



# Report on the WHO technical meeting on quantifying disease from inadequate housing

**Bonn, Germany, 28-30 November, 2005**

## ABSTRACT

The WHO European Centre for Environment and Health is addressing the question "how much human health is negatively affected by inadequate housing?" to support European policy-makers. Experts were invited to a meeting in Bonn on November 28-30, 2005 to review and discuss the feasibility of quantifying environmental burden of diseases (EBD) related to selected factors, with the support of the German Ministry of Environment. The main objective of the meeting was to identify the housing-health relationships that have sufficient evidence to be included in the first EBD assessment for inadequate housing. The experts provided documentation for the selected housing-health relationships as background material for the meeting, and presented the available evidence of the association between housing factors and health effects. Altogether, the meeting reviewed 25 housing-health relationships for the availability and weight of evidence. Among these relationships, 13 were considered to have sufficient evidence, 10 to have some evidence, and 2 to have insufficient evidence. The experts agreed to continue to review the available evidence for selected housing-health relationships. In order to look into the selected topics in more detail, five Working Groups were created to examine the physical, chemical, biological, building-related and social aspects of health impacts of inadequate housing conditions. The meeting recommended that the WHO/EURO continue to support European policy-makers by consolidating evidence of the health impact of inadequate housing conditions.

### Keywords

AIR POLLUTION, INDOOR - analysis - adverse effects  
HOUSING - standards  
ENVIRONMENTAL EXPOSURE - adverse effects  
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## CONTENTS

	<i>Page</i>
Executive summary .....	2
1. INTRODUCTION .....	3
2. THE MEETING .....	4
3. RECOMMENDATIONS.....	6
Review of evidence for housing-health linkages .....	6
The approaches to quantifying housing burden of disease .....	9
4. SUMMARY OF THE MEETING SESSIONS .....	11
SESSION 1: Physical housing conditions.....	11
SESSION 2: Chemical housing conditions .....	13
SESSION 3: Biological housing conditions.....	14
SESSION 4: Building & Equipment factors .....	15
SESSION 5: Social conditions of housing and mental effects .....	16
SESSION 6: Exposure models and housing risk profiles .....	17
SESSION 7: Assessment of data validity and evidence base.....	18
SESSION 8: Conclusion, challenges and the way forward .....	19
5. FOLLOW-UP ACTIONS AND COMMITMENTS .....	21
Annex 1 List of participants .....	22
Annex 2 List of presentations .....	29
Annex 3 Background documents distributed at the workshop .....	32
Annex 4 Causal web for housing burden of disease .....	147

## **Executive summary**

The WHO European Centre for Environment and Health is addressing the question “how much human health is negatively affected by inadequate housing?” Experts were invited to a meeting in Bonn on November 28-30, 2005 to review and discuss the feasibility of quantifying environmental burden of diseases related to inadequate housing conditions. The main objective of the meeting, organised with the support of the German Ministry of Environment, was to identify the housing-health relationships that have sufficient evidence to be included in the first assessment of burden of disease from inadequate housing. The experts provided documentation for the selected housing-health relationships as background material for the meeting. At the meeting the experts presented the available evidence of the association between housing factors and health effects, and selected housing-health relationships to assess the feasibility of quantifying housing burden of disease. In order to look into the selected topics in more detail, the following working groups were created:

- Working group 1: Physical effects of housing inadequacy,
- Working group 2: Chemical effects of housing inadequacy,
- Working group 3: Biological effects of housing inadequacy,
- Working group 4: Building / equipment-related effects of housing inadequacy, and
- Working group 5: Social effects of housing inadequacy.

Altogether, the meeting reviewed a total of 25 housing-health relationships that could potentially justify further analysis to estimate the burden of disease. Among these relationships, 13 were considered to have sufficient evidence, 10 to have some evidence, and 2 to have insufficient evidence. The meeting agreed that relationships with insufficient evidence should be re-examined before being discarded from further studies. It was agreed that the participants of the working groups would provide a more in-depth assessment of the selected housing-health relationships. The participants recommended that the WHO Regional Office for Europe continue to support European policy-makers by providing further evidence of the health impact of inadequate housing conditions in coming years.

## **1. INTRODUCTION**

Although it is essential to address the question “how much human health is negatively affected by inadequate housing?” in order for WHO to support policy-makers effectively and efficiently, few systematic reviews have been undertaken so far to explore the impact of inadequate housing on health. The WHO Regional Office for Europe (Bonn office) is assessing the "Environmental Burden of Disease" from inadequate housing based on the currently available scientific evidence on the relationship between housing and health. As an initial step, a preliminary model of a “causal web” has been designed which contains a variety of housing factors for which evidence of exposure-outcome linkages are potentially available. Experts were invited to a meeting on the housing burden of disease to review and discuss the feasibility of quantifying burden of diseases related to selected factors with the support of the German Ministry of Environment in Bonn on November 28-30, 2005. Evidence obtained at the meeting from expert reports or consensus opinions of the meeting participants was used to select the housing-health linkages for further estimation of burden of disease.

## 2. THE MEETING

The objective of the meeting was to select the housing-health relationships with sufficient evidence by answering to the following questions:

- For which linkages between housing factors and health outcomes do we have sufficient evidence for estimating the burden of disease?
- What are the informational and methodological gaps and how do we proceed to calculate the burden of disease due to inadequate housing?

On behalf of European office of WHO, Xavier Bonnefoy welcomed the participants, and gave an overview of the activities in the Housing and Health Program of the WHO Bonn office. A draft causal web linking housing condition with health outcomes prepared by WHO was presented during his introduction. He emphasized that the meeting is building the evidence base to discuss and prioritize the housing and health relationship, and will pave the way for a comprehensive approach to housing and health. Matthias Braubach (WHO) presented an overview of housing burden of disease project. The main objectives of the project are to conduct a comparative assessment of the burden of disease related to inadequate housing for the WHO European Region, to collect available knowledge and current “state-of-the-art” to promote a comprehensive understanding of housing and health, and to generate evidence-based support for policy-making and priority-setting. Rokho Kim (WHO) outlined the methodology of quantifying the environmental burden of disease (EBD). Quantifying disease burden is a great opportunity to place an issue on the political agenda, raise awareness in the population, estimate cost-effectiveness and understand health gains achieved by a policy. EBD is a method for quantification of disease from environmental risks following an established framework. Calculations are rather simple, but require data on exposure measurement and synthesizing toxicological and other evidence. It was emphasized the housing burden of disease project is intended to support policy based on ‘available evidence’ on exposure, exposure-response relationship, and disease prevalence. Therefore, the accuracy of quantification will depend mainly on accuracy of exposure assessment, available evidence and assumptions made.

In a keynote speech on the potential health effects of housing improvement, Hilary Thomson reported findings from a systematic review of world literature in 1900-2000, focusing on intervention studies which assessed health after housing improvement. There is a wealth of epidemiological evidence on both biological factors and social factors linking housing and health. However, the evidence of positive health impacts after housing improvements was limited in general. Small improvements in health, particularly in mental health, were identified, but long term health impacts were unknown. Other impacts on the individual and at the neighbourhood level are common after home improvement projects and may have a subsequent health impact. Possible adverse effects that may outweigh the potential health gain were delineated.. Identifying related impacts may help to minimise negative impacts and maximise the potential for housing improvement to lead to health improvement. It is necessary to incorporate knowledge of relationship between housing, health and socio-economic determinants of health (including changes to local housing environment / neighbourhood). Thomson's review showed that quantification of health effects of housing improvements is complicated because of many confounding variables, such as socioeconomic status and other environmental pollutants.

The main part of the meeting was organized into five sessions corresponding to the major proximal causes as outlined in the WHO-proposed causal web:

- (1) physical conditions,
- (2) chemical conditions,
- (3) biological conditions,
- (4) building and equipment factors,
- (5) social conditions of housing and mental effects.

Reviews of published literature on selected housing-health relationships were presented by the experts according to a template provided by WHO, which were used as background documents for the first meeting. The presentations focused on three major elements that are necessary to quantify the housing-health relationship: the exposure of the population, the health outcomes, and the dose-response relationship between exposure and outcomes. A total of 25 housing-health relationships was reviewed. For each topic, a designated expert provided an overview of the currently available evidence on exposure-response (ER) relationship and exposure data followed by an expert opinion on the adequacy and sufficiency of the evidence. Each relationship was discussed at the meeting to reach expert consensus on the feasibility of quantifying the housing burden of disease. At the end of the meeting, the initial assessment by individual experts was adjusted to reflect the recommendations at the meeting. The background documents as well as other documents (causal web drafts, meeting agenda, participant list etc.) prepared by the experts for the meeting are in the Annex.

### **3. RECOMMENDATIONS**

#### **Review of evidence for housing-health linkages**

##### ***Selection criteria***

Evidence for exposure-response relationships (“ER relationship”) and “exposure data” of the proposed housing-health linkages was presented by the topic-specific experts. The results of the assessment were summarized in three categories: sufficient evidence, some evidence, and insufficient evidence. After the individual presentations on the review of evidence, working groups corresponding to five housing factors (i.e., physical, chemical, biological, building, and social factors) revisited the initial assessments of the topic-specific experts and proposed revised assessments based on consensus. For the sake of summarizing the assessments, a score of 3 (sufficient evidence), 2 (some evidence), or 1 (insufficient evidence) was given to each criterion for each linkage. The sum of these scores on two criteria (ER relationship and exposure data) was used to select the linkages for quantifying burden of disease based on the overall sufficiency of evidence. Accordingly, the linkages with a total score of 6 or 5 were considered to have “sufficient evidence;” those with 4 or 3 were considered to have “some evidence;” and those with 2 or 1 were considered to have “insufficient evidence.” This summarizing methodology resulted in an initial screening of the available data provided by the experts in order to enable a first overview of the potential strength and weaknesses of the required housing data. Based on the results, areas of housing and health which need to be explored in more detail by the working groups could be defined, as well those areas for which the existing evidence is already sufficient.

