sufficient evidence to establish causality between cancer and radon exposure).

Apart from rocks and soils as radon sources, radon dissolved in water and radon emanating from building materials can contribute to increased indoor radon concentrations, but usually these sources are of less importance. Radon concentrations in buildings vary widely depending on the underlying geological formations, building structure, ventilation and other issues. Radioactive concentration is expressed in Becquerel per cubic meter or liter (Bq/m^3 or Bq/l). The worldwide average indoor radon concentration is estimated at 49 Bq/m^3 (UNSCEAR, 2000), but several countries in Europe including the Nordic countries, the Czech Republic and Switzerland have markedly higher average values (Table 1). As radon gas and its decay products quickly dissipate in outdoor air, outdoor concentrations around 10 Bq/m^3 are common, again with some regional variation.

2. Summary of the method

Lung cancer is the principal disease to assess the burden of disease associated with radon exposure. For this chapter, no formal calculations of the burden of lung cancer in Europe were performed. Detailed estimates for the population attributable fraction (PAF), which is the proportion of disease that can be associated with radon exposure, have been published for several European countries (Catelinois et al., 2006; Menzler et al., 2008). In this context, PAF represents the proportion of lung cancer in the population that would be prevented if exposure to radon in the home were reduced to zero Becquerel/m^3 or, more realistically, to the average outdoor radon concentration for the specific country. The PAF estimate is then multiplied by the total burden of lung cancer. This results in an estimate of the proportion of cases, deaths and disability adjusted life years (DALYs) that can be ascribed to radon in the home. As lung cancer is highly fatal, lung cancer deaths have been used as indicator of disease burden in most studies, including in a recent evaluation of radon prevention and mitigation strategies in the United Kingdom (Gray et al., 2009).

Table 1: Radon surveys in dwellings in some European countries

Country and population (millions)	No. of dwellings sampled	Period and approx. duration of measurement	Mean value Bq/m3	Geo-metric mean Bq/m3	Percent. >200 Bq/m3	Percent. >400 Bq/m3	Max. Bq/m3
Austria (8.2)	16 000	1991-2002, 3 months	97	61	12	4	8325
Belgium (10.4)	10 447	1995-present, 3 months	69	76	2.4	0.5	4500
Croatia (4.5)	782	2003-05, 1 year	68	n/a	7.2	1.8	751
Czech Republic (10.2)	>150 000	1984-present, 1 year	140	110	12.0-18.0	2.0-3.0	25 000
Denmark (5.5)	3120	1995-96, 1 year	53	64	2.9	0.2	590
Finland (5.2)	3074	1990-91, 1 year	120	84	12.3	3.6	33 000
France (62.2)	12 261	1980-2003, 3 months	89	53	8.5	2	4964
Germany (82.4)	>50 000	1978-2003, 1 year	50	40	3	<1	>10 000
Greece (10.8)	1277	1994-98, 1 year	55	44	3.1	1.1	1700
Ireland (4.2)	11 319	1992-99, 1 year	89	57	7.5	1.5	1924
Italy (58)	5361	1989-1998, 1 year	70	52	4.1	0.9	1036
Luxembourg (0.49)	2619	1993-2002, 3 months	115	n/a	n/a	3	2776
Netherlands (16.6)	952	1995-96, 1 year	30	25	0.3	<0.0001	382
Norway (4.6)	37 400	1990-99, 2 months	89	n/a	9	3	50 000
Poland (38.5)	2886	1992-94, 3 months	49	n/a	2	0.4	3261
Portugal (10.7)	3317	1988-1991, 2.5 months	86	39	n/a	n/a	3558
Slovenia (2)	892	1993-1995, 3 months	87	n/a	7.7	2	1890
Spain (40.5)	5600	1990-2005, 3 months	90	45	6	2	15 400
Sweden (9)	1360	1991-92, 3 months	108	56	9.0-13.0	3.0-4.0	3904
Switzerland (7.6)	55 000	1980-2005, 3 months	77	n/a	17	7	29 705
United Kingdom (61)	450 000	1980-2005, 3-12 months	20	n/a	0.5	0.1	17 000

Compiled from National Summary Reports at http://radonmapping.jrc.ec.europa.eu/

In general terms, the PAF is calculated as follows:

$$PAF = \frac{p(RR-1)}{p(RR-1)+1}$$

where p = proportion of the population exposed, and RR is the relative risk for the condition in those exposed. Using average country and regional radon concentrations implies that the whole population is exposed to this concentration, which reduces the formula to

$$PAF = \frac{RR-1}{RR}$$

The relative risk relates to the risk increase at the given average indoor radon concentration. Here, one may factor in a baseline radon concentration, e.g. the usual outdoor concentration in a country. In the case of radon, comprehensive burden of disease (BoD) estimates also include the prevalence of current country-specific smoking, because smoking is the leading lung cancer risk factor, and a large proportion of all radon-associated lung cancers occurs among smokers through an interaction mechanism.

Thus, a comprehensive radon Environmental Burden of Disease (EBD) assessment requires the following data:

- Exposure-risk relationship. Three summary analyses of case-control studies on lung cancer from radon exposure in the home are available. Data from studies among miners are useful for comparative purposes. Both indoor and miner studies have yielded risk models that can be applied to individual countries.
- Exposure assessment. Surveys of average indoor radon levels in European homes and UNSCEAR data on global radon indoor exposures are available.
- Smoking information. Gender-specific smoking prevalence data for the population under study are required for a detailed assessment and are available for several countries.
- Total burden of disease. Published estimates of the burden of disease from lung cancer the WHO Europe region are available.

The remainder of this chapter describes the key steps in obtaining this essential information. Because no comprehensive and detailed country-specific estimates can be provided, this chapter is an evidence summary. However, examples from detailed BoD studies in France, Germany and Switzerland are discussed.

3. Exposure-risk relationship for radon and lung cancer

3.1 Method for establishing the exposure-risk relationship

A number of comprehensive scientific reports on radon health effects have been published in the recent past, including reports by UNSCEAR (2000, 2008) and the United States National Research Council (1999). These reports are partially or exclusively based on epidemiological studies of miners. They were reviewed and information relevant for the current assessment was abstracted. A systematic PUBMED search revealed a small number of new publications on cohort updates, which were also included. All known international studies before 2005 have been analysed in three pooled analyses (Darby et al., 2005; Darby et al., 2006; Krewski et al., 2005; Krewski et al., 2006; Lubin et al., 2004). Together, these pooled analyses form a

comprehensive information base, which has been used for this report. A systematic literature search on non-lung cancer effects of radon supplements the assessment. The literature review on other health effects may not be complete, because single studies on the radon-disease relationship may have been missed. However, it should be noted that, with regard to radon, no other disease-exposure relationship apart from radon and lung cancer is currently regarded as firmly established.

3.2 Evidence of exposure-risk relationship

Evidence from studies of miners has been available for many decades. A review of the major studies of underground miners exposed to radon that were available in the 1990s was carried out by the Committee on the Biological Effects of Ionizing Radiation (BEIR VI, 1999). Eleven cohort studies including a total of 68 000 miners in Europe, North America, Asia and Australia were evaluated (n=2700 deaths from lung cancer in the combined cohorts). Lung cancer rates generally increased with increasing cumulative radon exposure. A joint RR of 1.44 per Working Level Month (WLM; ~ 3700 Bq/m^3 over 170 working hours; 1 WLM ~ 230 Bq/m^3 average radon concentration) was estimated. A large number of publications demonstrate the further work on the established cohorts, and three new cohorts (Poland, Brazil, Germany) have added to the evidence (Skowronek et al., 2003; Veiga et al., 2004; Kreuzer et al., 2002; Grosche et al., 2006).

As exposure conditions in mines and in the residential setting are quite different, a large number of epidemiological case-control studies has been carried out to directly assess risks of indoor radon. In these studies, the long-term radon concentration in homes occupied by lung cancer cases was compared with that of appropriately sampled control persons. Detailed smoking information was also collected. Most of these studies by themselves have not been large enough to provide clear evidence, so pooled analyses have been performed, notably of 13 European studies (Darby et al., 2005; Darby et al., 2006), 7 North American studies (Krewski et al., 2005; Krewski et al., 2006), and 2 Chinese studies (Lubin et al., 2004) (Table 2). The key results are as follows:

- In the European pooled analysis, the risk of lung cancer increased by 8% per 100 Bq/m^3 increase in measured radon concentration over an exposure time window of 5-34 years before the diagnosis or corresponding index date for controls. There were no statistically significant differences for the relative risk between smokers and non-smokers. When corrections were applied to account for the variability of measured radon concentrations, the risk coefficient increased to 16% per 100 Bq/m^3.
- The analysis of the North American studies yielded a pooled estimate of 11% per 100 Bq/m3 based on measured radon concentrations 5-30 years prior to the index date, with a linear exposure-response relationship similar to the European study and with no differences due to smoking history.
- The two Chinese studies showed a joint risk coefficient of 13% per 100 Bq/m3 and other results consistent with the European and North American studies.

In summary, the pooled studies provide consistent evidence of a relationship between lung cancer and indoor radon, although the direct comparability of the pooled studies is somewhat hampered by differences in the exposure time windows. No evidence of radon concentration thresholds below which there is no risk increase was found.

As expected, the miner and the indoor studies differ, because the exposures in mines are usually larger by several orders of magnitude than those in homes. Nevertheless, attempts have been made to investigate the consistency of results obtained in different study populations and exposure situations. Even with the lower exposures in the indoor air studies, there is a similar

dose-response relationship. This increases the confidence that radon is associated with an increased risk of lung cancer.

Table 2. Summary of pooled studies investigating the relationship between exposure to indoor radon and lung cancer

Reference	Study design, location	Study population	Exposure measure	Outcome measure	Adjusted covariates	Results (RR/OR, 95% CI)
Darby et al., 2005	Pooled case control study, Europe	7148 lung cancer cases, 14 208 controls	Measured indoor radon conc.	Relative risk for lung cancer	Study, age, gender smoking, region of residence	RR per 100Bq/m^3 = 1.08 (CI 1.03 – 1.15) Corrected for radon measurement error: RR per 100Bq/m^3 = 1.16 (CI 1.05-1.31)
Krewski et al., 2005	Pooled case-control, North America	3662 lung cancer cases, 4966 controls	Measured indoor radon conc.	Odds ratio for lung cancer	Study, age, gender smoking, number of homes	OR at 100 Bq/m^3 = 1.11 (95% CI 1.00-1.28)
Lubin et al., 2004	Pooled case control study, China	1050 lung cancer cases, 1996 controls	Measured indoor radon conc.	Odds ratio for lung cancer	Age, gender, smoking, number of homes, further study-specific factors	OR at 100 Bq/m^3 = 1.33 (95% CI 1.01-1.36)

Active smoking, radon and lung cancer

Tobacco smoke is the strongest known lung carcinogen, and the interaction between smoking and radon exposure is of interest for the lung cancer risk assessment. The BEIR VI Committee considered selected miner studies, for which information on smoking was available. In these studies the lung cancer death rate increased by 0.53% per WLM (95% confidence interval 0.20-1.38%), similar to the average percentage increase for all eleven studies. Separate analyses for those who never smoked and for those who did showed an increase in the lung cancer death rate of 1.02% per WLM (95% confidence interval 0.15-7.18%) for the former and 0.48% per WLM (95% confidence interval 0.18-1.27%) for the latter. The observed difference in the cancer risk according to smoking history was, however, not statistically significant (BEIR VI, 1999).

The pooled indoor studies are the other large source of information on combined effects of radon and smoking. In the European pooled study, no significant differences in the RR estimates were found among current-, ex- and those who never smoked, even though the RR per 100 Bq/m^3 was slightly higher for never smokers than for the other groups. Similar results were observed in the North American pooled study.

Second-hand smoke, radon and lung cancer

Detailed empirical assessments of possible synergies between second-hand smoke (SHS, also referred to as ETS – Environmental Tobacco Smoke) and radon have proven difficult in the past. This area of research has been deemed rather complex (Crawford-Brown, 1992), partly due to the uncertainties involved in establishing both long-term radon and long-term SHS exposure status in individuals. The BEIR VI report additionally notes that the statistical power for assessing joint effects would likely be extremely limited (BEIR VI, 1999). The European pooled study (Darby et al., 2006) provided data on nonsmokers exposed to tobacco smoke from their

spouses. Although the RR per 100 Bq/m^3 was clearly higher for SHS exposed as against non-SHS exposed individuals (RR = 1.19 vs. RR 1.04), the difference was not statistically significant.

Non-lung cancer effects of radon

Numerous diseases other than lung cancer have been studied in relation to radon. A possible link between leukemia and indoor radon has aroused the most interest. The evidence for a positive relationship has been deemed weak, as it was based on ecological studies with major methodological weaknesses (Laurier et al., 2001). However, more recent evidence from case-control studies (Rericha et al., 2007; Raaschou-Nielsen et al., 2008) and a new French ecological study (Evrard et al., 2008) has led to renewed interest in this issue. No leukemia risk increase was seen among German miners (Mohner et al., 2006; Kreuzer et al., 2008). Non-lung cancers have also been investigated among miners, with very little evidence for risk increases (Darby et al., 1995; Kreuzer et al., 2008). Cardiovascular diseases have not been found to be linked to radon exposure. For multiple sclerosis and Alzheimer´s disease, a small number of studies have failed to establish conclusive evidence of any association. In summary it is not possible to perform risk assessments for diseases other than lung cancer at this time.

4. Exposure assessment

4.1 Method for exposure assessment

Whereas in dedicated epidemiological studies individual radon measurements were performed in homes of participants to establish individual exposures over time, national or regional indoor radon surveys provide information on mean indoor radon concentrations. Due to the log-normal distribution of indoor radon concentrations, geometric mean and geometric standard deviation are used in surveys where possible, but arithmetic means are also provided. A comprehensive approach to provide an overview of radon mapping in Europe has recently been provided by the EC joint research centre (Dubois et al., 2005). A marked methodological heterogeneity in the assessment of indoor radon concentrations was noted. The United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) regularly compiles radon concentration estimates as supplied from national authorities worldwide.

For detailed BoD country-specific estimates, data on mean radon concentrations for defined regions or other geographical units are used.

- In the French study (Catelinois et al., 2006), a radon survey that included 12 261 radon indoor measurements homogenously distributed over a 36-49 km^2 grid was used to determine the average radon exposure level. Seasonally corrected averages were computed for an average radon concentration of 87 Bq/m^3.
- For Switzerland (Menzler et al., 2008), the national monitoring programme provided data from a survey of 45 361 homes. The seasonally corrected and population-weighted arithmetic mean was 78 Bq/m^3 (geometric mean: 55 Bq/m^3).
- For Germany, the average radon concentration was calculated from different indoor studies, because no nationwide survey was available. The assessment included data from 32 336 dwellings, with a seasonally corrected and population-weighted average mean of 49 Bq/m^3 (geometric mean 44 Bq/m^3) (Menzler et al., 2008).

4.2 Summary of exposure levels to radon

Radon indoor data are now available for many countries in Europe. National summaries are available at http://radonmapping.jrc.ec.europa.eu. Mean values range from high concentrations above 100 Bq/m^3 in countries such as the Czech Republic and Finland to markedly lower values

in the Netherlands and the United Kingdom, with many countries displaying values in the range of 50 – 90 Bq/m^3. In countries with comparatively high radon concentrations, the percentage of homes above 200 Bq/m^3 – used as a reference value in many countries – is correspondingly high and exceeds 10% in Austria, the Czech Republic, Finland, Sweden and Switzerland.

Whereas arithmetic means are available for all countries, the geometric mean is not always provided. As expected, arithmetic means are generally higher than the geometric means, and sometimes markedly so. However, it should be noted that data provided by different countries are not always directly comparable as they are based on different survey methods.

5. Total burden of disease from radon

5.1 Method for measuring the radon disease burden

As previously stated, lung cancer incident cases and deaths are generally assumed to be very similar in magnitude due to the continuous high fatality of the disease. Lung cancer mortality data per sex and age are available from national statistics in countries with functioning death registration systems.

5.2 Summary of disease burden

Globally, lung cancer is the most frequent cause of cancer mortality among men and the second for women and accounts for some 22% of all cancer deaths in the WHO European Region (WHO, 2005). Annually around 292 000 lung cancer deaths among men and 76 300 among women were estimated for the WHO European Region based on data for the year 2000 (Shibuya et al., 2002). 240 000 of these deaths occur in EU member countries[9]. The age-standardized incidence rate for cancers of the trachea, bronchus and lung for the WHO European Region varies markedly between subregions and is highest in the subregion Euro A among women (13.4/100 000) and in the subregion Euro C for men (52.2/100 000) (Shibuya et al., 2002). The number and rates concerning mortality are only slightly different due to the low survival of lung cancer sufferers (incidence/mortality ratio 1.07-1.10 globally) (Table 2).

6. Environmental burden of disease from radon exposure

The exposure-risk relationship for radon and lung cancer has been established in numerous studies. Different risk models have been established, notably those based on miner data and those using indoor study data. Radon levels in European homes are highly variable and have been measured systematically in several countries. Scientifically robust BoD assessments on the basis of a computed PAF of lung cancer from indoor radon have not been performed for all countries in Europe, and different methods (e.g risk models used, inclusion of smoking data) have been applied in available assessments. Studies from France, Switzerland and Germany serve as examples and indicate the range of potential EBD associated with radon. Similar data are available for several other countries in Europe. It should be noted that the vast majority of radon-associated deaths generally occurs among smokers.

To illustrate the numerical computation, a crude PAF for Switzerland is calculated using a RR of 1.16 per 100 Bq/m^3, a baseline radon concentration of 10 Bq/m^3 and a countrywide average indoor radon concentration of 78 Bq/m^3. The RR at 78 Bq/m^3 against a baseline (comparison) radon concentration of 10 Bq/m^3 would be 1.107.

[9] See http://www.euphix.org/object_document/ o4595n27172.html

$$PAF = \frac{0.107}{1.107} = 0.0967 = 9.7\%$$

The detailed calculations for lung cancer deaths in Switzerland, using the European indoor risk model, yield an overall PAF of 8.3%, with confidence limits for different categories of sex and smoking status ranging from about 3 – 24%. Due to the slightly lower average radon concentrations in Germany, the PAF was estimated to be 5%, with confidence intervals from about 2 – 13%.

Of the 25 134 lung cancer deaths in France in 1999, from 2% (90% confidence interval 0.3 – 4.4%) to 12% (11 – 13%) were estimated to be attributable to indoor radon exposure. The different values are derived from the application of different risk models, considering the entire log normal distribution of indoor radon concentration by administrative unit. With the risk model from the latest European pooled analysis a PAF of 5% (2.4 – 9%) was calculated for France, which is consistent with data from other countries.

The case studies illustrate comprehensive radon BoD assessments and show the range of PAFs that are to be expected in different countries and with the application of different risk models. Countries with high radon concentrations obviously will generally have higher PAFs whereas in countries such as the Netherlands, only a very small percentage of all lung cancer deaths are likely to be attributable to radon.

7. Uncertainty

7.1 Areas of uncertainty

Radon is a proven carcinogen (IARC, 1988), and there is strong evidence that radon concentrations typically found in European homes contribute to the lung cancer burden. Even though questions as to the mechanisms of disease causation are still an active area of research, the evidence from many studies in different populations consistently points to a linear relationship between radon concentration and lung cancer risk, without a threshold.

Areas of uncertainty include the quality and extent of radon surveys in Europe, the impact of changing living and housing conditions (multistorey buildings, effects of improving insulation and energy efficiency), and issues related to the radon-smoking interaction. In addition, possible interactions between radon and other indoor air pollutants are being discussed. More evidence is also needed on non – lung cancer health effects of radon. On the other hand, radon is one of the best-established environmental health hazards, and it is unlikely that major new evidence will lead to marked changes in the current assessment of health risks and associated disease burden.

7.2 Reducing uncertainty

There is a need to obtain more representative data on indoor radon concentrations in European homes. Currently, some surveys only focus on areas with suspected high radon concentrations. If the variability of radon concentrations with season and other factors can be taken into account in scientific studies, more realistic radon burden estimates will be generated. Similarly, updated smoking prevalence data are needed.

8. Conclusions

Radon gas is an established carcinogen and the major source of natural ionizing radiation exposure in most countries. The evidence available to date suggests that indoor exposure to

radon is a significant risk factor for lung cancer. Between 5 and 10% of all lung cancers can be attributed to radon, although varying local conditions may lead to even higher (or lower) estimates (Table 3).

Table 3. Summary of EBD of lung cancer caused by indoor radon exposure

Housing exposure	Radon, Bequerel/m^3			
Health outcome	Lung cancer			
Summary of EBD evidence	2-12% of all lung cancer deaths in the EU can be attributed to radon exposure. For France, Germany and Switzerland, the total number of radon-related lung cancer deaths is estimated to be about 3300 per year.			
	Level	**Range**	**Geographic scope**	**Source of information**
(a) Exposure risk relationship	RR 1.08/100 Bq/m^3	95% CI: 1.04 – 1.12 per 100 Bq/m^{3*}	European countries, United States, China	13 pooled case-control studies; further studies with similar results
(b) Exposure assessment	France 87 Bq/m^3 Switzl. 78 Bq/m^3 Germany 49 Bq/m^3	Mean values 20 – 140 Bq/m^3, depending on country (see table 1)	National level (data available for other countries)	National or regional surveys, variable methodology
(c) PAF	France 5% Switzerland: 8.3% Germany 5% EU countries: 2–12%	95% CI (%) France: 2.4–9 Switzerland: 3–24 Germany: 2.4–9 EU countries: 0.3–24	National level (data available for other countries)	Individual country studies
(d) Total burden of disease	Deaths per year: France: 25 134 (1999) Switzerland: 2780 (2001) Germany 37 900 (av. 1996-2000) WHO European Region: 368 200 (2000)	See table 2	National level (data available for other countries)	Individual country studies Country reports to European Office of WHO
(e) EBD from indoor radon exposure	Estimated deaths per year: France: 1234 (2.1/100 000) Switzerland: 231 (3.2/100 000) Germany: 1896 (2.3/100 000)		National level (data available for other countries)	Combined from (c) and (d) above
Main areas of uncertainty	Different risk models from etiological studies among miners and in the general population yield somewhat different results; exposure data (both on radon and smoking) are of differing quality across the WHO Regional Office for Europe member countries;			
Main implications	As the available evidence suggests that radon clearly contributes to the risk of lung cancer, actions to minimize indoor radon exposure including prevention in new homes and mitigation in existing homes are required. National radon programmes should be implemented where feasible.			

Bq = Becquerel, CI = Confidence interval, RR = Relative Risk

* for the "corrected European model": RR 1.16 (1.05 – 1.31) per 100 Bq/m^3

9. Policy implications

Because radon exposure is a significant risk factor for lung cancer, precautions should be taken to limit this exposure. Both prevention (in new homes) and mitigation (in existing homes) approaches should be promoted, ideally in a comprehensive radon programme that includes, among others, guidance on surveying, on measurements and on radon risk communication and evaluation (WHO, 2009). Several European countries have well-established radon programmes, and the numerous local, national and European radon projects need to be extended further to better protect the population of the WHO European Region from this well-controllable environmental hazard.

10. References

BEIR VI (1999). Health Effects of Exposure to Radon – BEIR VI. Washington, D.C., National Academy Press.

Catelinois O et al. (2006). Lung cancer attributable to indoor radon exposure in France: impact of the risk models and uncertainty analysis. *Environmental Health Perspectives*, 114:1361-1366.

Crawford-Brown DJ (1992). Modeling the modification of the risk of radon-induced lung cancer by environmental tobacco smoke. *Risk Analysis,* 12:483-493.

Darby S et al. (1995). Radon and cancers other than lung cancer in underground miners: a collaborative analyses of 11 studies. *Journal of the National Cancer Institute*, 87:378-384.

Darby S et al. (2005). Radon in homes and risk of lung cancer: collaborative analysis of individual data from 13 European case-control studies. *British Medical Journal*, 330:223-227.

Darby S et al. (2006). Residential Radon and Lung Cancer: Detailed Results of a Collaborative Analysis of Individual Data on 7,148 Subjects with Lung Cancer and 14,208 Subjects without Lung Cancer from 13 Epidemiological Studies in Europe. *Scandinavian Journal of Work, Environment & Health*, 32 (Suppl.1):1-83.

Evrard AS et al. (2006), Childhood leukemia incidence and exposure to indoor radon, terrestrial and cosmic gamma radiation. *Health Physics,* 90:569-579.

Gray A et al. (2009). Lung cancer deaths from indoor radon and the cost effectiveness and potential of policies to reduce them. *British Medical Journal*, 338:a3110.

Grosche B et al. (2006). Lung cancer risk among German male uranium miners: a cohort study, 1946-1998. *British Journal of Cancer*, 95:1280-1287.

International Agency for Research on Cancer (1988). *Monograph No 43 Man-made Mineral Fibres and Radon.* IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Lyon, IARC.

Kreuzer M et al. (2002). Characteristics of the German uranium miners cohort study. *Health Physics*, 83:26-34.

Kreuzer M et al. (2008). Radon and risk of extrapulmonary cancers – Results of the German uranium miners cohort study, 1960-2003. *British Journal of Cancer,* 99:1946-1953.

Krewski D et al. (2005). Residential radon and risk of lung cancer: A combined analysis of 7 North American case-control studies. *Epidemiology*, 16:137-145.

Krewski D et al. (2006). A combined analysis of North American case-control studies of residential radon and lung cancer. *Journal of Toxicology and Environmental Health A*, 69:533-597.

Laurier D, Valenty M, Tirmarche M (2001). Radon exposure and the risk of leukemia: A review of epidemiological studies. *Health Physics*, 81:272-288.

Lubin JH et al. (2004). Risk of lung cancer and residential radon in China: pooled results of two studies. *International Journal of Cancer*, 109:132-137.

Menzler S et al. (2008). Population attributable fraction for lung cancer due to residential radon in Switzerland and Germany. *Health Physics*, 95:179-189.

Mohner M et al. (2006). Leukemia and exposure to ionizing radiation among German uranium miners. *American Journal of Industrial Medicine,* 49:238-248.

Raaschou-Nielsen O et al. (2008). Domestic radon and childhood cancer in Denmark. *Epidemiology,* 19:536-543.

Rericha V et al. (2006). Incidence of leukemia, lymphoma, and multiple myeloma in Czech uranium miners: a case-cohort study. *Environmental Health Perspectives,* 114:818-822.

Shibuya K et al. (2002). Global and regional estimates of cancer mortality and incidence by site: II. results for the global burden of disease 2000. *BMC Cancer*, 2:37 doi:10.1186/1471-2407-2-37.

Skowronek J, Zemla B (2003). Epidemiology of lung and larynx cancers in coal mines in Upper Silesia–preliminary results. *Health Physics*, 85:365-370.

UNSCEAR (2000). *Sources and Effects of Ionizing Radiation.* Report to the General Assembly, with Scientific Annexes. United Nations Scientific Committee on the Effects of Atomic Radiation. New York, United Nations.

UNSCEAR (2008). *Sources-to-Effects Assessment for radon in homes and workplaces.* Report to the General Assembly, with Scientific Annexes. United Nations Scientific Committee on the Effects of Atomic Radiation. New York, United Nations.

Veiga LH et al. (2007). Feasibility study for a long-term follow-up in a historical cohort of Brazilian coal miners. *Journal of Radiological Protection*, 27:349-360.

WHO (2005). The European health report 2005: public health action for healthier children and populations. Copenhagen, WHO Regional Office for Europe. (http://www.euro.who.int/__data/assets/pdf_file/0004/82435/E87325.pdf, accessed 12 March 2008).

WHO (2009). WHO Handbook on Indoor Radon: A Public Health Perspective. Geneva, WHO. (http://whqlibdoc.who.int/publications/2009/9789241547673_eng.pdf, accessed 05 June 2010).

Residential second-hand smoke exposure and lower respiratory infections, asthma, heart disease and lung cancer

Maritta S. Jaakkola

1. Introduction

Second-hand tobacco smoke (SHS) exposure is defined as exposure to tobacco combustion products from smoking by others (Jaakkola, Jaakkola, 1997). It usually refers to exposure of non-smokers, although smokers themselves have high exposure to SHS in addition to their exposure to mainstream smoke. Commonly used synonyms in the research literature are 'passive smoking' and 'exposure to environmental tobacco smoke (ETS)'.

SHS is a combination of sidestream smoke (SS), which is released directly into the environment from the burning end of the cigarette between puffs, and exhaled mainstream smoke (MS). MS is smoke inhaled by the smoker during puff drawing. SS forms about 80% of SHS. Tobacco smoke is known to contain more than 4000 chemical compounds, including about 50 carcinogens and tens of irritant and toxic substances. The concentrations of many harmful substances are higher in undiluted SS than in MS due to differences in burning conditions (Jaakkola, Samet, 1999).

Population-based multicentre studies in Europe and the United States have measured cotinine, a major metabolite of nicotine, in body fluids of non-smokers. They have shown that some degree of exposure to SHS is very common among non-smoking populations, since more than 80% have had detectable levels of serum cotinine (Riboli et al., 1990; Pirkle et al., 1996). Indeed, SHS seems to be the most important preventable indoor pollutant in developed countries due to its well-established adverse health effects and high prevalence of exposure in many countries.

2. Summary of the method

The best available effect estimates in the form of relative risk, i.e., risk ratio (RR), odds ratio (OR), or incidence rate ratio (IRR), for different health outcomes were identified based on a literature review. Odds ratio was the risk estimate most commonly available. The effect estimate used here for the Environmental Burden of Disease (EBD) calculation is drawn from a recent meta-analysis and, if an estimate from a meta-analysis was not available, recent high quality individual studies. Only health outcomes for which there is a broad consensus on a causal relation to SHS in the recent reviews and for which there is reliable and quantifiable health statistics were used (Öberg et al., 2010a).

The prevalence estimates of SHS exposure in the early years of the 21st century were based on published surveys or on modeling of exposure data, mainly according to the proportion of children with at least one smoking parent as described in more detail in the assessment of the global burden of disease from SHS (Öberg et al., 2010b). Multicentre surveys of SHS exposure that included European countries were used when available. Published data included the Global Youth Tobacco Survey (GYTS) that included exposure data from 2002-2007 (CDC, 2009) and Eurobarometer data from 2009 (The Gallup Organization, 2009).

The following formulas are used to calculate the area-specific population attributable fraction (PAF) for each health outcome (Greenland, 1998):

Population attributable fraction

$$PAF = \frac{p\,(RR-1)}{p\,(RR-1)+1}$$

where p is the proportion of the population exposed to SHS at home and RR is the relative risk for the outcome of interest.

Finally, to calculate the EBD from home SHS exposure the following formula is used:

EBD = PAF x the number of deaths or Disability Adjusted Life Years (DALYs).

DALY is the sum of the years of life lost due to premature mortality in the population and the years lost due to disability for incident cases of the health condition (Murray et al., 2002).

As adult asthma, lung cancer and ischaemic heart disease are strongly related to active smoking, EBD from SHS exposure was calculated by subtracting the EBD due to active smoking from the disease burden. The calculations were performed with the WHO spreadsheet for estimating disease burden from SHS.[10]

For calculating EBD as number of deaths or DALYs due to SHS exposure, the data on disease statistics was from the Global Burden of Disease 2004 update[11].

3. Exposure-risk relation for the selected health effects

The first reports linking parents' smoking to respiratory infections of their children were reported in the 1970s (Norman-Taylor, Dickinson, 1972; Harlap, Davies, 1974). The first studies on health effects related to SHS exposure in adults were published in the early 1980s and showed increased risk of lung cancer in non-smoking women with a smoking spouse (Hirayama, 1981; Trichopoulos et al., 1981). Since then increasing amount of studies on different adverse health effects of SHS have accumulated and meta-analyses have been conducted on health outcomes such as children's respiratory infections and asthma, and adults' lung cancer and coronary heart disease.

The evidence on health effects of SHS in children and adults has been reviewed recently for a WHO guide on assessing the Environmental Burden of Disease from SHS (Öberg et al., 2010a). The best available estimates of relative risk for health outcomes were identified based on previous major reviews of SHS by the United States Department of Health and Human Services (2006) and California Environmental Protection Agency (2005), other recent meta-analyses, and reviews by individual scientists (Jaakkola, Jaakkola, 2002a; Jaakkola, Jaakkola, 2002b; Jaakkola, Jaakkola, 2006). For outcomes that have been studied mainly in the recent years, i.e. after the major reviews were conducted, a Medline database search was completed to identify the best risk estimate based on a high-quality individual study. For adult asthma and chronic obstructive pulmonary disease (COPD) the following terms were used: 'tobacco smoke pollution' AND 'asthma OR respiratory tract disease OR chronic obstructive pulmonary disease'.

[10] The WHO spreadsheet can be obtained by contacting EBDassessment@who.int
[11] More information on these can be found at the webpage address: www.who.int/healthinfo/global_burden_disease/en/index.html

Table 1 summarizes the selected estimates of relative risk for diseases/ conditions that are causally related to SHS exposure, i.e. for which the evidence has been judged as level 1, meaning that there is sufficient evidence of causality.

Table 1. The relative risk estimates for health effects related to SHS exposure

Health effects	Age group (years)	Exposure	RR/OR/IRR (95% CI)	Reference
Developmental effects in children				
Low birth weight (LBW)	0	Non-smoking mother with SHS exposure at home or at work	1.38 (1.13-1.69)	Windham, 1999 (meta-analysis)
Sudden infant death syndrome (SIDS)	<1	Any parental smoking	1.94 (1.55-2.43)	Andersson, 1999 (meta-analysis)
Respiratory effects in children				
Acute lower respiratory infections (ALRI)	<2	Any parental smoking	1.59 (1.47-1.73)	Department of Health and Human Services, 2006 (meta-analysis)
Chronic wheezing	<14	Any parental smoking	1.26 (1.20-1.33)	Department of Health and Human Services, 2006 (meta-analysis)
Chronic cough	<14	Any parental smoking	1.35 (1.27-1.43)	Department of Health and Human Services, 2006 (meta-analysis)
Acute/ recurrent otitis media	<4	Any parental smoking	1.38 (1.21–1.56)	California EPA (Cal-EPA), 2005 (review based on high-quality study from Etzel, 1992)
Onset of asthma	<14	Any parental smoking	1.32 (1.24-1.41)	Cal-EPA, 2005 (meta-analysis)
Respiratory effects in adults				
Onset of asthma	≥21	SHS exposure at home or at work	1.97 (1.19-3.25)	Jaakkola, 2003 (high-quality study)
Cancer				
Lung cancer	>15	SHS exposure at home	1.21 (1.13-1.30)	Department of Health and Human Services, 2006 (meta-analysis)
Cardiovascular effects				
Ischaemic heart disease (IHD)	>15	SHS exposure at home or at work	1.27 (1.19-1.36)	Department of Health and Human Services, 2006 (meta-analysis)

Adapted from: Öberg et al., 2010a

The EBD was not calculated for respiratory symptoms, because these are often related to respiratory diseases such as asthma, in order to avoid possible double counting of the burden. Low birth weight and sudden infant death syndrome were not included in the EBD calculation due to lack of reliable quantitative disease statistics.

4. Exposure assessment

4.1. Method for SHS exposure assessment

SHS exposure can be measured directly by measuring personal exposure to tobacco smoke constituents, such as nicotine and respirable suspended particles, with personal air sampling (Jaakkola, Jaakkola, 1997). Exposure can also be measured indirectly by questionnaires and interviews, and by measuring tobacco smoke constituents in different microenvironments. Measuring biomarkers, such as cotinine in body fluids, provides measures of dose rather than exposure (Jaakkola, Jaakkola, 1997). Health effect studies have usually used questionnaire-based assessment of SHS exposure. Questionnaires enable assessment of exposures in different microenvironments (including both home and work) and can also be used to measure past exposures, which may be most relevant for health effects such as lung cancer (Jaakkola, Samet, 1999). In addition, questionnaires are an inexpensive way to assess exposure in large populations. Some studies have combined the use of questionnaires with the use of biomarkers or indoor measurements of tobacco smoke constituents.

In order to quantify health impacts, questionnaire-based SHS exposure estimates were used here in order to ensure that the method to assess exposure prevalence is compatible with the exposure assessment method used in the health effect studies. Data from multicentre studies allow comparisons between populations and countries.

4.2 SHS exposure in Europe

There are differences in the prevalence of SHS exposure across European countries due to differences in smoking habits and traditions and differences in tobacco control legislation.