##### ***Results of evidence review***

The evidence necessary for quantifying the burden of disease reviewed and assessed by the topic-specific experts and by the working groups is summarised in Table 1. Thirteen housing-health relationships were selected as those with sufficient evidence. Two linkages, “Lighting conditions in the dwelling and mental and other health effects” and “Particulate matter in indoor air and respiratory and allergic effects,” were initially presented as having insufficient evidence. However, after a plenary discussion, the participants agreed that these two linkages should be reserved for more extensive review. Ten linkages were considered to have some evidence for quantifying burden of disease.

A: Linkages with *sufficient evidence* for estimating burden of disease

##### **Physical factors**

- o Heat and related cardiovascular effects and/or excess mortality
- o Cold indoor temperatures and winter excess mortality
- o Energy efficiency of housing and health
- o Radon exposure in dwellings and cancer
- o Neighbourhood and building noise and related health effects



**Chemical factors**

- o ETS exposure in dwellings and respiratory and allergic effects
- o Lead-related health effects

**Biological factors**

- o Humidity and mould in dwellings and related health effects
- o Hygrothermal conditions and house dust mite exposure

**Building factors**

- o Building and equipment factors and injuries / domestic accidents
- o Injury Database on domestic accidents and injuries
- o Estimating the number of home accidents from literature

**Social factors**

- o Multifamily housing, high-rise housing, and housing quality and mental health

B: Linkages with *some evidence* for estimating burden of disease

**Physical factors**

- o Ventilation in the dwelling and respiratory and allergic effects

**Chemical factors**

- o VOCs and respiratory, cardiovascular and allergic effects

**Biological factors**

- o Cockroaches and rodents in dwellings and respiratory and allergic effects
- o Cats, dogs, and mites in dwellings and respiratory and allergic effects
- o Pets and mites and respiratory, allergic or asthmatic effects

**Building factors**

- o Sanitation and hygiene conditions and related physical health effects

**Social factors**

- o Social conditions of housing and fear / fear of crime
- o Poverty and social exclusion and related health effects
- o Crowding and related health effects
- o Social factors / social climate and mental health

C: Linkages with insufficient evidence for estimating burden of disease

**Physical factors**

- o Lighting conditions in the dwelling and mental and other health effects
- o Particulate matter in indoor air and respiratory and allergic effects

Table 1. Assessment of feasibility of estimating evidence-based burden of disease from inadequate housing condition

	Experts initial assessment			Consensus at the meeting		
	ER Relation	Exposure data	Total	ER Relation	Exposure data	Total
<b>Physical factors</b>						
Heat and related cardiovascular effects and/or excess mortality	2	2	4	3	3	6
Cold indoor temperatures and winter excess mortality	2	3	5	3	3	6
Energy efficiency of housing and health	3	3	6	3	3	6
Radon exposure in dwellings and cancer	2	2	4	3	2	5
Neighbourhood and building noise and related health effects	2	3	5	2	3	5
Adverse health effects associated with inadequate light	1	1	2	?	?	?
Ventilation in the dwelling and respiratory and allergic effects	2	2	4	2	2	4
Particulate matter in indoor air and respiratory and allergic effects	1	1	2	?	?	?
<b>Chemical factors</b>						
VOCs and respiratory, cardiovascular and allergic effects	2	2	4	2	2	4
ETS exposure in dwellings and respiratory and allergic effects	3	3	6	3	3	6
Lead-related health effects	3	3	6	3	3	6
<b>Biological factors</b>						
Cockroaches and rodents in dwellings and respiratory and allergic effects	1	2	3	1	2	3
Hygrothermal conditions and house dust mite exposure	1	2	3	3	2	5
Cats, dogs, and mites in dwellings and respiratory and allergic effects	2	2	4	2	2	4
Pets and mites and respiratory, allergic or asthmatic effects	1	2	3	1	2	3
Humidity and mould in dwellings and related health effects	3	2	5	3	2	5
<b>Building factors</b>						
Building and equipment factors and injuries / domestic accidents	3	3	6	3	3	6
Injury Database on domestic accidents and injuries	3	3	6	3	3	6
Estimating the number of home accidents from literature	3	3	6	3	3	6
Sanitation and hygiene conditions	2	1	3	2	1	3

related physical health effects

**Social factors**

Social conditions of housing and fear / 2 fear of crime	2	4	2	2	<b>4</b>
Poverty and social exclusion and 3 related health effects	1	4	3	1	<b>4</b>
Crowding and related health effects 1	2	3	1	2	<b>3</b>
Multifamily housing, high-rise 2 housing, and housing quality and mental health	3	5	2	3	<b>5</b>
Social factors / social climate and 1 mental health	2	3	1	2	<b>3</b>

## The approaches to quantifying housing burden of disease

The meeting participants strongly supported the WHO/EURO effort to compile evidence of health impacts of inadequate housing conditions for European policy-makers. Participants also agreed to provide expert opinions into the process. In order to look into the selected topics in more detail, and provide the necessary data in a more structured way, the following working groups and their coordinators were created on specific topic areas:

Working group 1: Physical factors of housing inadequacy

Topics included: Thermal conditions, radon, noise, light, ventilation, PM etc.

Work group head: Claudia Weigert, Portugal

Working group 2: Chemical factors of housing inadequacy

Topics included: VOC, ETS, lead, etc.

Work group head: Jan Sundell, Denmark

Working group 3: Biological factors of housing inadequacy

Topics included: Pests and infestations, pets, mould growth, etc.

Work group head: Ian Matthews, UK

Working group 4: Building factors of housing inadequacy

Topics included: Sanitation equipment, home design and safety, etc.

Work group head: David Ormandy, UK

Working group 5: Social factors of housing inadequacy

Topics included: Fear of crime, poverty, crowding, etc.

Work group head: Hilary Thomson, UK

The working groups agreed to provide an in-depth assessment of the selected housing-health relationships for the next meeting in 2006. It was agreed that additional discussions on content and process could also take place in connection to the Healthy Buildings 2006 conference in Lisbon, Portugal.

Following the meeting, WHO agreed to – in its role as the secretariat for the working group on the burden of disease of housing – provide the final report to all participants, to post the results on the web for peer review by other housing experts, and to provide the working group with additional guidance on the next step towards the consolidation of evidence-base necessary to estimate housing burden of disease.

## 4. SUMMARY OF THE MEETING SESSIONS

### SESSION 1: Physical housing conditions

**Oliver Thommen** (Switzerland) presented "**Heat in dwellings and related cardiovascular effects and/or excess mortality.**" After reviewing the studies published in peer-reviewed journals, he initially concluded that there is some/partially sufficient evidence for the increased deaths from elevated ambient temperature. The data on exposure were also considered to be some/partially sufficient and reliable. Possible effects of climate change on extreme weather events (and health) were postulated such as altering frequency, timing, intensity and duration of extreme events, although the relationship between climate change and extreme weather events is not yet well understood. Building-related factors associated with heat-related mortality were identified as risk factors (living on higher floors of multi-storey buildings/ medical facilities; Outdoor temperature determines indoor temperature in naturally ventilated buildings; Urban heat island effect increases exposure) and protective factors (access to air conditioning in the home, living in a residence surrounded by trees or shrubs). Heat wave mortality may be mitigated by behavioural adaptation of the population and by protective housing factors. The burden of future heat wave deaths from inadequate housing is thus difficult to quantify. However, if the preventable fraction of heat wave mortality that can be preventable by protective housing factors such as air conditioning can be estimated, the burden of heat wave deaths from inadequate housing can be quantified conservatively. In this simplified approach, we do not need to estimate ER relations or exposure data.

**Jonathan Healy** (Ireland) presented "**Cold indoor temperatures and winter excess mortality (possibly circulatory and respiratory effects).**" Cold exposure/stress is a statistically significant factor in all-cause excess winter mortality, especially mortality from cardiovascular and respiratory diseases. The main causes of excess mortality in the winter season are: ischemic heart disease and cerebrovascular disease; these two causes of death account for approximately 85% of all mortality cases. The commonly-held assertion that pneumonia is a major cause of excess winter mortality is erroneous, accounting for less than 5% of excess winter deaths. Pan-European research indicates a strong inverse climatic gradient. Multivariate regression analysis has indicated that the gradient for levels of excess winter mortality is at its highest in countries with the mildest winters and lowest in the coldest countries. The thermal efficiency of housing has been attributed formally as a causal factor in the multivariate research. There is a relationship between cold indoor temperature and morbidity, though it is difficult to disentangle due to the ecological nature of studies. Irish research indicated that households enduring cold (or 'fuel-poor' households) were over three times as likely to report respiratory conditions and almost three times as likely to self-perceive ill-health caused by cold housing. After the discussion, the Working Group concluded that there is sufficient evidence for the linkage between cold indoor temperature and winter excess mortality, and that the exposure data are sufficient in Europe.

**Emma Hutchinson** (UK) presented "**Energy efficiency of housing and health**" and confirmed that the evidence for the relations between cold indoor temperatures and winter excess mortality is sufficient. As presented by Healy, there is a good inverse correlation

between the insulation of the house (such as double glazing and cavity wall insulation) and mortality. If the percentage of excess winter mortality that can be preventable by improved insulation and house heating can be estimated in Europe, the burden of excessive winter deaths from inadequate housing can be conservatively estimated.

**Olivier Catelinois** (France) presented “**Radon exposure in dwellings and cancer**”. The radon in homes would currently account for about 9% of the deaths from lung cancer and hence 2% of all cancer deaths in Europe, according to the excess risk of lung cancer from the joint analysis of case-controls studies published by S. Darby et al. (Radon in homes and risk of lung cancer: collaborative analysis of individual data from 13 European case-control studies. *BMJ*, 2005). For EBD study, it would be reasonable to use different sources of exposure-response relationship to perform an uncertainty analysis according to the model. Due to quite high variability of indoor radon concentration within the same country and the same region, the mean of exposure is not useful to perform a risk assessment. Thus, it would be reasonable to perform risk assessment at the adequate level and considering both the variability of indoor radon concentration within the same region and uncertainties related to the knowledge of indoor radon concentration. This can be done in two steps: (1) Definition of indoor radon concentration categories within a region; (2) Consideration of the entire indoor radon concentration distribution in each concentration categories weighted by the proportion of population concerned. After the discussion, the Working Group concluded that there is sufficient and some evidence for the radon - lung cancer relation and the exposure data in Europe, respectively.

**Hildegard Niemann** (Germany) presented “**Neighbourhood and building noise and related health effects.**” Based on the results of the LARES-study, the relative hypertension risk for adults (18-59 years) is increased by chronically strong annoyance by approximately 40 % in the European cities. If it is possible to keep away chronically strong annoyance by neighbourhood noise, 5 % of all hypertension treatments could be avoided. The attributable fraction for the exposed population is 30 %. Based on the results of the LARES-study it is to be assumed, that strong noise annoyance (strong and extremely from the ICBEN-scales) by neighbourhood noise, is detectable in approximately 8% of the inhabitants of European cities. The Work Group agreed that there is some evidence for ER relation, and sufficient evidence for exposure data.

**Mary Jean Brown** (USA) presented “**Adverse Health Effects Associated with Inadequate Light.**” Unipolar depression accounted for approximately 4.5% of DALYs worldwide with approximately 8.9% of DALYs attributed to depression in high income countries. The treatment effect found in these studies averages approximately 20-25% decrease in depressive symptoms. The LARES study suggested that improper lighting in dwellings is associated with increased risk of depression and falls. However, this linkage was found to have scarce data for the ER relation and exposure. The Working Group recommended that this topic should be revisited after an extensive search of evidence. It was also questioned whether the limited observations from the LARES study can be used for estimating DALYs related to inadequate light.

**Ian Matthews** (UK) presented “**Ventilation in the dwelling and respiratory and allergic effects.**” After a systematic review of available literature, it was concluded that none of the

ER relations were statistically significant. It was suggested that ventilation might be an effect modifier between indoor air quality and certain respiratory outcomes. The Working Group agreed that there are some evidence for both the ER relation and exposure data on ventilation.

**Jan Sundell** (Denmark) presented “**Particulate Matter & fine particles in indoor air and respiratory and allergic effects**” and concluded that no assessments can be made on the ER relation and indoor exposures in developed countries. The Working Group recommended that this topic should be revisited after extensive search of evidence.

## **SESSION 2: Chemical housing conditions**

**Bernhard Link** (Germany) presented “**VOC and chemical emissions in dwellings and respiratory, cardiovascular and allergic effects**” and concluded that there is some evidence of ER relation and exposure for making a valid assessment. Because the definition of respiratory and asthmatic symptoms is different across studies, interpretation of reported ER relation is not simple. Exposure to VOCs depends on factors like ventilation, temperature, humidity, season, and time after redecoration. Sampling methods and laboratory analyses are also variable. For example, active versus passive sampling (worst case / average situation), quantification of some versus all VOCs, and representative studies versus measurements after complaints of the inhabitants. The working group agreed that this linkage should be considered promising, but lacks sufficient evidence.

**Maritta Jaakkola** (UK) presented “**ETS exposure in dwellings and respiratory and allergic effects**” and concluded that there is sufficient and reliable evidence for making a valid assessment for asthma, respiratory infections, respiratory symptoms and low birth weight in children, and for lung cancer, asthma and respiratory symptoms in adults. There is also some / partially sufficient and reliable evidence for making a valid assessment for lung function impairment in children and for preterm delivery, and for lung function impairment, COPD and pneumonia in adults. The exposure is well documented in many European countries, and the prevalence of exposure is highly variable across European countries. The working group agreed that ETS and health linkage is among the most important topic in the estimation of burden of disease. However, questions were raised regarding the relation of ETS exposure to housing conditions. There should be further debates on whether change of behaviour of residents can be considered to be a housing issue. In the meantime, any *indoor* air quality should be considered in the estimation of housing burden of disease.

**David Jacobs** (USA) presented “**Lead in housing and lead poisoning** (co-authored with **Mary Jean Brown** (USA))”. In 2002, WHO ranked the lead-related burden of disease to be 16<sup>th</sup> in the DALYs. To estimate the housing burden of lead-related illnesses, housing-related lead exposure through paint, settled dust, bare soil, and drinking water should be partitioned properly. Airborne lead is now quite low, but historic use of lead gasoline still contaminates house dust and soil (together with deteriorating paint). Food and industrial emissions are thought to be low today. In Europe, there might be significant differences across countries in the portion of the population blood lead level that can be assigned to housing conditions, because each nation likely has a different regulatory history with regard to lead paint and

lead gasoline. European governments should be asked if unpublished nationwide assessments of lead in housing are available. According to the literature, France, Belgium (Brussels), Britain, Portugal, Germany, Netherlands, Poland, Italy, and Spain (Basque Region) have studies on exposure to lead paint, dust, soil or water in housing. There may be significant differences across countries in the portion of the population blood lead level that can be assigned to inadequate housing, due to different histories. If data are not available, but lead paint is known to be present, housing age may be a reasonable surrogate measure. The Working Group agreed that there is sufficient and reliable evidence in ER relationship and exposure data for making a valid assessment. Estimation of the housing-related proportion of total DALYs lost from lead exposure should be key process for this linkage.

### **SESSION 3: Biological housing conditions**

**Stephen Battersby** (UK) presented “**Rodents in dwellings and respiratory and allergic effects**” and concluded that as the result of inadequate surveillance there is no sufficient and reliable evidence for making a valid assessment for the total disease burden as the result of exposure to rodents in and around the home. Although there is some available exposure data, lack of ER relations limits the feasibility of quantifying housing burden of disease related rodents exposure in dwellings. It was also made clear in relation to the presentation that additional data would be needed on cockroaches, which have to be dealt with separately from rodents.

**David Crowther** (UK) presented “**Hygrothermal dwelling conditions and house dust mite exposure**”. He showed that the number of house dust mites present in dwellings can vary greatly, in ways that can be related to indoor hygrothermal conditions. More data on exposure in dwellings is becoming available, but it is still incomplete. Modelling of exposure based on climate and dwelling data will soon be possible but this too will require more data than is currently available. There is a large literature on the ill-health associated with an exposed population. There is some reliable evidence for making a valid assessment as to exposure but not yet for assessing actual disease burden.

**Joachim Heinrich** (Germany) presented “**Cats, dogs, and mites in dwellings and respiratory and allergic effects**”. Housing conditions influence pet ownership (strong evidence), and pet ownership influences allergen levels indoors (strong for cat allergen, moderate for dog, poor for mites). Pet ownership is associated with development of atopic disorders, while early exposure to dogs is protective (causal mechanism not understood). Pet-derived allergens (cat) causes specific allergic sensitization (strong evidence) and might consequently cause asthma (not clear). The effects of housing factors on cat and mite allergen levels are small. Other factors such as cat ownership (cat allergen) and climatic factors (Der p 1 allergen) have much stronger influences on allergen levels indoors. The Working Group agreed that there is some evidence on ER relation and exposure. The ECRHS II is the only study on a European level which used identical methods for each country-specific location. Within the ISAAC, indoor dust was collected using a standardized method and is assayed for allergens. These two studies should be used as the main reference in determining the feasibility of making an EBD estimate.



**Susanne Lau** (Germany, presented by Joachim Heinrich) presented **“Pets and mites in dwellings and respiratory, allergic or asthmatic effects in children”** and concluded that cat sensitization is a major risk factor for childhood asthma. However, the data on exposure to cats and sensitization were not available, while additional factors such as chemicals etc. are making it increasingly difficult to identify clear ER relationships. The Working Group accepted that evidence on this topic is rather rare and therefore difficult to integrate into the burden of disease work. Also, it was agreed to separate the discussions on pets and mites and deal with them individually.

**Aino Nevalainen** (Finland) presented **“Humidity and mould in dwellings and related health effects”**. Dampness and moisture problems are common in all climates, in all types of buildings (5-80%). Perhaps the strongest evidence exists on the association of dampness with cough, wheeze and on asthma; there is evidence on both onset of new asthma cases and increased asthma symptoms on previously sensitized individuals. The ORs vary between 1.4-2.2. For other health outcomes, the ORs vary remarkably from study to study and from symptom to symptom; approximately between 1.1-4.6. The literature has been carefully reviewed by several working groups during the recent years. Their conclusions are very similar: the evidence of the findings on cough, wheeze and asthma is strong. However, it is generally agreed that the evidence on many other health outcomes (see above) is still suggestive or insufficient. The problems with the exposure assessment of damp buildings are mainly attributed to methodological issues. Methods that adequately quantify microbial material are only now being validated. Working Group agreed that there is sufficient evidence for ER relation, and some evidence on exposure data. It was also decided to change the title of this topic to “Mould and dampness”.