Table 2 presents SHS exposure estimates for children from the Global Youth Tobacco Survey and for adults from the Eurobarometer survey. The GYTS data were developed by WHO and the United States' Centers for Disease Control and Prevention (CDC) to measure tobacco exposure among children 13-15 years (CDC, 2009). These data characterize well the eastern European countries as well as some central and southern European countries, but there are fewer data from western and northern European countries. For these countries data from other surveys, such as Pollution and The Youth (Pattenden et al., 2006) or a multicenter Nordic study (Lund et al., 1998), were used. The Eurobarometer data provided estimates for SHS exposure from living with someone who smokes inside the house and for daily SHS exposure at work across large regions of Europe (The Gallup Organization, 2009). In the total European assessment, exposure for those countries that were not included in any of the surveys was estimated as the mean exposure in that area. It should be noted that only SHS exposure at home was used in this calculation of the EBD from housing factors, but for the total burden of disease from SHS exposure the contribution from work exposure should also be taken into account.

Table 2: Exposure from second-hand smoke by country, data from two international surveys

		Global Youth Tobacco Survey #		Eurobarometer°		
Subregion*	**Country***	**Having one or more parents who smoke (%)**	**Year of survey**	**Living with someone who smokes inside the house (%)**	**Daily exposure at work (%)**	**Year of survey**
Euro A	Austria	NA	-	14	15	2008
Euro A	Belgium	NA	-	18	10	2008
Euro A	Croatia	59	2007	NA	NA	-
Euro A	Cyprus	56	2005	31	30	2008
Euro A	Czech Republic	50	2007	16	20	2008
Euro A	Denmark	NA	-	17	15	2008
Euro A	Finland	NA	-	2	9	2008
Euro A	France	NA	-	9	11	2008
Euro A	Germany	NA	-	13	12	2008
Euro A	Greece	67	2005	28	30	2008
Euro A	Ireland	NA	-	14	11	2008
Euro A	Italy	NA	-	11	9	2008
Euro A	Luxembourg	NA	-	8	8	2008
Euro A	Malta	NA	-	10	13	2008
Euro A	Netherlands	NA	-	15	9	2008
Euro A	Norway	NA	-	6	10	2008
Euro A	Portugal	NA	-	13	9	2008
Euro A	Slovenia	48	2003	15	11	2008
Euro A	Spain	NA	-	20	13	2008
Euro A	Sweden	NA	-	3	6	2008
Euro A	United Kingdom	NA	-	7	8	2008
Euro B	Albania	46	2003	NA	NA	-
Euro B	Armenia	68	2005	NA	NA	-
Euro B	Bosnia and Herzegovina	69	2003	NA	NA	-
Euro B	Bulgaria	76	2002	23	23	2008
Euro B	Georgia	73	2002	NA	NA	-
Euro B	Kyrgyzstan	37	2003	NA	NA	-
Euro B	Poland	59	2003	21	18	2008
Euro B	Romania	61	2004	23	24	2008
Euro B	Serbia and Montenegro	69	2003	NA	NA	-
Euro B	Slovakia	55	2002	13	21	2008
Euro B	Tajikistan	31	2007	NA	NA	-
Euro B	The former Yugoslav Republic of Macedonia	64	2002	NA	NA	-
Euro C	Belarus	60	2003	NA	NA	-
Euro C	Estonia	59	2002	16	13	2008
Euro C	Hungary	58	2002	12	13	2008
Euro C	Kazakhstan	54	2003	NA	NA	-
Euro C	Latvia	64	2002	12	18	2008
Euro C	Lithuania	59	2005	28	19	2008
Euro C	Republic of Moldova	50	2003	NA	NA	-
Euro C	Russian Federation	62	2004	NA	NA	-
Euro C	Ukraine	62	2005	NA	NA	-

Data for children of approximately 13-15 years (CDC, 2009)
° Data for non-smoking adults (The Gallup Organization, 2009)
* The list of countries for the European subregions is provided by Table 1 of the Introduction chapter. Countries are defined as they were in 2004 at the time that these data were collated.
NA: Not available

5. Environmental burden of disease from SHS in the home

The estimates of burden of disease for the three European subregions Euro A, B and C are given as the number of deaths (Table 3) and DALYs (Table 4) for the year 2004.

Table 3: Deaths from home exposure to second-hand smoke, in 2004

Disease (age group)	Region*			European Region
	Euro A	Euro B	Euro C	Total
Lower respiratory infections (children aged 0-2 years)[#]	60	5400	820	6 200
Otitis (children)[#]	<10	<10	<10	<10
Asthma (children)[#]	10	110	<10	120
Asthma (adults)[$]	500	750	1100	2400
Lung cancer (adults)[$]	1600	270	290	2200
Ischaemic heart disease (adults)[$]	12 500	14 300	25 400	52 200
Total	**14 700**	**22 300**	**27 700**	**64 700**

[#] Values for children: from Öberg et al., 2010b, Öberg et al., 2011

[$] Values for adults: Countries with missing exposure information were attributed weighted regional mean

* The list of countries for the European subregions is provided by Table 1 of the Introduction chapter

Table 4: DALYs from home exposure to second-hand smoke, in 2004

Disease (age group)	Region*			European Region
	Euro A	Euro B	Euro A	Total
Lower respiratory infections (children aged 0-2 years)[#]	2300	185 000	28 200	215 000
Otitis (children)[#]	810	700	500	2000
Asthma (children)[#]	35 800	22 600	8300	66 700
Asthma (adults)[$]	22 400	18 200	12 000	52 600
Lung cancer (adults)[$]	13 600	2700	2700	19 000
Ischaemic heart disease (adults)[$]	58 900	98 500	158 000	316 000
Total	**119 000**	**384 000**	**210 000**	**713 000**

[#] Values for children: from Öberg et al., 2010b, Öberg et al., 2011

[$] Values for adults: Countries with missing exposure information were attributed weighted regional mean

* The list of countries for the European subregions is provided by Table 1 of the Introduction chapter

Tables 3 and 4 show in 2004 in Europe, 64 700 deaths were attributable to SHS exposure at home and 713 000 DALYs were lost due to SHS exposure at home. The biggest amount of deaths attributable to SHS exposure was seen in European areas B and C, where also the largest loss of DALYs attributable to home SHS exposure was observed. Based on this assessment considerable health benefits could be achieved by eliminating SHS exposure from European homes. Tables 3 and 4 present EBD from SHS for the three WHO European subregions. It is recommended that each country calculates its own EBD based on the most recent survey data on

SHS exposure and disease statistics to get a more updated estimate. It is important to bear in mind that these estimates are for SHS exposure taking place at home, while SHS exposure at work contributes additional EBD and increases total EBD from SHS exposure considerably. Estimates of total burden of disease attributable to SHS exposure for other parts of the world are presented in Öberg et al. (2010b; 2011).

Among children, the burden of lower respiratory infections attributable to SHS represents 215 000 DALYS in Europe, with the highest burden of disease seen in the European subregion Euro B with countries in eastern Europe mainly. SHS exposure in Europe also attributes a total of 6200 deaths from acute respiratory infections annually. Asthma, which is the most common chronic disease in children, also has a high burden of disease from SHS exposure, accounting for a total of 66 700 DALYs and 120 deaths annually in Europe. Deaths due to asthma attributable to SHS are highest in subregion Euro B, while the highest DALYs due to asthma from SHS are observed in Euro A. Otitis media from SHS accounts for 2 000 DALYs in children, but fortunately only a small amount of deaths, as this is a disease for which good treatments are nowadays available.

Among adults, the highest burden of disease from SHS exposure is seen from ischaemic heart disease with a total of 316 000 DALYS and 52 200 deaths annually in Europe. The highest burden of disease from ischaemic heart disease attributable to SHS is seen in Euro C. Adult asthma attributable to SHS also contributes a significant amount of burden of disease with 52 600 DALYs and 2400 annual deaths in Europe. The highest burden of deaths from asthma attributable to SHS is seen in Euro C, while the largest amount of DALYs due to asthma from SHS is observed in Euro A. Lung cancer attributable to SHS also contributes significantly to the burden of disease with a total of 19 000 DALYs and 2200 deaths annually in Europe.

6. Uncertainty

6.1 Relative risk

The uncertainty of the risk estimates is discussed in more detail in the WHO guide on assessing the environmental burden of disease from SHS (Öberg et al., 2010a). To evaluate whether the relation between an exposure and a health outcome can be judged as causal (i.e. level 1 evidence) the following criteria were applied: consistency of effects in studies from different parts of the world; evidence of exposure-effect relation; meaningful temporal relation between exposure and outcome; lack of major biases; biologically plausible mechanisms; and adjustment for major confounders. When the risk estimate was derived from an individual high-quality study it reflects exposure conditions in a certain population/country, so the generalizability is not as good as for estimates from meta-analyses. However, the high quality study used to obtain the risk estimate for adult asthma was from a study conducted in Finland, so it is likely to reflect the situation in northern Europe as well as the current situation in many parts of western Europe. The study that formed the basis for the effect estimate of acute/recurrent otitis media was performed in United States, where the exposure prevalence was similar to those in northern and western Europe. However, in other areas of Europe the SHS exposure is often more prevalent and may also be of higher level, so these risk estimates may underestimate the health effects in subregions Euro B and C. The methods guide by Öberg et al. (2010a) also reviews evidence on other disease outcomes, such as breast cancer and stroke, for which there is increasing evidence of a role of SHS, but these were not included in this assessment, because level 1 evidence for a causal role was required for all health conditions included in this assessment.

6.2. Exposure estimates

Exposure estimates based on questionnaires carry larger uncertainties than those based on personal monitoring. However, questionnaire-based exposure assessment is often used for health effect studies. The advantage of using questionnaire data for assessing exposure in health effect studies include the ability to assess exposure during the time period most relevant for the health effect of interest as well as the possibility to assess exposures in different microenvironments (Jaakkola, Samet, 1999). Thus, using SHS exposure data from questionnaire-based surveys is compatible with the method used for assessing the health risk. In this assessment, SHS exposure data was derived from multicentre studies that had used standardized assessment tools to allow comparisons between the countries. Unfortunately, no survey was identified that covered all the European countries, so data from more than one multicentre survey had to be used. It should be noticed that any survey gives a picture of exposure situation at a specific point in time, so updating of surveys on SHS exposure and consequently EBD assessments should be conducted repeatedly. Other questions related to uncertainties in exposure estimates are discussed in the WHO report on Global estimate of the burden of disease from second hand smoke (Öberg et al., 2010b; Öberg et al., 2011).

6.3. Disease statistics

There are substantial data gaps and deficiencies, particularly for countries with limited death registration data. Information on these as well as on calculations of DALYs based on occurrence of diseases and long-term disability from these diseases is given in Annex B of www.who.int/healthinfo/global_burden_disease/en/index.html. The calculation of DALYs is approximated, which adds to uncertainty. In addition, there are no reliable disease statistics available for low birth weight or sudden infant deaths, which had to be excluded from this estimation, although there is strong evidence on the causal role of SHS for these conditions. Such exclusions suggest that our estimate is conservative and may well underestimate the true magnitude of the EBD from SHS.

7. Conclusions

The EBD from SHS is estimated as 713 000 DALYs and 64 700 annual deaths in Europe (Table 5), suggesting a significant adverse impact on public health. The highest burden of disease in terms of DALYs is seen in Euro B, followed by Euro C. SHS is one of the most important preventable indoor pollutants in Europe, attributing significantly to disability and deaths from lower respiratory infections and asthma in children and ischaemic heart disease, asthma and lung cancer in adults. With successful preventive actions those countries with high current SHS exposure prevalence will get proportionally a larger health benefit. These results show that there is a need for rather urgent preventive actions to reduce the burden of disease from SHS in Europe.

Table 5. Summary of EBD from residential SHS

Housing exposure	Residential second-hand smoke			
Health outcome	Lower respiratory infections (children aged 0-2 years); Otitis (children); Asthma (children); Asthma (adults); Lung cancer (adults); Ischaemic heart disease (adults)			
Summary of EBD evidence	For the WHO European Region, 64 700 deaths and 713 000 DALYs per year can be attributed to second-hand smoke. More detailed assessments for subregions, age groups and health outcomes are available (see Tables 3 and 4).			
	Level	**Range**	**Geographic scope**	**Source of information**
(a) Exposure risk relationship	LRI:1.59 Otitis: 1.38 Asthma (children): 1.32 Asthma (adults): 1.97 Lung cancer: 1.21 Ischaemic heart disease: 1.27	1.47-1.73 1.21–1.56 1.24-1.41 1.19-3.25 1.13-1.30 1.19-1.36	Mainly from meta-analyses based on studies from different countries; adult asthma: Finland	Jaakkola et al., 2003; California EPA report 2005; Department of Health and Human Services, 2006
(b) Exposure assessment	Varies by country (for details, see Table 2)	Varies by country	European countries	GYTS (CDC, 2009) Eurobarometer (The Gallup Organization, 2009), other multicenter surveys
(c) PAF	LRI 23% Otitis 12% Asthma (children) 10% Asthma (adults) 6% Lung cancer 0.6% Ischemic heart disease 2%	Varies by country	WHO European Region	Calculated based on (a) and (b)
(d) Total burden of disease	Lower respiratory infections: 235 000 deaths, 2.6 million DALYs; Otitis: 277 deaths, 94 000 DALYs; Asthma: 36 000 deaths, 1.3 million DALYs; Lung cancer: 371 000 deaths, 3.3 million DALYs; Ischaemic heart disease: 2 296 000 deaths, 16.8 million DALYs		WHO European Region	WHO Global BOD Report (WHO, 2008)
(e) EBD from second-hand smoke (in 2004)	64 700 deaths (7.3/100 000) 713 000 DALYs (80.7/100 000)		WHO European Region (for results by health outcome, region and age group see Tables 3 and 4)	WHO Global BOD Report (WHO, 2008) and calculations presented in this chapter.
Main areas of uncertainty	Potential misclassification of SHS exposure; area means used to approximate exposure in countries with no survey data			
Main implications	EBD from SHS has a significant adverse impact on public health in Europe. The highest EBD attributable to SHS is seen in subregion Euro B, followed by Euro C. SHS is one of the most important preventable indoor pollutants in Europe, attributing significantly to disability and deaths from lower respiratory infections and asthma in children and ischaemic heart disease, asthma and lung cancer in adults.			

8. Policy implications

Tobacco-free workplace legislation has been shown to reduce SHS exposure efficiently at work, with some data suggesting that such legislation also reduces to some extent SHS exposure at home, probably through promoting smoking cessation and increasing awareness of adverse health effects related to SHS (Jaakkola, Jaakkola, 2006). However, home SHS exposure should be targeted also by other types of interventions. In general, tobacco control laws reduce the prevalence of smoking and as a consequence of this exposure to SHS at home is al reduced. Educational programs on health risks related to SHS exposure are important to raise the awareness of the adverse effects related to passive smoking and to emphasize the importance of protecting non-smokers, especially children.

There are important factors related to housing that could be of importance for reducing SHS exposure in homes. Examples of such include: 1) in apartment buildings/houses maintenance charges could be set higher for smokers than for non-smokers, 2) landlords renting apartments or houses could rent them only to tenants signing an agreement not to smoke indoors, 3) houses and apartment buildings should be designed so that it is easy for smokers to smoke outdoors and away from intake vents for air, e.g. by providing a balcony on each floor, and 4) those smoking indoors could have extra taxes or a higher insurance premium due to increased risk of fire.

Smoking and exposing other people to tobacco smoke is more common in lower socioeconomic classes in Europe, especially where crowding is a problem, so improving housing standards in general is likely to lead to reduction in smoking and SHS exposure at homes.

9. References

Anderson HR, Cook DG (1997). Passive smoking and sudden infant death syndrome: review of epidemiological evidence. *Thorax,* 52:1003-1009.

California Environmental Protection Agency (2005). *Proposed identification of environmental tobacco smoke as a toxic air contaminant. Art B: Health effects assessment for environmental tobacco smoke*. California EPA, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Branch.

CDC. *Global Youth Tobacco Survey (GYTS)*. Atlanta, Centers for Disease Control and Prevention, Office on Smoking and Health. (http://www.cdc.gov/Tobacco/global/gyts/intro.htm, accessed 13 October 2009).

Department of Health and Human Services (2006). *The health consequences of involuntary exposure to tobacco smoke. A report of the Surgeon General*. Atlanta, U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, Coordinating Center for Health Promotion, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health.

Etzel RA et al. (1992). Passive smoking and middle ear effusion among children in day care. *Pediatrics,* 90:228-232.

The Gallup Organization (2009). *Survey on Tobacco. Analytical Report.* European Commission. (http://ec.europa.eu/public_opinion/flash/fl_253_en.pdf, accessed 4 November 2009).

Greenland S (1998). *Applications of stratified analysis methods. Modern epidemiology, 2nd ed*. Philadelphia, Lippincott-Raven.

Harlap S, Davies AM (1974). Infant admission to hospital and maternal smoking. *Lancet*, 1:529-532.

Hirayama T (1981). Non-smoking wives of heavy smokers have a higher risk of lung cancer: a study from Japan. *British Medical Journal,* 282:183-185.

Jaakkola MS, Jaakkola JJK (1997). Assessment of exposure to environmental tobacco smoke. *European Respiratory Journal,* 10:2384-2397.

Jaakkola JJK, Jaakkola MS (2002a). Effects of environmental tobacco smoke on the respiratory health of children. *Scandinavian Journal of Work, Environment & Health*, 28:71-83.

Jaakkola MS, Jaakkola JJK (2002b). Effects of environmental tobacco smoke on the respiratory health of adults. *Scandinavian Journal of Work, Environment & Health,* 28:52-70.

Jaakkola MS, Jaakkola JJK (2006). Impact of smoke-free workplace legislation on exposures and health: possibilities for prevention. *European Respiratory Journal,* 28:397-408.

Jaakkola MS, Samet JM (1999). Occupational exposure to environmental tobacco smoke and health risk assessment. *Environmental Health Perspectives,* 107:829-835.

Jaakkola MS et al. (2003). Environmental tobacco smoke and adult-onset asthma: A population-based incident case-control study. *American Journal of Public Health,* 93:2055-2060.

Lund KE et al. (1998). Children's residential exposure to environmental tobacco smoke varies greatly between the Nordic countries. *Scandinavian Journal of Social Medicine,* 26:115-120.

Murray CJL et al. (2002). *Summary measures of population health: concepts, ethics, measurement and applications*. Geneva, World Health Organization.

Norman-Taylor W, Dickinson VA (1972). Dangers for children in smoking families. *Community Medicine,* 128:32-33.

Öberg M et al. (2010a). *Second Hand Smoke. Assessing the environmental burden of disease at national and local levels.* WHO Methods guide. Geneva, World Health Organization. (http://www.who.int/quantifying_ehimpacts/publications/SHS.pdf).

Öberg M et al. (2010b). *Global estimate of the burden of disease from second-hand smoke*. Geneva, World Health Organization. (http://www.who.int/quantifying_ehimpacts/publications/shsarticle2010/en/index.html).

Öberg M et al. (2011). Worldwide burden of disease from exposure to second-hand smoke: a retrospective analysis of data from 192 countries. *Lancet*, 377:139-146.

Pattenden S et al. (2006). Parental smoking and children's respiratory health: independent effects of prenatal and postnatal exposure. *Tobacco Control,* 15:294-301.

Pirkle JL et al. (1996). Exposure of the US population to environmental tobacco smoke. The Third National Health and Nutrition Examination Survey 1988-1991. *Journal of the American Medical Association,* 275:1233-1240.

Riboli E et al. (1990). Exposure of non-smoking women to environmental tobacco smoke: a 10-country collaborative study. *Cancer Causes Control,* 1:243-252.

Trichopoulos D et al. (1981). Lung cancer and passive smoking. *International Journal of Cancer,* 27:1-4.

Windham GC, Eaton A, Hopkins B (1999). Evidence for an association between environmental tobacco smoke exposure and birth weight: a meta-analysis and new data. *Paediatric and Perinatal Epidemiology,* 13:35-57.

WHO (2008). *The Global burden of disease: Update 2004*. Geneva, World Health Organization. (http://www.who.int/healthinfo/global_burden_disease/GBD_report_2004update_full.pdf, accessed 25 September, 2009).

Health effects of lead in housing

David E. Jacobs

1. Introduction

The literature on lead toxicology is large and the evidence of adverse health effects from lead exposure associated with housing is substantial (Fewtrell et al., 2004; WHO, 2003). In children, the neurological, cognitive and developmental problems are of principal concern, although numerous other harmful effects have also been well documented (National Academy of Sciences, 1993). In adults, cardiovascular and long-term neurological effects are of greatest concern. Other important adverse health effects occurring at higher exposures include anaemia, decreased renal function, gastrointestinal effects, reduced reproductive health and at higher exposure levels, reduced stature, hearing loss, encephalopathy, seizures, coma and death. Finally, inorganic lead has been classified as a probable human carcinogen by both the International Agency for Research on Cancer and the United States Environmental Protection Agency.

Cognitive, developmental, neurological, behavioural and cardiovascular effects may occur at relatively common so-called "low" levels of exposure (e.g. from leaded gasoline, leaded paints, leaded water pipes, ceramics). However, several housing exposures such as ingestion of leaded paints, dust and soil may result in high exposures, leading to acute poisonings with very severe outcomes. While fatalities are now rare, several cases have been associated with exposure to residential lead-based paint hazards in housing in Great Britain (Elliot et al., 1999), France (Sinnaeve et al., 1999) and the United States (Caron et al., 2000) over the past few decades. The association between housing with lead paint and childhood lead poisoning is well-established (Jacobs 1995), as is the association between the presence of lead-based paint and lead-contaminated house dust and contaminated soil (Jacobs et al., 2002).

Neurological and behavioural problems include reduced IQ (Lanphear et al., 2005), attention deficits (Braun et al., 2006), hyperactivity, reduced organizational skills, and juvenile delinquency and criminal behaviour in later life (Dietrich et al., 2001; Needleman et al., 1996; Needleman et al., 2002; Nevin, 2000). One recent international study showed that trends in violent crime over a century were highly correlated with exposure to lead in house paint and gasoline in five European countries, the United States, Canada, Australia and New Zealand (Nevin, 2007). Because each of these countries phased out the use of lead in paint, gasoline and other sources at different times, the correlated trend lines between lead and crime over a substantial time period are compelling. The association between lead exposure and crime is biologically plausible, because studies have shown that lead exposure affects executive function and impulse control (Wright et al., 2008). While the precise mechanism remains uncertain, early exposure to lead can interfere with apoptosis, synaptogenesis, lower levels of serotonin, dopamine sensitivity, and mitochondrial energy metabolism (WHO, 1995).

WHO has published global blood lead levels for all WHO regions for the year 2000 and showed that 0.9% of the global burden of disease is due mostly to the mild mental retardation and cardiovascular effects of lead exposure. This is equivalent to 12.9 million DALYs, which places lead at the 16th position in leading risk factors at the global level (Prüss-Üstün et al., 2004). WHO provided updated global blood lead levels for 2005 for the purposes of this chapter and will make the data available in the future. In this chapter, these 2005 blood lead data and the

more recent evidence of the relationship between IQ and blood lead level are used to estimate the housing-related burden of disease associated with lead exposure in Europe.

2. Summary of the method

The main steps required for estimating the burden from exposure to lead in housing include:

- Assessing population blood lead levels using the 2005 data from WHO.
- Assessing the burden of population cardiovascular disease.
- Estimating the number of children that fall below 70 IQ points, resulting in mild mental retardation.
- Estimating the burden of cardiovascular disease in adults and mild mental retardation in children attributable to lead exposure from housing.

The population attributable fraction (PAF) is the proportion of mild mental retardation in children and cardiovascular disease in the population that would be prevented if exposure to lead from housing was controlled. The PAF is calculated as follows:

$$PAF = \frac{(pxOR) - 1}{(pxOR)}$$

Where p = proportion of the population exposed, and OR is the odds ratio for the condition in those exposed.

The remainder of this chapter describes the key steps in obtaining this essential information. Because no detailed country-specific estimates can be provided, this chapter is an evidence summary.

3. Exposure-risk relationship for lead

3.1 Method for establishing the exposure-risk relationship

The exposure-risk relationship for lead is established by reviewing the literature to quantify the magnitude of the effect on loss of IQ, increase in blood pressure and the association between lead exposure and violence.

3.2 Loss of IQ points and mild mental retardation

To estimate IQ loss from lead exposure, WHO previously used a linear relationship of 2.6 IQ points lost per 10 μg/dL blood lead interval for blood lead levels between 5 and 20 μg/dL and a loss of 3.5 IQ points for blood lead levels above 20 μg/dL, based on a meta analysis from 1994 (WHO, 2003). However, a more recent international pooled analysis published in 2005 (Lanphear et al., 2005) shows that the relationship is not linear and that IQ decrements are far higher in the first 10 μg/dL increment, where most exposures occur for European children and the bulk of the world's population.

Using a log-linear model, the new pooled analysis found an IQ decrement of 2.6 points for an increase in blood lead level from 5 μg/dL to 10 μg/dL and progressively smaller decrements at

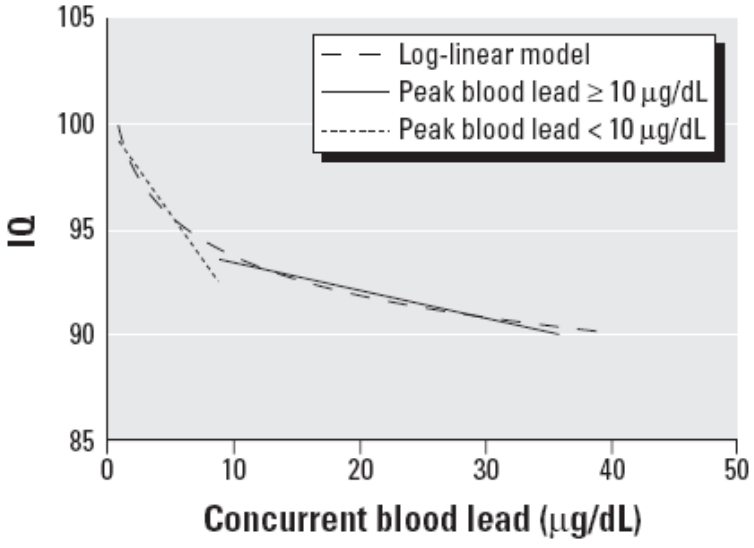
higher blood lead levels (see Figure 1). At the lower blood lead ranges, this represents an approximate increase of 300%, because the previous analysis was based on a decrease of 0.65 IQ points for an increase in blood lead levels from 5 μg/dL to 10 μg/dL (note that no effect was accounted for blood lead levels under 5 μg/dl in the earlier analysis).

While loss of IQ points does not indicate a lower health status per se, it is likely to significantly affect health in several ways. First, lower IQ is linked to lower educational achievements (Miranda et al., 2007). Lower education, in turn, is associated with higher premature mortality (Jemal et al., 2001). The association between childhood IQ and lower survival has also been studied directly (Whalley, Deary, 2001).

Second, one could estimate the impact of loss of IQ points only on those persons with an IQ that is already close to 70 IQ points, the level below which any further loss would result in the clinically defined range of mild mental retardation, i.e., up to 69 IQ points. Mild mental retardation is then a health outcome for which the disease burden can be quantified. This was done by WHO in 2003, resulting in 12.9 million DALYs (9.8 million DALYs were associated with mild mental retardation and 3.1 million DALYs were due to cardiovascular disease in that estimate) (Fewtrell et al., 2004). For Europe and the United States, that estimate can probably be considered to be an underestimate, because new evidence since that assessment now shows higher impacts on IQ levels at lower exposure levels (Lanphear et al., 2005).

Finally, the shift of IQ points of a large section of the population may have unexpected impacts on a given country more generally due to reduced productivity of those with lower incomes (in the form of lost lifetime earnings). Similarly, leadership and scholarship can be adversely affected, because lead exposure also affects those with high IQs, from which leaders and scholars are drawn.

Figure 1. Blood lead and IQ



Source: Lanphear et al., 2005

Peak blood lead means the child's maximum blood lead level between 1 and 6 years of age.

3.3 Cardiovascular effects

WHO found that the cardiovascular effects of lead exposure in adults are also significant (WHO, 2003). This estimate was based on evidence relating increased blood lead levels to increased blood pressure. A recent study revealed a positive association between stress and tibia lead on

systolic blood pressure, after adjusting for multiple covariates. Subjects reporting high stress were 2.66 times more likely to develop hypertension per standard deviation increase in tibia bone lead (Peters et al., 2007).

In the GBD 2000 assessment, the impact on cardiovascular diseases was estimated to amount to 3.1 million DALYs (Fewtrell et al., 2004). Consideration of the recent evidence could result in an alternative estimate, but a detailed analysis has not been carried out in the framework of this chapter.

3.4 Violence

Multiple studies have examined the relationship between childhood lead exposure and delinquency and crime in later life (Denno, 1990; Needleman et al., 2003; Nevin, 2000; Dietrich et al., 2001). Another study that examined crime trends and lead exposures in multiple countries over the course of a century has reported r-square values from 0.63 to as high as 0.95 (Nevin, 2007). Yet another study, which is the longest-term epidemiological study conducted to date that has examined the influence of early childhood exposure on self-reported delinquent behaviour in later life, while controlling for numerous confounding variables (such as birth weight, Home Observation for Measurement of the Environment (HOME) scores, socioeconomic status, parental IQ and others) reported adjusted total arrest rates for each 5 μg/dL increase in blood lead concentration, with a RR of 1.27 (95% CI = 1.03-1.57) for the blood lead level at age six (Wright et al., 2008). While it is feasible to use this relative risk estimate to calculate the burden of lead exposure related to violence (and the associated loss in DALYs), this chapter does not include violence in order to present a conservative burden estimate and to preserve methodological consistency with the earlier WHO estimate. Because this (and other adverse) health endpoint is not included, it is likely that this new calculation greatly underestimates the true burden of housing-related lead exposure.

4. Exposure Assessment

4.1 Method for exposure assessment

While lead contamination has been documented in many environmental media, food and thousands of children's consumer products (CDC, 2010), there is a compelling body of evidence that the major pathways of exposure in developed countries today are from residential lead-based paint, settled house dust, soil and drinking-water contaminated by old lead pipes and brass fixtures in housing (National Academy of Sciences, 1993). Airborne lead particulate levels are now quite low in Europe and the United States, due to the successful phase out of lead in gasoline and control of industrial emissions (WHO, 2009).[12] But historic deposition of lead particulate into residential soils and house dust from previous lead gasoline use and lead paint in older housing, particularly substandard low-income housing, remains a significant source (WHO, 2003). In addition, new residential lead-based paints are now being manufactured in China, India and other countries and are likely to contaminate houses there, as well as Europe and the United States as such paints are imported (Clark et al., 2006).

An international pooled analysis estimated the relationship between children's blood lead level and exposure to lead in settled house dust and soil. These two media were found to be the strongest predictors of childhood blood lead level (water lead and paint lead condition were also significant). The pooled analysis showed that, holding soil lead and other variables constant at a national average, an increase in floor dust lead from 1 μg/ft to 100 μg/ft^2 increases geometric mean blood lead levels in children from 2.8 μg/dL to 7.3 μg/dL. Holding interior floor dust lead

[12] See http://www.who.int/healthinfo/global_burden_disease/GlobalHealthRisks_report_Front.pdf for details

constant at 5 μg/ft^2, an increase in soil and exterior dust lead from 10 ppm to 1000 ppm increases blood lead levels from 3.2 to 5.3 μg/dL (Lanphear et al., 1998).

4.2 Summary of exposure levels to lead

Studies have documented exposures to lead-contaminated dust, soil, paint and water in housing in France (Nedelec et al., 1995), Brussels (Claeys et al., 2003), Portugal (Mayan et al., 2001), Poland (Zeida, 1995), Great Britain (Millstone, 1995; Duggan, 1985), Spain (Cambria et al., 1995), the United States (Jacobs et al., 2002) and other countries. By 1927, the following European nations had formally banned the use of residential lead paint: Austria, Belgium, Bulgaria, Czechoslovakia, Estonia, France, Great Britain, Greece, Latvia, Poland, Romania, Spain, and Sweden, although it is not known whether the ban was enforced in each country (International Labour Office, 1927). A paper from France indicates lead paint is prevalent in houses built before 1948 (Sinnaeve et al., 1999). This suggests that the 1927 ban may not have been fully effective in preventing the application of lead paint to housing in Europe.

Despite national differences, age and condition of housing have consistently been highly correlated with prevalence of deteriorated lead-based paint, lead contaminated dust and soil and lead in drinking-water in European studies. There are reliable data on age of housing in Europe (European Union, 2004). Because different countries banned the use of lead paint in housing at different times, older dilapidated housing is likely to be a reasonable surrogate for prevalence of lead hazards in housing. Lead in drinking-water is primarily housing-based, due to the contribution from leaded pipes and brass fixtures. In Germany, for example, 3.1% of water samples were found to exceed WHO guidelines (Zietz et al., 2001).

A major British review (Duggan, 1985) of lead in settled dust concluded that for each increment of 1000 μg/g in settled dust lead concentration, there is an average increase of about 5 μg/dL in blood lead level in young children. That review stated that European rural exterior dust lead levels were between 35 to 150 μg/g and in urban areas, the levels were more than 10 times higher. The review showed that dust wipe sample studies from the Netherlands, New Zealand, and the United States had average interior dust lead levels ranging from 5-1000 μg/ft^2.

In Brussels, Claeys et al. (2003) reported an odds ratio of 4.4 for blood lead level and pre-1940 housing; this increased to 7.2 for buildings undergoing renovation where old paint is likely to be disturbed. In Basque, Spain, blood lead levels were higher among children who lived in houses constructed prior to 1950 (Cambra et al., 1995). The geometric averages of lead in house dust, park soil, and park dust were 595, 299, and 136 μg/g, respectively.

In the United States, 25% of houses in 2000 had deteriorated lead paint, and/or dust and/or soil lead above government standards. For houses built after 1978, 1960-78, 1940-1959 and before 1940, the prevalence of these conditions is 3%, 8%, 43% and 68%, respectively. This trend is present because older housing has more surfaces with lead paint and the paint on those surfaces has higher concentrations of lead. The prevalence for households in poverty and not in poverty was 38% and 22% respectively (Jacobs et al., 2002).

In a study of 3000 housing units in the United States in which deteriorated lead paint, dust or soil were controlled, children's blood lead levels declined by 37% over a two-year period following intervention, which is similar to a number of other such studies (National Center for Healthy Housing, 2004; Wilson et al., 2006). A French lead paint abatement study also showed significant decreases in children's blood lead and dust lead levels (Nedelec et al., 1995). These and numerous other studies demonstrate that exposures to lead in housing can be controlled.

5. Total burden of disease from lead

WHO (2003) previously estimate the number of Disability Adjusted Life Years (DALYs) from mild mental retardation and cardiovascular outcomes. The new estimated blood lead/IQ slope from Lanphear et al., 2005 and the WHO updated blood lead distribution are combined to estimate the updated total IQ loss due to lead exposure in the three WHO European subregions Euro A, B and C (Table 1).

The 2002 World Health report included an estimate of over 20 million total DALYs worldwide due to injuries caused by intentional violence (excluding war and self-inflicted injuries) (WHO, 2002). For the European Region, the total DALYs from intentional violence amount to 1 916 000 in subregion Euro C, 320 000 in Euro B and 144 000 in Euro A, for a total of 2 380 000 DALYs. The relative risk for adjusted arrest rates for violent crimes for each increase of 5 µg/dL was found to be 1.48 for six year old children (Wright et al., 2008)

Table 1. DALYs associated with lead exposure in Europe and European subregions

	Euro A*	Euro B*	Euro C*	European Region
Mild Mental Retardation	20 000	98 000	110 000	228 000
Cardiovascular Outcomes	63 000	280 000	482 000	825 000
Total	**83 000**	**378 000**	**592 000**	**1 053 000**

* The list of countries for the European subregions is provided by Table 1 of the Introduction chapter

This is likely to be a conservative (low) estimate, for several reasons. First, it does not include the other health effects from lead described above. Second, the decline in IQ related to lead exposure that does not result in mild mental retardation is not included in this total. Third, the influence of lead exposure in childhood on violence in later life is not included.

6. Environmental burden of disease from housing related lead exposure

The proportion of lead exposure that is related to older housing is difficult to quantify precisely, because lead is a multimedia pollutant. Nevertheless, the evidence is clear that most exposure in the advanced world is associated with older housing, because the use of leaded gasoline has now been phased out across Europe and industrial lead emissions are well controlled, except for a few notable but confined areas near smelters or mines. The portion of lead exposure from residential yard soil, which is contaminated by earlier use of leaded gasoline and exterior lead-based paint, and the portion associated with contamination of residential drinking-water from leaded pipes, should both be included as housing-related lead exposures, because they occur in the housing environment.

The portion of lead exposure that is related to older housing can be estimated as follows. One study in France showed that 74% of older houses had high dust lead and/or paint lead content (Ginot et al., 1995). This is similar to the percentage of older houses in the United States with lead paint (68%). Another study in Brussels showed that the odds of elevated blood lead for children in dwellings constructed before 1940 were four times greater compared to children in other dwellings (Claeys et al., 2003). A study in Spain showed that geometric mean dust lead levels in housing and parks were 595 µg/g and 136 µg/g, respectively, suggesting that mean exposures in housing were 4.3 times greater than those from the outdoor environment (Cambra et al., 1995). Altogether, the results of these studies converge, suggesting that on average

elevated lead exposures are 4 times greater in older houses, including exposures from leaded paint, dust, soil or other sources, compared to newer houses.

If the proportion of older housing with elevated lead levels is 74%, and if the odds of children in these houses of having an elevated blood lead level is about 4 (as documented above), the population attributable fraction (PAF) of elevated lead due to housing is the following:

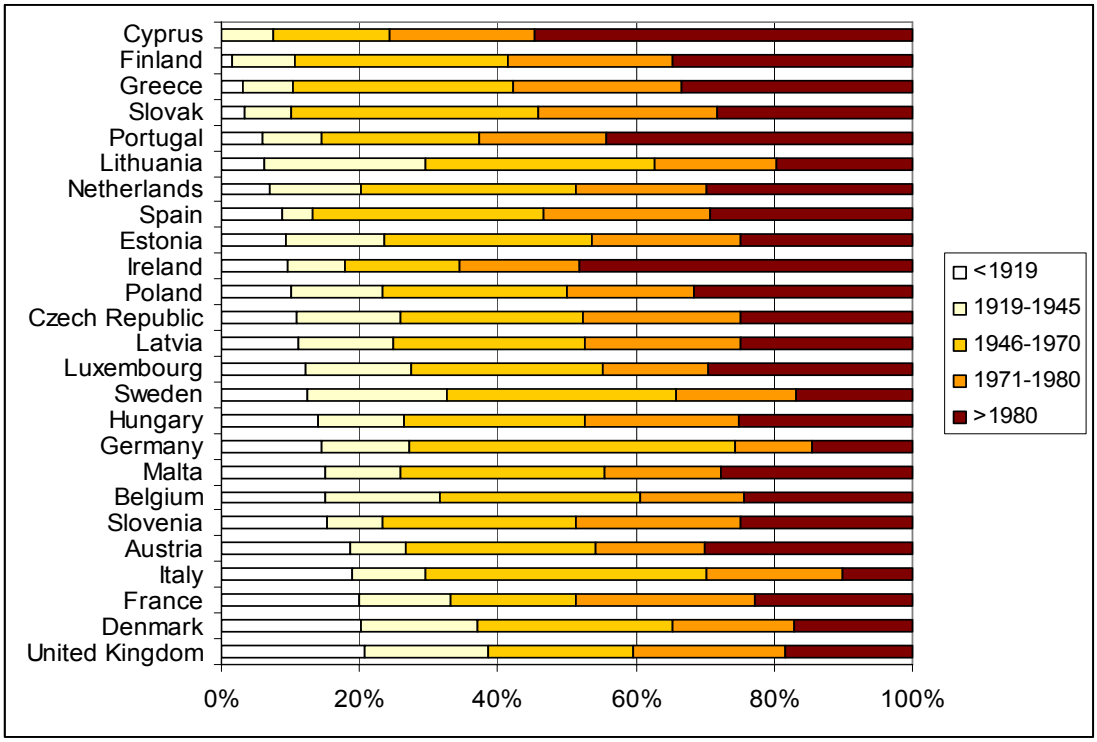
$$PAF = \frac{(pxOR)-1}{(pxOR)} = \frac{(0.74x4)-1}{(0.74x4)} = 0.66$$

Where p = proportion exposed (in this case to houses with high lead levels in dust and/or soil and/or paint), and OR is the odds ratio of elevated blood lead for children in dwellings constructed before 1940 compared to other dwellings.

In the United States, 68% of older housing built before 1940 has lead paint hazards (Jacobs et al., 2002), which is similar to the percentage from France cited previously (74%). Approximately 70% of lead poisoned children in the United States have lead-based paint hazards in their housing, which is similar to the population attributable fraction shown above (Levin et al., 2009). Data on the age of the European housing stock also suggests that as much as 40% of European houses were built before 1940, although there are differences in nations (see Figure 2). In short, the population attributable fraction shown above is consistent with other data. Therefore, the overall DALYs associated with housing-related lead exposure in Europe can be calculated as follows:

1 053 000 DALYs x 0.66 = 694 980 DALYs

Figure 2. Age distribution of European Housing Stock



Data source: Data compiled from national statistical institutes (National Board of Housing, Building and Planning, 2005)

7. Uncertainty

IQ is the most commonly used measure of cognitive function, but it is unlikely to reflect the full effect of lead exposure. Many other variables are related to IQ, including socioeconomic status, parental IQ, nutrition and others, all of which could confound the blood lead/IQ relationship. Many well-conducted studies have controlled for these confounding influences (Lanphear et al., 2005). It is noteworthy that most studies show an inverse relationship between blood lead and IQ and those that do not lacked statistical power. In other words, no study to date has demonstrated that lead improves IQ, so if there was no relationship between IQ and blood lead, one would expect studies to be randomly distributed. Some think the reported effect size of blood levels less than 10 μg/dL is improbably large. The reasons that the effect could be overestimated could include the fact that the results are based on the experience of only a few children as well as concerns that residual confounding may have resulted in some level of misclassification. Another limitation is that blood lead level is not a good measure of lifetime lead exposure and is susceptible to short exposures, because the half-life of lead in blood is approximately 30 days. Other uncertainties include the IQ measurement process, the portion of IQ loss that is associated with other non-lead related factors and the definition of mild mental retardation

8. Conclusions

Lead exposure remains a significant health problem in European and United States housing, especially in older housing where exposures to lead in paint, dust, soil and water are likely to be higher and where housing repairs and renovations that disturb lead paint are more likely to occur. Approximately 66% of the burden of lead exposure (and its influence on mild mental retardation and cardiovascular effects) is due to housing-based exposures, which includes lead-based paint and contaminated dust and soil, and is estimated to cause about 695 000 DALYs per year (Table 2). The continuing manufacture of new lead-based paint poses an emerging threat to the entire global housing stock that could increase this percentage further, unless action is taken to ban it. The environmental burden of disease related to lead exposure from housing is significant.

Table 2. Summary of EBD of lead poisoning caused by older housing

Housing exposure	Lead contaminated paint, dust soil and drinking-water		
Health outcome	Mental retardation, cardiovascular disease, behavioural problems		
Summary of EBD evidence	Elevated blood lead levels associated with lead contained in older residential buildings cause about 650 000 DALYs in the European population per year.		
	Level	**Geographic scope**	**Source of information**
(a) Exposure risk relationship	IQ decrement of 2.6 points for an increase in blood lead level from 5- 10 µg/dL and smaller decrements at higher blood lead levels.	European countries, United States	Lanphear et al., 2005; Fewtrell et al., 2004
(b) Exposure assessment	OR = 4.4 for blood lead level and pre-1940 housing. 74% of older housing has lead paint.	France, Spain, United Kingdom, United States	Claeys et al., 2003; Cambra et al., 1995; Jacobs et al., 2002; Duggin, 1985
(c) PAF	66%	WHO European Region	European housing stock age, prevalence of lead paint
(d) Total EBD	1 053 000 DALYs		See table 1
(e) EBD from lead exposure in older housing	694 980 DALYs (79.2/100 000)	WHO European Region	Blood lead levels and housing data
Main areas of uncertainty	Many other variables are related to IQ, including socioeconomic status, parental IQ, nutrition and others, all of which could confound the blood lead/IQ relationship, the IQ measurement process, the portion of IQ loss that is associated with other non-lead related factors and the definition of mild mental retardation. National surveys of lead paint prevalence available through limited studies.		
Main implications	Methods of controlling exposures to lead in housing are known to be effective and should be implemented widely to protect public health, especially the health of children who are at greatest risk. Eliminating lead-based paint, dust and soil lead hazards can be accomplished by a combination of cleaning, covering and/or removing lead painted or contaminated surfaces in housing.		

9. Policy implications

Methods of controlling exposures to lead in housing are known to be effective and should be implemented widely to protect public health, especially the health of children who are at greatest risk. Eliminating lead-based paint, dust and soil lead hazards can be accomplished by a combination of cleaning, covering and/or removing lead painted or other contaminated surfaces in housing. In a large study of such measures in several thousand housing units in the United States, blood lead levels declined by 38% two years following abatement (National Center for Healthy Housing, 2004) and dust lead levels declined by 66-90% over a six year period (Wilson et al., 2006). Methods of controlling such hazards should be integrated into existing housing finance, maintenance, rehabilitation, property turn-over and construction systems. Nations should also undertake studies to more precisely determine which housing poses risks to children. While blood lead level measurements and case management are still needed, further efforts are needed to eliminate exposures before they occur (primary prevention), rather than responding only after a rise in childhood blood lead level has occurred. Elimination of all non-essential uses of lead (American Public Health Association, 2007) will help to eliminate exposures and improve the public health the world over.

10. References

American Public Health Association (2007). *Calling for a Global Ban on Lead Use in Residential Indoor and Outdoor Paints, Children's Products, and All Nonessential Uses in Consumer Products*. (http://www.apha.org/advocacy/policy/policysearch/default.htm?id=1348, accessed 12 December 2010)

Bellinger DC, Needleman HL (2003). Intellectual impairment and blood lead levels. *New England Journal of Medicine*, 349:500-502.

Braun J et al. (2006). Exposures to environmental toxicants and attention deficit disorder in US children. *Environmental Health Perspectives*, 114:1904-1909.

Cambra K, Alonso E (1995). Blood lead levels in 2- to 3-year-old children in the Greater Bilbao Area (Basque Country, Spain). *Archives of Environmental Health*, 50:362-366.

Caron RM et al. (2000). Fatal Pediatric Lead Poisoning – New Hampshire, 2000. Morbidity and Mortality Weekly Report, 50:457-459.

CDC (2010). *Lead Recalls*. Atlanta, Centers for Disease Control and Prevention. (http://www.cdc.gov/nceh/lead/Recalls/allhazards.htm, accessed 2 November 2009).

Claeys F et al. (2003). Childhood lead poisoning in Brussels. Prevalence study and etiological factors. *Journal de Physique*, 107:307-310.

Clark CS et al. (2006). The lead content of currently available new residential paint in several Asian countries. *Environmental Research*, 102:9-12.

Denno DW (1990). *Biology and Violence*. New York, Cambridge University Press.

Dietrich K et al. (2001). Early exposure to lead and juvenile delinquency. *Neurotoxicology and Teratology*, 23:511-518.

Duggan MJ, Inskip MJ (1985). Childhood exposure to lead in surface dust and soil. *Public Health Review*, 13:1-54.

Elliot P et al. (1999). Clinical lead poisoning in England: an analysis of routine sources of data. *Occupational and Environmental Medicine*, 56:820-824.

Fewtrell L et al. (2004). Estimating the global burden of disease of mild mental retardation and cardiovascular disease from environmental lead exposure. *Environmental Research*, 94:120-133.

Ginot L et al. (1995). Screening for lead poisoning in children by measuring lead levels in housing: A study of the Paris region. *Revue d'Epidémiologie et de Santé Publique,* 43:477-484.

International Labour Office (1927): *White Lead*. Studies and Reports Series F (Industrial Hygiene), No. 11. Geneva, ILO.

Jacobs DE (1995). Lead Paint as a Major Source of Childhood Lead Poisoning: A Review of the Evidence. In: Beard ME, Iske SDA, eds. *Lead in Paint, Soil and Dust: Health Risks, Exposure Studies, Control Measures and Quality Assurance*. Philadelphia, American Society for Testing and Materials:175-187.

Jacobs DE et al. (2002). The Prevalence of Lead-Based Paint Hazards in U.S. Housing. *Environmental Health Perspectives,* 110:A599-A606.

Jemal A et al. (2001). Mortality from leading causes by education and race in the United States. *American Journal of Preventive Medicine,* 34:1-8.

Lanphear BP et al. (1998). The Contribution of Lead-Contaminated House Dust and Residential Soil to Children's Blood Lead Levels: A Pooled Analysis of 12 Epidemiological Studies. *Environmental Research*, 79:51-68.

Lanphear BP et al. (2005). Low-level environmental lead exposure and children's intellectual function: An international pooled analysis. *Environmental Health Perspectives*, 113:894-899.

Levin R et al. (2008). Lead Exposure in US Children, 2008: Implications for Prevention. *Environmental Health Perspectives*, 116:1285-1293.

Mayan ON et al. (2001). Childhood lead exposure in Oporto, Portugal. *International Journal of Occupational and Environmental Health*, 7:209-216.

Millstone E, Russel J (1995). Lead toxicity and public health policy. *Journal of the Royal Society of Health*, 115:347-350.

Miranda ML et al. (2007). The relationship between early childhood blood lead levels and performance on end-of-grade tests. *Environmental Health Perspectives,* 115:1242-1247.

National Academy of Sciences (1993). *Measuring Lead Exposure in Infants, Children, and Other Sensitive Populations*. Washington DC, National Academy Press.

National Board of Housing, Building and Planning (2005): *Housing Statistics in the European Union 2004*. Karlskrona, Boverket. (http://www.iut.nu/EU/HousingStatistics2004.pdf, accessed 19 November 2009).

National Center for Healthy Housing (2004). *Evaluation of the HUD Lead Hazard Control Grant Program. Final Report.* National Center for Healthy Housing and University of Cincinnati, Columbia, MD.

Nedelec V et al. (1995). Evaluation of decontamination interventions in 59 homes of children with lead poisoning. *Revue d'Epidémiologie et de Santé Publique,* 43:485-93.

Needleman H et al. (1996). Bone lead levels and delinquent behavior. *Journal of the American Medical Association,* 257:363-369.

Needleman HL et al. (2002). Bone lead levels in adjudicated delinquents. A case control study. *Neurotoxicology and Teratology*, 24:711-717.

Nevin R (2000). How lead exposure relates to temporal changes in IQ, violent crime and unwed pregnancy. *Environmental Research*, 83:1-22.

Nevin R (2007). Understanding International Crime Trends: The Legacy of Pre-school Lead Exposure. *Environmental Research*, 104:315-336.

Peters J et al. (2007). Stress as a potential modifier of the impact of lead levels on blood pressure. *Environmental Health Perspectives*, 115:1154-1159.

Prüss-Üstün A et al. (2004). Lead exposure. In: Ezzati M et al., eds. *Comparative quantification of health risks. Volume 2*. Geneva, WHO. (http://www.who.int/healthinfo/global_burden_disease/cra/en/, accessed 23 September 2008)

Sinnaeve O et al. (1999). Chronic lead poisoning in children today. *Archives of Pediatrics & Adolescent Medicine,* 6:762-767.

Whalley LJ, Deary IJ (2001). Longitudinal cohort study of childhood IQ and survival up to age 76. *British Medical Journal* 322:1-5.

WHO (1995). *Environmental health criteria 165: Inorganic lead*. WHO International Programme on Chemical Safety. Geneva, WHO. (http://www.inchem.org/documents/ehc/ehc/ehc165.htm, accessed 24 April 2009)

WHO (2002). *World Health Report 2002. Annex Table 3: Burden of disease in DALYs by cause, sex and mortality stratum in WHO regions, estimates for 2001.* Geneva, WHO. (http://www.who.int/whr/2002/en/whr2002_annex3.pdf, accessed 24 April 2009)

WHO (2003). *Lead: Assessing the environmental burden of disease at national and local levels.* World Health Organization Environmental Burden of Disease, Series 2. Geneva, WHO. (http://www.who.int/quantifying_ehimpacts/publications/en/leadebd2.pdf, accessed 24 April 2009)

WHO (2004). *Comparative Quantification of Health Risks web site*. Geneva, WHO. (www.who.int/publications/cra, accessed 24 April 2009)

WHO (2009). *Global Burden of Disease web site.* Geneva, WHO. (http://www.who.int/healthinfo/global_burden_disease/en/, accessed 24 April 2009)

Wilson J et al. (2006). Evaluation of HUD-funded lead hazard control treatments at 6 years post-intervention. *Environmental Research*, 102:237-248.

Wright JP et al. (2008). Association of prenatal and childhood blood lead concentrations with criminal arrests in early adulthood. PLoS Medicine, 5:732-740.

Zeida JE (1995) Blood lead levels in urban children of Katowice Voivodship, Poland. *Central European Journal of Public Health*, 5:60-64.

Zietz B et al. (2001). Lead contamination in tap water of households with children in Lower Saxony, Germany. *Science of the Total Environment,* 275:19-26.

Household carbon monoxide poisoning

Stefanos N. Kales, Tanzima Islam, Min Kim

1. Introduction

Carbon monoxide (CO) is a toxic gas that is colorless, odorless, tasteless and non-irritating, and thus without warning properties (ATSDR, 2009). CO is produced by the incomplete combustion of carbonaceous materials including vehicle and heating fuels. Without appropriate ventilation, indoor levels of CO can reach harmful or even life-threatening concentrations, sometimes within minutes. CO inhalation leads to tissue hypoxia and toxicity through several mechanisms. The best recognized is the impairment of oxygen transport. CO preferentially binds haemoglobin, which displaces oxygen and adversely affects the delivery of oxygen to the tissues.

CO intoxication is the number one cause of unintentional, non-drug poisoning in developed countries. The case fatality rate is about 3% among persons seeking/receiving hospital care for CO poisoning (Sam-Lai et al., 2003; CDC, 2005). Ambient CO concentration in outside air is not a good predictor of poisoning incidence (diMarco et al., 2005). Instead, accumulations of CO in indoor air are the most common cause of intoxication. In several developed countries, 50-64% of CO poisoning occurs in the home (Sam-Lai et al., 2003; CDC, 2005; Clifton et al., 2001; European Center for Injury Prevention, 2007).

Accordingly, CO is a highly relevant risk related to inadequate housing conditions. Unintentional CO poisoning in the home – as considered in this chapter – is related to inappropriate or faulty heating, cooking or other combustion appliances and the entry of vehicle exhaust from attached garages. Intentional CO inhalation (suicides and suicide attempts), occupational CO inhalation or CO inhalation as a consequence of smoke inhalation due to structure fires are beyond the scope of this chapter.

Individuals with greater susceptibility to CO exposure include pregnant women, infants and small children, the elderly and persons with underlying cardiopulmonary disease. Additionally, certain homes or residential areas (e.g., those with older/poorly maintained heating systems) are at significantly higher risk for both episodic CO elevations and/or chronically higher CO concentrations.

Gas heating and cooking can be significant contributors to CO concentration in homes (Bruinen et al., 2004). The climate of most developed regions is such that heating is used in most homes at least part of the year. Additionally in Europe, for example, the WHO LARES study, which included 3300 homes in 8 European cities (Angers, Bonn, Bratislava, Budapest, Ferreira, Forli, Geneva, and Vilnius), revealed that two thirds (67%) of these houses relied on gas energy for cooking (WHO LARES database). Thus, CO is an important potential household hazard throughout most developed countries.

2. Summary of the method

Data on CO cases are tracked using the International Classification of Disease (ICD-10; T58) and the data on non-drug poisoning use ICD-10 (X47 or Y17).

Death and delayed/persistent neurological effects are the principal adverse health effects used to assess the burden of disease associated with CO exposure. Detailed estimates for the population

attributable fraction (PAF), which is the proportion of disease or death that can be associated with CO are reviewed below. In this context, PAF represents the proportion of CO poisoning in the population that would be prevented if exposure to CO in the home were reduced to the level outdoors. The PAF estimate is then multiplied by the total burden of CO poisoning. This results in an estimate of the proportion of CO cases, deaths and disability adjusted life years (DALYs) that can be ascribed to CO exposure in the home.

The steps required for estimating the household disease and fatality burden from CO is as follows:

- Retrieve available health-care statistics for CO intoxications receiving hospital care;
- Assess the incidence of significant CO poisonings and/or deaths for countries with available data;
- The PAF of CO poisonings associated with the household or housing-related exposures is then calculated by multiplying the incidence of CO poisonings/deaths by 60%. The literature demonstrates that the proportion of unintentional CO poisonings due to housing conditions ranges from 50-64% (Sam-Lai et al., 2003; CDC, 2005; Clifton et al., 2001; European Center for Injury Prevention, 2007).
- Select the health effects or outcomes for study: unintentional poisoning death and delayed/persistent neurologic sequelae (DNS/PNS);
- Apply the mean case-fatality rate of 3% to the incidence of serious CO exposure;
- Apply the rate of DNS/PNS incidence (3-40%) to the incidence of serious CO exposure incidence (Raub et al., 2000).

3. Exposure assessment

This section provides a short overview of typical exposures encountered in Europe.

Table 1 demonstrates examples of typical household CO concentrations as measured in Europe. As expected, average indoor CO levels for a large majority of homes are less than the WHO guidelines of 10 mg/m3 (9 ppm) as an eight-hour, time-weighted average and 35 mg/m3 (30.6 ppm) for 60 minutes.

While average CO concentrations in the home are quite low, significant short-term CO exposures can develop quickly with changes in conditions, such as: turning on a heating system; the blockage of a vent or a chimney; some other appliance malfunction; and the use of supplemental heating appliances or use of electrical power generators indoors following a power outage. Significant exposures are episodic and predominantly occur during autumn and winter months. Certain types of housing are at much higher risk for these episodic CO elevations.

Risk factors include: older/poorly maintained heating systems; combustion-powered space heaters; and housing found in low-income areas. In a survey of gas appliances in low-income English homes, 23% had some type of problem with a gas appliance; 5% were at high risk of CO exposure and 3% were at “very” high risk (Croxford, 2006). Furthermore, 0.7% of appliances were deemed “immediately dangerous” and disconnected on the spot. Almost 20% of homes had CO concentrations that exceeded the current WHO 8-hour limit of 10 mg/m^3 (9 ppm) as a time-weighted average at least once during the monitoring period, and about 4% exceeded the previous 2000 WHO short-term, 30 minute guideline of 60 mg/m^3 (52 ppm) (WHO, 2000).

Table 1: Typical household CO concentrations.

Reference	Country	Type of housing; Surveyed exposure parameters	Mean concentration [mg/m^3]	Maximum concentration [mg/m3]	Percentage of households with CO > 10 mg/m^3
Bruinen de Bruin et al., 2004	Italy	Homes of 46 Milan office workers: ▪ No home CO source; ▪ Gas cooking in home; ▪ 1 hour maximum cooking exposures	 2.1 +/- 1.5 2.8 +/- 1.7	 21*	0% (8 hour exposure)* 30% (1 hour exposure)*
Raw et al., 2004	United Kingdom (England)	830 randomly selected homes 14 day average concentrations Gas cooking oven; winter	0.45 (bedrooms) 0.54 (kitchen)	4.5 (bedrooms) 5.1 (kitchen)	Short-term peaks not measured
Croxford, 2006	United Kingdom (England)	Indoor CO monitored in 270 homes. Appliances examined in 597 low income homes for CO, estimation made that ~6% of homes will exceed WHO 1hour guideline.	2.0 +/- 1.8	95	18% (exceeded for an 8 hour exposure at least once during monitoring period of 1 – 4 weeks) (4% > 60 mg/m^3 for a 30 minute exposure)
Willers et al., 2004	Nether-lands	72 homes (kitchens) 7 day average	0.5 +/- 1.2	6.0	Not reported
diMarco et al., 2005	Finland	Mean of > 250 000 one minute CO exposures in homes based on personal sampling from 201 residents in Helsinki area	1.2 +/- 0.6	2.3 (95%ile)	Not reported

*Based on personal sampling.

4. Exposure-risk relationship for CO and adverse health

There is a direct correlation between the concentration of CO in ambient air and the carboxyhaemoglobin saturation (COHb%) in the human body (Table 2). However, the measured COHb% does not reliably predict individual clinical presentations, especially the incidence of neurologic sequelae. Susceptible persons (see above) may experience more serious health effects, up to and including death, at lower levels of exposure than shown in the table. However, the table provides examples of probable health effects in "average" adults for each range of increasing exposure.

Based on the exposure-response relationship, serious health effects due to housing-related CO poisoning are most likely to occur in two situations of "very significant exposures":

- the acute production of several hundred to thousand mg/m^3, and
- chronic exposures of 80-230 mg/m^3 (75-200 ppm).

A functioning warning system (e.g. CO detector or alarm) would be expected to prevent the consequences of such exposure scenarios, otherwise, they are expected to result in poisonings of varying severity when occupants are present (~100% incidence).

Table 2: Health effects associated with increasing CO concentrations

CO inhaled mg/m^3 (ppm)	COHb% Saturation	Health Effects
1.2-3.4 (1-3)	0.49-0.81	None expected
8-58 (7- 50)	1.46- 8	Reduced time to angina, arrhythmia and/or ischaemia in adults with coronary heart disease.
80-115 (70-100)	11-14	Diminished performance of complex tasks, cardiac ischaemia in susceptible persons, mild headache.
115- 230 (100-200)	15-25	Severe headache, nausea, vomiting, syncope
345-575 (300-500)	33-45	Confusion; collapse on exercise
805-1150 (700-1000)	54-62	Seizures, coma, loss of consciousness, death

Adapted from Stewart (1975) and Beckett (1998). This table assumes that equilibration of haemoglobin with CO has occurred after constant exposure for 5 or more hours.

Reliable data on the population's exposure to episodically elevated household CO levels are not available. Therefore, we do not recommend using a relative risk in an exposure-based approach for estimating the PAF.

5. Total burden of disease from CO

While reliable data on the population's exposure to episodically elevated household CO levels are not available, serious cases of CO poisoning are almost always recorded in health-care statistics, such as emergency department or hospital discharge data. Here, the approach for estimating the disease burden of CO poisoning uses the rates of serious neurologic complications and the case fatality rate. The incidence rates of serious CO poisoning from various countries are conservative estimates based on health care statistics for persons seeking hospital care for CO poisoning. The best estimate of the case-fatality rate for cases receiving hospital care is 3% based on United States and French data (Sam-Lai et al., 2003; CDC, 2005). In addition, some severe CO exposures will result in DNS/PNS. This permits an estimate of longer term disease burden, in addition to deaths. DNS/PNS refers to persons who survive CO intoxication, but suffer longer term neurologic complications ranging from more subtle deficits of cognition and/or affective disorders to severe neurologic impairment. The incidence of DNS/PNS has been assessed by various studies and varies widely (3-40%) in the literature depending on how it is defined (including only more severe cases or also including cases with only subtle deficits).

6. Environmental burden of housing-related CO poisoning in Europe

The steps required for estimating household disease burden from CO following the alternative approach are summarized below, based on the work by the Apollo project (European Center for Injury Prevention, 2007):

- Retrieve available health-care statistics for CO intoxications receiving hospital care;
- Assess the incidence of significant CO poisonings and/or deaths for countries with available data;

- Adjust for the proportion of CO poisonings associated with the household or housing-related exposures (PAF) by applying a factor of 0.6, based on the findings in the literature that the proportion of unintentional CO poisonings due to housing conditions ranges from 50-64%;
- Select the health effects or outcomes for study: unintentional poisoning death and DNS/PNS;
- Apply the mean case-fatality rate of 3% to the incidence of serious CO exposure;
- Apply the rate of DNS/PNS incidence (3-40%) to the incidence of serious CO exposure incidence.

As a result of unintentional, very significant household CO exposures, 27-366 per 100 million people (0.03 – 0.4/100 000) will suffer delayed or persistent neurologic sequelae; and on average 27 ± 23 persons will die of their poisoning (0.03 ± 0.02 deaths/100 000) (Table 3). The best estimate of the PAF is that household exposures account for 50-64% of CO poisoning.

Table 3. Estimates of non-fatal and fatal cases of CO poisoning due to inadequate housing conditions in western and central Europe

Country	**Reference**	**Serious CO exposures*/year (cases)**	**Non-fatal DNS/PNS**/year (cases)**	**Mortality***** **/year (cases)**
France	Sam-Lai et al., 2003	2300†	69-920	69
Bulgaria	European Center for Injury Prevention, 2007	541	16-216	16
Denmark	European Center for Injury Prevention, 2007	2101	63-840	63
Hungary	European Center for Injury Prevention, 2007	1857†	56-743	56
Latvia	European Center for Injury Prevention 2007	78	2-31	2
Malta	European Center for Injury Prevention, 2007	0	0	0
Norway	European Center for Injury Prevention, 2007	655	20-262	20
Portugal	European Center for Injury Prevention, 2007	587	18-235	18
Slovenia	European Center for Injury Prevention, 2007	1111	33-444	33
Spain	European Center for Injury Prevention, 2007	579	17-232	17
Sweden	European Center for Injury Prevention, 2007	553	16-221	16
Netherlands	European Center for Injury Prevention, 2007	620	19-248	19
Total	**Mean (SD)**	**915 (765)**	**27 (23)-366 (306)**	**27 (23)**

* Estimates are based on standard populations of 100 million and reported proportion of household cases († France and Hungary) or using 60% to estimate household incidents.

**Assumes 3-40% incidence rate for DNS/PNS among the cases in the adjacent column.

***Assumes 3% case fatality rate.

7. Uncertainty

The principal sources of uncertainty in the assessment include the following:

- Lack of data for most countries: mortality, poison center calls, hospital discharges and other statistics that might allow a direct estimate of disease burden due to CO poisoning are often not available or cannot easily be located.
- For countries with some available information: the sensitivity of the statistic or study for capturing the actual number of serious CO poisoning cases, as well as their associated rates of mortality, DNS/PNS and the proportion attributable to inadequate housing. For example regarding the European data, Hungary was the only country in the APOLLO project with an estimate (50%) for the proportion of cases that are household-related (European Center for Injury Prevention, 2007).

Uncertainty could be reduced by mandating CO poisoning as a reportable disease to public health authorities, as household cases almost always justify some type of public health response.

8. Conclusions

While CO poisoning is relatively rare, it has a high case-fatality rate, is highly preventable and therefore, is an important concern in developed countries. The CO data show that 27-366 people per 100 million people will suffer delayed or persistent neurologic sequelae and 27 ± 23 persons will die of their poisoning. The best estimate of the PAF is that household exposures account for 50-64% of CO poisoning (Table 4). To better estimate the total disease burden caused by CO, health care facilities, fire and emergency medical services, and utility companies (e.g. gas companies) should be mandated to report significant CO exposures. Additionally, improved collection, synthesis and analysis of this exposure information at the national and international levels are also needed.

Table 4. Summary of EBD of housing-related CO poisoning

Housing exposure	Indoor exposure to CO		
Health outcome	Headache, nausea, cardiovascular ischaemia/insufficiency, seizures, coma, loss of consciousness, death		
Summary of EBD evidence	As a result of unintentional, very significant household CO exposures, 114 – 1545 persons (27-366 per 100 million population) will suffer delayed or persistent neurologic sequelae per year in Euro A*; and on average 114 ± 97 persons will die of their CO poisoning.		
	Level	**Geographic scope**	**Source of information**
(a) Exposure risk relationship	Case-fatality rate of 3% to the incidence of serious CO exposure DNS/PNS incidence (3-40%) to the incidence of serious CO exposure	Euro A*	Based on Raub et al., 2000; Sam-Lai et al., 2003; CDC, 2005
(b) Exposure assessment	Varies largely by country	Euro A*	For details, see Table 1 and WHO, 2010
(c) PAF	50-64%	Euro A*	See section 6
(d) Total EBD from CO poisoning	Not available		See section 5.

(e) EBD from indoor CO poisoning	114 – 1545 persons with delayed or persistent neurologic sequelae (0.03 – 0.4/100 000) 114 ± 97 deaths (0.03 ± 0.02/100 000)	Euro A*	Extrapolated for Euro A population based on section 6
Main areas of uncertainty	The principal sources of uncertainty relate to the lack of data in many countries, and – in case of data being available – the difficult association with health effects. Data on real exposure to CO in European homes is also rare. Available data for western Europe cannot be extrapolated to the eastern part of the region.		
Main implications	Effective policy measures and regulations need to be installed, such as laws and economic incentives regarding the use of CO detectors in residential units. Periodic testing and maintenance of combustion-powered heating systems and home appliances capable of emitting CO is necessary as well.		

* The list of countries for the European subregions is provided by Table 1 of the Introduction chapter

9. Policy implications and prevention measures

Prevention measures at the policy level

While there is still a need for research examining the effectiveness of household CO detectors as a prevention measure, effective policy measures may include laws and economic incentives favoring the proper placement and maintenance of CO detectors in residential units. Another useful policy would require periodic testing and maintenance of combustion-powered heating systems and home appliances capable of emitting CO.

When an incident is discovered or when a patient reaches the health-care system due to CO exposure, checking for other victims and corrective actions regarding the appliance or other source of CO exposure are necessary actions.

Prevention measures at the household level (CDC, 2005)

- A qualified technician should service the heating system, water heater and every other gas, oil, or coal burning appliance annually.
- Battery-operated CO detectors should be placed in the home (batteries should be checked twice a year). When a detector alarms, the home should be left immediately and the appropriate emergency number should be called.
- Upon suspicion of CO poisoning and feeling dizzy, light-headed, or nauseous, seek urgent medical attention.
- Never use a generator, charcoal grill, camp stove, or other gasoline or charcoal-burning device inside the home, basement, garage or near a window.
- Never run (idle) a car or truck inside a garage attached to the house, even if the garage door is open.
- Never burn anything in a stove or fireplace that is not vented.
- Never heat your house with a gas oven.

10. References

ATSDR (2009). *Toxicological profile for carbon monoxide*. Agency for Toxic Substances and Disease Registry, Atlanta. (http://www.atsdr.cdc.gov/toxprofiles/tp201.pdf, accessed 27 February 2011).

Beckett WS. Chemical Asphyxiants. In: Rom WN, ed. *Environmental & Occupational Medicine*, 3rd ed. Lipincott-Raven, New York:657-665.

Bruinen de Bruin Y et al. (2004). Personal carbon monoxide exposure levels: contribution of local sources to exposures and microenvironment concentrations in Milan. *Journal of Exposure Analysis and Environmental Epidemiology,* 14:312-322.

CDC (2005). Unintentional, non-fire related Carbon Monoxide exposures-United States, 2001-2003. *Morbidity and Mortality Weekly Report*, 54:36-39.

Clifton JC et al. (2001). Surveillance for carbon monoxide poisoning using a national media clipping service. *American Journal of Emergency Medicine*, 19:106-108.

Croxford B (2006). *Gas appliance check project*. London, Bartlett School of Graduate Studies. (http://www.hse.gov.uk/gas/domestic/uclpresentation.pdf).

diMarco GS et al. (2005). Personal carbon monoxide exposure in Helsinki, Finland. *Atmospheric Environment,* 39:2697-2707.

European Center for Injury Prevention. *Analysis of Hospital Discharge Data gathered under the "Burden of Injuries" APOLLO WP2 project.* Unpublished data. April 2007 (personal communication).

Raub JA et al. (2000). Carbon Monoxide Poisoning- a public health perspective. *Toxicology,* 145:1-14.

Raw GJ et al. (2004). Exposure to air pollutants in English homes. *Journal of Exposure Analysis and Environmental Epidemiology,* 14:85-94.

Sam-Lai NF, Saviuc P, Daniel V (2003). Carbon Monoxide Poisoning Monitoring Network: A Five-Year Experience of Household Poisonings in Two French Regions. *Clinical Toxicology,* 41:349-353.

Stewart RD (1975). The effects of carbon monoxide on humans. *Annual Review of Pharmacology and Toxicology,* 15:409-423.

WHO (2000). *Carbon Monoxide*. Air quality guidelines for Europe, 2nd ed. WHO Regional Publications, European Series, 91:75-79 (http://www.euro.who.int/__data/assets/pdf_file/0005/74732/E71922.pdf, accessed 27 February 2011).

WHO (2010). *Carbon Monoxide*. WHO guidelines for indoor air quality: selected pollutants, Copenhagen, WHO Regional Office for Europe: 55-101 (http://www.euro.who.int/__data/assets/pdf_file/0009/128169/e94535.pdf, accessed 27 February 2011).

WHO LARES Database. *Unpublished data analysis*. Copenhagen, WHO Regional Office for Europe.

Willers SM et al. (2004). Gas cooking, kitchen ventilation and exposure to combustion products. *Indoor Air*, 16:65-73.

Formaldehyde and respiratory symptoms in children

Nicolas L. Gilbert, Mireille Guay

1. Introduction

At room temperature, formaldehyde is a reactive gas. Formaldehyde sources that influence indoor concentrations of this chemical fall into two categories: combustion and off-gassing. Combustion sources include open fireplaces and smoke from cigarette and other tobacco products. Off-gassing sources include wood-based products assembled with formaldehyde-containing resins such as plywood and particle board, as well as some latex paints, varnishes, and floor finishes (WHO, 1989; ATSDR, 1999).