#### **SESSION 4: Building & Equipment factors**

**David Ormandy** (UK) presented **“Building and equipment factors and unintentional injuries / domestic accidents”** and reviewed two large studies linking dwelling factors and domestic accidents. In England, each year, on average, housing conditions are implicated in up to 50,000 deaths; and round 0.5 million injuries and illnesses requiring medical attention (HHSRS 2000). There is a question whether and how we integrate the behavioral factors with the dwelling factors per se. Because most injury data are available in relation to the place of injury, Working Group agreed that this linkage has sufficient evidence for ER relation and exposure data.

**Mathilde Sengölge** (Austria) presented **“The European Injury Database (EU IDB)”** and showed the European Injury Database evidence on housing threats in unintentional domestic injuries. The Injury Database is a unique data source in the EU about nonfatal home and leisure injuries based on data collected at accident & emergency departments, presently in 10 countries in the EU-15. Injuries account for 9% of deaths and 12% of the burden of disease worldwide. In the EU 51% of injuries occur in the home and leisure place. The EU IDB shows that the top injury location within the home is the living room/bedroom with the highest risk group being children 0 to 4 years old, followed by persons above 80 years of age. The EU IDB can be a tool for the monitoring of home injuries in EU as well as a tool for investigating in-depth questions about home injuries (place, activity, body part injured, type

of injury, product involved or causing the injury, etc), but no information is collected on home design. The EU IDB is an excellent evidence base for the development of injury prevention measures in housing safety in EU. The Working Group agreed that there is sufficient evidence for this linkage to quantify the burden of disease using EU IDB.

**Dinesh Sethi** (WHO Rome office) presented “**Estimating the number of home accidents from literature and data sources**” and suggested an alternative way to quantify the health burden of home injuries, or to extend and scrutinize its accuracy. Specifically, drawing from the few identified surveys from US, UK and The Netherlands, it could be possible to extract reliable data on the various home accident types if available data does not specify this. However, the major limitation seems to be the direct link to the health outcome, which is best documented for mortality but much less for morbidity, leading to a strong underestimation of health effects. The Working Group agreed that in any case, additional data from literature and case studies will be needed to validate the evidence for this linkage.

**Thomas Kistemann** (WHO-CC, Germany) presented “**Sanitation and hygiene conditions in dwellings and related physical health effects**” and focused on the quality of water supply, as sufficient data on the quality and direct health effects of inadequate sanitation equipment are not available. Looking into water quality, populations are relevantly exposed to water-related causes of disease at their homes but despite the fact that the health outcomes may be severe, data to calculate a burden of disease is rarely available (especially linking exposure and health effect often proves difficult). The key issue for the quantification would be whether exposure data are available, as dose-response relationships between exposure and outcomes are partly well established. The Working Group agreed that this linkage should be examined in more detail, trying to identify the availability of exposure data and the opportunities to apply established ER relationships for selected health outcomes.

## **SESSION 5: Social conditions of housing and mental effects**

**Edmond D. Shenassa** (USA) reported about “**Social conditions of housing and fear / fear of crime**”. He provided the theoretical background about this relationship and provided an overview of European as well as non-European studies from which data could possibly be drawn. Potential linkages to health outcomes could be identified for the amount of physical exercise which is associated with the perceived safety of the residential area. The Working Group decided to keep the issue of fear on the agenda, trying to identify other, more direct health endpoints than physical activity (such as depression, etc.).

**Philippa Howden-Chapman** (New Zealand) provided expertise on the area of “**Poverty and social exclusion and related health effects**”. She listed housing quality, housing tenure / social housing, energy issues and crowding as some of the most relevant poverty-related housing effects on health, but cautioned that in many cases there is no exposure data on individual level. Best evidence as required for the burden of disease approach could be available in the area of crowding and thermal comfort / energy efficiency aspects, for which linkages to health effects may exist. The Working Group therefore agreed to look especially into these recommended areas.

**James Dunn** (Canada) discussed “**Crowding (by area or by rooms) and related health effects**”. He provided evidence that the exposure to crowding is known for many European countries. The challenge lies within the linkage with crowding-related health effects, some of which are available (self-reported health, anxiety etc.) but need to be adapted to the burden of disease approach. He also mentioned that the issue of crowding is strongly linked to the issue of affordability, making a large part of the crowding problem a socio-economic problem and therefore mostly affecting vulnerable populations, which should possibly be considered during the quantification. The Working Group agreed that more time should be spent on identifying recent projects from which a more clear association between crowding and health impacts could be drawn.

**Gary Evans** (USA) presented an overview on “**Building and architectural aspects (“hardware”) and related mental health effects**”, looking into the mental effects of multi-family housing, high-rise housing, and housing quality. Various studies do confirm that housing features are associated with mental symptoms, often enabling the identification of an OR for the selected outcomes. Still, the challenge is in clearly defining the mental symptoms in a way that is compatible with the burden of disease methodology (which is based on ICD codes). Self reports of psychological illness will be considered in the evidence review as long as they are valid and reliable. Based on the wealth of existing studies, the Working Group agreed that a more detailed review of such studies could lead to some sufficient results for specific housing features and selected health outcomes.

**Jérôme Fredouille** (France) discussed the relevance of “**Social factors and social climate (“software”) and related mental health effects**”. He identified various issues with relevance to mental effects, such as neighbourhood quality and social capital, but also personal issues such as socio-economic status (SES), unemployment or life events. From the evidence provided, it seemed difficult to identify clear ER relationships attributable to housing or urban conditions. The Working Group agreed that more evidence would be necessary for quantifying this aspect of housing and health, but also doubted that sufficient data in the required format would be available.

## **SESSION 6: Exposure models and housing risk profiles**

**Nathalie Tchilian** (France) then went from the discussion of existing evidence to the tools for collecting evidence, and discussed the French approach towards “**Indoor air quality exposure assessments and the estimation of health effects**”. She presented the French Observatory on Indoor Air Quality (IAQ) as a part of the National Environment and Health Action Plan and introduced the structure of the observatory, measuring IAQ and exposure conditions at the national level. Mrs. Tchilian showed that with the project focusing on exposure conditions, it could provide helpful data on exposure but it would not help identify or strengthen the evidence between indoor air pollutants and health effects as required for the burden of disease approach. Therefore, it will be necessary to identify other national projects which can provide data on health effects in addition to exposure.

**Maggie Davidson** (UK) finally gave some ideas on the challenge of comparing and extrapolating data on housing from the European perspective and discussed “**European**

**housing typologies and specific health risk profiles of housing types”**. She showed the large number of problems that affect data collection and the comparability of statistics on international level, and recommended the use of various data sources to limit the likelihood of mistakes. Further, she listed the publicly available data sources on dwelling conditions, and provided a first impression of the variability of housing stock data both on the national and international level. She concluded that there is very little reliable and consistent housing quality information on the international level and thereby brought up the issue of modelling as an useful means for quantifying and assessing housing problems.

## **SESSION 7: Assessment of data validity and evidence base**

The last session focused on a review of the presentations and data collections that had been compiled for the meeting in order to provide a first rough assessment of the data at hand.

**Mounir Mesbah** (France) provided some **“Comments on the statistical validity of the presented data and recommendations for further work”**. He made it very clear that after seeing the presentations and the collected data, the key issue for success would much less be the statistical validity but the causal link between housing conditions, exposure, and specific health effects. He reminded the meeting participants of the essential causality criteria when selecting the studies for the burden of disease project.

During the group discussion, there was consensus that for the burden of disease of housing, it was necessary to identify the strong and well-documented ER relationships in order to accomplish the project successfully. Special attention would need to be paid towards the “attributable fraction” that can really be related to housing conditions, meaning that improvement of a specific housing characteristic would lead to a measurable decrease of a specific health effect.

Finally, **Rokho Kim** (WHO) attempted a first crude overview of the presented data and suggested a **“WHO summary and preliminary evaluation of evidence-based overviews”**. He summarized the results of the expert assessments in three categories: sufficient evidence, some evidence, and insufficient evidence.

Thirteen housing-health relationships were selected as those with sufficient evidence. Two linkages, “Lighting conditions in the dwelling and mental and other health effects” and “Particulate matters in indoor air and respiratory and allergic effects,” were initially presented as having insufficient evidence. However, after a plenary discussion, the participants agreed that these two linkages should be reserved for more extensive review. Ten linkages were considered having some evidence for quantifying burden of disease.

Details for the first assessment of available evidence can be found in Table 1 in this report.

The crude and preliminary assessment led to intense debate and it became clear that – this meeting being the very first – there was still much effort needed to provide the required solid base of evidence that is needed to make an informed decision on whether a housing-health relationship is sufficiently documented for matching the burden of disease requirements.

Still, the presentations showed that the assessment of the burden of disease due to inadequate housing is a realistic task, and there was agreement within the Working Group that in various cases where some evidence exists that does not comply with the data requirements, footnotes or annexes could provide additional information on housing and health issues that cannot yet be fully quantified. This was seen as especially relevant for health outcomes often used in housing studies which cannot be applied for the burden of disease, such as self-reported health or undefined symptoms.