Average formaldehyde concentrations measured in Europe in the 1980s and 1990s ranged from 20 to 41 $\mu g/m^3$ in homes and between 1 and 4 $\mu g/m^3$ outdoors (WHO, 2010), indicating that indoor exposure is a much bigger concern than exposure in ambient air.

The main established health effect from formaldehyde exposure is an increased prevalence of lower respiratory symptoms in infants and young children. Consequently, this chapter focuses on this relationship. Because indoor concentrations in European houses are still poorly characterized, this chapter is restricted to an evidence summary.

2. Summary of the method

Although the evidence of the relationship between formaldehyde and acute respiratory symptoms in infants and young children is still limited, it is possible to develop an approximate estimation of the fraction of disease burden attributable to indoor exposure to formaldehyde using this process:

- Review the literature on the effect of formaldehyde on respiratory health.
- Use the best available odds ratio from case control studies to characterize the exposure-risk relationship.
- Derive an attributable risk from the odds ratio and combine it with measurements of the concentration of formaldehyde in the home to calculate the population attributable fraction (PAF).
- Apply the resulting fraction to the incidence of acute lower respiratory symptoms in the study population (in this case children in Europe).

The PAF is the proportion of acute respiratory symptoms in infants and young children. The PAF is calculated as shown in section 6 below.

3. Exposure-risk relationship

3.1 Method for establishing exposure-risk relationship

A review of literature was used to identify and evaluate studies that investigated the relationship between formaldehyde exposure and respiratory symptoms.

3.2 Evidence of exposure-risk relationship

Suitable studies are summarized in Table 1. Associations between residential or school exposure to formaldehyde and respiratory symptoms have been found in several observational epidemiologic studies:

In Arizona, a 1.23 μg/m^3 (1 ppb) increase in residential indoor formaldehyde was associated with a 1.28 L/min decrease (standard error 0.46 L/min, $p<0.05$) in peak expiratory flow among 298 children aged less than 15 years (Krzyzanowski et al., 1990)

In Australia, among 148 children aged 7 to 14 years, no association was found between formaldehyde and physician-diagnosed asthma, although in children suffering from asthma-related symptoms, formaldehyde exposure *was* associated with more frequent symptoms (Garrett et al., 1999)

In Sweden, an increased incidence of physician-diagnosed asthma was found in children attending classrooms with increased concentration of formaldehyde. The odds ratio for a 10 μg/m^3 increase in formaldehyde was 1.7 (95% CI 1.1 to 2.6) in a cohort of 1347 children followed for 4 years (Smedje, Norbäck, 2001).

In Australia, 88 children discharged from an emergency department with an asthma diagnosis were compared with 104 controls. All children were aged between 6 months and 3 years. A non-significant increase in the risk (OR=1.20) was observed in children exposed to formaldehyde concentrations ranging from 50 to 59 μg/m^3, and a significant increase of risk (OR=1.39) was observed in those exposed to 60 μg/m^3 and over, compared to those exposed to less than 10 μg/m^3 (Rumchev et al., 2002).

Similar to the Garret study cited above, another case control study in the United Kingdom showed no difference in formaldehyde exposure between children suffering from wheezing and controls, but there was a significant association between formaldehyde concentration in bedroom and the frequency of symptoms (Venn et al., 2004).

Association of formaldehyde exposure with increase allergic sensitization and with increase prevalence of respiratory symptoms are consistent with the observation that the inhalation of 100 μg/m^3 formaldehyde for 30 min increases bronchial responsiveness to the house dust mite *Dermatophagoides pteronyssus* allergen in asthmatics sensitized to this allergen (Casset et al., 2006).

The Rumchev et al. study (2002) appears to be the most suitable for assessing the burden of diseases from formaldehyde present in homes because:

- the exposure considered is formaldehyde in homes
- the outcome is a medical condition, i.e. asthma as the primary diagnosis in the Emergency Department, not a change in a physiological parameter such as lung function
- odds ratios were determined for several exposure ranges

Table 1. Studies on exposure to formaldehyde and children's respiratory symptoms

Reference	Study design, location	Study population	Exposure measure	Outcome measure	Covariates	Results
Krzyza-nowski et al., 1990	Cross-sectional study, United States	298 Children aged 6-15 yr and 613 adults aged > 15 yr.	Formaldehyde in bedrooms	Respiratory symptoms questionnaire (all participants) and peak expiratory flow in a subsample of 208 children and 526 adults	SHS, NO_2	In children, peak expiratory flow negatively correlated with bedroom formaldehyde
Garrett et al., 1999	Cross-sectional study, Australia, 1994-1995	148 children aged 7-14 yr	Formaldehyde in child's bedroom, kitchen and living room	Physician-diagnosed asthma, respiratory symptoms score	Parental asthma and allergy	No association between formaldehyde and doctor-diagnosed asthma, but significant association with respiratory symptoms score (p=0.03)
Smedje, Norbäck, 2001	Cohort study, Sweden, 1993-1997	1347 children (mean age at entry 10.3 yr)	Formaldehyde in classrooms	Physician-diagnosed asthma	Age, sex, smoking	In children that were not atopic at entry: OR for a 10 $\mu g/m^3$ increase in formaldehyde 1.7 (95% CI 1.1 to 2.6)
Rumchev et al., 2002	Case-control study, Australia, 1997-1999	Children aged between 6 months and 3 yr Cases discharged from hospital with asthma diagnosis (n=88), Controls with no asthma (n=104)	Formaldehyde in child's bedroom and living room	Physician-diagnosed asthma	Dust mite allergens, humidity, indoor temperature, atopy, family history of asthma, SES, SHS, pets, air conditioning, humidifier, gas appliances	Houses with formaldehyde ≥60 $\mu g/m^3$, compared to <10 $\mu g/m^3$: OR 1.39, 95% CI 1.09 to 1.69.
Venn et al., 2003	Case-control study, United Kingdom, 1998-1999	Children aged 9-11 yr identified from a previous cross-sectional study Cases: reported wheezing Controls: no reported wheezing	Formaldehyde in child's bedroom	Wheezing (Y/N), intensity of symptoms	Age, sex, SES	No difference between cases and controls with respect toformaldehyde exposure. Among cases, more frequent night-time symptoms in those exposed to higher formaldehyde: OR for > 32 $\mu g/m^3$ vs. 0-16 $\mu g/m^3$: 3.33, 95% CI 1.23 to 9.01 (similar but non significant trend for daytime symptoms)

4. Exposure assessment

4.1 Method for exposure assessment

In order to estimate the proportion of the population of European countries exposed to formaldehyde concentrations associated with various levels of health risk, the arithmetic or geometric means and standard deviations of each study were extracted. When geometric mean and geometric standard deviation were provided, the log-normal distribution with these parameters was used to calculate the various proportions of population. The same method was implemented using the normal distribution when only arithmetic descriptive statistics were available.

4.2 Summary of exposures

Exposure estimates could be computed for Finland, France, Hungary and Sweden (see Table 2). In Hungary, the proportion of homes where formaldehyde concentrations exceeded 60 µg/m^3 appears to be negligible; in France and in Sweden, this proportion ranged between less than 0.1% and 13%. The highest proportion (19%) was found in Finland. The estimated proportion of homes with concentrations exceeding 100 µg/m^3 was below 0.4%, except in France where they ranged from 0.9 to 2.1%.

Table 2. Exposure to formaldehyde in Europe

Percentage of population exposed to formaldehyde concentrations associated with various health effects						
				Percent of Homes by Exposure Category		
Reference	**Country, year**	**Distribution assumed, *n***	**Sampling location**	**<60 µg/m^3 (minimal health effects)**	**>60 µg/m^3 (increased risk of respiratory symptoms in children)**	**>100 µg/m^3 (increased responsiveness to inhaled allergens in asthmatics)**
Jurvelin et al., 2003	Finland, 1997	Normal, *n*=15	Not specified	80.6	19.4	0.4
Clarisse et al., 2003	France, 2001	Log-normal, *n*=61	Kitchen	94.3	5.7	0.9
			Living Room	92.0	8.0	1.4
			Bedroom	90.2	9.8	2.1
Erdei et al., 2003	Hungary, 1998	Normal, *n*=123	Bedroom	100.0	<0.1	<0.1
Gustafson et al., 2005	Sweden, 1999	Normal, *n*=24	Bedroom	99.2	0.8	<0.1
	Sweden, 2000	Normal, *n*=40	Bedroom	87.2	12.8	0.2
Sakai et al., 2004	Sweden, 1998	Log-normal, *n*=27	Bedroom	100.0	<0.1	<0.1

Most of these estimates are based on statistics assuming a normal distribution because the original papers did not present a geometric standard deviation. This is particularly true for the Finnish studies for which, given the large difference between the arithmetic and geometric mean in this study, the distribution of formaldehyde concentrations appears to be far from normal.

If we restrict our analysis to data for which geometric means and standard deviations were computed, i.e., the Clarisse et al. (2003) study in France and the Sakai et al. (2004) study in Sweden, the proportion of homes where formaldehyde concentrations exceed 60 µg/m^3 is up to 9.8%, and the proportion of homes where formaldehyde concentrations exceed 100 µg/m^3 is up to 2%. These estimates are to be taken with caution since they are based on two small studies adding up to a total of less than 100 homes. However, the use of geometric means only is a conservative approach, because many of the homes in the other studies had higher levels of formaldehyde. Thus, it is possible that exposures are higher than those presented here.

It should also be kept in mind that for diseases that are not rare, such as asthma and allergies, odds ratios are systematically greater than relative risks.

5. Total burden of disease

5.1 Method for measuring asthma and respiratory symptoms in children

The International Study of Asthma and Allergies in Childhood (ISAAC) was conducted between 1991 and 1995 in 58 countries. The standardized questionnaire included questions on the occurrence of wheeze in the past 12 months and on ever having asthma. Ten countries in western Europe participated in the study: Austria, Belgium, France, Germany, Greece, Italy, Portugal, the Republic of Ireland, Spain, and the United Kingdom; in these countries, 68 460 children aged 6-7 years and 135 559 children aged 13-14 years and participated in the study (Asher et al., 1995; ISAAC Steering Committee, 1998).

5.2 Summary of asthma and respiratory symptoms burden in Europe

Based on the ISAAC study, the prevalence of wheezing in western Europe in the early 1990s was 8.1% in children aged 6-7 years, and 16.7% in children aged 13-14 years. The percentage of children reporting ever having had asthma was 7.2% in children aged 6-7 years and 13.0% in those aged 13-14 years (ISAAC Steering Committee, 1998).

6. Environmental burden of disease

EBD estimate for Europe

The formaldehyde concentrations in the bedroom in the Clarisse et al. study are the exposure metrics most similar to those used in the epidemiologic study from which the odds ratios are derived (highest formaldehyde concentration in the room). According to the currently available evidence, up to 9.8% of children may be exposed to formaldehyde concentrations associated with a 39% increase of the risk of lower respiratory symptoms. Therefore, the population attributable fraction (PAF) of formaldehyde health effects on respiratory symptoms associated with housing can be calculated as follows using the standard formula:

$$PAF = \frac{\sum(P_i \times OR_i) - 1}{\sum(P_i \times OR_i)} = \frac{(0.098 \times 1.39 + 0.902 \times 1.00) - 1}{(0.098 \times 1.39 + 0.902 \times 1.00)} = 0.037 = 3.7\%$$

where P is the proportion of the population associated with a given odds ratio and OR is the odds ratio. In the equation above, the proportion of those exposed is multiplied by the odds ratio of those at risk and then added to the proportion not exposed and not at risk to arrive at a population attributable fraction that is expressed as a percentage.

In addition, up to 2% of asthmatics may be exposed to formaldehyde concentrations enhancing their responsiveness to inhaled allergens, thus increasing their risk of suffering from respiratory symptoms when they are simultaneously or shortly later exposed to these allergens. Thus, the estimate provided above may be a conservative underestimate.

There are many asthma triggers in the home environment, including exposure to allergens and mould. While it is difficult to determine the relative contributions of each, the Environmental Burden of Disease from housing-related formaldehyde exposures in children associated with respiratory symptoms such as wheezing can be approximated by multiplying the population attributable fraction calculated above by the range of children having reported wheezing (8.1% to 16.7%). In short, formaldehyde exposure in the home environment may account for between 0.30% and 0.62% of wheezing in children. This estimate does not include other lower respiratory symptoms and is therefore likely to be a conservative underestimate.

7. Uncertainty

7.1 Areas of uncertainty

While there is consistent evidence from several studies conducted in different countries that long-term exposure to high formaldehyde concentration can induce respiratory symptoms in children, the exposure assessment is based on a small study. On the other hand, using only those studies with log-normal distributions could lead to an underestimate.

7.2 Approaches for reducing uncertainty

The best way to reduce uncertainty regarding the contribution of formaldehyde to the prevalence of asthma symptoms is to conduct a multicentre cross-sectional study in which current respiratory symptoms, exposure to formaldehyde and covariates would be assessed by standardized methods. Other housing factors suspected to be linked to the same symptoms (e.g., moulds and nitrogen dioxide) could be assessed at the same time to arrive at a better measure of all housing factors associated with asthma and related health outcomes.

8. Conclusion

There is the need for a better characterization of indoor sources and concentrations for formaldehyde in European countries. The evidence available to date suggests that indoor exposure to formaldehyde is a significant risk factor for lower respiratory symptoms such as wheezing (Table 3).

Table 3. Summary of EBD of respiratory symptoms in children from formaldehyde exposure

Housing exposure	Formaldehyde, μg/m^3			
Health outcome	Lower respiratory symptoms in children			
Summary of EBD evidence	Formaldehyde exposure in the home environment may account for between 0.30% and 0.62% of wheezing in children.			
	Level	**Range**	**Geographic scope**	**Source of information**
(a) Exposure risk relationship	OR 1.39	95% CI 1.09 to 1.69	Developed countries	Rumchev et al., 2002 with support from other studies

(b) Exposure assessment	up to 9.8%		Euro A*	Clarisse et al., 2003 with support from other studies
(c) PAF	3.7%			Section 6
(d) Total burden of formaldehyde-related disease	Wheezing: 8.1% in children aged 6-7 yr 16.7% in children aged 13-14 yr	Wheezing: 6-7 yr: 6.2% (Spain) to 18.4% (United Kingdom) 13-14 yr: 3.7% (Greece) to 32.2% (United Kingdom)	Euro A*	ISAAC Survey, 1991-1995
(e) EBD from formaldehyde exposures in the home		0.3% to 0.6% of current wheezing in children	Euro A*	Section 6
Main areas of uncertainty	Few etiological studies to establish exposure-risk relationship and limited exposure data. Assessment is only possible for western European countries (Euro A*).			
Main implications	Because indoor exposure to formaldehyde is a significant risk factor for lower respiratory symptoms, precautions should be taken to limit this exposure.			

* The list of countries for the European subregions is provided by Table 1 of the Introduction chapter

9. Policy implications

Because indoor exposure to formaldehyde is a significant risk factor for lower respiratory symptoms, especially in asthmatics, precautions should be taken to limit this exposure. In addition, more research is needed to assess concentrations and sources of formaldehyde in European homes, and the association between formaldehyde exposure and the risk for lower respiratory symptoms (in particular wheezing) in children.

10. References

ATSDR (1999). *Toxicological Profile for Formaldehyde*. Atlanta, Agency for Toxic Substances and Disease Registry. (http://www.atsdr.cdc.gov/ToxProfiles/tp.asp?id=220&tid=39, accessed 12 May 2008).

Asher MI et al. (1995). International Study of Asthma and Allergies in Childhood (ISAAC): rationale and methods. *European Respiratory Journal,* 8:483-491.

Casset A et al. (2006). Inhaled formaldehyde exposure: effect on bronchial response to mite allergen in sensitized asthma patients. *Allergy,* 61:1344-1350.

Clarisse B et al. (2003). Indoor aldehydes: measurement of contamination levels and identification of their determinants in Paris dwellings. *Environmental Research,* 92:245-253.

Committee on the medical effects of air pollutants (1997): *Handbook on air pollution and health*. Department of health, committee on the medical effects of air pollutants. London, Her Majesty's Stationary Office.

Erdei E et al. (2003). Indoor air pollutants and immune biomarkers among Hungarian asthmatic children. *Archives of Environmental Health,* 58:337-347.

Garrett MH et al. (1999). Increased risk of allergy in children due to formaldehyde exposure in homes. *Allergy,* 54:330-337.

Gustafson P (2005). Formaldehyde levels in Sweden: personal exposure, indoor, and outdoor concentrations. *Journal of Exposure Analysis and Environmental Epidemiology,* 15:252-260.

ISAAC Steering Committee (1998). Worldwide variations in the prevalence of asthma symptoms: the International Study of Asthma and Allergy in Children (ISAAC). *European Respiratory Journal,* 12:315-335.

Jurvelin JA et al. (2003). Residential indoor, outdoor, and workplace concentrations of carbonyl compounds: relationships with personal exposure concentrations and correlation with sources. *Journal of the Air & Waste Management Association,* 53:560-573.

Krzyzanowski M, Quackenboss JJ, Lebowitz MD (1990). Chronic respiratory effects of indoor formaldehyde exposure. *Environmental Research,* 52:117-125.

Prüss-Üstün A (2003*). Introduction and methods: assessing the environmental burden of disease at national and local levels*. WHO Environmental Burden of Disease Series, No. 1. Geneva, World Health Organization.

Rumchev KB et al. (2002). Domestic exposure to formaldehyde significantly increases the risk of asthma in young children. *European Respiratory Journal*, 20:403-408.

Smedje G, Norback D (2001). Incidence of asthma diagnosis and self-reported allergy in relation to the school environment – a four-year follow-up study in schoolchildren. *International Journal of Tuberculosis and Lung Disease,* 5:1059-1066.

Venn AJ (2003). Effects of volatile organic compounds, damp, and other environmental exposures in the home on wheezing illness in children. *Thorax,* 58:955-960.

WHO (1989). *Formaldehyde. Environmental Health Criteria 89*. International Programme on Chemical Safety. Geneva, World Health Organization.

WHO (2010). *WHO Guidelines for Indoor Air Quality: selected pollutants.* Copenhagen, WHO Regional Office for Europe (http://www.euro.who.int/__data/assets/pdf_file/0009/128169/e94535.pdf, accessed 15 January 2011).

Indoor smoke from solid fuel use

Manish Desai, Eva Rehfuess, Sumi Mehta and Kirk R. Smith

1. Introduction

Burning solid fuels indoors for cooking or heating, particularly in open fires or stoves with poor ventilation, generates high concentrations of air pollutants. This indoor smoke is associated with a variety of health outcomes. Globally, more than 1.5 million deaths are caused by solid fuel use every year, mainly from pneumonia in children, as well as chronic obstructive pulmonary disease (COPD) and lung cancer in adults.

About 50% of households worldwide use solid fuels, mainly in rural and poor urban areas of low- and middle-income countries. Solid fuel use (SFU) is defined as the combustion of biomass fuels (such as charcoal, dung, wood, or crop residues), or coal for cooking and/or heating. In reality, however, nearly all the studies used to develop the risk database for household SFU relate only to cooking, although of course heating with solid fuels also produces air pollution exposures.

The disease burden attributable to SFU has been fully assessed elsewhere (Smith et al., 2004) and the method for assessment at a national level has been published in detail (Desai et al., 2004). Therefore, despite the strong evidence for major health impacts worldwide from this housing risk, the method for assessment of disease burden and the estimate of impacts for Europe are only summarized in this guide. Full details can be found in the two referenced documents (Smith et al., 2004; Desai et al., 2004).

2. Summary of the method

The main steps required for estimating disease burden from SFU include:

- Assessing the percentage of households using solid fuels for cooking (as a proxy for exposure to indoor smoke from SFU), and of these households, the fraction using improved stoves that possess chimneys versus traditional stoves (which typically do not possess chimneys).
- Estimating the adjusted proportion exposed to SFU, which is the sum of the percentage of households using solid fuels with traditional stoves plus the percentage of households using solid fuels with improved stoves multiplied by a ventilation factor of 0.25 to account for lower exposure. This procedure is expressed by the formula:

  $p = FSF ((1-FIS) + (FIS \times 0.25))$

where: p: adjusted proportion exposed

FSF: fraction of households using solid fuels for cooking

FIS: fraction of solid fuel using households using improved stoves.

A ventilation factor of 1 corresponds to solid fuels burnt in a traditional stove with little or no room ventilation. A ventilation factor of 0 would correspond to biomass or clean coal burnt in a clean-burning and well-ventilated stove.[13]

- Compiling health statistics for the relevant health outcomes (national statistics for the selected diseases; alternatively, WHO also provides disease estimates for countries[14].
- Calculating the population attributable fraction (PAF) for each outcome and population group. The PAF is estimated using the following formula:

$$PAF = \frac{(p \cdot RR) - 1}{(p \cdot RR)}$$

where: p: adjusted proportion exposed

RR: relative risk for the disease and population group

- The attributable fraction is multiplied by the total annual burden of the disease of concern (in deaths, DALYS, or other indicator) for the population of concern to estimate the burden attributable to SFU.

3. Exposure-response relationship

Smith et al. (2004) reviewed the evidence linking SFU to various health outcomes. Nearly all of the epidemiological research has utilized some form of binary exposure classification (i.e. 'uses solid fuels', or 'does not use solid fuels'), which can be made with more confidence than estimates of actual pollutant exposures. Moreover, at present exposure-response relationships based on indoor concentrations of air pollutants are not well-established. A summary of the relative risks and their confidence intervals in relation to a binary exposure classification, according to level of evidence, is provided in Table 1.

It is recommended to only quantify those health outcomes with the strongest evidence – those in the 'Strong' and 'Moderate I' categories. The outcomes in the 'Moderate II' categories may be quantified for indicative purposes only, or after additional evidence becomes available. It should be noted that research on these less-studied outcomes (e.g. tuberculosis, perinatal health outcomes) is currently under way, and updates will be made available on the WHO web site.[15]

[13] Achieving clean burning requires special stove designs with combustion control, but there are types of dirty coal that cannot be burned cleanly with any stove. Because such cookstoves are currently rare, however, 0.25 is the suggested ventilation factor for improved stoves relying mainly on chimneys. In most of Europe, for example, heating with solid fuels is generally done with vented furnaces, which would be assigned a ventilation factor of 0.25. If local knowledge is unavailable, a ventilation factor of 1 should be considered the default value.

[14] See http://www.who.int/healthinfo/bodestimates/en/index.html, under "Death and DALY estimates for 2002 by cause for WHO Member States"

[15] See http://www.who.int/indoorair/en/

Table 1. Relative risks for strong and moderate health outcomes

Evidence	Health outcome[a]	Group (sex, age in years)	Relative risk	Confidence interval
Strong	ALRI	Children <5	2.3	1.9–2.7
	COPD	Women ≥30	3.2	2.3–4.8
	Lung cancer (from exposure to coal smoke)	Women ≥30	1.9	1.1–3.5
Moderate-I	COPD	Men ≥30	1.8	1.0–3.2
	Lung cancer (from exposure to coal smoke)	Men ≥30	1.5	1.0–2.5
Moderate-II	Lung cancer (from exposure to biomass smoke)	Women ≥30	1.5	1.0–2.1
	Asthma	Children 5-14	1.6	1.0–2.5
	Asthma	All ≥15	1.2	1.0–1.5
	Cataracts	All ≥15	1.3	1.0–1.7
	Tuberculosis	All ≥15	1.5	1.0–2.4

Data source: Desai et al., 2004

[a] Abbreviations: ALRI = acute lower respiratory infection; CI = confidence interval; COPD = chronic obstructive pulmonary disease. Strong evidence: Many studies of SFU in developing countries, support by evidence from studies of active and passive smoking, urban air pollution and biochemical or laboratory studies. Moderate evidence: At least three studies of SFU in developing countries, supported by evidence from studies on active smoking and on animals. Moderate I: strong evidence for specific age/sex groups. Moderate II: limited evidence.

4. Exposure assessment

Exposure to smoke from SFU is approximated by household use of solid fuels for cooking (and, to a lesser extent, for heating). The proportion of households using solid fuels is generally assessed by national censuses, national energy statistics, and international household surveys (such as USAID/MEASURE Demographic and Health Surveys, WHO's World Health Survey, the World Bank's Living Standards Measurement Study and UNICEF's Multiple Indicator Cluster Survey).

The proportion of households using solid fuels is also an indicator to monitor progress towards achievement of the Millennium Development Goals (Mehta et al., 2006; Rehfuess et al., 2006). The most up-to-date estimates for countries are available through the World Health Statistics[16].

5. Uncertainty

The uncertainty of the estimate of disease burden attributable to solid fuels mainly depends on the estimate of exposure and the lack of knowledge about the exposure-response relationship. The estimate of exposure critically depends on the type of stove, kitchen location and room ventilation; the ventilation factor is an imperfect approximation of these characteristics. Although it is generally not possible to perform a formal uncertainty estimate, a recommended approach is to explore the uncertainty of estimated disease burdens by creating alternative scenarios with different input data (i.e. confidence intervals around relative risks and, if feasible, around exposure estimates, and by applying other reasonable ventilation factors).

[16] See http://www.who.int/whosis/whostat/

6. Estimates of health impacts due to solid fuel use in Europe

The disease burden caused by solid fuels was estimated globally and for WHO's 14 epidemiological subregions for the year 2000 (Smith et al., 2004). These estimates were updated (WHO, 2006) and expanded to a country-by-country assessment for the year 2002 (WHO, 2007). The results for the three European subregions are shown in Tables 2 and 3. Of the 14 280 deaths due to SFU in Europe, 53% occur in children, and another 36% in adult women, as these are the population groups that spend the most time in households.

Table 2. Burden of disease attributable to indoor air pollution from SFU, 2002

Country*	% SFU	Data source type	SFU-attributable ALRI deaths in children <5 years	SFU-attributable COPD deaths in adults ≥30 years	SFU-attributable lung cancer deaths in adults ≥30 years	Total SFU-attributable deaths	Total SFU-attributable DALYs	% BoD
Albania	50	m	40	<10	-	<100	1500	0.3%
Andorra	<5	a	-	-	-	–	-	-
Armenia	26	s	40	80	-	100	2400	0.5%
Austria	<5	a	-	-	-	–	-	-
Azerbaijan	49	m	1550	270	-	1800	59 400	3.8%
Belarus	19	m	<10	150	-	200	2100	0.1%
Belgium	<5	a	-	-	-	–	-	-
Bosnia and Herzegovina	50	m	<10	<10	<10	<100	300	0.1
Bulgaria	17	m	<10	20	-	<100	500	0.0%
Croatia	12	s	-	-	-	–	200	0.0%
Cyprus	<5	a	-	-	-	–	-	-
Czech Republic	<5	s	-	-	-	–	<100	0.0%
Denmark	<5	a	-	-	-	–	-	-
Estonia	16	s	-	-	-	–	<100	0.0%
Finland	<5	a	-	-	-	–	-	-
France	<5	a	-	-	-	–	-	-
Georgia	43	s	70	30	-	100	2900	0.3%
Germany	<5	a	-	-	-	–	-	-
Greece	<5	a	-	-	-	–	-	-
Hungary	<5	a	-	-	-	–	-	-
Iceland	<5	a	-	-	-	–	-	-
Ireland	<5	a	-	-	-	–	-	-
Israel	<5	a	-	-	-	–	-	-
Italy	<5	a	-	-	-	–	-	-
Kazakhstan	<5	s	20	30	<10	<100	1500	0.0%
Kyrgyzstan	76	m	750	820	-	1600	38 200	3.3%
Latvia	10	s	-	-	-	–	<100	0.0%
Lithuania	<5	a	-	-	-	–	-	-
Luxembourg	<5	a	-	-	-	–	-	-
Malta	<5	a	-	-	-	–	-	-
Monaco	<5	a	-	-	-	–	-	-
Netherlands	<5	a	-	-	-	–	-	-
Norway	<5	a	-	-	-	–	-	-
Poland	<5	a	-	-	-	–	-	-
Portugal	<5	a	-	-	-	–	-	-
Republic of Moldova	63	m	30	130	-	200	3000	0.3%

Country*	% SFU	Data source type	SFU-attributable ALRI deaths in children <5 years	SFU-attributable COPD deaths in adults ≥30 years	SFU-attributable lung cancer deaths in adults ≥30 years	Total SFU-attributable deaths	Total SFU-attributable DALYs	% BoD
Romania	23	s	90	170	-	300	4600	0.1%
Russian Federation	9	s	30	320	10	400	5900	0.0%
San Marino	<5	a	-	-	-	–	-	-
Serbia and Montenegro*	no data	NA	-	-	-	–	-	-
Slovakia	<5	s	-	-	-	–	<100	0.0%
Slovenia	8	s	-	-	-	–	<100	0.0%
Spain	<5	s	-	-	-	–	-	-
Sweden	<5	s	-	-	-	–	-	-
Switzerland	<5	a	-	-	-	–	-	-
Tajikistan	75	s	1150	410	-	1600	48 700	3.5%
The former Yugoslav Republic of Macedonia	30	m	-	-	-	–	200	0.0%
Turkey	11	s	820	1720	-	2500	62 100	0.5%
Turkmenistan	<5	s	-	-	-	–	300	0.0%
Ukraine	6	s	<10	230	<10	200	3500	0.0%
United Kingdom	<5	a	-	-	-	–	-	-
Uzbekistan	72	m	3860	1390	-	5300	157 600	3.7%
Subregion Euro A*	0		0	0	0	0	0	0.0%
Subregion Euro B*	24		8400	4940	<10	13 300	378 600	0.9%
Subregion Euro C*	9		<100	860	<100	980	16 000	0.03%
European Region	**9**		**8490**	**5800**	**<100**	**14 280**	**394 600**	**0.12%**

SFU: Solid fuel use
%SFU: Proportion of population using solid fuels
ALRI: Acute lower respiratory infections
COPD: Chronic obstructive pulmonary disease
%BoD: Percent of the country's total burden of disease in DALYs
m: SFU modeled, according to percentage of rural population and Gross National Income (GNI)
a: SFU assumed as lower than 5% for countries with Gross National Income (GNI) > 10 500US$/year
s: SFU assessed in surveys
NA: not applicable

* The list of countries for the European subregions is provided by Table 1 of the Introduction chapter. Countries are defined as they were in 2002 at the time that these data were collated.

Table 3. DALYs due to SFU for the year 2002 in European subregions

Disease	Euro A*	Euro B*	Euro C*	European Region
ALRI (children < 5 years)	0	290 400	3200	293 600
COPD (adults >30 years)	0	88 200	12 500	100 700
Lung cancer (adults >30 years)	0	<10	200	200
Total	**0**	**378 600**	**15 900**	**394 600**

* The list of countries for the European subregions is provided by Table 1 of the Introduction chapter

These estimates allow countries with a high disease burden attributable to SFU to be identified. Countries where SFU currently claims more than 1% of the total disease burden include Azerbaijan, Kyrgyzstan, Tajikistan and Uzbekistan. Expanding the methodology to subnational

levels would provide a basis for drawing attention to geographical or population groups within a country for which interventions should be prioritized.

7. Conclusions

In western Europe, cleaner fuels, such as gas and electricity, represent the norm for cooking and heating. Where solid fuels are used, they are either efficiently burning processed biomass, such as briquettes, or are combusted in clean-burning and well-ventilated modern stoves. In sharp contrast, solid fuels still pose significant health threats in some central and eastern European countries, with 24% and 9% SFU for cooking in subregions Euro B and Euro C, respectively. The total respiratory health impacts associated with these cooking practices are estimated to cause about 14 280 deaths per year in the European Region (Table 4). In addition, several countries of the former Soviet Union, the Balkans and central Asia may require special considerations as they meet their cooking needs almost exclusively through gas and other cleaner fuels but may heavily rely on solid fuels for heating. SFU therefore emerges as one of the main risks to health in the context of housing in several European countries. Estimating the disease burden provides a basis for priority action for interventions to reduce exposure to indoor smoke from solid fuels.

Table 4. Summary of EBD of solid fuel use

Housing exposure	Household use of solid fuels for cooking			
Health outcome	ALRI deaths and DALYs among children <5 years of age COPD deaths and DALYs among adults ≥30 years of age Lung cancer deaths and DALYs among adults ≥30 years of age			
Summary of EBD assessment	24% and 9% of the populations in Euro B and Euro C respectively rely on solid fuels for cooking. In Euro B, approximately 8400 ALRI deaths among children and more than 4940 COPD among adults are attributable to SFU. While Euro A is not affected, approximately 100 ALRI and 860 COPD deaths are attributable to this risk factor in Euro C. *			
	Level	**Range**	**Geographic scope**	**Source of information**
(a) Exposure risk relationship	ALRI (children): RR 2.3 COPD (women): RR 3.2 COPD (men): RR 1.8 Lung cancer (women): RR 1.9 Lung cancer (men): RR 1.5	1.9–2.7 2.3–4.8 1.0–3.2 1.1–3.5 1.0–2.5	Developing countries, United States	RCT, cohort, case-control and cross-sectional studies
(b) Exposure assessment	8%	<5-75%	WHO European Region	Nationally representative household surveys and modelling
(c) PAF	ALRI (children) 0.09 COPD (women) 0.15 COPD (men) 0.06	0.07-0.12 0.09-0.23 0.00-0.15	WHO European Region	Derived from (a) and (b)
(d) Total burden of disease	Deaths= 14 280 DALYs= 394 600		WHO European Region	See tables 2 and 3
(e) EBD from exposure	8490 ALRI deaths in children <5 (16.7/100 000) 293 600 ALRI DALYs in children <5 (577/100 000)		WHO European Region	See tables 2 and 3

	5800 COPD deaths in adults ≥30 (1.1/100 000) 100 700 COPD DALYs in adults ≥30 (19.3/100 000)
Main areas of uncertainty	Exposure-risk relationships are primarily derived from studies conducted in the developing world. Heating with solid fuels, the primary source of solid fuel use in Europe, is not taken into account. There are stark differences in exposure and related health outcomes between different European subregions.
Main implications	Reducing exposure to indoor smoke from solid fuel use through a switch to cleaner fuels or an investment in cleaner-burning stoves could significantly contribute to reducing deaths and DALYs due to ALRI and COPD in Euro B and, to a lesser extent, in Euro C.

* The list of countries for the European subregions is provided by Table 1 of the Introduction chapter
# These values are calculated for an average solid fuel use of 8% with a ventilation factor of 0.25. Existing variations between countries and subregions would need to be considered for a more accurate estimate.

8. Policy implications

Effective solutions to reduce the disease burden caused by solid fuels used for cooking exist. Efforts centre on the following measures (Bruce et al., 2006; Smith, 1987; Smith, 1989; Barnes et al., 1993; Ezzati, Kammen, 2001; WHO, 2002; WHO, 2006):

- use of "cleaner" liquid or gaseous fuels (e.g. kerosene, liquefied petroleum gas, or biogas and so-called modern biofuels).
- improvements to cooking stoves (e.g. cleaner and more efficient combustion, or good ventilation by flues with chimneys or hoods);

Improved ventilation and behavioural changes (e.g. encouraging mothers to keep their children away from the fire) are unlikely to bring about large reductions in exposure to indoor air pollution by themselves although they play an important role in accompanying interventions.

Beyond their ability to reduce respiratory diseases among children and adults, such interventions can result in financial and time savings, limit deforestation and reduce greenhouse gas emissions, in particular carbon dioxide and methane (Hutton et al., 2006). It should be noted, however, that traditional cooking practices are often at the heart of local culture and changing such a complex social picture is not straightforward. Clearly, the extent to which a specific intervention can be applied successfully and in a sustainable way varies across different populations, depending on local circumstances of income, housing, availability of and access to different fuel types and climate (Bruce et al., 2006).

9. References

Barnes DF et al. (1993). The design and diffusion of improved cookstoves. *World Bank Research Observer*, 8:119–141.

Bruce N et al. (2006). Indoor air pollution. In: Jamison DT et al., eds. *Disease Control Priorities in Developing Countries. 2nd edition.* New York, Oxford University Press.

Desai MA, Mehta S, Smith KR (2004). Indoor smoke from solid fuels – Assessing the burden of disease at national and local levels. Environmental Disease Burden Series No.4. Geneva, World Health Organization. (http://www.who.int/quantifying_ehimpacts/publications/en/Indoorsmoke.pdf, accessed 12 December 2009)

Ezzati M, Kammen DM (2001). Quantifying the effects of exposure to indoor air pollution from biomass combustion on acute respiratory infections in developing countries. *Environmental Health Perspectives*, 109:481–488.

Hutton G et al. (2006). Evaluation of the costs and benefits of household energy and health interventions at global and regional levels. Geneva, World Health Organization. (http://www.who.int/indoorair/publications/household_energy_health_intervention.pdf, accessed 12 December 2009).