## **SESSION 8: Conclusion, challenges and the way forward**

### ***Conclusion***

In general terms, the meeting participants agreed that with the currently available data a first numeric quantification of the burden of disease of inadequate housing is feasible. Also, the group agreed that the burden of disease of inadequate housing – considering the variety of housing factors with relevance to health – may be rather large. Still, it was felt that future studies are required to enhance the knowledge base and thereby allow more detailed and accurate estimates.

### ***Challenges***

The major challenges that were identified by the experts in preparing the summary documents for the meeting were mostly related to

- general lack of studies on specific housing topics, or a lack of high-quality studies with reliable results
- the use of unspecified and self-reported health outcomes as evidence base for housing burden of disease which may not be compatible with the EBD methodology
- difficulty in deriving exposure-response relationships
- missing information on specific exposure situations of the population at national or European level

For various housing topics, it will be challenging to provide sufficient data on the effect of and the exposure to inadequate housing conditions. While for some aspects, European assessments may be feasible, other aspects will only allow for local or national assessments due to limited data availability. Therefore, one key challenge of the EBD project for housing inadequacy will be the methodological approach on how to quantify the EBD internationally securing comparability. Specific attention will also have to be paid to the selection of studies used for the quantification.

### ***Way forward***

The final discussion focused on the way forward for calculating the burden of disease. Xavier Bonnefoy (WHO) stated that there are two ways for the assessment to be done: at national level, or at the European level. As WHO was interested in an international approach, it was suggested that a network of experts be formed to collect the necessary data and provide the best-possible assessment of the currently existing evidence on the documented housing-health relationships drawing from international studies. Reflecting the different

sessions, five Working Groups were formed to focus on the chemical, physical, biological, social and building-related aspects of housing inadequacy, respectively. Within each Working Group, a volunteer agreed to facilitate the coordination of future work within the group in liaison with the WHO secretariat. These volunteers will be called “Working Group facilitators” and will be provided with a specific terms of reference. The Working Groups agreed to provide an in-depth assessment of the selected housing-health relationships for their next meeting in 2006.

Following the meeting, WHO committed to functioning as the secretariat for this project, coordinating all work to be done and providing the overall methodological guidance, while the responsibility for making scientific decisions for a specific topic would be in the hands of the individual experts and the Working Groups.

It was agreed that the task of each Working Group was to:

- (a) validate and enhance the evidence base for the housing and health ER relationships that fulfill the requirements of the burden of disease methodology
- (b) revisit and extend the evidence base for those ER relationships which were insufficient but promising at the current stage
- (c) identify potential housing-health relationships with sufficiently documented evidence that may not have been covered by the meeting
- (d) confirm and justify the deletion of the topics with insufficient evidence base for housing-health relationships which are not yet documented well enough for the burden of disease.

It was agreed that the identification of the current status of knowledge was the key issue, and that it must be done in a structured and conservative way. WHO asked all present experts and institutions for their support in moving ahead on this project despite the technical and methodological challenges.

Finally, Xavier Bonnefoy thanked the participating experts and institutions – on behalf of WHO and the housing and health team – for their support and expressed his hope for a fruitful and efficient collaboration on the quantification of the health effects of housing inadequacy in the near future.

## 5. FOLLOW-UP ACTIONS AND COMMITMENTS

WHO will, in its role as the secretariat for the project, provide the following guidance to the experts:

- List of key documents on the burden of disease methodology and the identification of epidemiological evidence for environmental health risk assessment
- A general and specific description of responsibilities and working relationship of WHO secretariat, the Working Group facilitators, and the individual experts.

WHO will also commit to identifying experts on housing topics not yet covered by this meeting. The following issues have been considered for addition so far, and the international experts on these topics are being invited to participate in this project. Drs Stefanos Kales (USA) and Nicolas Gilbert (Canada) agreed to contribute to the topics of CO, NO<sub>2</sub>, and Formaldehyde in the project.

<b>Housing factor</b>	<b>Health effect</b>
CO, NO <sub>2</sub> , Formaldehyde	Respiratory effects and poisoning
Pesticides and chemical household products	Allergies, respiratory effects, poisoning
Cockroaches	Allergic effects
Accessibility	Accidents and social effects
Home fires / fire detectors	Fire-related injuries and mortality
Sanitation and hygiene equipment	Infectious diseases, diarrhoea, etc.

The inputs from experts received after the meeting which have not been incorporated in this report will be discussed with the group before, during and after an informal meeting with the attendants at the Healthy Building 2006 in June in Lisbon. WHO will convene the expert group, including new experts covering additional topics, for the second meeting with the support of the German Ministry of Environment. *The proposed date for the second meeting is 15-17 November 2006.*

### *Annex 1 List of participants*

Mr Stephen Battersby  
Chartered Environmental Health Practitioner and Environmental Health and Housing Consultant  
353 Hillcross avenue  
Morden, Surrey SM4 4BX  
UK  
Telephone + 44 208 286 6484  
Fax + 44 208 286 6484  
Email [sbattersby@blueyonder.co.uk](mailto:sbattersby@blueyonder.co.uk)

Dr Mary Jean Brown  
Chief, Lead Poisoning Prevention Branch  
Centers for Disease Control and Prevention  
4770 Buford Highway NE MS F-40  
Atlanta, GA 30341  
USA  
Telephone No. +1 770-488-7492 2  
Fax No +1 770-488-3635  
Email: [MJB5@cdc.gov](mailto:MJB5@cdc.gov)

Dr Olivier Catelinois  
IRSN, Laboratoire d'épidémiologie  
des rayonnements ionisants  
BP 17  
92262 Fontenay aux Roses Cedex  
France  
Telephone No +33 (0) 1 58 35 71 94  
Email [olivier.catelinois@irsn.fr](mailto:olivier.catelinois@irsn.fr)

Dr David Crowther  
The Martin Center, University of Cambridge  
6 Chaucer Road  
Cambridge CB2 2EB  
United Kingdom  
Telephone No +44 1223 331713  
Fax No +44 1223 331701  
Email [drgc2@cam.ac.uk](mailto:drgc2@cam.ac.uk)

Dr Maggie Davidson  
Building Research Establishment  
Garson  
Watford WD25 9XX  
United Kingdom  
Telephone No +44 (1923) 664 410  
Fax No +44 1923 664201  
Email [davidsonm@bre.co.uk](mailto:davidsonm@bre.co.uk)

Dr James Dunn  
Centre for Research on Inner City Health, St. Michael's Hospital  
Assistant Professor, Geography, University of Toronto  
70 Richmond St. E., 4th floor  
Toronto, ON M5C 1N8  
Canada  
Telephone No +1-416 864-5486



Fax No +1-416 864-5485  
Email [jim.dunn@utoronto.ca](mailto:jim.dunn@utoronto.ca)

Prof Gary Evans  
E306 Martha van Rensselaer Hall  
Cornell University  
Ithaca NY 14853  
USA  
Telephone No +1 (607) 255 4775  
Fax No +1 (607) 255 0305  
Email [gwe1@cornell.edu](mailto:gwe1@cornell.edu)

Dr Jérôme Fredouille  
Médecin Psychiatre  
Hôpital du Vinatier  
95 Bld Pinel  
69 677 BRON cedex  
France  
Telephone No +33 437 91 52 90  
Email [jerome.fredouille@ch-le-vinatier.fr](mailto:jerome.fredouille@ch-le-vinatier.fr)

Mr Giulio Gallo  
DG SANCO C4  
JMO C5/90 Rue Alcide de Gaspari  
2920 Luxembourg  
Telephone +352 4301 36642  
Email : [giulio.gallo@cec.eu.int](mailto:giulio.gallo@cec.eu.int)

Dr Jonathan Healy  
Policy and Research Analyst  
Combat Poverty Agency  
Bridgewater Centre, Conyngham Road, Islandbridge  
Dublin 8  
Ireland  
Telephone No +353-1-602 6612  
Fax No +353-1-670 6760  
Email [healyj@cpa.ie](mailto:healyj@cpa.ie)

Dr Joachim Heinrich  
Institut für Epidemiologie  
GSF-Forschungszentrum für Umwelt und Gesundheit, GmbH  
Ingolstädter Landstraße 1  
D-85764 Neuherberg  
Germany  
Telephone No +49-89 3187-4150  
Fax No +49-89 3187-3380  
Email [joachim.heinrich@gsf.de](mailto:joachim.heinrich@gsf.de)

Prof. Philippa Howden-Chapman  
Wellington School of Medicine and Health Sciences  
PO Box 7343  
6000 Wellington  
New Zealand  
Email: [howdenc@wnmeds.ac.nz](mailto:howdenc@wnmeds.ac.nz)

Mrs Emma Hutchinson

London School of Hygiene & Tropical Medicine  
Public & Environmental Health Research Unit  
Keppel Street  
London WC1E 7HT  
United Kingdom  
Telephone No. +44 20 7927 2007  
Fax No +44 20 7580 4524  
Email [Emma.Hutchinson@lshtm.ac.uk](mailto:Emma.Hutchinson@lshtm.ac.uk)

David E. Jacobs, PhD, CIH  
U.S. National Center for Healthy Housing  
10227 Wincopin Circle, Suite 200  
Columbia, MD 21044  
USA  
Telephone No: + 1 202-607-0938  
Fax: No +1 202-237-2875 (call first)  
Email: [dejacobs@starpower.net](mailto:dejacobs@starpower.net)