Mehta S et al. (2006). Modeling household fuel use toward reporting of the Millennium Development Goal indicator. *Energy for Sustainable Development*, 10:36-45.

Rehfuess EA, Mehta S, Prüss-Üstün A (2006). Assessing household solid fuel use – multiple implications for the millennium development goals. *Environmental Health Perspectives*, 114:373–378.

Smith KR (1987). *Biofuels, air pollution, and health: a global review*. New York, Plenum.

Smith KR, Mehta S, Mäusezahl-Feuz M (2004). Indoor air pollution from household use of solid fuels. In: Ezzati M, Lopez AD, Rodgers A, Murray CJL, eds. *Comparative quantification of health risks*. Geneva, World Health Organization. (http://www.who.int/healthinfo/global_burden_disease/cra/en/, accessed 12 December 2009).

WHO (2002). *Addressing the links between indoor air pollution, household energy, and human health.* Meeting report of the WHO-USAID global consultation on the health impact of indoor air pollution and household energy in developing countries. Geneva, World Health Organization. (http://www.who.int/mediacentre/events/HSD_Plaq_10.pdf, accessed 12 December 2009).

WHO (2006). *Fuel for life: household energy and health*. Geneva, World Health Organization. (http://www.who.int/indoorair/publications/fuelforlife.pdf, accessed 12 December 2009).

WHO (2007) *Indoor air pollution: national burden of disease estimates*. Geneva, World Health Organization. (http://www.who.int/indoorair/publications/indoor_air_national_burden_estimate_revised.pdf, accessed 12 December 2009).

Housing quality and mental health

Gary W. Evans

1. Introduction

There are several potential reasons why poor housing quality might impact mental health. Housing symbolizes self identity and thus inadequate housing may lead to stigmatization and feelings of inadequacy. Poor housing is stressful in several respects: more worries about hazards and safety (particularly if children or frail elderly are involved), hassles with maintenance, and financial worries related not only to housing itself but also things like utility bills. Some types of housing (e.g., high rise buildings) may foster social isolation. Two key aspects of parenting, responsiveness to children's needs and monitoring, can be disrupted when parents must contend with chronic housing difficulties. Many of the features of poor quality housing are beyond the control of the occupants and thus may lower self- efficacy and feelings of mastery over the environment. Lastly for many people, their home is a refuge, a place to recover from the stress and strain of daily life and work. But for those with inadequate housing, the home may mean more difficulties, not a place of refuge.

2. Summary of the method

Numerous investigators have examined the mental health correlates of housing (Evans et al., 2003; Freeman, 2008). While a few authors have restricted their definition of mental health to psychiatric illness, most studies have operationalized mental illness as more mild forms of psychological distress such as symptoms of anxiety, depression, and in the case of children, behaviour conduct disorders.

Unfortunately, many housing and mental health studies suffer methodological problems, principally self-selection bias, limiting confidence in the evidence. Furthermore, nearly all studies employ continuous outcome variables making it difficult to quantitatively estimate health impacts at the population level. The present review focuses on the subset of more scientifically rigorous studies addressing three topics: multiple family versus single family housing, building height, and housing quality. There are two types of research designs that allow stronger causal inferences to be drawn. The best but rare option for a more scientifically rigorous study design is when residents are randomly assigned to various housing conditions rather than choosing where they live. The second and more common option is a longitudinal study design where the same individuals are tracked over time as their housing conditions changes. Research on homelessness is not included in this review.

3. Exposure-risk relationship for inadequate housing and mental health

Multifamily housing represents several dwellings and households sharing a common building, often with common features, such as staircases or elevators, basement and, if available, outside spaces. Incidence rates for physician visits for psychoneurotic disorders were 36.1 and 17.9 per 1000 among wives of British serviceman randomly assigned to multifamily units versus

detached single family residences (Fanning, 1967). This difference was especially large for women with young children.

High rise housing. Four studies with random assignment of families to higher floors in housing complexes have been conducted. Residence on higher floors was associated with more physician visits for psychological symptoms (63.0 vs. 127.3 per thousand for ground vs. third floor) among wives of British and Canadian serviceman living in Germany (Fanning, 1967). Another found that low-income, public housing residents living in 3 storey vs. 14 storey buildings differed on self-reported measures (scaled from 1-6) of perceived crowding (*mean* = 2.43 vs. 4.77), control over their living environment (*M* = 4.97 vs. 3.00), and level of social activity in their building (*mean* = 3.48 vs. 2.19) (McCarthy, Saegert, 1976). The third study uncovered differences in social support (1-7 scales) in low-rise (*M* =6.4) compared to high-rise dormitories (*mean* =4.4) and in social involvement with co-residents (*mean* = 6.6 vs. 4.2) (Wilcox, Holahan, 1976). Low-income, primary school aged boys but not girls who were residents of high-rise public housing in New York City had higher levels of psychological symptoms (*mean* =20.17) compared to boys living in low-rise, public housing (*mean* = 13.99) (Saegert, 1982). In that study, low and hi-rise buildings were in the same neighbourhoods.

Housing quality. Wilner et al. (1962) studied African American female residents of public housing, half of whom moved to improved quality housing. Adults who moved to housing of better structural quality had better mental health: 56% vs. 52% enhanced mood, 53% vs. 46% higher self efficacy, 66% vs. 54% less aggressive, 48% vs. 39% more optimistic and 59% vs. 49% high life satisfaction. Groups were equivalent on all of these mental health outcomes pre-relocation. Children of families who moved to better housing also did better in school. In a national study of 10 000 children from birth to age 7 in the United Kingdom, the presence of basic housing amenities (hot water, own bath, indoor bath) was associated with about a 30% difference in teacher ratings of the child's adjustment to school. Significant differences were also found for reading and mathematics performance (Davie et al., 1972). Among married women with at least one child in Canada, structural deficiencies were correlated (*r* =.08) with use of tranquilizers and with psychiatric impairment (*r* =.15) (Duvall, Booth, 1978). Residents of council housing in the United Kingdom with psychological problems who were relocated to better housing manifested less depression and anxiety (*mean* = 2.5 and 1.2, respectively) immediately after the move compared to those who did not move (*m* = 7.6 and 6.5, respectively) (Elton,Packer, 1986). Defining mental health improvement as a 50% reduction in symptoms of anxiety and depression combined, 82% of council housing residents who received better housing achieved mental health improvement, compared to 29% of those who remained in their previous housing. Moreover these improvements in mental health persisted one year later. Anxiety and depression improved among those whose United Kingdom council homes were remodeled, compared to residents whose housing quality remained constant (Halpern, 1995). For example, 89% and 32% had clinically significant levels of anxiety and depression, respectively, before remodeling compared to 29% and 4% after housing improvements. A particularly striking aspect of Halpern's data is evidence also showing a dose-response effect with data after partial remodeling showing significant but more modest improvements in mental health. Another study showed the opposite effect (Thomas et al., 2005). Yet another demonstrated a significant influence of housing on mental health, with R^2 =.13 for both cross-sectional and prospective, longitudinal designs among a sample of American women (Evans et al., 2000). A cross-sectional study of 10 000 United Kingdom adults documented 1.4 increased odds for common mental disorders among those in housing with structural problems (Welch, Lewis, 1997). Finally, a study of Canadian children aged 9-12 found that housing quality was significantly correlated (r =.39) to mental health (Gifford, LaCombe, 2006).

Due to the design of the studies referred to above, socioeconomic status (SES) is unlikely to be a significant confounding factor. Mental health sequelae of housing conditions in these studies are

independent of residents' SES. These effects have been shown by intervention studies measuring health impacts before and after housing changes in the same individuals. Alternatively a few studies have been able to take advantage of natural experiments with random assignment of residents to different housing conditions (see also next chapter on housing improvements and their health effects).

Given the information on exposure-response relationships, the exposure variables currently known for which we can estimate mental health impacts from housing include:

- Proportion of households living in multifamily residential buildings
- Proportion of households living high above the ground (e.g., on the 4th floor or higher)
- Proportion of households living in inadequate or low-quality housing (e.g., lacking the following amenities: flush-toilet, hot running water, bath or shower, private cooking facility, or being in bad physical condition that affects comfort and safety in the home).

Still, the current estimates on the exposure-risk relationship are considered not robust enough to be applied for a quantitative assessment as studies have relied primarily on continuous outcome measures of health thus precluding a reliable estimate of a dichotomous outcome required for EBD assessment.

4. Exposure Assessment

The following section provides an overview of exposure data in Europe and the United States.

Multifamily housing. In the European Union countries reporting housing units separately for Total Structures and Multifamily Structures, the percentages range from 9% multifamily (Ireland) to 75% multifamily (Italy). The average across the EU countries is 56% multifamily housing (National Board of Housing, 2004), defined as buildings with three or more residential units or dwellings. In America, approximately 22% of the total 118 000 000 housing units consist of three or more units (Department of Commerce, 2001).

High rise housing. For 15 of the EU countries providing data on this issue, an average of 15% per country have residences of 4 stories or higher with a range of 2.4% (United Kingdom) to 39% (Poland) (National Board of Housing, 2004). In America of the 118 000 000 housing units, 26% are 3 or more stories high and 7% are four stories or higher (Department of Commerce, 2001).

Housing quality. In 27 EU countries, 7% of the population are exposed to severe housing deprivation (defined as household with overcrowding and at least one of the following: leaking roof, no bath/shower, no indoor toilet and dwelling considered too dark (Eurostat, 2010). In America, 5% of 118 million housing units have open cracks or holes in the interior, 3% have broken plaster or peeling paint, and 12% have roofs that leak. Two percent of all residences were characterized as having serious physical problems (defined as one or more of (but not limited to) the following conditions: lack of indoor plumbing, uncomfortably cold for at least 24 hrs, major electrical problems, serious water damage and others) and another 4% as moderate physical problems (e.g., no serious problems but one or more of following: unvented heater as primary source, lack of private cooking facilities and others) (Department of Commerce, 2001). The full definition of housing units with severe and moderate deficiencies is provided elsewhere (Eggers et al., 2007).

5. EBD estimate for Europe

Not appropriate since no robust estimate could be made.

6. Uncertainty

Few of the studies have large samples and many of the housing experiments with random assignment of residents rely upon specialized samples, such as wives of military personnel (Fanning 1967), low-income public housing residents (McCarthy, Saegert, 1976; Saegert, 1982) and college undergraduates in student housing (Wilcox, Holahan, 1976), respectively.

A much larger number of studies with weaker research designs converge with the conclusion that multi family housing and living on the upper floors of high rise housing is endemic to mental health (Evans et al., 2003, Freeman, 2008). The data on housing quality and mental health are robust across countries and include several longitudinal studies with mental health improving in concert with improved housing quality. EBD estimates are not possible since the research to date has used continuous indices of mental health rather than dichotomous indicators of risk as required by EBD methodology.

7. Conclusions

People living in multiple family housing or on the upper floors of high rise buildings have greater mental health problems than comparison groups. These effects are probably larger for women with young children. Effects may exist for children as well, but the size of the effect cannot be estimated at this time. The negative impacts of poor quality housing on mental health are stronger and based on solid evidence.

Given that nearly all of the data on housing and mental health rely on well validated but continuous indices of mental health, quantification of health impacts at population level using the environmental burden of disease methodology is difficult given the current evidence.

More studies with random assignment of residents to varying housing conditions or longitudinal studies tracking individuals over time as they change housing are necessary. In both cases, larger and more representative samples from different countries are needed. More work is also needed to understand the underlying psychological processes that give rise to the housing quality to mental health link.

8. Policy implications

The primary policy implication of work on housing and mental health is that housing policy needs to expand the definition of health outcomes to include mental health. This also means that measures of housing quality need to consider factors that might be relevant to mental health issues. Most current indicators of housing quality were developed with physical health (principally respiratory diseases) in mind. It is unlikely that housing itself will precipitate serious mental disorder. However, there are two likely ways in which housing can contribute to mental health. One, it can directly affect chronic stress which is known to affect nonclinical symptoms of anxiety, depression, and hostility and frustration. More mild indices of mental health are likely sensitive to housing unlike more serious mental health outcomes. Two, poor quality housing may be an additional risk factor that often covaries with poverty and thus is associated with other physical (e.g., pollution, toxins) and social (e.g., family instability,

violence) risk factors. Considerable work in clinical psychology and psychiatry reveals that exposure to multiple risk factors dramatically escalates the probability of psychological distress.

9. References

Davie R, Butler N, Goldstein H (1972). *From birth to seven*. London: Longman.

Department of Commerce (2001). *American Housing Survey* U.S. Census. Washington, Department of Commerce.

Duvall D, Booth A (1978). The housing environment and women's health. *Journal of Health and Social Behavior,* 19*:*410-417.

Eggers FJ, Thackery A (2007). *32 Years of Housing Data*. Prepared for the US Department of Housing and Urban Development, Office of Policy Development and Research, Contract No. C-CHI-00839.

Elton PJ, Packer JM (1986). A prospective randomized trial of the value of rehousing on ground of mental illness. *Journal of Chronic Diseases,* 39*:*221-227.

Eurostat (2010). Combating poverty and social exclusion. A statistical portrait of the European Union 2010. Luxembourg, Publications Office of the European Union. (http://epp.eurostat.ec.europa.eu/cache/ITY_OFFPUB/KS-EP-09-001/EN/KS-EP-09-001-EN.PDF, accessed 03 September 2010)

Evans GW, Wells NM, Moch A (2003). Housing and mental health: A review of the evidence and a methodological and conceptual critique. *Journal of Social Issues,* 59:475-500.

Evans GW et al. (2000). Housing quality and mental health. *Journal of Consulting and Clinical Psychology* 68:526-530.

Fanning DM (1967). Families in flats. *British Medical Journal,* 4:382-386.

Freeman H (2008). Housing and mental health. In: Freeman H, Stansfeld S, eds. *The impact of the environment on psychiatric disorder*. London, Routledge:210-241.

Gifford R, LaCombe C (2006). Children's socioemotional health and housing quality. *Journal of Housing and Built Environment,* 21:177-189.

Halpern D (1995). *Mental health and the built environment*. London, Taylor & Francis.

McCarthy D, Saegert S (1976). Residential density, social overload, and social withdrawal. *Human Ecology,* 6:253-272.

National Board of Housing, Building and Planning (2005): *Housing Statistics in the European Union 2004*. Karlskrona, Boverket. (http://www.iut.nu/EU/HousingStatistics2004.pdf, accessed 19 November 2009)

Saegert S (1982). Environment and children's mental health: Residential density and low income children. In: Baum A, Singer JE, eds. *Handbook of psychology and health, vol. II*. Hillsdale, Erlbaum:247-271.

Thomas R et al. (2005). Housing improvement and self-reported mental distress among council estate residents. *Social Science and Medicine,* 60:2773-2783.

Welch S, Lewis G (1997). Material standard of living, social class, and the prevalence of the common mental disorders in Great Britain. *Journal of Epidemiology and Community Health,* 52:8-14.

Wilcox BL Holahan CJ (1976). Social ecology of the megadorm in university student housing. *Journal of Educational Psychology*, 68:453-458.

Wilner DM et al. (1962). *The housing environment and family life*. Baltimore, Johns Hopkins Press.

Housing improvements and their health effects

Hilary Thomson

1. Introduction

The link between poor housing and poor health is well established. Many, possibly hundreds, of cross-sectional studies have reported consistent and statistically significant associations between poor housing conditions and poor health. Those living in poor housing are most likely to be socioeconomically deprived and have long-standing illness. In addition, vulnerable groups such as the sick, the elderly, and the unemployed, are among those most likely to live in poor housing and also tend to spend large amounts of time in their homes exposed to potentially hazardous environments (British Medical Association, 2003). Investment to improve housing conditions is a means of improving the living conditions of low income groups at high risk of poor health and is therefore a potential means through which public policy might improve health and also reduce health and social inequalities.

This chapter presents a summary of the best available research evidence on the health impacts of housing improvements. The evidence summaries presented have been prepared following an assessment of the quality of the evidence and risk of bias to prioritize best available evidence. There are two main components to this chapter. Section 2 draws on evidence syntheses carried out by the author (Thomson et al., 2001; Thomson et al., 2002; Thomson et al., 2009) and presents a review of housing improvements that substantially alter the fabric of the house with the aim of improving indoor living conditions. This includes warmth and energy efficiency improvements and refurbishment or retrofitting. Sections 3 and 4 draw on the best available evidence identified in other literature reviews, where available Sections 3 and 4 draw on Cochrane Collaboration reviews. Sections 3 and 4 summarize a variety of diverse and less substantial housing improvements such as equipment, furniture and behavioural measures to reduce accidents, fires, exposure to allergens etc. A summary list of the housing interventions included in the research reviewed in this chapter is provided in Box 1.

2. Health evidence of large-scale housing rehabilitation projects

This section presents a synthesis of data on the health impacts of two key types of housing improvement. Section 2.1 reviews evidence on the health impacts of warmth and energy efficiency improvements, and Section 2.2 reviews evidence on the health impacts of rehousing/refurbishment (retrofitting) in the context of wider neighbourhood regeneration or renewal. The presented data draws on the best available evidence identified in a systematic review of housing improvement published in 2009 (Thomson et al., 2009). This review draws on both quantitative and qualitative data. For full details of the scope and methods of the review please refer to the full paper (Thomson et al., 2009). While this chapter refers to a number of studies published after the review date of 2008 subsequent studies were not extensively searched for. Thus the conclusions reported in the review and in this chapter relate largely to studies published by 2008.

There is extreme diversity between the studies included in this synthesis. The studies varied widely with respect to study design, outcomes assessed and the nature of the intervention. For this reason the reported health impacts have not been statistically pooled in a meta-analysis;

Box 1: Housing interventions reviewed in this chapter

Warmth & Energy Efficiency improvements

Insulation- various measures (cavity wall, loft insulation, hot water tank)

Installation/repair/upgrading of heating system

Installation of double glazing

Some interventions included advice on benefit uptake as well as completion general domestic repair needs, provision of energy efficient appliances, security measures, safety equipment e.g. smoke alarms

Housing-led neighbourhood renewal

Rehousing or partial/total retrofitting of houses accompanied by wider physical neighbourhood improvement and often also accompanied by socio-economic initiatives across the target area e.g. employment initiatives, health promotion, promotion of benefit uptake. In relation to the changes to housing conditions the improvements often vary widely where existing houses are retrofitted and may include installation of new kitchen and bathroom equipment or redecoration. In most programmes of rehousing and retrofitting the programme would include installation/repair/upgrading of heating systems and energy efficiency measures such as insulation double glazing.

Interventions to reduce exposure to specific indoor hazards

House dust mite - various physical (e.g. vacuuming, mattress covers, air filters etc) and chemical measures (e.g. acaricide)

Unintentional injury, fires & falls - Safety equipment, smoke alarms (including free give-aways), education, promotion campaigns, balancing exercises

Lead reduction - removal and public awareness

Noise reduction

rather the data have been synthesised narratively. Data on the characteristics and reported quantitative outcomes of the studies included in this review have been tabulated to provide a visual summary for the reader (Table 1). Where possible standardised effect estimates have been calculated; however, poor reporting of suitable data mean that this was not possible for all studies (Table 2). The synthesis draws on qualitative and quantitative data from 19 studies of warmth and energy efficiency improvements, (Allen, 2005a; Allen, 2005b; Barton et al., 2007; Braubach et al., 2008; Caldwell et al., 2001; Eick et al., 2004; Green, Gilbertson, 1999; Health Action Calderdale Kirklees and Wakefield, 2005; Heyman et al., 2011; Hopton, Hunt, 1996; Howden-Chapman et al., 2008; Howden-Chapman et al., 2007; Iversen et al., 1986; Platt et al., 2007; Shortt, Rugkasa, 2007; Lloyd et al., 2008; Somerville et al., 2000; Warm Front Study Group, 2006; Winder, Armstrong, 2003) and ten United Kingdom studies of housing-led neighbourhood renewal (Ambrose, 2000; Barnes, 2003; Blackman, Harvey, 2001; Critchley et al., 2004; Ellaway et al., 2000; Evans, Laysell, 2000; Halpern, 1995; Kearns et al., 2008; Thomas et al., 2005; Thomson et al., 2007). A greater weight and emphasis is attached to the studies which had a lower risk of bias as indicated by “Study Quality” “Grade A” or “Grade B” (Table 2). The timescale for assessment of health impacts varied widely, from 3 to 24 months after the intervention.

Table 1: Summary of included studies and reported quantitative health impacts

Reference	Study design	Study quality	Housing condition	Final sample Int/Cont	Time since intervion	General health	Respiratory effects	Mental effects	Illness/ symptoms
Intervention: Warmth and Energy Efficiency improvements (post 1985)									
Heyman et al., 2011	RCT	A	▲	~96/82	2 years	<> 2			
Howden-Chapman et al., 2008 ***	RCT	A	▲	175/174	4-5 months	▲	▲ 11		◄► 4
Barton et al., 2007 **	RCT	A	◄►	14/13	<2 years		▲ a 7		◄► a 2
Howden-Chapman et al., 2007 **	RCT	A	▲	1689/1623	<1 year	▲ 3	▲ 5	▲ 3	
Braubach et al., 2008	PC	A	Λ	~210/165	5-8 months	Λ	Λ	▲ 4	
Platt et al., 2007	PC	A	▲	1281/1084	1-2 years	▲ 2	◄► 2		▲ 2
Lloyd et al., 2008	PC	B		9/27	1-2.5 years				▲ a
Shortt et al., 2007	PC	B	▲	46/54	1-3.5 years		◄► b 3	▲ b	▲ b 3
Somerville et al., 2000 ***	P	B	▲	72	3 months		▲ 7		
Hopton et al., 1996 ***	PC	B	▲	55/77	5-11 months			◄► b 2	◄► b 11
Warm Front Study Group, 2006	RC	C	Λ	1561/619	~3-5 mths	◄►	◄►	▲ ₸	◄►
Allen, 2005 a	P	C	Λ	16	<1 year			▲	
Allen, 2005 b	P	C	Λ	24	<3 years	▼		▲ 3	
Health Action Kirklees, 2005	R	C	Λ	102	2-8 months				Λ
Eick et al., 2004 ***	RCT	C	◄►	41	4-12 months		▲ b 2		
Winder et al., 2003 *	P	C	Λ	72	14 months	<>			
Caldwell et al., 2001 **	PC	C	▲ a	302/110	6-12 months		▲ b		◄► 4
Green et al., 1999	RC	C	Λ	111/94	~2-4 years	◄► 3			
Iversen et al., 1986	PC	C	Λ	106/535	3-6 months		▲		▲ 3
Intervention: Rehousing/retrofitting +/- neighbourhood renewal (post 1995)									
Kearns et al., 2008 **	PC	A	▲	262/284	24 months	▲	▼	▲ 4	◄► 3
Thomson et al., 2007	PC	A	▲	50/50	12 months	▲ 2		◄► a	
Critchley et al., 2004 ₸	PC	A	Λ	246	1-12 months	<> a		<> a	
Thomas et al., 2005 ₸	PC	B		585/759	22 months			◄► b	
Barnes et al., 2003	PC	B	◄►	45/45	18 months	▲ 4		▲	▲
Evans et al., 2002	PC	B	<>	17/17	6-18 months	Λ b		<> b	V b

Reference	Study design	Study quality	Housing condition	Final sample Int/Cont	Time since intervion	General health	Respiratory effects	Mental effects	Illness/ symptoms
Halpern, 1995	P	B		27	10 months			▲2	
Blackman et al., 2001 **	P	C	<>	166	5 years	▼	▼2	▲	
Ambrose, 1999	P	C	▲	227	4 years		◄►2	▲	◄►2

Source: Thomson et al., 2009

* narrative only, no data reported; ** data for children also available; *** children only; **** area level data not relating to study population alone; [T] subgroup analysis (presented in favour of main analysis where there were high levels of contamination within control group, or where data only available for a subgroup)

Study design: RCT: Randomized Controlled Trial; PC: Prospective controlled study; P: Prospective uncontrolled study; RC: Retrospective controlled study; R: Retrospective uncontrolled study

Effect direction: upward arrow= improved outcome, downward arrow= deterioration in outcome, sideways arrow= mixed effects/conflicting findings

Sample size: Final sample size (individuals) in intervention group Large arrow >300; medium arrow 50-300; small arrow <50

Statistical significance: Black arrow p<0.05; grey arrow p>0.05; empty arrow= no statistics/data reported

Statistical tests: Controlled studies- Difference between control and intervention group at follow-up (unless stated); [a] Difference in change between control and intervention group; [b] Change within intervention group only; Uncontrolled studies: Change since baseline

Number of outcomes within each category synthesis is 1 unless indicated in subscript beside effect direction, showing the number of similar outcomes reported in this study.

Synthesis of multiple outcomes within same outcome category

Where multiple outcomes all report an effect in the same direction and with the same level of statistical significance, this effect direction is reported and overall level of statistical significance indicated

Where direction of effect varies across multiple outcomes:

- Where 70% of outcomes reported a similar effect direction and level of statistical significance, the summary arrow measure reflects that effect direction and the overall statistical significance
- Where <70% of outcomes report a consistent direction of effect, the summary arrow measure indicates no clear effect/conflicting findings ◄► (size to reflect sample size)

Where statistical significance varies:

- If direction of effect is similar and >60% of outcomes are statistically significant, the summary arrow measure indicates the overall effect as statistically significant.
- If one conflicting outcome was judged to be distinct from other outcomes or of less value/validity, it was not included, or reported separately.

Table 2: Summary of standardised measure of health effect (Odds Ratios) following housing improvement: Odds Ratio for Intervention group compared to control group at study endpoint

Outcome Category	Reference/Year	Study grade	Specific outcome	OR for intervention group (95% CI)
Intervention: Warmth and Energy Efficiency improvements (post 1985)				
General health	Howden-Chapman et al., 2008[Ψ]	A	Poor/fair self-reported health	0.480 (0.310 to 0.740)**
	Howden-Chapman et al., 2007	A	Poor/fair self-reported health	0.589 (0.467 to 0.743)***
Respiratory	Howden-Chapman et al., 2008 [Ψ]	A	Sleep disturbed by wheeze	0.550 (0.350 to 0.850)**
			Speech disturbed by wheezing	0.690 (0.400 to 1.180)
			Dry cough at night	0.520 (0.320 to 0.830)*
			Wheeze during exercise	0.670 (0.420 to 1.060)
	Howden-Chapman et al., 2007	A	Morning phlegm	0.640 (0.523 to 0.784)***
			Wheezing in past 3 months	0.570 (0.467 to 0.696)***
			Cold/flu	0.545 (0.430 to 0.691)***
			Sleep disturbed by wheeze	0.570 (0400 to 0.812)***
			Speech disturbed by wheezing	0.514 (0.310 to 0.852)*
	Barton et al., 2007	A	Asthma	~0.946 (0.598 to 1.496)
			Bronchitis	~1.007 (0.477 to 2.127)
			Other respiratory symptoms	~1.010 (0.560 to 1.820)
	Platt et al., 2007	A	First diagnosis nasal allergy	1.520 (1.050 to 2.200)*
	Shortt et al., 2007	B [◊]	Asthma [Ŧ]	~0.568 (0.099 to 3.254)
			Chest infection/bronchitis [Ŧ]	~1.875 (0.495 to 7.102)
			Pneumonia/hypothermia [Ŧ]	~3.593 (0.143 to 90.361)
Mental health	Braubach et al., 2008	A	Depression	1.404 (0.329 to 5.987)
	Howden-Chapman et al., 2007	A	Low happiness (SF-36)	0.560 (0.409 to 0.767)***
		A	Low vitality (SF-36)	0.510 (0.408 to 0.637)***
	Shortt et al., 2007	B [◊]	Stress/Mental illness	~0.261 (0.053 to 1.299)
Illness / symptom	Howden-Chapman et al., 2008[Ψ]	A	Diarrhoea	0.720 (0.450 to 1.160)
			Ear infection	1.160 (0.680 to 1.990)
			Vomiting	0.880 (0.550 to 1.400)
	Barton et al., 2007	A	Arthritis	~1.058 (0.533 to 2.100)
			Rheumatism	~1.908 (0.829 to 4.395)
	Platt et al., 2007	A	First diagnosis hypertension	0.770 (0.610 to 0.972)*
			First diagnosis heart disease	0.690 (0.520 to 0.916)*
	Shortt et al., 2007	B [◊]	'Other' illnesses [Ŧ]	~0.568 (0.099 to 3.254)
			Arthritis [Ŧ]	~1.619 (0.343 to 7.641)
			Angina [Ŧ]	~0.200 (0.041 to 0.966)*

Outcome Category	Reference/Year	Study grade	Specific outcome	OR for intervention group (95% CI)
Intervention: Rehousing/Retrofitting +/- neighbourhood renewal (post 1995)				
General health	Kearns et al., 2008	A◊	Self-reported poor health	0.769 (0.500 to 1.176)
			Long standing illness	0.680 (0.440 to 1.050)
			Health not improved since 1 year ago	0.787 (0.541 to 1.163)
	Thomson et al., 2007	A	Fair/poor health	1.757 (0.777 to 3.973)
			Lower SF-36 Physical Component Score	0.960 (0.437 to 2.110)
	Barnes et al., 2003	B◊	Fair/poor health	~0.273 (0.110 to 0.682)*
			Health somewhat/much worse than 1 year ago	~0.356 (0.135 to 0.942)
			Health interferes with daily activities	~0.516 (0.617 to 3.730)
			Physical/emotional problems with daily life (in past 4 weeks)	~0.338 (0.138 to 0.829)
Mental health	Thomson et al., 2007	A	Lower SF-36 Mental Component Score	0.733 (0.333 to 1.613)
	Barnes et al., 2003	B◊	Anxiety/Depression self reported	~0.361 (0.152 to 0.856)*
Respiratory	Kearns et al., 2008	A◊	Wheezing in past year	1.040 (0.690 to 1.560)
	Kearns et al., 2008 Ψ	A◊	Asthma	1.039 (0.650 to 1.661)
			Breathlessness	1.185 (0.459 to 3.063)
			Persistent cough	1.093 (0.663 to 1.800)
			Bronchitis	0.311 (0.032 to 3.010)
			Sinus/catarrh	0.890 (0.480 to 1.650)
Illness / symptom	Kearns et al., 2008	A◊	Smoker	1.470 (0.849 to 2.546)
			Heavy drinker	0.610 (0.300 to 1.240)
			Less than 5 portions fruit/vegetable per day	0.794 (0.519 to 1.215)
	Kearns et al., 2008 Ψ	A◊	Chronic illness	1.039 (0.549 to 1.966)
			Headaches	0.991 (0.604 to 1.626)
			Indigestion	0.941 (0.058 to 15.145)
			Sleeping problems	1.128 (0.618 to 2.059)
			Eczema	1.148 (0.683 to 1.931)
			Hay fever	0.990 (0.513 to 1.913)
	Barnes et al., 2003	B◊	Pain and discomfort	~0.400 (0.170 to 0.940)
			Mobility	~0.533 (0.215 to 1.322)

Source: Thomson et al., 2009

* p<0.05, ** p<0.005, *** p<0.000 Ψ children only ∓ proportion of households as opposed to individuals

◊ Inadequate control for confounding Grade C/key confounder emerged in analysis

~ estimated as no indication of missing data for specific outcomes

2.1 Warmth and energy efficiency improvements (post 1985)

We found 19 studies which had assessed health impacts following warmth and/or energy efficiency improvements (Health Action Calderdale Kirklees and Wakefield, 2005; Allen, 2005b; Allen, 2005a; Barton et al., 2007; Caldwell et al., 2001; Eick et al., 2004; Green, Gilbertson, 1999; Heyman et al. submitted 2008; Hopton and Hunt, 1996; Howden-Chapman et al., 2007; Howden-Chapman et al., 2008; Iversen et al., 1986; Platt et al., 2007; Shortt, Rugkasa, 2007; Somerville et al., 2000; Warm Front Study Group, 2006; Winder, Armstrong, 2003; Lloyd et al., 2008; Braubach et al., 2008).
Six of these studies reported both quantitative and qualitative data (Heyman et al., 2011 [(Harrington et al., 2005; Heyman et al., 2005)]; Barton et al., 2007 [(Basham et al., 2004)]; Shortt, Rugkasa, 2007 [(Rugkasa et al., 2004)]; Warm Front Study Group, 2006 [(Gilbertson et al., 2006)]; Allen, 2005a [(Allen, 2005b)]; Caldwell et al., 2001).[17]

Intervention, context and population
The type of interventions varied but included at least one of the following; insulation (roof and/or cavity wall), installation/upgrade of central heating system, replacement of an unflued with an improved flued (vented) combustion heat source. Some programmes included additional energy efficiency measures, e.g. light bulbs, domestic repairs and welfare advice. In many cases the nature and extent of housing improvement was tailored according to individual need, leading to considerable variation in the intervention delivered within a single study. For example, within the same programme the energy efficiency measures varied, ranging from minor heating repairs to installation of central heating and insulation measures.

All the studies reviewed below were set in developed countries and implemented since 1985. Most of the interventions were set in deprived areas, and some of the interventions were targeted at vulnerable groups such as children (Howden-Chapman et al., 2008; Somerville et al., 2000; Hopton, Hunt, 1996, Eick et al., 2004), the elderly (Platt et al., 2007; Allen, 2005b; Winder, Armstrong, 2003), or people with cardiac or respiratory conditions (Howden-Chapman et al., 2008; Howden-Chapman et al., 2007; Allen, 2005b; Allen, 2005a; Health Action Calderdale Kirklees and Wakefield, 2005; Eick et al., 2004).

General health impacts (9 studies)
After installation of warmth and energy efficiency measures, general health outcomes were better in the intervention group compared to the control group in four well conducted studies (Howden-Chapman et al., 2008; Howden-Chapman et al., 2007; Platt et al., 2007; Braubach et al., 2008); these differences were statistically significant. In two randomized controlled trials (RCT) from New Zealand (Howden-Chapman et al., 2008; Howden-Chapman et al., 2007), general health was improved (poor/fair self-reported health: OR 0.480, 95% CI 0.310 to 0.740 (Howden-Chapman et al., 2008); OR 0.589, 95% CI 0.467 to 0.743 (Howden-Chapman et al., 2007)). And in one United Kingdom study two SF-36 scores (100 point scale) were improved (general health: 2.570, 95% CI 0.870 to 7.592; physical functioning: 2.510, 95% CI 0.620 to 10.161 (Platt et al., 2007), but this is unlikely to be of clinical significance. Changes in general health outcomes in the less rigorous studies were unclear (Warm Front Study Group, 2006; Allen, 2005a; Winder, Armstrong, 2003; Caldwell et al., 2001).

Respiratory health impacts (11 studies)
When compared to the control group, there was improved respiratory health in the intervention group in the two New Zealand studies (Howden-Chapman et al., 2008; Howden-Chapman et al., 2007). Improvement was reported for all the respiratory measures, mainly asthma symptoms, assessed for both adults and children. These differences were statistically significant for most

[17] Where the qualitative and quantitative data are reported separately, the square bracketed references indicate the source of the qualitative data associated with the immediately preceding reference.

measures, for example 'sleep disturbed by wheeze' in children (OR 0.550 (95% CI 0.350 to 0.850) (Howden-Chapman et al., 2008); OR 0.57 (95% CI 0.400 to 0.812) (Howden-Chapman et al., 2007)). Amongst the remaining European studies a mix of positive, unclear or conflicting respiratory impacts were reported regardless of study quality (Barton et al., 2007; Platt et al., 2007; Shortt, Rugkasa, 2007; Somerville et al., 2000; Warm Front Study Group, 2006; Eick et al., 2004; Caldwell et al., 2001; Iversen et al., 1986; Braubach et al., 2008).

Mental health impacts (7 studies)
All (Howden-Chapman et al., 2007; Shortt, Rugkasa, 2007; Warm Front Study Group, 2006; Allen, 2005a; Allen, 2005bv Braubach et al., 2008) but one (Hopton, Hunt, 1996) of the studies assessing mental health reported a positive impact; one of these studies was a well conducted RCT (SF-36 low happiness: OR 0.560, 95% (CI 0.409 to 0.767)) (Howden-Chapman et al., 2007).

Other illness/symptom impacts (10 studies)
The range of outcomes reported within this category was diverse and there was no consistent effect reported for similar outcomes between studies. Within studies the overall impact was unclear due to conflicting findings across different outcomes.

Socioeconomic impacts (7 studies)
Improved warmth and energy efficiency measures can lead to reduced fuel bills and less time off school or work for illness episodes. (Howden-Chapman et al., 2007; Somerville et al., 2000; Eick et al., 2004; Caldwell et al., 2001; Lloyd et al., 2008) Changes in fuel bills are also highly influenced by changes in market fuel prices. There is also some indication from qualitative analysis of residents' reports that improving warmth and energy efficiency leads to increased use of the home for studying and leisure, inviting friends into the home, increased privacy and improved relationships between household members (Basham et al., 2004; Caldwell et al., 2001; Gilbertson et al., 2006).