Dr Thomas Kistemann  
WHO CC Health Promoting Water Management and Risk Communication  
University of Bonn  
Sigmund-Freud-Straße 25  
53105 Bonn  
Germany  
Telephone No +49 (228) 287 55 34  
Fax No +49 (228) 287 9516  
Email: [thomas.kistemann@ukb.uni-bonn.de](mailto:thomas.kistemann@ukb.uni-bonn.de)

Dr Bernhard Link  
Regierungspräsidium Stuttgart, Landesgesundheitsamt  
Wiederholdstrasse 15  
70174 Stuttgart  
Germany  
Telephone No +49 0711/1849 309  
Fax No +49 0711/ 1849 242 or 369  
Email [bernhard.link@rps.bwl.de](mailto:bernhard.link@rps.bwl.de)

Mrs Monika Luxem  
Referat IG II 2  
Umwelteinwirkungen auf die menschliche Gesundheit  
Robert-Schuman-Platz 3  
53175 Bonn  
Germany  
Telephone No+49-228-305 2722  
Fax No +49-228-305 3524  
Email: [monika.luxem@bmu.bund.de](mailto:monika.luxem@bmu.bund.de)

Prof Ian Matthews  
Department of Epidemiology, Statistics and Public Health  
Cardiff University  
Heath Park  
Cardiff CF14 4XN  
United Kingdom  
Telephone No +44-29 20 742324  
Fax No+44-29 20 742898  
Email [matthewsip@Cardiff.ac.uk](mailto:matthewsip@Cardiff.ac.uk)

Prof Mounir Mesbah  
Université Pierre Et Marie Curie (UPMC)  
Laboratoire de Statistique Theorique et Appliquée (LSTA)  
175 rue du Chevaleret, Boîte 158, - Bureau 8A1 - Plateau A  
75013 Paris  
France  
Telephone No. : +33 1 44 27 33 51  
Fax No. : +33 1 44 27 33 42  
Email: [mesbah@ccr.jussieu.fr](mailto:mesbah@ccr.jussieu.fr)

Dr Aino Nevalainen  
National Public Health Institute, Department of Environmental Health  
Neulaniementie 4  
PO Box 95  
70701 Kuopio  
Finland  
Telephone No +358 17 201 342  
Email [Aino.Nevalainen@ktl.fi](mailto:Aino.Nevalainen@ktl.fi)

Dr Hildegard Niemann  
Berliner Zentrum Public Health  
Ernst-Reuter-Platz 7  
10587 Berlin  
Germany  
Telephone No +49 30314 28 995  
Fax No +49 30314 29 269  
Email: [hildegard.niemann@tu-berlin.de](mailto:hildegard.niemann@tu-berlin.de)

Prof David Ormandy  
School of Law  
University of Warwick  
Coventry CV4 7AL  
United Kingdom  
Telephone No +44 24 7652 4936  
Fax No +44 24 7652 4105  
Email [david.ormandy@warwick.ac.uk](mailto:david.ormandy@warwick.ac.uk)

Mrs Rita Pazdrazdyte  
Ministry of Health  
Vilnius str. 33  
2001 Vilnius  
Lithuania  
Tel. No.: +370 (5) 266 14 63  
Fax No.: +370 (5) 273 73 97  
E-mail: [rita.pazdrazdyte@sam.lt](mailto:rita.pazdrazdyte@sam.lt)

Dr Manfred H.J. Schmitz  
Head of Division „International Affairs“  
Ministry of Labour, Health and Social Affairs  
of the State of North Rhine-Westphalia  
Fürstenwall 25  
40219 Düsseldorf,  
Germany  
Telephone No +49 211 855-3317  
Email [manfred.schmitz@mags.nrw.de](mailto:manfred.schmitz@mags.nrw.de)

Ms Mathilde Sengölge  
Kuratorium für Verkehrssicherheit  
Schleiergasse 18  
A- 1100 Vienna  
Austria  
Telephone No + 43 (0) 577 077 1341  
Fax No + 43 (0) 577 077 1399  
Email [Mathilde.Sengoelge@kfv.at](mailto:Mathilde.Sengoelge@kfv.at)

Prof. Edmond D. Shenassa  
Brown Medical School  
Centers for Behavioral and Preventive Medicine  
Coro Building, Suite 500, One Hoppin Street  
Providence, Rhode Island 02903  
USA  
Telephone No +1 (401) 793-8120  
Fax No +1 (401) 793-8059  
Email [Edmond\\_Shenassa@Brown.edu](mailto:Edmond_Shenassa@Brown.edu)

Mrs Simone Strecker  
Bundesministerium für Gesundheit und Soziale Sicherung  
Am Propsthof 78a  
53121 Bonn  
Germany  
Tel +49 (1888) -441-3281  
Fax +49 (1888) 441 4888  
Email [simone.strecker@bmgs.bund.de](mailto:simone.strecker@bmgs.bund.de)

Prof. Jan Sundell  
International Centre for Indoor Environment and Energy  
Technical University of Denmark, Building 402  
2800 Lyngby  
Denmark  
Telephone No +45 45 25 40 27  
Fax No +45 45 93 21 66  
Email [jas@mek.dtu.dk](mailto:jas@mek.dtu.dk)

Mrs Nathalie Tchilian  
Direction générale de la santé  
8, avenue de Ségur  
75350 Paris 07 SP  
France  
Telephone No +33 1 40 5665 02  
Fax No + 33 1 140565056  
Email [nathalie.tchilian@sante.gouv.fr](mailto:nathalie.tchilian@sante.gouv.fr)

Dr Oliver Thommen  
Institut für Sozial- und Präventivmedizin  
Universität Basel, Medizinische Fakultät  
Steinengraben 49  
4051 Basel  
Switzerland  
Telephone No +41 (0) 61 270 22 14  
Email [oliver.thommen@unibas.ch](mailto:oliver.thommen@unibas.ch)

Dr Hilary Thomson  
MRC Social and Public Health Sciences Unit

4 Lilybank Gardens  
Glasgow G12 8RZ  
Scotland  
United Kingdom  
Telephone No +44 141-  
Email [hilary@msoc.mrc.gla.ac.uk](mailto:hilary@msoc.mrc.gla.ac.uk)

Mrs Claudia Weigert  
Ministry of Health, Public Health Department  
Alameda D. Afonso Henriques, 45  
1049-05 Lisboa Codex  
Portugal  
Telephone +351 (2184) 30701  
Fax +351 (2184) 30600  
Email [claudiaweigert@dgsaude.min-saude.pt](mailto:claudiaweigert@dgsaude.min-saude.pt)

## **WORLD HEALTH ORGANIZATION, REGIONAL OFFICE FOR EUROPE**

Dr Dinesh Sethi  
Via Francesco Crispi 10  
00187 Roma  
Italy  
Tel. +39 -06 – 4877526  
E-mail: [din@ecr.euro.who.int](mailto:din@ecr.euro.who.int)

Mr Xavier Bonnefoy  
Regional Adviser  
Görresstr. 15  
D-53113 Bonn  
Germany  
Telephone No +49 2282094 415  
Fax No +49 228 2094 201  
Email: [xbo@ecehbonn.euro.who.int](mailto:xbo@ecehbonn.euro.who.int)

Mr Matthias Braubach  
Technical Officer Housing and Health  
Görresstr. 15  
D-53113 Bonn  
Germany  
Telephone No +49 2282094 410  
Fax No +49 228 2094 201  
Email: [mbr@ecehbonn.euro.who.int](mailto:mbr@ecehbonn.euro.who.int)

Dr Rokho Kim  
Housing and Health Scientist  
Görresstr. 15  
D-53113 Bonn  
Germany  
Telephone No +49 2282094 432  
Fax No +49 228 2094 201  
Email [rki@ecehbonn.euro.who.int](mailto:rki@ecehbonn.euro.who.int)

Dr Nathalie Röbbel  
Technical Officer Housing and Health  
Görresstr. 15

EUR/00/50  
page 28

D-53113 Bonn  
Germany  
Telephone No +49 2282094 414  
Fax No +49 228 2094 201  
Email: nat@ecehbonn.euro.who.int

***Secretariat:***

Mrs Nuria Aznar Marti  
Programme Assistant Noise, Housing and Health  
Görresstr. 15  
D-53113 Bonn  
Germany  
Telephone No +49 2282094 415  
Fax No +49 228 2094 201  
Email: naz@ecehbonn.euro.who.int

## *Annex 2 List of presentations*

### **SESSION 1: PHYSICAL FACTORS**

Oliver Thommen

“Heat in dwellings and related cardiovascular effects and/or excess mortality”

Jonathan Healy

“Cold indoor temperatures and winter excess mortality (possibly circulatory and respiratory effects)”

Emma Hutchinson

“Cold indoor temperatures, health effects, and their attributable fraction to housing”

Olivier Catelinois

“Radon exposure in dwellings and cancer”

Hildegard Niemann

“Neighbourhood and building noise and related health effects”

Mary Jean Brown

“Lighting conditions in the dwelling and mental and other health effects”

Ian Matthews

“Ventilation in the dwelling and respiratory and allergic effects”

Jan Sundell

“Particulate Matter & fine particles in indoor air and respiratory and allergic effects”

### **SESSION 2: CHEMICAL HOUSING CONDITIONS**

Bernhard Link

“VOC and chemical emissions in dwellings and respiratory, cardiovascular and allergic effects”

Maritta Jaakkola

“ETS exposure in dwellings and respiratory and allergic effects”