Summary: Health impacts of warmth and energy efficiency improvements
Improvements in provision of affordable warmth can lead to health improvement in the short term, in particular respiratory and mental health. The greatest potential for health improvements is for those with existing respiratory illness who are living in houses that are difficult and costly to heat.

Health improvements were more consistently reported in the New Zealand studies than in the United Kingdom studies. This may reflect differences in housing conditions at baseline, with greater potential to improve warmth in New Zealand housing. Despite higher summer temperatures in New Zealand compared with the United Kingdom, New Zealand still experiences cold winters and levels of excess winter mortality are similar to those in the United Kingdom. (Davie et al., 2007) Measures such as insulation and central heating to protect residents from the cold are rare in New Zealand housing and many houses are constructed from poorly insulated weatherboard (Howden-Chapman et al., 2007). There is no indication from the better quality studies that warmth improvements have adverse health impacts.

Improved warmth in the home may also impact positively on socioeconomic health determinants. Following these types of improvements, residents reported less time off work/school, and increased social and educational opportunities; these impacts may be health promoting in the long term.

2.2 Rehousing/retrofitting +/-neighbourhood renewal (post 1995)

Ten studies of rehousing or retrofitting were identified (Kearns et al., 2008; Thomson et al., 2007; Critchley et al., 2004; Thomas et al., 2005; Barnes, 2003; Evans, Layzell, 2000; Halpern, 1995; Blackman, Harvey, 2001; Ambrose, 2000; Ellaway et al., 2000). One study reported a mix

of quantitative and qualitative data (Kearns et al., 2008) and one study reported only qualitative data (Ellaway et al., 2000).

Intervention, context and population

These studies evaluated programmes of housing-led neighbourhood renewal in the United Kingdom; relocation to a new neighbourhood was not part of this intervention. While it is likely that warmth improvement measures were part of the intervention in each study, only four studies specifically reported that the intervention included upgrading or installation of heating and energy efficiency measures (Evans, Layzell, 2000; Blackman, Harvey, 2001; Critchley et al., 2004; Thomas et al., 2005). Programmes of housing and neighbourhood improvement are delivered to whole neighbourhoods, and it is likely that the extent of improvement in housing conditions varied considerably between individual households within the same study sample. Nine studies assessed changes in housing conditions; six studies reported improved conditions, while three studies reported no change (Barnes, 2003; Evans, Layzell, 2000; Blackman, Harvey, 2001).

Each of these studies was set in socioeconomically deprived United Kingdom neighbourhoods and included adults. Only one study reported impacts for children as well as adults (Blackman, Harvey, 2001).

General health impacts (6 studies)

Impacts on general health outcomes were unclear. The better quality studies (Grade A and B) either reported small improvements which were not statistically significant (Thomson et al., 2007; Kearns et al., 2008; Barnes, 2003) or were not accompanied by supporting data or statistics (Critchley et al., 2004; Evans, Layzell, 2000). One study (Grade C) reported a statistically significant increase in self-reported poor health (+12.3%) among adults but not children (Blackman, Harvey, 2001).

Respiratory health impacts (3 studies)

There was little clear evidence of improvements in respiratory health; some of the respiratory outcomes were better in the control group following the housing improvement.

Mental health impacts (9 studies)

The nine quantitative studies assessed changes in mental health. In the three better quality studies (Kearns et al., 2008; Thomson et al., 2007; Critchley et al., 2004) there was no clear impact on SF-36 measures of mental health, while in the less rigorous studies statistically significant improvements were reported across a range of measures (Thomas et al., 2005; Barnes, 2003; Evans, Layzell, 2000; Halpern, 1995; Blackman, Harvey, 2001; Ambrose, 2000).

Other illness/symptoms impacts (3 studies)

The range of outcomes assessed was diverse; a mix of positive and negative impacts was reported and there was no clear overall indication of benefit or harm (Kearns et al., 2008; Barnes, 2003; Evans, Layzell, 2000).

Socioeconomic impacts (2 studies)

In two studies residents reported that they were more able to afford basic essentials suggesting reduced financial strain (Critchley et al., 2004; Kearns et al., 2008). No other studies investigated the possibility of changes in socioeconomic outcomes associated with housing improvement.

Summary: Health impacts of housing-led neighbourhood renewal

Despite programmes of housing-led renewal delivering major improvements to housing and the outdoor housing environment, it would appear that there is little evidence of associated improvements in health. There is some suggestion from poorer quality studies that mental health may improve. Very little is known about possible impacts on respiratory health or other specific

symptoms. It is important to note that there is little evidence that rehousing leads to deterioration in physical or mental health.

3. Health evidence of focused rehabilitation projects

This section provides an overview of evidence on the health impacts of interventions to reduce exposure to a range of specific domestic hazards.

3.1 Interventions to reduce House Dust Mite allergens

The major allergen in house dust comes from the house dust mite and this is the allergen to which asthmatics are most often sensitive. A systematic review (Götzsche, Johansen, 2008) of the effectiveness of house dust mite control measures in the management of asthma has been carried out. Measures used included vacuuming and acaricidal chemical measures. The authors concluded that current chemical and physical measures to reduce exposure to house dust mite allergens seem to be ineffective in the management of asthma. This finding may be largely explained by the ineffectiveness of the interventions assessed to actually reduce house dust mite and associated faecal pellets and also because asthma sufferers are often sensitive to other allergens as well as house dust mite.

A further recent review (Krieger et al., 2010) examining measures to control asthma-related indoor biologic agents cite four United States studies of multifaceted tailored interventions to reduce exposure to allergens (Carter et al., 2001; Eggleston et al., 2005; Krieger et al., 2005; Morgan et al., 2004). Multifaceted interventions typically involve provision of allergen reduction and avoidance measures as well as related education and support in the form of home visits. There was little evidence that these multifaceted interventions led to significant improvements in the key asthma measures and or significant reductions in exposure to house dust mite or other key domestic allergens.

Interventions which could significantly reduce exposure to house dust mite may have the potential to reduce asthma symptoms, but as yet these interventions which can effectively reduce levels of house dust mite have not been identified.

Summary: health impacts of measures to reduce house dust mite allergen
Although house dust mite is known to be one of the most common allergens to exacerbate symptoms among asthmatics, current measures to reduce house dust mite have not been shown to be effective at either reducing asthma symptoms or reducing exposure to house dust mite allergens.

3.2 Interventions to reduce unintentional injuries, fires, and falls in the home

The home is an important location for unintentional injury and death, most commonly resulting from falls, poisoning and fires; children and the elderly are particularly at risk.

The use of safety devices in the home, particularly child resistant packaging on poisonous products, may reduce the risk of unintentional injury. Targeted programmes of free distribution of devices along with education and home visits do lead to increased use of safety equipment and implementation of safety practices in the home but the subsequent impact on domestic injury is not known (Kendrick et al., 2007). For example, homes provided with a safety kit and a water temperature card have been reported to be more likely to have bath water at a safe temperature but the impact on reported scalds and injury from hot bath water following community distribution of safety advice of equipment is not yet established (Turner et al., 2004). Pool fencing has been recommended to prevent child drowning. Pool fences should separate the pool from the house and should surround the pool on all sides (Thompson, Rivara, 1998).

Programmes of environmental modification or free distribution of safety appliances must be accompanied by education and home visits for their potential to benefit to be realized. In addition, devices which are affordable and easy to use may be more likely to be used and therefore increase effectiveness (Saegert et al., 2003).

Homes with working smoke alarms installed are associated with a reduced risk of injury and death from residential fires, and residential fires detected by a smoke alarm are less likely to result in fatality (Marshall et al., 1998; DiGuiseppi et al., 1998). However, promotion programmes, including mass media, education and free smoke alarm give-aways, have not been shown to be an effective way to increase ownership and correct use or maintenance of a smoke alarm or to reduce fire or fire-related injury (DiGuiseppi et al., 2001). Proper installation and maintenance are essential if the potential prevention of fire-related injury is to be achieved (DiGuiseppi et al., 2002). Smoke alarms using an ionization sensor and powered by a 10 year battery are most likely to be functioning one year after installation (Rowland et al., 2002).

Effective interventions to reduce the risk of falling among the elderly include exercise, balance training and tailored interventions for those on sedative/hypnotic drugs or suffering from postural hypotension (Gillespie et al., 2009; Gillespie et al., 1996). The potential for benefit with these programmes will depend on the different components of the intervention, implementation, and may vary considerably between individuals. It is unclear whether environmental modifications to the home, i.e. removing clutter and electrical cords, securing rugs, reduce injury but are likely to reduce falls in the elderly (Gillespie et al., 2009; Lyons et al., 2006). There is some preliminary evidence suggesting that community based initiatives aimed a preventing falls and fall-related injury among the elderly may be effective (McClure et al., 2005).

Summary: interventions to reduce unintentional injury, falls and fires in the home
The level of injury in homes with specific devices such as a working smoke alarm or four-sided pool fencing may be lower than in those homes without such devices, suggesting that these devices may be valuable injury prevention interventions. Although provision of safety equipment accompanied by education can improve safety practices in the home, but it is not known whether or not levels of domestic injury are reduced. Exercise, balance training and hazard removal may help prevent falls among ‘at-risk’ elderly people. Programmes to promote and even provide free smoke alarms do not always lead to effective installation and use of a smoke alarm, and the eventual impact on incidence of fires and fire-related injury is not known.

3.3 Interventions to reduce lead

Many countries have successfully invested heavily in lead reduction (Saegert et al., 2003; Guilart et al., 2003) but exposure to lead in some countries remains a common domestic hazard with significant health impacts, those most at risk are low income groups living in poor housing (Canfield et al., 2003; Margai, Henry, 2003; Leighton et al., 2003; Prüss-Üstün et al., 2003). Adverse physical, mental, intellectual and developmental effects have been associated with lead exposure even at low levels of exposure (Lanphear et al., 2005). The impacts are most often reported among children. Evaluations of interventions to reduce lead exposure have, therefore, most often focussed on outcomes among children.

Widespread public awareness, government and private action to reduce childhood exposure to lead has led to sharp declines in blood lead concentrations in children (Soldin et al., 2003). There is increasing evidence also of the growing breadth of effective prevention and treatment (Cohen, 2001). For example, controlling dust within contaminated homes can reduce blood lead concentrations significantly in children (Haynes et al., 2002). Such measures to reduce or eliminate lead exposure and poisoning have excellent benefit-to-cost ratios. Strict enforcement can lead to actual cost savings through medical and education savings and increased productivity for protected children (Brown, 2002).

Summary: child health impacts of lead reduction in homes
Multiple efforts to control childhood exposure have been successful in reducing blood lead concentrations, and adverse health effects of lead hazards.

3.4 Interventions to reduce exposure to noise

Indoor noise from neighbours and traffic is a common irritant to residents. Typically indoor noise will not lead to hearing loss but is likely to cause annoyance and sleep disturbance; impacts on other long term health outcomes including blood pressure and mental health are less clear (MRC Institute of Environment and Health, 1997). Improved sound insulation can reduce exposure to outdoor noise and this may reduce levels of annoyance. This review did not identify any studies which have investigated the health impacts of such measures.

Summary: health impact of measures to reduce noise in the home
Although effective measures to reduce noise may reduce disturbance and annoyance caused by noise, little is known about the health impacts of measures to reduce occupants' exposure to noise.

3.5 Interventions to adapt housing or rehouse residents to meet medical or mobility needs

Various housing adaptations are available to help those with a specific medical or mobility need to live independently in their own home, for example specially adapted toilets, shower facilities, and stair lifts. There is a growing literature assessing the health impacts of these measures. A detailed synthesis of this literature is beyond the scope of this chapter. These types of housing improvement are primarily used to promote independence and prevent accidents due to housing that is inappropriate to the resident's needs. Following housing adaptations residents are reported to have a reduced need for personal care (Baba et al., 1994; Heywood, 2007).

In some countries social housing tenants can apply to be rehoused to more appropriate housing on medical grounds. In the United Kingdom the most common reason to apply for medical rehousing is a mobility problem, but other reasons include dampness and mould, and links between mental health and problems with neighbours and the quality of the local neighbourhood (Blackman et al., 2003). Reports of the health impacts of rehousing on medical grounds are unclear. In most studies residents report improvements in both physical and mental health (Blackman et al., 2003; Cole, Farries, 1986; Elton, Packer, 1987; Kuroda et al., 1994; Smith, 1990).

Summary: health impact of housing adaptations and rehousing to meet medical or mobility needs
Housing adaptations to promote independent living and rehousing to meet medical or mobility needs can have health benefits for residents.

4. Conclusion

Both the quantity and the quality of research evidence on the health impacts of housing improvements have grown in recent years, in particular for warmth and energy efficiency improvements. Investment in affordable warmth measures that are targeted at those in poor housing, and with pre-existing illness can lead to health improvements, in particular respiratory improvements. Health improvements following area-based programmes of housing-led neighbourhood renewal are less clear. There is little suggestion that housing improvement leads to negative health impacts.

Interventions most likely to lead to measurable health improvements are those that target groups in most need where the potential to benefit is greatest, i.e. residents in the poorest housing who

are also most vulnerable to the detrimental health effects of poor housing. The differential health impacts of warmth improvements reported by the large New Zealand study and other United Kingdom studies is also likely to be a reflection of the potential to benefit at baseline, i.e. poorer health and worse housing conditions at baseline. Programmes delivering warmth and energy efficiency improvements are more likely to target individual households and tailor the improvement according to individual need. This contrasts with programmes of housing-led renewal which are rolled out across whole areas or neighbourhoods and are less discriminating about the varying needs of individual households within a geographical area.

5. Interpreting lack of clear research evidence and uncertainty around the health impacts of housing improvement

Although many studies have investigated the links between housing and health, it is apparent that there are far fewer studies which have investigated the health impacts of housing improvement. The evidence available on the health impacts of housing improvements is limited with respect to both the quantity of studies and the quality of evidence. This means that there is insufficient evidence to be confident that housing improvements will lead to health improvement.

The lack of research evidence is disappointing, and needs to be interpreted carefully. The lack of research evidence points to uncertainty and a gap in knowledge about the health impacts of housing improvement. Despite the uncertainty, the lack of evidence should not be used to conclude that housing improvements do not lead to health improvement. There are strong justifications to continue to invest in improved housing and to evaluate the potential for improved housing as a health promoting investment. The links between poor housing and poor health are sufficiently well established to argue for housing improvements on health grounds. It may be more realistic to expect health impacts some or many years, or even in the next generation, following a housing improvement, these longer term impacts cannot be detected by relatively short-term evaluations of 6-18 months, and are likely to remain largely unconfirmed by research. It may also be that, among populations experiencing various aspects of socioeconomic deprivation, improving housing alone is unlikely to lead to tangible health benefits in the short-term. Moreover, besides the pursuit of improved health there are other reasons to provide good quality housing, such as social justice, improved access, global energy conservation and general comfort. These improvements need to be carefully evaluated if the potential for health gain is to be validated and understood.

6. Policy implications

The above evidence synthesis suggests that improving housing conditions, in particular improved provision of affordable warmth, to vulnerable residents in very poor housing can lead to health improvement: The health impacts of smaller, relative improvements in housing conditions are less clear. Poor housing, poverty, and poor health are inextricably linked to each other and it may be that improvements to housing conditions alone are insufficient to lead to measurable health improvements, especially in the short term. The possibility of long-term health impacts and prevention of poor health among future generations remains largely unknown.

A range of other types of housing improvements have been implemented to reduce exposure to specific hazards in the home. National programmes of multifaceted approaches to remove lead and reduce exposure to lead among children have had health benefits. Many other interventions

aim to reduce unintentional injury, fires, exposure to house dust mite, and noise. However, the tangible health benefits of these interventions are unclear.

7. References

Allen T (2005a). *Evaluation of the housing for healthier hearts project April 2003-March 2005*. Bradford, University of Bradford.

Allen T (2005b). Private sector housing improvement in the United Kingdom and the chronically ill: implications for collaborative working. *Housing Studies*, 20:63-80.

Ambrose P (2000). *A drop in the ocean; the health gain from the Central Stepney SRB in the context of national health inequalities*. London, The Health and Social Policy Research Centre (University of Brighton).

Baba K et al. (1994). Research into the achievement and impact of 'Assistance Programme for Healthy Housing' of Edogawa Ward. *Annals of Japanese Public Health*, 41:404-413.

Barne, R (2003). *Housing and health uncovered*. London, Shepherds Bush Housing Association. (http://www.sbhg.co.uk/NR/rdonlyres/3291AD65-81F9-4409-BBC9-DB27C40AC2AF/1015/Healthand Housing Uncovered.pdf).

Barton A et al. (2007). The Watcombe Housing Study: the short term effect of improving housing conditions on the health of residents. *Journal of Epidemiology and Community Health,* 61:771-777.

Basham M et al. (2004). *Central heating: Uncovering the impact on social relationships in household management.* Exeter: Peninsula Medical School, Eaga partnership charitable trust. (http://www.eaga.com/downloads/pdf/central_heating.pdf, accessed 2 August 2007).

Berglund B, Lindvall T, Schwela DH (1999). *Guidelines for community noise*. Geneva, World Health Organization. (http://whqlibdoc.who.int/hq/1999/a68672.pdf, accessed 31 August 2010).

Blackman T, Aanderson J, Pye P (2003). Change in adult health following medical priority rehousing: a longitudinal study. *Journal of Public Health Medicine*, 25:22-28.

Blackman T, Harvey J (2001). Housing renewal and mental health: a case study. *Journal of Mental Health,* 10:571-583.

Braubach M, Heinen D, Dame J (2008). *Preliminary results of the WHO Frankfurt housing intervention project*. Copenhagen, World Health Organization.

British Medical Association Board of Science and Education (2003). *Housing and health: building for the future*. London, British Medical Association Board of Science and Education. (http://www.bma.org.uk/ap.nsf/AttachmentsByTitle/PDFhousinghealth/$FILE/Housinghealth.pdf, accessed October 2007),

Brown M (2002). Costs and benefits of enforcing housing policies to prevent childhood lead poisoning. *Medical Decision-making*, 22:482-492.

Caldwell J et al. (2001). *Glasgow Warm Homes Study: Final Report Glasgow*. Glasgow, Glasgow City Council Housing Services. (http://www.glasgow.gov.uk/NR/rdonlyres/BDA67F07-0A84-4F2A-924E-FB6BA8EFB4D9/0/final_report.pdf, accessed 1 August 2007).

Canfield R et al. (2003). Intellectual impairment in children with blood lead concentrations below 10 microg per deciliter. *New England Journal of Medicine,* 348:1517-1526.

Carter M (2001). Home intervention in the treatment of asthma among inner-city children. *Journal of Allergy & Clinical Immunology,* 108:732-777.

Cohen S (2001). Lead poisoning: prevention and treatment. *Pediatric Nursing*, 27:129-130.

Cole O, Farries JS (1986). Rehousing on medical grounds – assessment of its effectiveness. *Public Health*, 100:229-235.

Critchley R et al. (2004). *Housing investment and health in Liverpool*. Sheffield, Center for Regional Economic and Social Research (Sheffield-Hallam University).

Davie G et al. (2007). Trends and determinants of excess winter mortality in New Zealand: 1980 to 2000. *BMC Public Health*, 7:263.

Diguiseppi C, Goss C, Higgins JPT (2001). *Interventions for promoting smoke alarm ownership and function*. Cochrane Database of Systematic Reviews. (http://www.mrw.interscience.wiley.com/cochrane/clsysrev/articles/CD002246/frame.html).

DiGuiseppi C, Roberts I, Li L (1998). Smoke alarm ownership and house fire death rates in children. *Journal of Epidemiology and Community Health* 52:760-761.

DiGuiseppi C (2002). Incidence of fires and related injuries after giving out free smoke alarms: cluster randomised controlled trial. *British Medical Journal,* 325:995.

Eggleston PA et al. (2005). Home environmental intervention in inner-city asthma: a randomized controlled clinical trial. *Annals of Allergy, Asthma & Immunology*, 95:518-524.

Eick SA, Houghton N, Richardson G (2004). *The breath of fresh air project: Draft report for comments.* September 2004. Plymouth, AC & T England Ltd.

Ellaway A, Macintyre S, Fairley A (2000). Mums on Prozac, kids on inhalers: the need for research on the potential for improving health through housing interventions. *Health Bulletin,* 58:336-339.

Elton PJ, Packer JM (1987). Neurotic illness as grounds for medical priority for rehousing. *Public Health,* 101:233-242.

Evans M, Layzell J (2000). *The effect of housing renewal on health: the Riverside project* (end of grant report). Cardiff, University of Wales College of Medicine.

Gilbertson J et al. (2006). Home is where the hearth is: Grant recipients' views of England's home energy efficiency scheme (Warm Front). *Social Science & Medicine*, 63:946-956.

Gillespie L et al. (2009). *Interventions for preventing falls in older people living in the community*. Cochrane Database of Systematic Reviews**,** Art. No.: CD007146.

Gøtzsche P, Johansen H (2008). House dust mite control measures for asthma. Cochrane Database of Systematic Reviews**,** Art. No.: CD001187.

Green G, Gilbertson J (1999). Housing, poverty and health: the impact of housing investment on the health and quality of life of low income residents. *Open House International,* 24:41-53.

Guilart T et al. (2003). Environmental enrichment reverses cognitive and molecular deficits induced by developmental lead exposure. *Annals of Neurology*, 53:50-56.

Halpern D (1995). *Mental health and the built environment: More than bricks and mortar?* Philadelphia, Taylor & Francis, 1995.

Harrington BE (2005). Keeping warm and staying well: findings from the qualitative arm of the Warm Homes Project. *Health & Social Care in the Community***,** 13:259-267.

Haynes E (2002). The effect of interior lead hazard controls on Children's blood lead concentration: a systematic evaluation. *Environmental Health Perspectives*, 110:103-107.

Health Action Calderdale Kirklees and Wakefield (2005). *Initial analysis on the Health Action Calderdale Kirklees and Wakefield Project. Initial analysis and project repor*t. Kirklees & Wakefield, Kirklee, Calderdale, and Wakefield Council.

Heyman B et al. (2005). Keeping warm and staying well. Does home energy efficiency mediate the relationship between socio-economic status and the risk of poorer health? *Housing Studies*, 20**:**649-664.

Heyman B, Harrington B, Heyman A (2011). The National Energy Action Research Group. A Randomised Controlled Trial of an Energy Efficiency Intervention for Families Living in Fuel Poverty. *Housing Studies,* 26:117-132.

Heywood F (2007). *Better Outcomes, lower costs: Implications for health and social care budgets of investment in housing adaptation, improvements and equipment: A review of evidence*. London, School of Policy Studies, University of Bristol.

Hopton J, Hunt S (1996). The health effects of improvements to housing: a longitudinal study. *Housing Studies,* 11:271-286.

Howden-Chapman P et al. (2007). Effect of insulating existing houses on health inequality: cluster randomised study in the community. *British Medical Journal*, 334:434-435.

Howden-Chapman P et al. (2008). Effects of improved home heating on asthma in community dwelling children: randomisrandomized controlled trial. *British Medical Journal,* 337:1411a.

Iversen M, Bach E, Lundqvist GR (1986). Health and comfort changes among tenants after retrofitting of their housing. *Environment International*, 12:161-166.

Jacobs DE et al. (2010). A Systematic Review of Housing Interventions and Health: Introduction, Methods, and Summary Findings. *Journal of Public Health Management and Practice*, 16:5-10.

Kearns A et al. (2008). *SHARP survey findings: physical health and health behaviour outcomes.* Edinburgh, Scottish Government. Social Research, Department of Urban Studies, University of Glasgow & MRC Social & Public Health Sciences Unit. (http://www.scotland.gov.uk/Resource/Doc/246083/0069429.pdf, accessed 12 May 2008).

Kendrick D et al. (2007). *Home safety education and provision of safety equipment for injury prevention. Cochrane Database of Systematic Reviews*. (http://www.mrw.interscience.wiley.com/cochrane/clsysrev/articles/CD005014/frame.html, accessed 12 May 2008).

Krieger JW et al. (2005). The Seattle-King county healthy homes project: A randomized, controlled trial of a community health worker intervention to decrease exposure to indoor asthma triggers. *American Journal of Public Health*, 95:652-659.

Krieger JW et al. (2010). Housing interventions and control of ashtma-related indoor biologic agents: a review of the evidence. *Journal of Public Health Management & Practice,* 16:11-20.

Kuroda K et al. (1994). Effectiveness of a programme to improve housing conditions for elderly requiring supportive care in Edogawa-ku, Tokyo. *Japanese Journal of Public Health*, 41:404-414.

Landphear BP et al. (2005). Low-level environmental lead exposure and children's intellectual function: An international pooled analysis. *Environmental Health Perspectives*, 113:894-899.

Leighton J et al. (2003). The effect of lead-based paint hazard on blood levels of lead poisoned children in New York City. *Environmental Research*, 92:182-190.

Lloyd EL et al. (2008). The effect of improving the thermal quality of cold housing on blood pressure and general health: a research note. *Journal of Epidemiology and Community Health*, 62:793-797.

Lyons RA et al. (2006). *Modification of the home environment for the reduction of injuries*. Cochrane Database of Systematic Reviews.

Margai F, Henry N (2003). A community-basd assessment of learning disabilities using environmental and contextual risk factors. *Social Science & Medicine*, 56:1073-1085.

Marshall S et al. (1998). Fatal residential fires: who dies and who survives? *Journal of American Medical Association,* 279:1633-1637.

McClure R et al. (2005). *Population-based interventions for the prevention of fall-related injuries in older people*. Cochrane Database of Systematic Reviews.

Morgan WJ et al. (2004). Results of a home-based environmental intervention among urban children with asthma. *New England Journal of Medicine,* 351:1068-1080.

MRC Institute of Environment and Health (1997). *The non-auditory effects of noise (R10)*. Leicester, MRC Institute for Environment and Health, University of Leicester. (http://www.cranfield.ac.uk/health/researchareas/environmenthealth/ieh/ieh%20publications/r10.pdf, accessed 12 January 2010).

Nuffield Institute for Health (1996). *Preventing falls and subsequent injury in older people*. Effective Health Care. New Zork, University of Leeds & NHS CRD.

Platt, S et al. (2007). *The Scottish Executive Central Heating Programme: assessing impacts on health*. Edinburgh, Scottish Executive, Social Research Development Department.

Prüss-Üstün A et al. (2003). *Comparative quantification of health risks*. Lead exposure. Geneva, World Health Organization.

Rowland D et al. (2002). Prevalence of working smoke alarms in local authority inner city housing: randomised controlled trial. *British Medical Journal*, 325:998-1001.

Rugkasa J, Shortt N, Boydell L (2004). Engaging *communities: an evaluation of a community development model for tackling rural fuel poverty*. Dublin, Institute of Public Health in Ireland. (http://www.inispho.org/phis/catalogue/resdetails.php?resID=245, accessed 2 August 2007)

Saegert SC et al. (2003). Healthy housing: a structured review of published evaluations of US interventions to improve health by modifying housing in the United States, 1990-2001. *American Journal of Public Health*, 93:1471-1477.

Shortt N, Rugkasa J (2007). "The walls were so damp and cold" fuel poverty and ill health in Northern Ireland: Results from a housing intervention. *Health & Place*, 13**:**99-110.

Smith SJ (1990). Health status and the housing system. *Social Science & Medicine* 31:753-762.

Soldin O et al. (2003). Changing trends in the epidemiology of pediatric lead exposure: interrelationship of blood lead and ZPP concentrations and a comparison to the US population. *Therapeutic Drug Monitoring*, 25:415-420.

Somerville M et al. (2000). Housing and health: does installing heating in their homes improve the health of children with asthma? *Public Health*, 114:434-439.

The Warm Front Study Group (2006). *Health impact evaluation of England's home energy efficiency scheme (Warm Front)*. Headline results. Report to Energy Saving Trust/DEFRA.

Thomas R et al. (2005). Housing improvement and self-reported mental distress among council estate residents. *Social Science & Medicine*, 60:2773-2783.

Thompson D, Rivara F (1998). *Pool fencing for preventing drowning in children*. Cochrane Database of Systematic Reviews**.**

Thomson H, Morrison D, Petticrew M (2001). Health effects of housing improvement: systematic review of intervention studies. *British Medical Journal,* 323:187-190.

Thomson H, Morrison D, Petticrew M (2002). *Housing improvement and health gain: a summary and systematic review*. Glasgow, MRC Social & Public Health Sciences Unit.

Thomson H, Morrison D, Petticrew M (2007). The health impacts of housing-led regeneration: a prospective controlled study. *Journal of Epidemiology and Community Health,* 61:211-214.

Thomson H, Thomas S, Sellstrom E, Petticrew M (2009). The Health Impacts of Housing Improvement: A Systematic Review of Intervention Studies From 1887 to 2007**.** *American Journal of Public Health*, 99:681-692.

Turner C et al. (2004). *Community-based interventions for the prevention of burns and scalds in children*. Cochrane Database of Systematic Reviews 3, No.: CD004335

Winder R, Armstrong D (2003). *The Lambeth Study: Heating and Well-being in Older People (Final report).* London, King's College.

Quantifying the economic cost of unhealthy housing – a case study from England

Simon Nicol, Mike Roys, Maggie Davidson, David Ormandy, Peter Ambrose

1. Introduction

This chapter reports the results of a project commissioned by the BRE Trust (the charitable arm of the Building Research Establishment (BRE)) that developed a method to quantify the cost of unhealthy housing, and the cost–benefit of interventions to improve such housing (Roys et al., 2010).

There is a long established, recognized relationship between poor housing and poor health. In Victorian England diseases such as tuberculosis, cholera, and typhus were known to be associated with insanitary, cold, damp and overcrowded housing. Campaigns led by public figures including Chadwick, Snow and Southwood Smith highlighted the links between the appalling housing conditions in the poorer areas and health. These led to the introduction of legislation to establish standards of new housing and to deal with the conditions in existing housing. The problems of disease associated with the so-called 'slum' housing, such as cholera and tuberculosis, have now largely been eradicated but there remains a significant number of housing health and safety hazards, compounded by the fact that England has one of the oldest housing stocks in the developed world and one of the lowest rates of housing replacement.

Many studies have investigated the relationship between housing and health and there is now a large and growing body of evidence systematically linking adverse health effects with poor housing conditions. These conditions include: dampness; the effects of living in a cold home; unintentional domestic injuries; noise; insecurity; crowding and fire safety.

Warwick University, the BRE and the London School of Hygiene and Tropical Medicine were involved in the development of the Housing Health and Safety Rating System (HHSRS), which has since 2006 been the prescribed method for assessing housing conditions in England (ODPM, 2006). Assessments using the HHSRS produce numerical scores for dwellings based on the threats to health posed by 29 potential housing hazards.

Through the English House Condition Survey (EHCS) which now includes assessment of HHSRS hazards, we are able to quantify the national risk from unhealthy housing and the cost of remedial action (CLG, 2008). However, up until now, it has not been possible to link this information to costs incurred by the health sector and to society attributable to unhealthy housing.

2. Summary of the Method

The approach is to:

- Define unhealthy housing
- Quantify the number and type of unhealthy dwellings in England
- Quantify the cost of improving these unhealthy dwellings to an acceptable level
- Quantify the costs to society of the health outcomes from living in these unhealthy dwellings.
- Develop a cost–benefit tool to quantify the health impact of different housing interventions.

2.1 Defining unhealthy housing

'Unhealthy housing' can be defined in a number of different ways. However, for the purpose of this project it has been defined as those dwellings assessed to have one or more Category 1 HHSRS Hazard (on which see below). As HHSRS hazards are assessed as part of the EHCS, this means there are data on unhealthy housing at a national level.

The Housing Health and Safety Rating System (HHSRS)
The HHSRS was developed over ten years and was informed by a large body of research and statistical evidence on the link between housing conditions and health. It shifts the emphasis from defects and deficiencies to their potential effect on the health and safety of people.

The HHSRS provides a means of evaluating the potential effect of any defects on the health and safety of occupants, visitors, neighbours and passers-by. Using the HHSRS grades the seriousness of potential hazards and takes account of the frequency of occurrence and the severity of the outcome. This logical approach allows hazardous occurrences that may happen often but with minor outcomes to be compared with those that happen rarely but will have major health outcomes.

There are 29 potential HHSRS hazards identified, which fall into four groups (see Table 1).

Table 1: The 29 HHSRS Potential Housing Hazards

A. Physiological Requirements	**C. Protection Against Infection**
Damp and mould growth etc	Domestic hygiene, Pests and Refuse
Excessive cold	Food Safety
Excessive heat	Personal Hygiene Sanitation and Drainage
Asbestos (and Manufactured Mineral Fibre)	Water supply
Biocides	
Carbon Monoxide and Fuel combustion products	**D. Protection Against Accidents**
Lead	Falls associated with baths etc
Radiation	Falling on level surfaces
Uncombusted fuel gas	Falling on stairs etc
Volatile Organic Compounds	Falling between levels
	Electrical Hazards
B. Psychological Requirements	Fire
Crowding and Space	Flames and hot surfaces
Entry by intruders	Collision and entrapment
Lighting	Explosions
Noise	Position and operability of amenities
	Structural collapse and falling elements

Based on ODPM, 2006

For each hazard identified at a dwelling, a formula is used to generate a numerical score. A numerical score allows the widely differing hazards to be compared – the higher the score, the greater is the severity of the hazard. Three sets of numbers are used in the formula; one set is fixed (the weighting given to the health outcome), the other sets reflect the surveyor's judgments of:

- the likelihood of an occurrence (an event or exposure), which could result in a harm over the following 12 month period (the likelihood is to be given as a ratio – e.g. 1 in 10, 1 in 560).
- the most probable and other possible health outcomes, or harms, that would result from the occurrence.

From any occurrence there may be a most probable outcome, and other possible ones which may be more or less severe. For example, a fall from a second floor window could result in a 60% chance of severe concussion, but there may also be a 30% chance of a more serious injury, and a 10% chance of something less serious. The four classes of harms are listed in Table 2.

Table 2: Examples of the HHSRS Classes of Harms

Class I covers the most extreme harm outcomes including – Death from any cause; Lung cancer; Mesothelioma and other malignant lung tumours; Permanent paralysis below the neck; Regular severe pneumonia; Permanent loss of consciousness; and 80% burn injuries.	**Class II** covers severe harm outcomes, including – Cardio-respiratory disease; Asthma; Non-malignant respiratory diseases; Lead poisoning; Anaphylactic shock; Crytosporidiosis; Legionnaires disease; Myocardial infarction; Mild stroke; Chronic confusion; Regular severe fever; Loss of a hand or foot; Serious fractures; Serious burns; Loss of consciousness for days.
Class III covers serious harm outcomes, including – Eye disorders; Rhinitis; Hypertension; Sleep disturbance; Neuro-psychological impairment; Sick building syndrome; Regular and persistent dermatitis, including contact dermatitis; Allergy; Gastro-enteritis; Diarrhoea; Vomiting; Chronic severe stress; Mild heart attack; Malignant but treatable skin cancer; Loss of a finger; Fractured skull and severe concussion; Serious puncture wounds to head or body; Severe burns to hands; Serious strain or sprain injuries; Regular and severe migraine.	**Class IV** includes moderate harm outcomes which are still significant enough to warrant medical attention. Examples are – Pleural plaques; Occasional severe discomfort; Benign tumours; Occasional mild pneumonia; Broken finger; Slight concussion; Moderate cuts to face or body; Severe bruising to body; Regular serious coughs or colds.

Based on ODPM, 2006

These Classes of Harm are based on previous work by the BRE (Raw et al., 2000), and the weightings are intended to reflect the degree of incapacity suffered by a victim – Class I = 10 000; Class II = 1000; Class III = 300; and Class IV = 10.

From the judgments made by the surveyor, the HHSRS formula can generate a hazard score as illustrated in Table 3, using the example of falling between levels.

Table 3: Example Hazard Score for falls between levels.

Class	Weighting	Likelihood (1 in)	Spread of harm	Total
Class I	10 000	100	0	0
Class II	1000	100	30	300
Class III	300	100	60	180
Class IV	10	100	10	1
				Hazard Score = 481

Using this approach, hazard scores can range from 1 (very safe) to over 5000 (very dangerous). To avoid too much emphasis being placed on the numerical score, and to make the vast range more manageable, the scores have been put into 10 Bands (see Table 4). While there will be potential hazards in every dwelling (such as doors, windows, stairs, cooking facilities), the

Bands have been divided into two Categories, and Category 1 hazards (those in Bands A, B or C, with a hazard score of 1000 or more) are considered totally unacceptable. It is these Category 1 hazards that we have taken to be our definition of unhealthy housing.

Table 4: The HHSRS Hazard Bands

Hazard Bands	Hazard Score Range
A	5000 or more
B	2000 to 4999
C	1000 to 1999
D	500 to 999
E	200 to 499
F	100 to 199
G	50 to 99
H	20 to 49
I	10 to 19
J	9 or less

Source: ODPM, 2006

2.2 Quantifying unhealthy housing

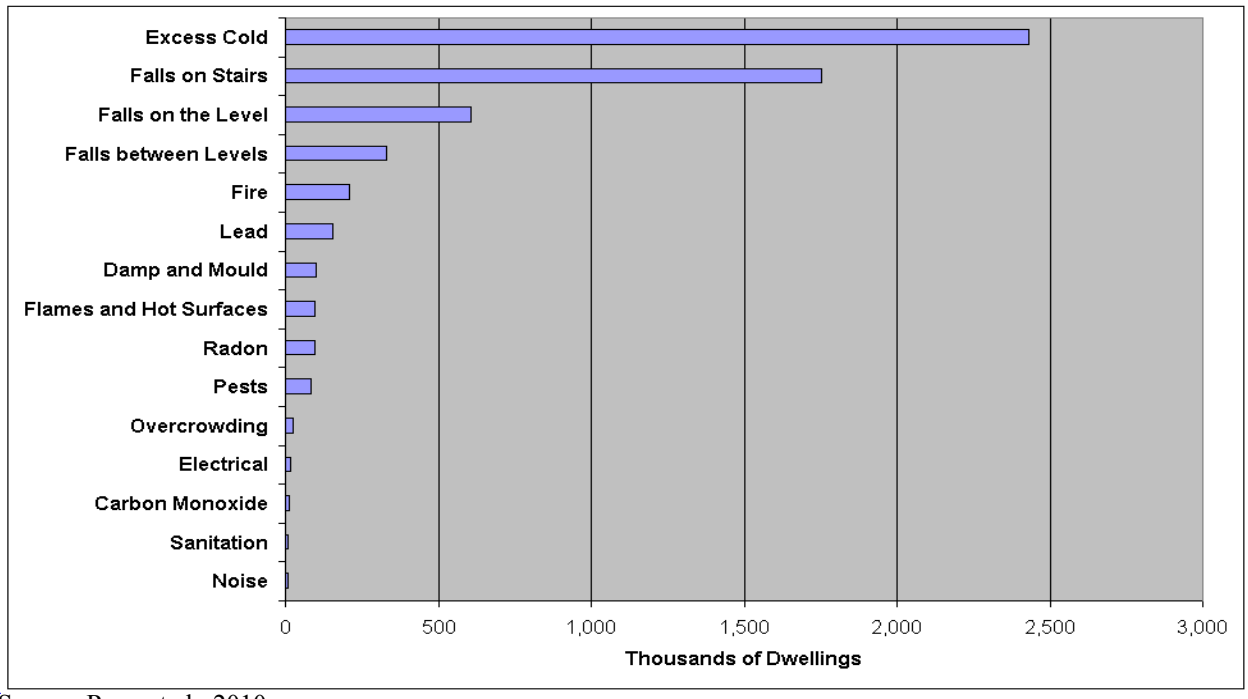
The EHCS is a national sample survey of housing conditions and energy efficiency carried out by the Government department responsible for the development and monitoring of housing policies (see CLG, 2008). The first 5 yearly EHCS was undertaken in 1971. Since 2001 it has been continuous, with an annual sample of 8000 dwellings taken randomly from across the housing stock of all types and tenures. In 2008 the EHCS merged with the Survey of English Housing to become the English Housing Survey (EHS) and so enabled it to collect comprehensive information on households as well as the dwellings they live in.

The EHCS collects information on the presence of 26 of the 29 HHSRS hazards for each dwelling sampled. Ten HHSRS hazards are fully assessed (the surveyor giving judgments on the likelihood and the outcomes); for 16 hazards the surveyor gives a judgment on whether there is a Category 1 hazard without going through the full assessment process; and for three hazards no data are collected – asbestos, biocides; volatile organic compounds – as these are both uncommon in their extreme form and cannot be deduced from a non-intrusive survey.

Results of the 2006 EHCS

At the time of this project, the latest EHCS results available were from the 2006 survey. Some 4.8 million (22%) of England's 22 million dwellings were identified as having a Category 1 HHSRS hazard and thus by our definition deemed to be 'unhealthy housing'. Over half of these dwellings were considered to be unhealthy because of the exposure of the household to a Category 1 risk from excess cold (see Fig. 1). The great majority of the remaining Category 1 hazards relate to 'falls' while some of the 29 hazards hardly figure at all in their extreme form.

Fig. 1: The Frequency of HHSRS Category 1 hazards



Source: Roys et al., 2010

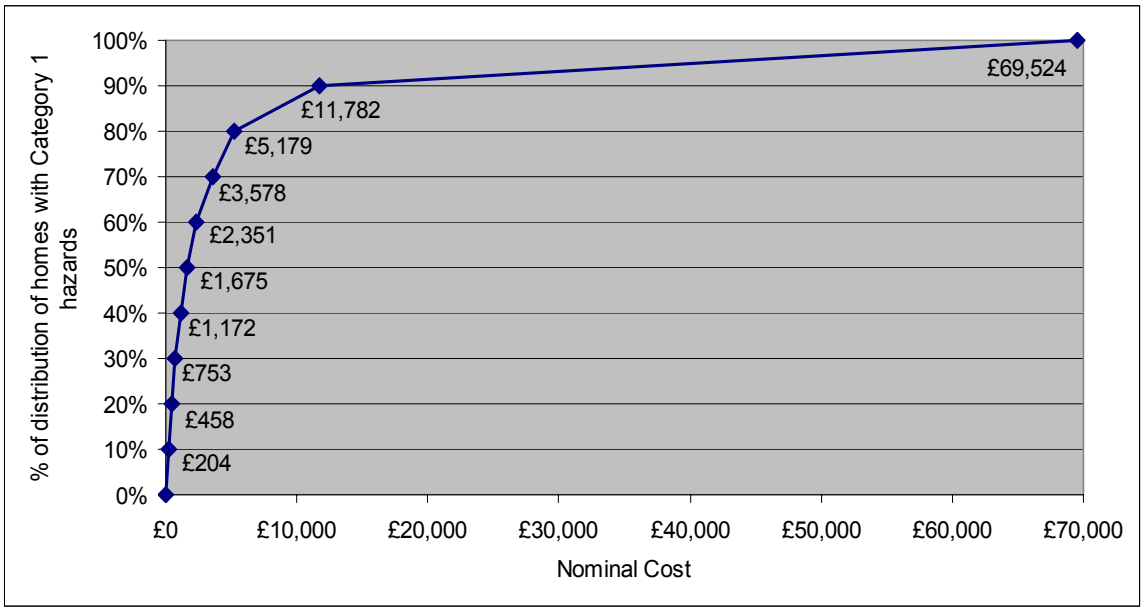
Note: Dwellings can have more than one HHSRS hazard, so the table above will not add up to the total number of dwellings with Category 1 hazards (4.8 million)

2.3 The cost of improving these homes to an acceptable level

The EHCS also collects the data on the remedial work considered necessary when a HHSRS hazard has been identified. These works are not for the eradication of the hazard altogether, but to reduce it to an acceptable level – this level usually being the average for the type of dwelling. From these data, standard costs are applied.

The range of costs to mitigate the Category 1 HHSRS hazards is presented in Fig. 2.

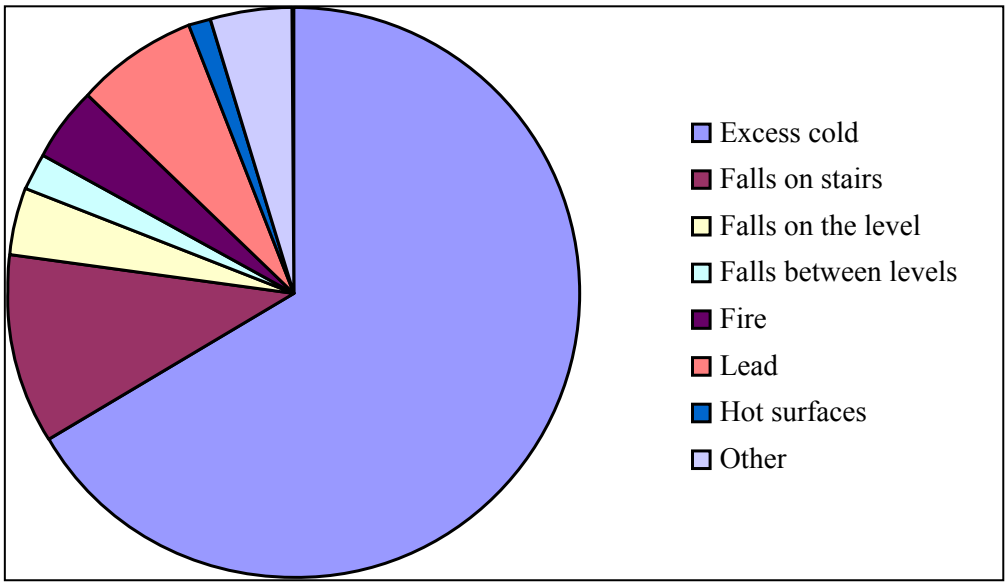
Fig. 2: Distribution of costs for remedial action on HHSRS Category 1 hazards



Source: Roys et al., 2010

This demonstrates that in many cases the cost of remedial work is not that high – with around a quarter of all Category 1 hazards being made acceptable for a cost of less than £600 (approx. 717 €), and the average cost for making Category 1 hazards acceptable being £3710 (approx. 4435 €) (CLG, 2008).The total cost of dealing with HHSRS Category 1 hazards in the English housing stock is some £17.6 billion (approx 21 billion €). These costs are dominated by those for making cold homes more comfortable (see Fig. 3), work that includes updating heating systems and providing insulation.

Fig. 3: The cost of dealing with HHSRS Category 1 hazards



Source: Roys et al., 2010

2.4 Quantifying the cost to society of unhealthy housing

Work on the task of measuring the 'exported costs' of unhealthy housing has been developed independently in a number of countries. Lawson (1997) argues that the United Kingdom National Health Service (NHS) spends about one fifth of its clinical budget on trying to cure illness that is actually caused by unemployment, poverty, bad housing and environmental pollution. More specifically, the costs to the NHS of treating ill health resulting from substandard housing has been estimated at £2.4 billion per year (National Housing Federation, 1997).

The issue of quantifying the effect of poor housing in Australia has been taken up by Berry (2002) who comments that "sufficient evidence exists to suggest that by seriously attacking the issue of insufficient affordable housing … government can materially alleviate a range of economic and social problems, while reducing the cost to tax payers, in the longer term". A paper on home injuries in the United States (Zaloshnja et al., 2005) calculated the medical costs of home injuries to be some £11.8 billion (approx 14.1 billion €) per annum, of which some 16% could be attributed to falls on stairs and steps. Another paper from the United States shows that remediation of lead paint hazards in housing yields a net benefit of $67 billion (Nevin et al., 2008).

What costs should be included?

This is a key question as some types of cost can be estimated or modelled more reliably than others. One of the most comprehensive reviews of poor housing (Ambrose, 2001) provides a matrix of costs, categorising them in terms of their measurability – costs that can be quantified

(H); costs that could be quantified given better data (M); and costs that exist but are probably non-quantifiable (NQ) (see Table 5).

Table 5: The costs to society of poor housing

Residents' costs	**External costs**
Annual loss of asset value if owned (H)	Annual loss of asset value if rented (H)
Poor physical health (H)	Higher health service treatment cost (H)
Poor mental health (M)	Higher health service treatment cost (H)
Social isolation (NQ)	Higher care service treatment cost (M)
Higher home fuel bills (H)	Higher building heating costs (H)
Higher insurance premiums (H)	Higher external insurance premiums (NQ)
Uninsured content losses (M)	Uninsured external losses (M)
Under achievement at school (NQ)	Extra school costs/homework classes (H)
Loss of future earnings (M)	Loss of talents to society (NQ)
Personal insecurity (NQ)	High policing cost (H)
More accidents (M)	High emergency service costs (H)
Poor hygienic conditions (NQ)	High environmental health costs (H)
Costs of moving (M)	Disruption to service providers (M)
Adopting self-harming habits (M)	Special health care responses (H)
	Government and EU programmes (H)

Source: Ambrose, 2011

H: costs that can be quantified

M: costs that could be quantified given better data

NQ: costs that exist but are probably non-quantifiable

Following a review of data sources and attempts to estimate costs of these factors and link them directly to hazards in the home, it was decided that we should focus on the treatment costs to the heath system alone (external costs triggered by "Poor physical health", "Poor mental health" and "Social isolation" as shown in Table 5 above). This is because:

- It is a transparent method of selecting a typical outcome for each level of harm of each hazard;
- Robust data are available to estimate the medical and care costs;
- It is a conservative approach, i.e., the benefits are likely to be underestimated.

However, our studies enabled us to conclude that the annual cost to the NHS of treating the health outcomes attributable to Category 1 HHSRS hazards in English housing accounts for a maximum of 40% of the total cost to society.

Having determined this methodology, our next step was to provide descriptions of the NHS treatments for the different outcomes for the different hazards. These could then be estimated; using NHS data (see Table 6).

Table 6: Typical outcomes and first year treatment cost for selected HHSRS hazards

Hazard	Class I	Class II	Class III	Class IV
Damp and mould growth	Not applicable -	Type 1 allergy (£1998)	Severe asthma (£1120)	Mild asthma (£180)
Excess cold	Heart attack, care, death (£19 851)	Heart attack (£22 295)*	Respiratory condition (£519)	Mild pneumonia (£84)
Radon (radiation)	Lung cancer, then death (£13 247)	Lung cancer, survival (£13 247)*	Not applicable -	Not applicable -
Falls on the level	Quadraplegic (£59 246)*	Femur fracture (£25 424)*	Wrist fracture (£745)	Treated cut or bruise (£67)
Falls on stairs and steps	Quadraplegic (£59 246)*	Femur fracture (£25 424)*	Wrist fracture (£745)	Treated cut or bruise (£67)
Falls between levels	Quadraplegic (£59 246)*	Head injury (£6464)*	Serious hand wound (£1693)	Treated cut or bruise (£67)
Fire	Burn,smoke, care, death (£11 754)*	Burn, smoke, care (£7878)*	Serious burn to hand (£2188)	Burn to hand (£107)
Hot surfaces and materials	Not applicable -	Serious burns (£4652)	Minor burn (£1234)	Treated very minor burn (£107)
Collision and entrapment	Not applicable -	Not applicable -	Punctured lung -	Treated cut or bruise -

Source: Roys et al., 2010

Not applicable = HHSRS class of harm outcome very rare or non existent

* Costs after 1 year are likely to occur, these are not modelled

The total cost of unhealthy housing

Earlier, we calculated that the total cost of reducing the Category 1 hazards in English dwellings to an acceptable level (the average for their age and type) was some £17.6 billion. For the hazards that were fully measured through the EHCS, we have a 'likelihood' score for all dwellings with a Category 1 hazard, and we have a national average likelihood score for the same home for its age and type (calculated during the development of the HHSRS, see ODPM, 2003). Using the difference between the actual score and the national average for the whole stock, an estimate for the total annual treatment cost to the NHS can be calculated, which in this case is just over £600 million (approx 717 million €) per year if the dwellings are left unimproved (Table 7). Using this information, the direct payback period for all hazards can be calculated at 29 years, if the repairs are all made up front.

Table 7: The costs, and benefits to the NHS, of reducing HHSRS Category 1 hazards to an acceptable level

HHSRS Hazard	Total number of HHSRS Category 1 hazards in English housing stock	Average one-off cost per dwelling	Total one-off cost	Annual Savings to the NHS if hazard mitigated
Falls between levels	332 000	£1276	£423 715 000	£36 059 696
Excess cold	2 346 500	£4994	£11 717 151 475	£21 433 443
Carbon monoxide	12 000	£1000	£12 000 000	£970 923
Overcrowding	23 000	£700	£16 100 000	£21 815 546
Dampness	99 000	£5000	£495 000 000	£8 794 064
Electrical problems	15 000	£4000	£60 000 000	£2 264 248
Fire	210 000	£1756	£368 900 000	£25 391 915
Flames, hot surfaces	98 000	£2200	£215 551 000	£8 967 969
Lead	154 000	£800	£1 232 000 000	£21 815 546
Noise	9000	£4000	£36 000 000	£1 270 750
Falls on the level	607 000	£1050	£634 673 130	£85 144 902
Domestic hygiene	82 000	£1400	£114 800 000	£7 902 858
Radon	96 000	£800	£76 800 000	£7 605 943
Falls on stairs	1 755 000	£1084	£1 902 420 000	£371 049 778
Personal hygiene	9000	£1300	£12 600 000	£1 208 064
Any Hazard	4 752 000	£3710	£17 644 252 905	£601 888 565

Source: Roys et al., 2010

It should be noted that the direct costs to the NHS used in this calculation, at best, only account for 40% of the total cost to society. By multiplying this saving up to 100%, the total cost to society is estimated at some £1.5 billion (approx 1.8 billion €) per year and the payback period for all hazards would be reduced from 29 years to 12 years.

The benefits to the NHS of bringing dwellings with excess cold hazards back to an acceptable level (the average for the age and type of dwelling) are disappointing. This is because the average excess cold HHSRS hazard score of 926 (the upper end of band D) is currently close to being a Category 1 hazard. Most improvement schemes would hope to improve homes to much better than this national average in terms of their energy efficiency and this would make a significant improvement to the NHS cost savings.

2.5 The health cost benefit of housing interventions

Clearly, England cannot afford £17.6 billion to go out and fix every HHSRS Category 1 hazard immediately. But the assumptions and outputs of this research have enabled us to design a cost-benefit spreadsheet to inform strategies for dealing with unhealthy housing, and to target improvement works. For example, the EHCS estimates that it would cost £3834 (approx 4584€) to re-design a dangerous staircase. But for a similar investment, the Category 1 hazards of more than 20 dwellings with basic handrail or guarding repairs could be addressed and thus save the NHS considerably more in potential diagnosis and treatment costs.

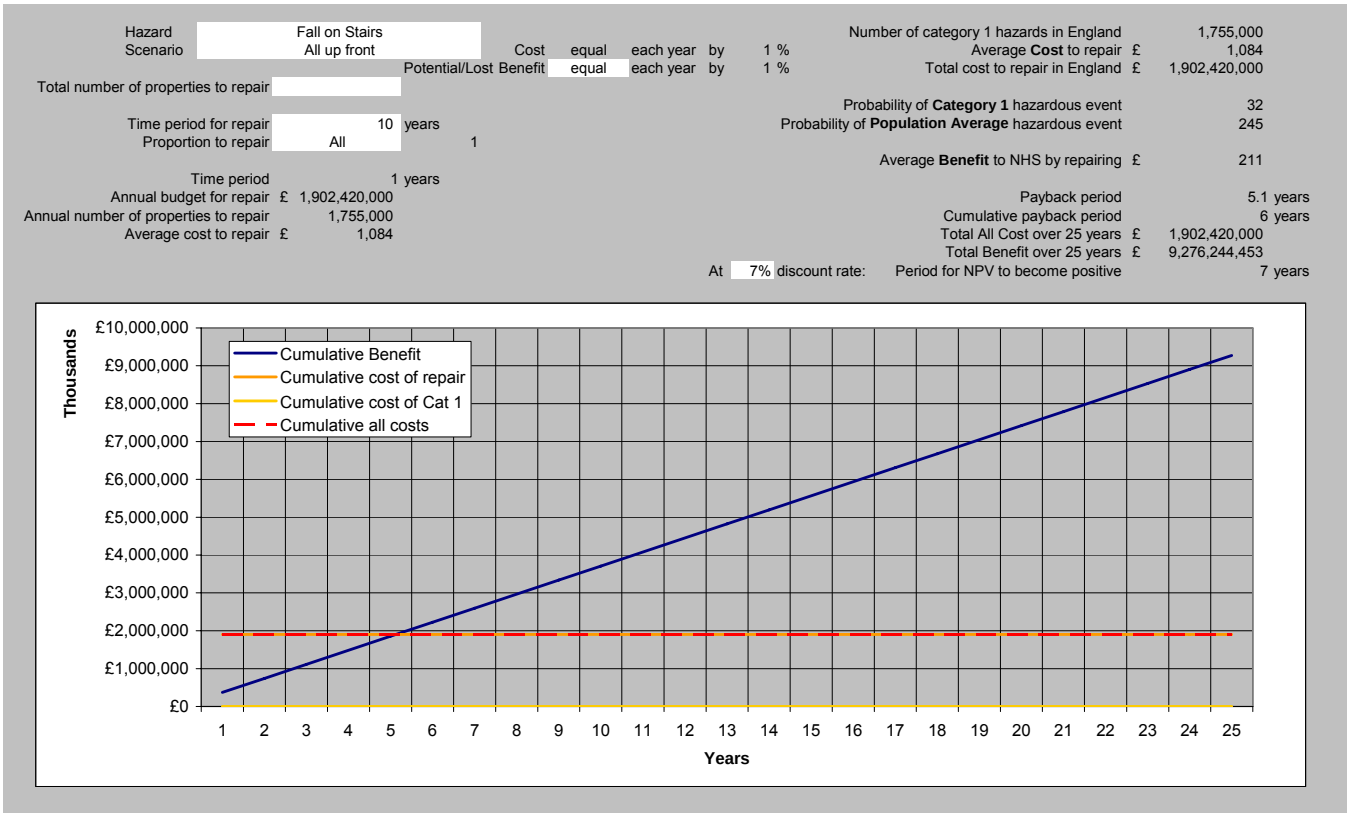
The example spreadsheet shown in Fig. 5 has been developed in Excel and uses the EHCS derived data for ‘falls on stairs’ and the other assumptions of this research. It is very flexible and

can be used to calculate the health costs benefit of a number of different scenarios since it is possible to change the following:

- The hazard to be considered
- The scenario to be applied (mitigation costs all up front, annual payment, no improvement works)
- The number of properties to be improved
- The proportion of properties to improve (all, cheapest 20%, cheapest 50%)
- Flexibility in value of costs and benefits
- Different discount rates for Net Present Value calculations.

In Fig. 4 the scenario 'all up front' has been applied. This shows that if all of the £1.9 billion (approx 2.3 billion €) required to reduce the Category 1 HHSRS stair hazards in England was spent immediately, the treatment costs to the NHS would be recouped within 5 years, and there would be a cumulative benefit of over £9 billion (approx 10.7 billion €) in 25 years.

Fig. 4: Example cost–benefit spreadsheet for falls on stairs



Source: Roys et al., 2010

These spreadsheets have now been developed for a number of HHSRS hazards and are being used to demonstrate to English local authorities the health cost–benefits of their schemes to deal with unhealthy housing. All that is required is the HHSRS assessment of dwellings prior to improvement, the condition following improvement, and the cost of the work.

Warwick University and the Building Research Establishment were recently commissioned by the Regional Leaders Board of the North West of England to investigate the health impact of housing interventions in six pilot authorities (Ormandy et al., 2010), using the methodology and Excel spreadsheets from this research. In one authority it was estimated that the annual benefit

to the health service of works undertaken to reduce the hazards in 30 sample homes was £34 900 (approx 41 000 €) against a total single cost of £310 000 (approx 370 600 €). This means that the payback period (the period when the cost of these housing interventions will be recovered) would be 9 years.

Some of the pilot authorities had lower payback periods than this because of the highly targeted nature of the works. The single lowest cost was £10 (approx 10 €) to address a 'falling on level surfaces' hazard; and in this case the benefit to the NHS was £21 (approx 25 €) per year. Other low mitigation works dealt with 'falls associated with baths' and 'falling on stairs'. The longest payback periods were for the hazards associated with fire safety, dampness and mould growth, and food safety.

3. Conclusions

Because of the way that information on HHSRS hazards is now collected through the EHCS, and the availability of good quality data from the NHS on the costs of treating the outcomes of HHSRS hazards, it has been possible, for the first time, to quantify the cost to the health sector of unhealthy housing in England. The total cost to the NHS attributable to the health outcomes from unhealthy housing is some £600 million (approx 717 million €) per year out of a total annual budget of £100 billion (approx 120 billion €). This cost would be 'saved' if the hazards were removed, or at least reduced to an acceptable level. The full cost of unhealthy housing to society is estimated to be some £1.5 billion (approx 1.8 billion €) per year.

This cost may actually seem quite low when compared to some of the estimates that have been made in the past. However, it should be stressed that this research has focused on reducing the effect of the 'worst' (the unacceptable) hazards, rather than eradicating them altogether or raising the standard of all housing to an optimum level. This pragmatic 'sticking plaster' approach has the earliest payback period in terms of cost savings, although some may regard it to be unambitious in the longer term where we should be aiming for ever higher housing standards.

One of the particular results of this project is to show that simple home safety improvements – handrails on dangerous stairs, hard wired smoke detectors, better home security etc – are very cost-effective. Because of the approach adopted (reducing hazards to the national average), the health gains from undertaking basic energy improvements to Category 1 HHSRS cold homes do not appear to be cost-effective. However, a further project is being undertaken by the BRE Trust to quantify the health cost–benefits of more comprehensive energy improvements, which are likely to be substantial.

The cost benefit tool is a real practical outcome of this research and it is already helping local authorities to justify expenditure on private sector housing renewal and to target the most cost-effective improvements on vulnerable people in unhealthy housing.

Finally, this research is being used to present a more informed case to governments for investment in housing, on the basis that it not only improves people's health and life chances but that it makes sound economic sense and can actually save money in the longer term.

4. References

Ambrose P (2001). Living Conditions and Health Promotion Strategies. *Journal of the Royal Society for the Promotion of Health*, 121:9-15.

Berry M (2002). *Affordable Housing. A Project for the Brotherhood of St Laurence in partnership with the Committee for Economic Development*. Melbourne, Australian Housing and Urban Research Institute.

CLG (2008). *English House Condition Survey 2006 – Annual Report*. London, Department of Communities and Local Government.

Lawson R (1997). *Bills of Health.* Oxford, Radcliffe Medical Press.

National Housing Federation (1997). *Housing for Health*. London, National Housing Federation.

Nevin R et al. (2008). Monetary benefits of preventing childhood lead poisoning with lead-safe window replacement. *Environmental Research*, 106:410-419.

ODPM (2003). *Statistical Evidence to Support the Housing Health and Safety Rating System. Volumes I, II, and III.* London, Office of the Deputy Prime Minister.

ODPM (2006). *Housing Health and Safety Rating System – Operating Guidance.* London, Office of the Deputy Prime Minister.

Ormandy D et al. (2010). *Linking Housing Conditions to Health*. Coventry, The University of Warwick.

Raw G et al. (2000). *A Risk Assessment Procedure for Health and Safety in Buildings*. London, CRC.

Roys M et al. (2010). The Real Cost of Poor Housing. Watford, Building Research Establishment BRE Trust.

Zaloshnja E et al. (2005). The Cost of Unintentional Home Injuries. *American Journal of Preventative Medicine*, 28:88-94.

Conclusions and Perspectives

David Ormandy, Matthias Braubach, David E. Jacobs

Introduction

The chapters in this method guide focus on the quantification of health impacts of particular housing conditions and present methodological approaches that can be applied. Although most chapters quantify the health impacts according to the environmental burden of disease approach, several chapters provide different approaches depending on the available data. In summary, this report provides various toolkits that can be applied for the quantification of health impacts of housing risks at both the national and local level. The editors hope that housing, health and environmental professionals will use these examples for developing further work to quantify and report on the health impacts of inadequate housing conditions.

In all chapters, the relationship of a single housing risk factor with one or more health outcomes is presented. This segmented approach was taken for practical and evidential reasons and was necessary to assess the quality of the evidence for a given housing/health relationship. Nevertheless, this approach is likely to underestimate the true extent, because many dwellings do not have only one defect related to health. Therefore, the possibility (or even probability) of a dwelling suffering from two or more health threatening conditions should not be ignored. For some conditions there is a direct link; for example, energy inefficient dwellings will be prone to dampness and mould. For others, the link may be indirect but the effect still quite large. For example, the presence of both environmental tobacco smoke and radon in a dwelling synergistically increases the risks from each.

It is also important to recognize the possible inter-relationship between aspects of housing. This means that improvements to one aspect of housing may affect another, and that effect may be either negative or positive. For example, improvements to energy efficiency may include draught-proofing to reduce heat wasted through excessive air changes, but this could have a negative impact on indoor air quality. However, as some conditions may be linked, it may mean that dealing with one problem may also resolve another; for example, improving energy efficiency and ventilation should also reduce the likelihood of damp and mould.

Health Outcomes Associated with Housing Conditions

This work has reviewed the evidence of the links between particular health outcomes and housing conditions, including asthma and other respiratory conditions, lung cancer, physical injuries, the spread of infections, cardiovascular conditions, neurological development, and negative mental health conditions. The importance of these and the links are reviewed below; however, it should be noted that categorization of health outcomes with specific housing conditions is not simple and straight-forward, as the outcomes can be linked with more than one condition, and vice-versa.

Respiratory Conditions

One of the most significant childhood chronic conditions in developed countries is asthma. A considerable proportion of childhood asthma cases is attributable to exposure to indoor dampness and mould: based on data for 45 countries of the WHO European Region, Jaakkola

and colleagues estimate that 0.07 deaths and 50 Disability-Adjusted Life Years (DALYs) per 100 000 children from asthma are associated with exposure to dampness in dwellings, and that 0.06 deaths and 40 DALYs per 100 000 children from asthma are associated with exposure to mould. In total numbers, mould exposure is associated with 83 deaths per year. Reducing the possibility of exposure would be extremely beneficial to public health and prevent or reduce a large proportion of asthma among adolescents and adults. The cause of dampness and associated mould can be related to the design, construction, maintenance, and use of the building. Good design and proper construction can help prevent problems from occurring; timely maintenance, including speedy response to traumatic events (such as a plumbing malfunction) will help keep the dwelling in a sound condition; and making occupiers aware of how and when moisture is generated, and how the use of means of ventilation can contribute to avoiding a build-up of moisture. It may not be possible to avoid acute occurrences such as extreme weather events (eg, storms and floods), but there should be effective responses to deal with the aftermath.

Exposure to radon is known to be a significant risk factor for lung cancer, and Zeeb shows that that the number of deaths per year attributable to exposure to radon in France, Switzerland and Germany is 1234, 231, and 1896, respectively (ranging between 2.1 – 3.2 deaths/100 000). Although the presence of radon may be geographically limited, new dwellings should be designed and constructed to prevent its penetration where exposure is possible, and mitigation measures should be carried out in existing houses in high radon areas.

It is well established that tobacco smoking damages the health of the smoker, and since the early 1970s it has been shown that exposure to environmental or second-hand tobacco smoke (SHS) causes respiratory infections and asthma in children, and also lung cancer and coronary heart disease in adults. In total, Jaakkola calculates the number of deaths and DALYs caused by second hand smoke to be 7.3/100 000 and 80.7/100 000 respectively in the WHO European Region, causing an annual 64 700 deaths per year. It is also known that smoking increases the risks from radon, and this may extend to the exposure to SHS. Smoking and exposure to SHS is not attributable to the design, construction or maintenance of housing. However, occupiers, particularly parents should be made aware of the threats to health and programmes should be initiated to discourage smoking in dwellings.

Formaldehyde has been shown to be associated with lower respiratory symptoms and can, as Gilbert et al. indicate, affect up to one percent of wheezing in children. The use of products containing or associated with formaldehyde should not be used in housing.

The use of solid fuel for cooking or heating in open fires or inappropriate stoves, and in dwellings with poor ventilation produces high concentrations of air pollutants. These pollutants have been linked to chronic obstructive pulmonary disease and lung cancer in adults and to pneumonia in children. Desai and colleagues calculate that, for the WHO European Region, there are 14 280 deaths and 394 600 DALYs per year related to exposure to indoor pollutants from solid fuel use. The largest health impact is reported for acute lower respiratory infections in children below the age of 5, which are estimated to cause 16.7 deaths/100 000 children per year. The health burden is, however, exclusively found for the subregions Euro B and C. There are effective solutions to reduce the possibilities of production of the indoor pollutants, including improvements to the stoves and the ventilation, and the use of cleaner fuels.

Cardiovascular Conditions

Noise is considered a stressor that has physical and psychological traits, and it has effects on both the nervous and the endocrine systems. There is evidence of an association between persistent noise and an increased risk of stress-related diseases, including immunosuppressive, gastrointestinal and cardiovascular disorders. In his chapter on the environmental burden of disease of traffic noise, Babisch focuses on cardiovascular effects and, applying an exemplatory EBD assessment for Germany, estimates 4.8 myocardial infarcts and 30.1 ischaemic heart

disease cases/100 000 population. There are three possible approaches to protect residents from road traffic noise; the first directed at reducing the noise sources, the second at the modification of housing, and the third at reducing the possibility of noise reaching the housing. (Although not covered in this work, air pollution from road traffic may also affect the indoor air quality of housing.)

Deaths from cardiovascular diseases are directly linked to exposure to excessively low indoor temperatures for long periods. It appears that between 50% and 70% of excess winter deaths are attributed to cardiovascular conditions, and about 15% to 33% to respiratory disease. Rudge estimates that per year, 38 200 excess winter deaths in 11 selected European countries are related to low indoor temperatures, representing 12.8 excess deaths/100 000 due to indoor cold. Low indoor temperatures are a result of energy inefficiency of the dwelling (poor thermal insulation and/or inefficient or inappropriate provision for heating), the social or economic status of the household, the cost of energy, or, more probably, a combination of these factors. New dwellings should be designed and constructed to meet energy efficient standards. For existing dwellings, there are two possible solutions – financial subsidies to those households struggling to meet the cost of energy required to maintain adequate temperatures, and energy efficiency measures (additional insulation and efficient provision for heating). The first is a short term solution, but necessary to protect health; while the second will provide a long term solution.

Injuries and poisoning at home

There are many features of dwellings that increase the likelihood of an accident that could result in a physical injury and features that could increase the severity of such injuries. The injuries can range from relatively minor cuts or bruises, through broken bones, to paralysis and even death. They can also include burns and scalds and drowning or near-drowning. The causes include slips, trips and falls, entrapments, collisions, poor lighting, and poor ergonomics. Keall and colleagues calculate that, using data for the whole WHO European Region, there are 7500 deaths and 200 000 DALYS attributable to a lack of window guards and smoke detectors, with the by far largest number of deaths caused by the lack of smoke detectors (0.9 deaths/100 000). It is possible to avoid most dangerous features, and to make potentially dangerous elements safer, such as a safe layout of kitchens and the installation of restrictors to openable windows. For existing housing it is possible to carry out works (often minor) to improve safety, such as provision of smoke detectors/alarms, fencing of pools and ponds, fitting window catches and restrictors, provision of adequate guarding to balconies, the fitting of handrails to stairs, and provision of carbon monoxide detectors.

The chapter by Kales et al. addresses the specific issue of CO poisoning which is a major cause of home poisoning related to the combustion of gas or solid fuels. CO exposure in indoor settings can quickly reach lethal levels but reliable, measured data on domestic CO exposure are rare. Thus, the assessment provides only the potential range of health outcomes expected for countries of the subregion Euro A (114 – 1545 persons with delayed or persistent neurologic sequelae; equalling 0.03 – 0.4/100 000) for which relevant data is available. Considering the household energy sources in subregions Euro B and C, it can be assumed that the environmental burden of disease may even be higher in countries of these European subregions but lack of data restricts the assessment to Euro A only. Essential elements of effective preventive measures against CO poisoning are: regular maintenance of gas-driven and other fossil fuel combustion equipment, improved exhaust ventilation, education of residents on the proper use of combustion devices, and the (in some countries already mandatory) installation of CO detectors in homes with gas heating appliances.

Spread of infections

For this work, the focus is on the relationship between tuberculosis (TB) and crowding. Acknowledging that there must be an infected person present in the dwelling, the spread of infection within the dwelling is more likely when that dwelling is crowded. Using TB as an example, it seems logical to argue that the spread of all infectious diseases transmitted from person to person will be more likely in crowded conditions. TB associated with household crowding in the subregions Euro B and C is estimated by Baker and colleagues to result in 0.8 deaths/100 000 and 17.6 DALYs/100 000 (a total of 15 351 additional tuberculosis cases, and 3518 additional deaths in Euro B and C). Crowding is a result of a mis-match between the household and the dwelling, and policies directed to promoting an adequate supply of affordable, and suitable sized, houses, together with speedy diagnosis and treatment of infectious diseases would help reduce the possibility of the spread of such diseases.