Irene van Kamp

“Chemical products and pesticides and related health effects”

David Jacobs

“Lead in housing and lead poisoning”

### **SESSION 3: BIOLOGICAL HOUSING CONDITIONS**

Stephen Battersby

“Cockroaches and rodents in dwellings and respiratory and allergic effects”

David Crowther

“Hygrothermal dwelling conditions and house dust mite exposure”

Joachim Heinrich

“Cats, dogs, and mites in dwellings and respiratory and allergic effects”

Susanne Lau (presented by Joachim Heinrich)

“Pets and mites in dwellings and respiratory, allergic or asthmatic effects in children”

Aino Nevalainen

“Humidity and mould in dwellings and related health effects”

#### **SESSION 4: BUILDING & EQUIPMENT FACTORS**

David Ormandy

“Building and equipment factors and unintentional injuries / domestic accidents”

Mathilde Sengölge

“Injury Database evidence on housing threats and unintentional injuries / domestic accidents”

Dinesh Sethi

“Estimating the number of home accidents from literature and data sources”

Thomas Kistemann

“Sanitation and hygiene conditions in dwellings and related physical health effects”

#### **SESSION 5: SOCIAL CONDITIONS OF HOUSING AND MENTAL EFFECTS**

Edmond D. Shenassa

“Social conditions of housing and fear / fear of crime”

Philippa Howden-Chapman

“Poverty and social exclusion and related health effects”

James Dunn

“Crowding (by sqm or by rooms) and related health effects”

Gary Evans

“Building / architectural aspects (“hardware”) and related mental health effects”

Jérôme Fredouille

“Social factors / social climate (“software”) and related mental health effects”



**SESSION 6: EXPOSURE MODELS AND HOUSING RISK PROFILES**

Nathalie Tchilian

“Indoor air quality exposure assessments and the estimation of health effects”

Maggie Davidson

“European housing typologies and specific health risk profiles of housing types”

**SESSION 7: ASSESSMENT OF DATA VALIDITY AND EVIDENCE BASE**

Mounir Mesbah

“Comments on the statistical validity of the presented data and recommendations for further work”

Rokho Kim

“WHO summary and preliminary evaluation of evidence-based overviews”

*Annex 3 Background documents distributed at the workshop*

**(by alphabetic order of the contributor)**

<b>Housing factor</b>	<b>Health effect</b>	<b>Contributor</b>
Rodents and pests	Diseases and stress	Battersby, UK
Inadequate Residential Light	Depression	Brown, USA
Radon	Lung cancer mortality	Catelinois, France
House Dust Mites	Asthma and allergies	Crowther, UK
Social Factors and Crowding	General health status	Dunn, Canada
Housing conditions	Mental Health	Evans, USA
Cold (indoor) temperatures	Excess winter mortality / seasonality; cold-related mortality (cardiovascular / respiratory deaths)	Healy, Ireland
Cats, dogs, mites	Respiratory and allergic effects	Heinrich, Germany
Poverty and housing deprivation	General health effects	Howden-Chapman, New Zealand
Environmental tobacco smoke	Respiratory diseases and pregnancy outcomes	Jaakkola, Finland / UK
Lead	Reduced IQ, Cognitive Deficits, Adverse Neurobehaviorial Effects, Increased Hypertension, Probable Human Carcinogen, Criminality and others	Jacobs, USA
VOC and chemical emissions in dwellings	Respiratory and allergic effects	Link, Germany
Ventilation in the dwelling	Respiratory and allergic effects	Matthews, UK
Dampness and mould	Respiratory symptoms, asthma	Nevalainen, Finland
Neighbourhood noise	Unspecific health effects	Niemann, Germany
Home design and low-standard housing	Falls and general home injuries	Sengölge, Austria
Fear of crime and housing	Mental health effects	Shenassa, USA
Particulate Matter and fine particles in indoor air	Respiratory and allergic effects	Sundell, Denmark
Heat in the dwelling	Cardiovascular effects/ excess mortality	Thommen, Switzerland
Housing improvements	Physical and mental health effects	Thomson, UK

## Summary of main element of evidence between:

**Cause/ Housing Factor: Rodent pests**  
**Health Effects: diseases and stress**

Contributor: Stephen Battersby, UK

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Warning: The contents of this paper are a very brief and limited summary. The work involved in preparing a more detailed assessment was not available and there may be significant differences between the health impacts of mice and rats in and around housing.

### Part I Health Effects/attribution risk/possible exposure-response relationship

#### 1) Data Sources Used

Authors	Year	Title	Journal/book	Location
Battersby SA, Parsons R, Webster JP	2002	Urban rat infestations and the risk to public health	Journal of Environmental Health Research	London
Cohn, RD, Arbes, SJ, Yin, M, Jaramillo, R,	2004	National prevalence and exposure risk for mouse allergen in US households	Journal of Allergy & Clinical Immunology	Milwaukee USA
Community Reference Laboratory on the Epidemiology of Zoonoses,	2005	Trends and sources of zoonotic agents in the European Union and Norway in 2003,	Report: European Commission Health & Consumer Protection Directorate-General SANCO/339/2005	Berlin
Hirschorn, R,	2005	Public Health, Public Health Systems and Urban Pests	WHO Urban Pests and Health Project, Experts Meeting,	Bonn, Germany
Perry, T, Matsui, E, Merriman, B,	2003	The prevalence of rat allergen in inner-city homes and its relationship to sensitisation and asthma morbidity	Journal of Allergy and Clinical Immunology	Milwaukee USA

#### 2) General evidence

There is evidence that rats (*Rattus norvegicus*) and to some extent house mice (*Mus musculus* (*M. domesticus*)) are infected with parasites, and are a reservoir of infection beyond those historically associated with rodents in Europe (e.g. *Leptospira spp.*) However there is relatively little direct evidence to demonstrate any exposure-response relationship. Indeed until recently there had been little work on the parasites infecting rodents.

#### 3) Specific evidence

Little specific evidence as most evidence of rodents and disease has been gained from occupational rather than domestic exposure. Carrer *et al.* (2001) suggested that the presence of rodents in the home might contribute to increased levels of indoor allergens, causing

allergic asthma and rhinoconjunctivitis. Cohn et al (2004) reported that exposure to mouse allergen causes asthma in occupational settings and exhibits high prevalence and association with allergic sensitisation in inner-city homes. It was reported that household mouse allergen is widespread in many settings in the USA at levels that might contribute to asthma morbidity. Perry et al (2003) have also reported that rat allergen sensitisation and exposure are associated with increased asthma morbidity in inner-city children in the USA, with 33% of inner-city homes having detectable rat allergen, compared with 95% of such homes having mouse allergen.

Hirschorn (2005) in the study of the incidence of rat bites over a 22-year period in Philadelphia did not report on the incidence of rat-bite fever (*Streptobacillary* or Haverhill fever) caused by infection with *Streptobacillus moniliformis*. However such rat bites will have caused stress and emotional upset.

#### 4) Limitations

Many of the parasitic diseases are not notifiable in humans. For example Yersiniosis is only notifiable in Norway in Sweden. Laboratory-based systems exist in Belgium Spain and the UK (where it is notifiable only if thought to be the cause of 'food poisoning'), but in the Netherlands the surveillance system stopped in 1997 (Community Reference Laboratory, 2005). Worm infections are not notifiable at all, and it unlikely that clinicians will consider exposure to rodents when considering causes of any diseases or symptoms presented to them.

The results of a survey in the UK indicated a high level of ignorance of rat borne infections on the part of Directors of Public Health (Battersby et al 2002). Investigations of diseases have either been of limited extent, or rats have not been seen as important vectors of disease. It is likely that investigations of disease do not assess whether there has been any possible exposure to rats. The situation may be little different in other modern developed countries). Rats may represent reservoirs of infection but only diseases known by practitioners to be specifically associated with rats might prompt any further investigation. This is likely to be undertaken by the local authority, and it was shown that there is minimal liaison between the DPH and the environmental health practitioner charged with ensuring effective rodent control in the local authority.

#### 5) Suggested incidence for exposed population

It is not possible to suggest an incidence for the exposed population, due to rodent infestations in and around homes. This is the result of inadequate surveillance or any specific studies relating the health of communities to their exposure to domestic rodent infestations. This would in any event be a difficult exercise.

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#### Final recommendation

As the result of inadequate surveillance there is no sufficient and reliable evidence for making a valid assessment for the total disease burden as the result of exposure to rodents in and around the home.