Mental Health and Development

Poor housing is stressful in several respects, as Evans shows in his evidence summary. These include concerns about hazards and safety, financial worries about mortgage or rent payments, and about meeting running costs (costs of maintenance and utility bills). The design of some types of housing (such as high rise buildings) may encourage social isolation. For those on low income, they may feel a lack of control over their environment. One finding in this work is that there is a need to recognize fully the mental health outcomes from inadequate housing, which is difficult to assess based on the environmental burden of disease approach. While it is unlikely that housing alone will precipitate serious mental disorder, it can induce chronic stress with symptoms of anxiety, depression, and hostility and frustration. And, poor quality housing may also be associated with poverty, exacerbating the mental stress of each.

Low level exposure to lead can have cognitive, developmental, neurological, behavioural and cardiovascular effects, and higher exposure levels can result in acute poisoning. The association between housing and lead-based paint, lead contaminated dust and soil lead and childhood lead poisoning is now well-established. Exposure to lead in housing in the WHO European Region is calculated by Jacobs to result in at least 652 860 DALYs (74.4/100 000). Controlling exposures to lead in housing is known to be effective; these include a combination of cleaning, covering and/or removing lead painted surfaces, and removing lead contaminated dust and soil.

The Public Health Significance of Housing Conditions

This work provides substantial evidence that unsatisfactory and inadequate housing is directly and indirectly associated with diseases, injuries and other health conditions. These negative health outcomes have a cost to the individuals, their households and to society. They lead to decreased quality of life, pain and discomfort, increased demands on the health services, absenteeism from work and from school, and education underachieving.

Although the threats to health and safety covered here do not include all potential housing threats, it is quite clear that the scale of the burden is considerable as summarized in Table 1. What is not clear at present is the effect of exposure to more than one condition, each associated with one health outcome, such as asthma, or the effect of exposure to two or more conditions associated with different health outcomes. This means that, although Table 1 provides a summary of the findings, the deaths and DALYs should not be simply summed together as many health outcomes may have more than one cause, and also because some effects may be synergistic or additive. Simply totalling the figures may therefore be either double-counting, or an underestimation.

Table 1: Summary of exposure, PAF and EBD from inadequate housing conditions

Exposure	**Health outcome**	**Exposure/ Risk relation-ship**	**Population Attributable Fraction**	**Environmental Burden From Housing per year**
Mould	Asthma deaths and DALYs in Children (0-14 years)	RR=2.4	12.3%	45 countries of WHO European Region: 83 deaths (0.06/100 000) 55 842 DALYs (40/100 000)
Dampness	Asthma deaths and DALYs in Children (0-14 years)	RR=2.2	15.3%	45 countries of WHO European Region: 103 deaths (0.07/100 000) 69 462 DALYs (50/100 000)
Lack of window guards	Injury deaths and DALYs (0-14 years)	RR=2.0	33-47%	WHO European Region: ~10 deaths (0.007/100 000) ~3310 DALYs (2.0/100 000)
Lack of smoke detectors	Injury deaths and DALYs (all ages)	RR=2.0	2-50%	WHO European Region: 7523 deaths (0.9/100 000) 197 565 DALYs (22.4/100 000)
Crowding	Tuberculosis	RR=1.5	4.8%	WHO Euro B and C subregions:* 15 351 TB cases (3.3/100 000) 3518 deaths (0.8/100 000) 81 210 DALYs (17.6/100 000)
Indoor cold	Excess winter mortality	0.15% increased mortality/°C	30%	11 European countries: 38 203 excess winter deaths (12.8/100 000)
Traffic noise	Ischaemic heart disease including myocardial infarction	RR=1.17/10 dB(A)	2.9%	Germany only: 3900 myocardial infarcts (4.8/100 000) 24 700 ischaemic heart disease cases (30.1/100 000) 25 300 DALYs (30.8/100 000)
Radon	Lung cancer	RR=1.08/ 100 Bq/m3	2-12%	Three western European countries: France: 1234 deaths (2.1/100 000) Germany: 1896 deaths (2.3/100 000) Switzerland: 231 deaths (3.2/100 000)
Residential Second Hand Smoke	Lower respiratory infections, asthma, heart disease and lung cancer	Risk estimates range from 1.2 to 2.0	PAF estimates range from 0.6% to 23%	WHO European Region: 64 700 deaths (7.3/100 000) 713 000 DALYs (80.7/100 000)

Exposure	Health outcome	Exposure/ Risk relation-ship	Population Attributable Fraction	Environmental Burden From Housing per year
Lead	Mental retardation, cardiovascular disease, behavioural problems	OR=4.4	66%	WHO European Region: 694 980 DALYs (79.2/100 000)
Indoor carbon monoxide	Headache, nausea, cardiovascular ischaemia/insuffi-ciency, seizures, coma, loss of consciousness, death	Case-fatality rate 3%; DNS/PNS incidence 3-40%	50-64%	WHO Euro A subregion:* 114 – 1545 persons with DNS/PNS (0.03 – 0.4/100 000) 114 ± 97 deaths (0.03 ± 0.02/100 000)
Formaldehyde	Lower respiratory symptoms in children	OR=1.4	3.7%	WHO Euro A subregion:* 0.3 to 0.6% of wheezing in children
Indoor solid fuel use	COPD, ALRI, Lung Cancer	RR=1.5-3.2	6-15%	WHO European Region: 8490 ALRI deaths in children <5 (16.7/100 000) 293 600 ALRI DALYs in children <5 (577/100 000) 5800 COPD deaths in adults ≥30 (1.1/100 000) 100 700 COPD DALYs in adults ≥30 (19.3/100 000)

OR= Odds Ratio; RR= Relative Risk; DALYs = Disability Adjusted Life Years; N/A= Not available; COPD = Chronic Obstructive Pulmonary Disease; ALRI=Acute Lower Respiratory Infections, DNS/PNS= Delayed or Persistent Neurologic Sequelae

* The list of countries for the European subregions is provided by Table 1 of the Introduction chapter

Presenting the data in this way may suggest the possibility of comparing the health burden related to specific housing conditions. However, despite the quantification of each respective burden of disease, the individual chapters applied different methodological approaches and different assumptions. In consequence, each chapter has specific restrictions and uncertainties. Therefore, a comparison of the results of the individual chapters is affected by these described uncertainty levels and, therefore, is simplistic and not recommended.

While additional data and evidence will strengthen the assertion that the environmental burden of disease from poor housing is huge, the conclusion must be that ensuring housing is as safe and healthy as possible will benefit public health and society generally. Achieving this objective will involve policies and actions of a wide range of individuals, bodies and agencies concerned with housing (in the widest meaning of that term). It will involve policy-makers at local and national level, legislators, architects, constructors, housing owners and managers, those concerned with housing renovation and improvement, and (of course) occupiers. It should also involve the health sector, which, as well as responding to the demands of individuals, should consider whether housing conditions could be implicated with a patient's condition. A coordinated, multidisciplinary approach will yield substantial benefits to both the housing and health sectors.

Policy implications

David Ormandy, Matthias Braubach

Poverty, poor housing, and poor health are usually linked, and this means that it is difficult to measure health gains from improvements to housing conditions alone. Although there is a need for more sound evidence of the health gains associated with housing interventions, the chapters of this report have shown that inadequate housing conditions are directly and indirectly linked to negative health outcomes. Inadequate housing conditions most often affect the less wealthy and the disadvantaged, and are therefore most often suffered by the more vulnerable population groups. In addition, those who make the most use of, and most demands on, housing are the very young, the elderly, and the sick, and these are population subgroups most vulnerable to environmental risks. Satisfactory, safe and healthy housing should therefore be a basic requirement for any society. The environmental burden of disease attributable to inadequate housing in Europe thus more than justifies the introduction of health based housing policies and actions as a means to achieve better housing, and provides clear evidence that housing is an important public health issue.

Housing strategies and policies are complex and include planning and construction to residential use followed by improvement, renovation and reconstruction. This means that strategies and policies for healthy housing need to be comprehensive and need to involve a wide range of professions.

Control of New Housing

The control of the design and construction of new dwellings should ensure that the necessary and appropriate precautions are incorporated to protect against the identified potential threats to health and safety. In particular, adequate design and construction of buildings should include:

- adequate damp-proofing
- radon protection measures (where necessary)
- prohibition of products associated with off-gassing of compounds (e.g. formaldehyde or similar volatile organic compounds) or other harmful material (e.g. lead or asbestos)
- provision to maintain effective controllable ventilation
- effective protection against noise penetration
- energy efficiency (thermal insulation, efficient provision of heating and draught-proofing)
- mandatory installation of smoke and CO detectors/alarms
- fencing of pools and ponds
- safety catches and restrictors to windows
- guarding to stairs and balconies (such as balustrades, handrails etc.)
- safe design and layout of kitchens

The matters mentioned above are those covered by the various report chapters, but clearly the control of housing should ensure the full range of safe and healthy conditions, with all necessary facilities and amenities. Aspects not covered in this report include e.g. water supply and sanitation, asbestos and other building-related pollutants, heat exposure, ventilation, infestations, fine particles, and hygiene requirements. Further aspects affecting housing conditions to be considered would be the location of settlements in safe areas, avoiding potential natural disasters

(flooding, landslides etc.) and well away from other risks such as e.g. waste sites, incineration plants or industries with harmful emissions.

To be effective, the controls should be in the form of legislative codes, capable of being properly applied and enforced, and backed by effective sanctions for non-compliance. While it is usual practice to set clear standards, care should be taken to avoid minimum standards becoming the norm.

As well as controls on the design and construction of individual dwellings, there should be effective planning controls. These should include ensuring housing is protected from noise sources, either by separation of roads (and other potential sources) or by the provision of barriers.

There should be programmes directed to the provision of affordable housing. The number and size of the housing should take account of local and national demands and trends, one intention being to limit the possibility of crowding and the associated risks to health which are not caused by the building itself, but by a mismatch between the size and characteristics of the household and the capacity of the dwelling.

Control of Conditions in Existing Housing

New dwellings only make up a very small proportion of the housing stock; the vast majority of the stock already exists and some of it is old and built to standards unacceptable today. For example, in the United Kingdom (England), 50% of dwellings are over 50 years old, and 20% are over 100 years old. It is the existing housing, therefore, where health based policies and actions will have the biggest impact and guidance should be developed for ensuring the improvement and rehabilitation of this housing.

For existing housing, it is not always possible to set specific standards or standards that can be required for new dwellings, although it should be possible require improvements. Some improvements can be achieved through retrofitting and mitigation measures; while for others it may be necessary to carry out investigations to determine whether and what action is necessary (such as for the presence of lead-based paint or of mould growth). For rented housing, there should be a responsibility placed on the landlord to properly maintain all elements of dwellings.

Clearly, it will not be possible to improve all the housing stock at once. To ensure that existing dwellings are improved or replaced according to the needs, there should be national and local policies and programmes with defined target areas where the most serious conditions are likely to exist.

One obvious problem associated with renovation and improvement programmes is the question of funding. What is now clear from the research is that there is a cost to society from the health outcomes attributable to inadequate housing, particularly to the health sector. This means that the one-off (single) cost of remedying or at least reducing potential threats to health from inadequate housing will produce a continuing benefit to society. While it should be the owner of the dwelling who pays for the maintenance and improvement of that dwelling (the owner's asset), where the owner cannot afford the cost of works, there is good reason for some form of State subsidy in the form of a grant or a loan, or an option to release equity through the purchase of a share in the property.

For energy inefficient dwellings, consideration should be given to the introduction of financial subsidies to help those households struggling to meet the cost of energy required to maintain adequate temperatures. While this is only a short-term, but necessary solution to protect the health of those households at a given time, the long term solution of improving energy inefficient dwellings should be the major goal of national policies.

Housing Designers, Constructors and Managers

While it may be considered necessary to have in place effective controls on new and existing dwellings, all those involved in the design, construction, management, maintenance and repair/rehabilitation of housing and building-related equipment should be made aware of the links between housing conditions and health. As well as understanding the links, these housing professionals have in the past and can in the future contribute to making housing safer and healthier. For example, it was the industry that developed child safety catches for windows, that developed safety cut-off devices for gas appliances, and that developed safer stairs. For other health threats, architects and building designers may be the responsible profession for action. By involving different sectors and approaches it is likely that new and innovative solutions to avoid health and safety threatening housing conditions will be developed.

Consideration should especially be given to campaigns and publicity to inform housing owners and managers (in both the public and private sectors) of the benefits of good maintenance and improvement, including the protection of their property from deterioration. While awareness raising campaigns are important, they should be backed by effective legal measures placing duties and responsibilities for the effective maintenance of their housing on landlords, housing companies and managers. Such duties and responsibilities should be framed in such a way that they are capable of enforcement, backed by meaningful sanctions, and supported by systems and resources to monitor their compliance.

Policy-makers and regulators

Housing-related regulations and codes fill thousands of pages in each country. Most often, they inform about required conditions and standards that need to be kept but leave options for interpretation and how, in technical terms, building and engineering solutions can satisfy the requirements. While many housing standards and codes may have been based on health principles when originally devised, in most cases there has been a lack of continued health input. The result is that, over the years, they have developed from the perspective of the building and the equipment, with little if any account being taken of health based evidence.

Policies and regulations affecting the construction and management of housing can be found on different levels and with varying legal status. On a global scale, Article 25(1) of the Universal Declaration of Human Rights (United Nations, 1948) states that

> Everyone has the right to a standard of living adequate for the health and well-being of himself and of his family, including food, clothing, housing and medical care and necessary social services, and the right to security in the event of unemployment, sickness, disability, widowhood, old age or other lack of livelihood in circumstances beyond his control.

The Istanbul Declaration on Human Settlements (UN Habitat, 1996) features a commitment by Member States to "endorse the universal goals of ensuring adequate shelter for all and making human settlements safer, healthier and more liveable, equitable, sustainable and productive." However, these definitions have no direct impact on the quality of housing in the field as they represent no legally binding controls, but they do provide aspirational objectives to be met.

Within Europe, the European Social Charter identifies the right to adequate housing under article 31 (Council of Europe, 2008). However, only about half of the European countries that have signed the charter per se have also signed this article and committed themselves to recognizing that right.

The European Commission issues Directives, including ones that directly and indirectly affect housing, such as the Construction Product Directive (Council of the European Communities, 1989) and the Energy Performance of Building Directive (Council of the European Communities, 2003). Each Member State must adopt the principles of the Directives and follow the framework given. However, how each MS satisfies the Directive will depend on its own

legal and administrative arrangements, so the mechanisms for ensuring the principles are met will vary. It is not clear that the health implications of Directives are always fully recognized during the drafting. For example, it is not apparent that the possible effects on indoor environmental quality of increased energy performance (through tighter buildings) have been taken into account. The current Directives therefore, although they may make progress towards better housing, cannot be considered healthy housing regulations.

At national levels, governments have adopted a variety of regulations covering such matters as energy, safety, personal hygiene, food safety, design and lay-out, and so on, however, the formulation of the standards and regulations may differ from country to country. One approach will be to set quantitative and specific standards referring to characteristics of housing features that should or should not be present (i.e. sink, cooking facilities, no dampness, etc.). Another approach is to set qualitative standards stating what should be taken into consideration when designing or assessing housing features in order to mitigate the threats to health and/or safety from the condition of the housing. The advantages of specific standards are that they are clear and easily understood, can be applied by relatively untrained staff, and are relatively inexpensive to implement. Such standards are particularly suitable for controlling standards in new buildings. Nevertheless this type of standard has disadvantages as they are difficult to update and extend, are building focused and are not ideal for the existing housing stock. On the other hand, qualitative standards can be human (and health) focused, relatively easy to update and more suited to existing dwellings. Unfortunately, because trained staff is needed for applying and enforcing them, these standards are expensive to implement and are not readily understandable by everyone.

There is a wide range of disciplines and professionals who have or should have an interest in housing standards and conditions, but these are not always brought together. Ideally, current building codes and regulations should be regularly reviewed and critically evaluated against the modern construction techniques, the use of the buildings, and the available health evidence.

Finally, on the subnational level, local and regional authorities can set strategies and policies directed to the identification of inadequate housing and assuring that (and also how) necessary action is taken. In particular, local authorities can, if accepting the challenge, become a relevant stakeholder in setting health based housing policies and controlling their effective implementation. This is especially the case in countries where local authorities administer a large proportion of the public or social housing that aims at providing housing for the less wealthy population groups.

Occupation

It seems obvious that the way a dwelling is used can contribute to (or even cause) unhealthy conditions. There should be campaigns and publicity to inform occupiers of potential dangers (such as carbon monoxide, and the threats to others from second hand tobacco smoke) and of important precautions (such as effective ventilation). Occupiers should also be made aware of any subsidies they may be entitled to, such as financial assistance toward energy costs.

It is in the interest of housing owners to encourage occupiers to report any disrepair or other problems (such as leaking pipes) so that effective action can be taken before there is damage to the property.

Any campaigns should take account of the particular target audience and the messages conveyed. This should include what media is used – television, radio, newspapers, internet – to ensure it reaches the target audience, and the clarity of the message and language used to try to ensure the message(s) are understood.

Health Professionals

There is a need to increase the involvement of the health sector in the development and implementation of policies and programmes directed at dealing with inadequate housing. Health professionals should especially contribute to setting standards and requirements for housing based on health evidence. As there will be data on illnesses, injuries and other health conditions that could be linked with particular housing conditions, this data could usefully inform decisions on priorities for, and the targeting of, housing programmes and actions. Health data can also be used to monitor the effectiveness of housing programmes and policies and should thus be considered an integral element of any approach that aims at the assessment of housing conditions and quality.

Systems should be set in place enabling doctors and Accident and Emergency Departments to refer patients (with their consent) for housing advice where patients present with health conditions (including injuries) that could be related to housing conditions. There are examples of such systems, including Conseiller Médical en Environnement Intérieur in France or the Green Ambulance in Brussels (and various other places), where a referral is made when a patient presents with a certain health condition that could be associated with the housing environment. Such a referral then triggers an investigation of possible exposures and risks in the home environment as a potential cause of the respective health effects.

Local Authorities/Municipalities

There are several functions and activities of local authorities that can promote healthier housing and alleviate conditions that could threaten health and safety.

First, urban planning in general should take account of the housing environment in the widest sense. Housing should be protected from noise sources and from sources of air pollution. The immediate environment should facilitate walking and give easy access to open space. Social services should recognize the role poor housing can have on the health and welfare of occupiers and where appropriate should coordinate with other agencies and departments.

Second, local authorities usually have responsibilities for reviewing the housing needs of their area and for developing and implementing housing strategies. Such strategies will cover the provision of new housing, schemes for the replacement of obsolete housing, and the application and enforcement of standards. The health relevance of housing means that there should be input from public health professionals into the local strategies, and an intense cooperation between the health, social and housing departments on municipal level.

Third, public housing bodies are in a position that requires adequate management for the benefit of the health and quality of life of their tenants, often including some of the most vulnerable population groups.

Research

The contributions to this work are limited to those areas where sufficient robust data is available. It seems clear that additional research and additional data will improve and enhance the existing evidence base, especially if it goes beyond the infrastructural, engineering or finance-related aspects of housing and considers the main function of housing – the provision of a safe and healthy residential environment for the occupiers. The lack of evidence is particularly high in relation to the health and cost benefits of housing interventions, for which systems should be developed to evaluate and monitor the effects both short- and long-term. The findings from such exercises will provide valuable evidence to inform policies and practices, helping to ensure effective targeting of resources.

References

Council of Europe (2008). *European Social Charter. Collected texts (6th edition)*. Strasbourg, Council of Europe (http://www.coe.int/t/dghl/monitoring/socialcharter/Presentation/ ESCCollectedTexts_en.pdf, accessed 17 November 2010).

Council of the European Communities (1989). *Council Directive 89/106/EEC of 21 December 1988 on the approximation of laws, regulations and administrative provisions of the Member States relating to construction products*. Official Journal L 040:12-26.

Council of the European Communities (2003). *Directive 2002/91/EC of the European Parliament and of the Council of 16 December 2002 on the energy performance of buildings*. Official Journal L 001:65–71.

UN Habitat (1996). *Istanbul Declaration on Human Settlements*. Habitat II Conference, 1996 (http://www.unhabitat.org/declarations/ist-dec.htm, accessed 17 November 2010).

United Nations (1948). *Universal Declaration of Human Rights*. New York, United Nations (http://www.un.org/Overview/rights.html, accessed 17 November 2010).

Abbreviations

ALRI	Acute Lower Respiratory Infection
aOR	Adjusted Odds Ratio
BCG	Bacille Calmette-Guérin *(strain of the tubercle bacillus used as basis for TB vaccination)*
BoD	Burden of Disease
BRE	Building Research Establishment
CDC	Centers for Disease Control and Prevention, United States
CI	Confidence Interval
CO	Carbon Monoxide
COHb%	Carboxyhaemoglobin saturation
COPD	Chronic Obstructive Pulmonary Disease
CVD	Cardiovascular Disease
DALY	Disability Adjusted Life Year
DNS	Delayed Neurologic Sequelae
EBD	Environmental Burden of Disease
EHCS	English House Condition Survey
EU	European Union
EUROSTAT	Statistical Office of the European Union
EWD	Excess Winter Deaths
GBD	Global Burden of Disease
GYTS	Global Youth Tobacco Survey
HDM	House Dust Mite
HHSRS	Housing Health and Safety Rating System
ICD	International Classification of Disease
IDB	EU Injury Database
IHD	Ischaemic Heart Disease
IRR	Incidence Rate Ratio
LTBI	Latent tuberculosis infection
MI	Myocardial Infarction
MVOC	Microbial Volatile Organic Compound
NHS	National Health Service
OR	Odds Ratio
PAF	Population Attributable Fraction
PNS	Persistent Neurologic Sequelae
ppm	Parts per Million

PPR	Person per Room
RCT	Randomized Controlled Trial
RD	Respiratory Disease
RR	Relative Risk or Rate Ratio
SES	Socioeconomic Status
SFU	Solid Fuel Use
SHS	Second-hand Smoke (“passive smoking”)
TB	Tuberculosis
TST	Tuberculin skin test
VOC	Volatile Organic Compound
WDI	World Development Indicators
WLM	Working Level Month
WHO	World Health Organization

Contributors

PETER AMBROSE, University of Brighton, United Kingdom (Author)

WOLFGANG BABISCH, Department of Environmental Hygiene, Federal Environment Agency, Germany (Author)

MICHAEL G. BAKER, He Kainga Oranga/Housing and Health Research Programme, University of Otago, Wellington, New Zealand (Author, Project meeting participant)

STEPHEN BATTERSBY, Chartered Environmental Health Practitioner and Environmental Health and Housing Consultant, United Kingdom (Project meeting participant)

ROBERT BAUER, Department of Research and Knowledge Management, Austrian Road Safety Board, Austria (Project meeting participant, Reviewer)

TIM BLACKMAN, Wolfson Research Institute, Durham University, United Kingdom (Reviewer)

HANS BÖGLI, Federal Office for the Environment, Switzerland (Reviewer)

MARY JEAN BROWN, Healthy Homes and Lead Poisoning Prevention Branch, US Centers for Disease Control and Prevention, United States of America (Project meeting participant)

PAOLO CARRER, Department of Occupational and Environmental Health, University of Milan, Italy (Reviewer)

OLIVIER CATELINOIS, IRSN, Laboratoire d'épidémiologie des rayonnements ionisants, France (Project meeting participant)

DAVID COWAN, Central Science Laboratory, York, United Kingdom (Reviewer)

DAVID CROWTHER, Department of Architecture, University of Cambridge, United Kingdom (Project meeting participant)

BEN CROXFORD, Bartlett School of Graduate Studies, University College London, United Kingdom (Reviewer)

DERRICK CRUMP, Institute of Environment and Health, Cranfield University, United Kingdom (Reviewer)

MAGGIE DAVIDSON, Housing Group, Building Research Establishment, United Kingdom (Author, Project meeting participant)

MANISH DESAI, School of Public Health, University of California, USA (Author)

GERT DOEKES, Institute for Risk Assessment Sciences, University Utrecht, The Netherlands (P, Reviewer)

JEROEN DOUWES, Centre for Public Health Research, Wellington, New Zealand (Author)

JAMES R. DUNN, Department of Health, Aging & Society, McMaster University, Canada (Project meeting participant)

GARY W. EVANS, Departments of Design and Environmental Analysis and of Human Development, Cornell University, United States of America (Author, Project meeting participant)

CHERYL FORCHUK, Department of Psychiatry, Schulich School of Medicine & Dentistry, University of Western Ontario & Lawson Health Research Institute, Canada (Reviewer)

JÉRÔME FREDOUILLE, Médecin Psychiatre Hôpital du Vinatier, France (Project meeting participant, Reviewer)

MARIA JOÃO FREITAS, Housing and Urban Rehabilitation Institute, Portugal (Project meeting participant, Reviewer)

LIN FRITSCHI, Western Australian Institute for Medical Research, Australia (Project meeting participant, Reviewer)

NICOLAS GILBERT, Air Quality Programs Division, Health Canada, Canada (Author, Project meeting participant)

MIREILLE GUAY, Biostatistics and Epidemiology Division, Health Canada, Canada (Author)

LARS GUNNARSEN, Danish Building Research Institute, Aalborg University, Denmark (Reviewer)

OTTO HÄNNINEN, Department of Environmental Health, National Institute for Health and Welfare (THL), Finland (Reviewer)

EVERT HASSELAAR, Delft University of Technology, Netherlands (Reviewer)

ULLA HAVERINEN-SHAUGHNESSY, Department of Environmental Health, National Institute for Health and Welfare, Kuopio, Finland (Author)

JONATHAN HEALY, Policy and Research Analyst Combat Poverty Agency, Ireland (Project meeting participant)

JOACHIM HEINRICH, Institute of Epidemiology, Helmholtz Zentrum München, Germany (Project meeting participant)

CLAUDIA HORNBERG, University of Bielefeld, Germany (Reviewer)

PHILIPPA HOWDEN-CHAPMAN, He Kainga Oranga/Housing and Health Research Programme, University of Otago, Wellington, New Zealand (Author, Project meeting participant)

EMMA HUTCHINSON, Public and Environmental Health Research Unit, Department of Public Health and Policy, London School of Hygiene & Tropical Medicine, United Kingdom (Project meeting participant)

TANZIMA ISLAM, Employee & Industrial Medicine, Cambridge Health Alliance, USA (Author)

MARITTA S. JAAKKOLA, Respiratory Medicine Unit, Center for Environmental and Respiratory Health Research and Institute for Clinical Medicine, University of Oulu, Oulu, Finland (Author, Project meeting participant)

DAVID E. JACOBS, National Center for Healthy Housing, Washington DC, United States of America (Author, Editor)

MATTI JANTUNEN, Department of Environmental Health, National Institute for Health and Welfare (THL), Finland (Project meeting participant, Reviewer)

LARS JARUP, Imperial College London, United Kingdom (Reviewer)[18]

STEFANOS N. KALES, Department of Environmental Health/ Employee & Industrial Medicine, Harvard School of Public Health/ Cambridge Health Alliance, USA (Author, Project meeting participant)

[18] Deceased.

MICHAEL KEALL, He Kainga Oranga/Housing and Health Research Programme, University of Otago, Wellington, New Zealand (Author)

VLADIMIR KENDROVSKI, Sector for Environmental Health, National Institute of Public Health, The former Yugoslav Republic of Macedonia (Reviewer)

MIN KIM, Employee & Industrial Medicine, Cambridge Health Alliance, USA (Author)

THOMAS KISTEMANN, Institute for Hygiene & Public Health/WHO CC for Health Promoting Water Management & Risk Communication, University of Bonn, Germany (Project meeting participant)

JENS KORSGAARD, Institute of Hygiene, University of Aarhus, Denmark (Reviewer)

LINDA LARCOMBE, Faculty of Medicine, Department of Internal Medicine, University of Manitoba, Canada (Reviewer)

BERNHARD LINK, Baden-Württemberg State Health Office, Regierungspräsidium Stuttgart, Germany (Project meeting participant, Reviewer)

IAN MATTHEWS, Department of Epidemiology, Statistics and Public Health, United Kingdom (Project meeting participant)

SUMI MEHTA, Health Effects Institute, USA (Author)

MARK MENDELL, Indoor Environment Department, Lawrence Berkeley National Laboratory, United States (Reviewer)

MOUNIR MESBAH, Laboratoire de Statistique Théorique et Appliquée, Université Pierre et Marie Curie, Parie 6, Paris, France (Project meeting participant)

DAVID MICHAUD, Faculty of Social Sciences, University of Ottawa, Canada (Reviewer)

LIDIA MORAWSKA, International Laboratory for Air Quality and Health, Queensland University of Technology, Australia (Project meeting participant)

AINO NEVALAINEN, Department of Environmental Health, National Institute for Health and Welfare, Kuopio, Finland (Author, Project meeting participant)

SIMON NICOL, Building Research Establishment, United Kingdom (Author)

MATS NILSSON, Department of Psychology, Stockholm University, Sweden (Reviewer)

HILDEGARD NIEMANN, Berliner Zentrum Public Health, Germany (Project meeting participant)

DAVID ORMANDY, WHO Collaborating Centre for Housing Standards and Health, School of Health and Social Studies, University of Warwick, United Kingdom (Author, Editor, Project meeting participant)

BIRGIT RASMUSSEN, Danish Building Research Institute, Denmark (Reviewer)

ANNE ROGERS, School of Medicine, University of Manchester, United Kingdom (Reviewer)

MIKE ROYS, Building Research Establishment, United Kingdom (Author)

JANET RUDGE, Research Fellow Human Rights and Social Justice Research Institute London Metropolitan University Ladbroke House, United Kingdom (Author, Project meeting participant)

JÜRGEN SCHNEIDER, Umweltbundesamt, Austria (Reviewer)

MATHILDE SENGOELGE, Austrian Road Safety Board, Austria (Project meeting participant)

EDMOND D. SHENASSA, Brown Medical School Centers for Behavioral and Preventive Medicine, USA (Project meeting participant)

KIRK R. SMITH, School of Public Health, University of California, USA (Author)

JAN SUNDELL, International Centre for Indoor Environment and Energy, Denmark (Project meeting participant)

HILARY THOMSON, Social & Public Health Sciences Unit, Medical Research Council, Glasgow, United Kingdom (Author)

OLIVER THOMMEN, Institut für Sozial- und Präventivmedizin Universität Basel, Medizinische Fakultät, Switzerland (Project meeting participant)

KAREN TOCQUE, Centre for Public Health, John Moores University, United Kingdom (Reviewer)

RAINER ULRICH, Friedrich-Löffler-Institut, Greifswald, Germany (Reviewer)

KAMALESH VENUGOPAL, University of Otago, Wellington, New Zealand (Author)

JOANNE VINCENTEN, European Child Safety Alliance, Netherlands (Reviewer)

MAGNUS WICKMAN, Institute of Environmental Medicine, Karolinska Institute, Stockholm, Sweden (Reviewer)

PAUL WILKINSON, London School of Hygiene & Tropical Medicine, United Kingdom (Reviewer)

HAJO ZEEB, Bremen Institute for Prevention Research and Social Medicine, University of Bremen, Germany (Author)

Project meeting observers

GIULIO GALLO, DG SANCO, Luxembourg

MONIKA LUXEM, Federal Ministry of the Environment, Germany

GUDRUN LÜCKE-BRUNK, Federal Ministry of the Environment, Germany

RITA PAZDRAZDYTE, Ministry of Health, Lithuania

MANFRED SCHMITZ, Ministry of Labour, Health and Social Affairs, North Rhine-Westphalia, Germany

SIMONE STRECKER, Federal Ministry of Health, Germany

NATHALIE TCHILIAN, Ministry of Health, France

CLAUDIA WEIGERT, Ministry of Health, Portugal

World Health Organization

XAVIER BONNEFOY, Regional Advisor Noise and Housing, WHO European Centre for Environment and Health, Bonn, Germany (Project meeting participant, Project initiator)[19]

MATTHIAS BRAUBACH, Technical Officer Housing and Health, WHO European Centre for Environment and Health, Bonn, Germany (Author, Editor, Project meeting participant, Project leader)

ROKHO KIM, Scientist, WHO European Centre for Environment and Health, Bonn, Germany (Author, Project meeting participant, Reviewer)

MICHAL KRZYZANOWSKI, Regional Adviser, WHO European Centre for Environment and Health, Bonn, Germany (Project meeting participant, Reviewer)

HOJOON DANIEL LEE, Intern, WHO European Centre for Environment and Health, Bonn, Germany (Secretariat)

KNUT LONNROTH, Stop TB Department, WHO, Switzerland (Reviewer)

NURIA AZNAR MARTI, Programme Assistant Noise, Housing and Health, WHO European Centre for Environment and Health, Bonn, Germany (Secretariat)

FRANCESCO MITIS, WHO European Centre for Environment and Health, Rome, Italy (Reviewer)

ANNETTE PRÜSS-ÜSTÜN, Department of Public Health and Environment, WHO, Switzerland (Project meeting participant, Reviewer)

EVA REHFUESS, Department of Public Health and Environment, WHO, Switzerland (Author)

ANDREA RHEIN, Programme Assistant, Housing and Health, WHO European Centre for Environment and Health, Bonn, Germany (Secretariat)

NATHALIE RÖBBEL, Technical Officer Housing and Health, WHO European Centre for Environment and Health, Bonn, Germany (Project meeting participant)

DEEPIKA SACHDEVA, Programme Assistant, Living and Working Environments, WHO European Centre for Environment and Health, Bonn, Germany (Secretariat)

FERID SHANNOUN, Department of Public Health and Environment, WHO, Switzerland (Reviewer)

FRANCESCO ZAMBON, WHO Country Office, Moscow, Russian Federation (Reviewer)

[19] Deceased.

EBD associated with inadequate housing

This guide describes how to estimate the disease burden caused at national and subregional levels by inadequate housing conditions typically encountered in the WHO European region. It contributes to the WHO series of guides that describe how to estimate the burden of disease caused by environmental and occupational risk factors. An introductory volume to the series outlines the general methodology.

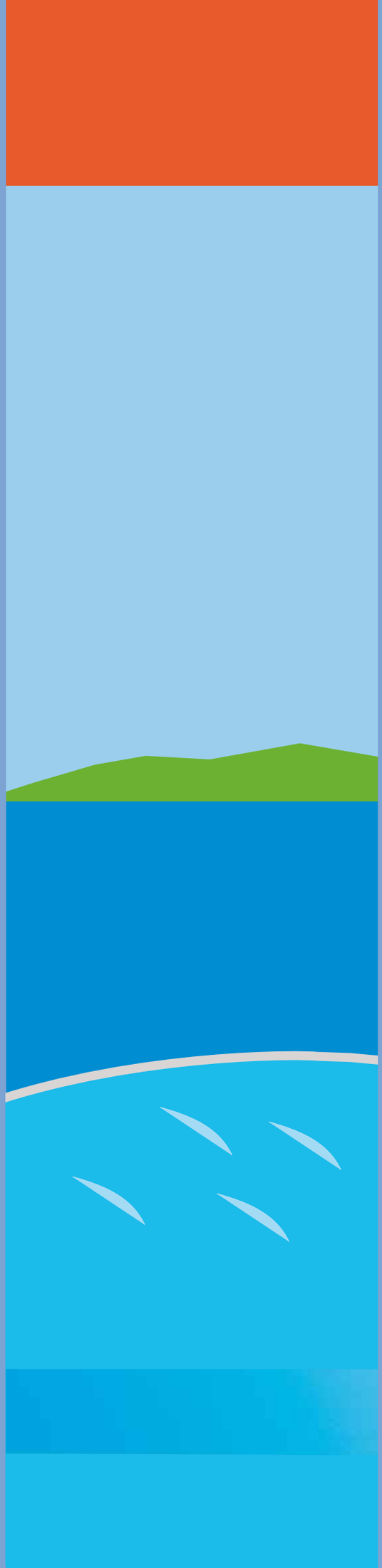
In this context, the WHO Regional Office for Europe took up the challenge to quantify the health effects of inadequate housing and convened an international working group to quantify the health impacts of selected housing risk factors, applying in particular the environmental burden of disease (EBD) approach.

The guide outlines, using European data, the evidence linking housing conditions to health, and the methods for assessing housing impacts on a population basis. This is done for twelve housing risk factors in a practical step-by-step approach that can be adapted to local circumstances and knowledge. This guide also summarizes the recent evidence on the health implications of housing renewal, and provides a national example on assessing the economic implications of inadequate housing.

The findings confirm that housing is a significant public health issue. However, to realize the large health potential associated with adequate, safe and healthy homes, joint action of health and non-health sectors is required.

Quantitative assessment of the size and distribution of health risks can be an important tool in identifying which actions will be most effective to reduce disease and injury.

The web site www.who.int/phe provides additional information on the environmental burden of disease and the guides already published. For further information: EBDAssessment@who.int



Environmental burden of disease associated with inadequate housing

WHO Europe