### **Part II Exposure situation**

6) Data sources used

<b>Authors</b>	<b>Year</b>	<b>Title</b>	<b>Journal/book</b>	<b>Location</b>
Carrer, P., Maroni, M., Alcini, D & Cavallo, D	2001	Allergens in indoor air environmental assessment and health effects	The science of the total environment	Amsterdam
Colvin B,	2001	Opinion: Interview, The rat catcher	New Scientist,	London
Ceruti, R, Sonzogni, O, Origgi, F, Vezzoli, F, Cammarata, S, Giusti, AM, Scanziani, E	2001	<i>Capillaria hepatica</i> infection in wild brown rats ( <i>Rattus norvegicus</i> ) from the urban area of Milan, Italy	Journal of Veterinary Medicine Series B	Oxford
Central Science Laboratory, York	2005	Rodent infestations in domestic properties in England, 2001 - A report arising from the 2001 English House Condition Survey	Report prepared for European Wildlife Division, Defra	London
Battersby SA, Parsons R, Webster JP	2002	Urban rat infestations and the risk to public health	Journal of Environmental Health Research	London
Hirschorn, R,	2005	Public Health, Public Health Systems and Urban Pests	WHO Urban Pests and Health Project, Experts Meeting,	Bonn, Germany
Langton, SD, Cowan, DP, Meyer, AN,	2001,	The occurrence of commensal rodents in dwellings as revealed by the 1996 English House Condition Survey	Journal of Applied Ecology	London
Murphy R. G. Oldbury D. J.	2002	Rat control by local authorities within the UK.	Proceedings of the fourth International Conference on Urban Pests	Charleston, South Carolina, USA
Murphy, RG, & Marshall, PA	2003	House conditions and the likelihood of domestic rodent infestations in an inner city area of Manchester	Conference Proceedings, <i>Healthy Housing: promoting good health</i> , University of Warwick	Coventry
Webster, JP, MacDonald DW	1995	Parasites of wild brown rats ( <i>Rattus norvegicus</i> ) on UK Farms	Parasitology	Cambridge
Williams RH, Murphy RG, Hughes, JM & Hide G	2005	The Urban Mouse ( <i>Mus domesticus</i> ) and its role in the transmission of <i>Toxoplasma gondii</i> infection	ICUP	Singapore

7) General evidence

In the USA the presence of rats in urban areas is taken to be a common indicator of a degraded environment (Colvin 2001). In the USA it has also been found that the more deprived communities in urban areas are less likely to be concerned about the presence of rats, and take less action to counter the presence of rats. Langton *et al.* (2001) found in England that the issue of housing density was more important in urban than rural areas for rat infestations. It seems likely that the higher the density of dwellings, the more likely it is that a nearby dwelling can be a source of infestation. The home range of rats may well encompass more than one dwelling at a time and dispersal by both rats and mice is more likely to be successful over short distances. Rat infestations were found to be significantly more common in older properties and there was a higher frequency in the dwellings falling into the 'unfit' category.

Rodents have been identified as vectors of disease but until recently the parasitic burden of wild brown rats in rural and urban environments has not been examined. Recent studies have aimed to rectify the lack of baseline data on rat-borne infection. A survey of a wide range of parasites of wild brown rats on UK farms (Webster & Macdonald, 1995), found them to be infected with 13 zoonotic species with a range of 2-9 simultaneously per rat, and up to 10 non-zoonotic species. Concerns arise because the incidence of diseases associated with brown rats could return if rodent numbers and population densities become sufficiently high (Battersby et al 2002).

The prevalence of zoonotic species in brown rats in the urban environment has been found generally to be lower than in rural rats. The only species that has been found with significantly higher prevalence levels amongst urban rats was *Trichuris spp* (Battersby et al 2002).

Murphy and Oldbury, (2002) found that domestic mouse infestations were most likely to occur where there was poor structural maintenance, poor hygiene and ample internal harbourage. This species tends to live almost entirely inside buildings, increasing the opportunities for contact with people and poses a potential threat to public health through the diseases it may carry. In a study of an area of Manchester (England) 50% of the terraced properties were found to have mouse infestations (Murphy & Marshall 2002).

#### 8) Specific evidence

In Philadelphia a study examined 662 rat bite victim reports within an urban population over a 22-year span (Hirschorn, 2005). Although numerically a large number, with the population of the city estimated at 4.389 million, this represents 0.007 bites per year per thousand total population. However, it is likely that the incidence of rat bites was restricted to certain parts of the population, and do not occur evenly throughout the population. Rat bite victims tended to be less than one-year old living in sub-standard housing in close proximity to brown rat infestations. It was reported that brown rats are often found in substandard dwellings where the building density is high and the construction type denies rats outdoor burrowing opportunities. Hirschorn (2005) considered that rat bites show the threats to public health not only have a medical, but also a social and emotional dimension.

Dwelling type contributed to the risk of receiving a rat bite. Most bites occurred in the home. For all rat bites reported, 67% occurred in single-family dwellings, 24% in multiple family

dwellings, with children less than one-year old were most likely to be bitten. Socio-economic factors were important in determining the risk of a rat bite. Most victims were living in poverty as defined by the U.S. Population Census.

Carrer *et al.* (2001) suggested that the presence of rodents in the home might contribute to increased levels of indoor allergens, causing allergic asthma and rhinoconjunctivitis

In Milan (Italy) out of a sample of 47 wild brown rats 17 (36%) were found to have liver lesions consistent with *Capillaria hepatica* infection (Ceruti *et al.*, 2001) and it was suggested that the potential transmission of *C. hepatica* to children in the area of Milan covered by that study should be considered an important health issue.

In the area of Manchester referred to above an infection rate of 58.5% was found for *T gondii* in the mice when determined by detection of *T. gondii*-specific DNA. This compares with a prevalence of 35% of *T gondii* in rats on farms identified by Webster & Macdonald (1995)

#### 9) Limitations

All published studies have concentrated on either the level of rodent infestations around homes or on the level and nature of parasitic infection within rodent populations. They have highlighted a relationship between poor housing and environmental conditions and rodent infestations and also the range of zoonotic agents infecting rodents. However there has been little published assessment of the link between rodent infestations and disease, beyond the link with asthma.

#### 10) Suggested exposure rate in the European population

This is difficult to assess and requires further study. The majority of mouse infestations are within dwellings. The average infestation rate of dwellings in England in 2001 for house mice infestations inside dwellings was 1.4% and 0.3% for rats inside, with 2.9% of dwellings having rats outside. The majority of rat infestations are outside the dwelling in the yards and gardens (CSL, 2005). However whatever the average infestation rates, there will be areas of considerably higher infestation rates as reported above. It is not known what the average infestation rates are for other states in the European region.

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#### Final recommendation

There is some reliable evidence for making a valid assessment as to exposure.

**Summary of main element of evidence between:**

**Cause/ Housing Factor: Inadequate Residential Light  
Health Effects: Depression**

Contributor: Mary Jean Brown, USA

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**Part I: Health effects / Attributable risk / possible exposure-response relationship**

**Data sources used**

<b>Author(s)</b>	<b>Year</b>	<b>Title</b>	<b>Journal / Book</b>	<b>Location</b>
Wirz-Justice A	1998	Beginning to see the light	Arch Gen Psychiatry	Vol 55 Pg 861-862.
Rosenthal NE, Sacks DA, Gillin JC, Lewy AJ, Goodwin FK, Davenport Y, Mueller PS, Newsome DA, Wehr TA.	1984	Seasonal affective disorder: A description of the syndrome and preliminary findings with light therapy.	Arch Gen Psychiatry;	Vol. 41 Pg 72-80.
Golden RN, Gaynes BN, Ekstrom RD, Hamer RM, Jacobsen FM, Suppes T, Wisner KL, Nemeroff CB.	2005	The efficacy of light therapy in the treatment of mood disorders: A review and meta-analysis of the evidence	Am J Psychiatry	Vol. 162: Pg 656-662.
Kripe KF. J;.	1998	. Light treatment for nonseasonal depression: speed, efficacy and combined treatment	Affec Disorders	Vol 49 Pg:109-117
Partonen T, Loonqvist J.	<sup>1</sup> 2000	Bright light improves vitality and alleviated distress in healthy people.	J Affec Disorders	Vol 57 Pg 55-61.
NA		Basics of Bright Light Therapy Devices	available at <a href="http://www.lighttherapyproducts.com/sadinfo.html">http://www.lighttherapyproducts.com/sadinfo.html</a> Accessed 9/24/05	
Wirtz-Justice A, Graw P, Krauchi K, Sarrafzadeh A, English J, Arendt J and Sand L.	1996	Natural light treatment of seasonal affective disorder’.	J Affective Disorder	Vol 37 Pg 109-120
<sup>1</sup> Haynes PL, Ancoli-Israel, S and McQuiad J.	2005	Illuminating the impact of habitual behaviors in depression.	Chronobiology International	Vol .22 Pg 279-297.



## 2) General Evidence- Depression

The relationship between lack of light and depression has been well documented and the evidence that light is a potent neurobiological agent seems clear. Light therapy has been used to treat seasonal affective disorder at least since the 1980's when Rosenthal et al found that artificial light was effective in treating the disorder. A consensus has been reached concerning the efficacy of light to treat seasonal depression based on independent studies from various centers around the world. The treatment effect found in these studies averages approximately a decrease of 20-25% in depressive symptoms.

## 3) Specific Evidence-

The effectiveness of bright light to decrease depressive symptoms is not limited to patients with seasonal affective disorder (SAD). Subsequently investigators have found that bright light therapy was also efficacious in the treatment other forms of depression as well. A meta-analysis of 20 randomized, controlled trials of light therapy for mood disorders found a significant decrease in depression severity in patients undergoing bright light treatment. Even among depressed patients receiving standard antidepressant medication, application of bright light resulted in a greater improvement in symptoms for patients receiving both therapies. This suggests that light may be acting through a pathway independent of that affected by pharmacotherapy. In addition application of bright light in light deprived volunteers who were not clinically depressed resulted in increased vitality and decreased depressive symptoms. When exposure to the light was stopped subjects rebounded to baseline levels within two weeks.

The light intensity of 2,500-10,000 lux used during therapy is much brighter than normal indoor light which is usually 300-500 lux, but not as bright as summer sunlight which can be as bright as 100,000 lux. In most studies light was administered through a device such as a light box or visor that confined the light to the participant's visual field.

Few studies have compared artificial with natural light. However, in a study conducted in Switzerland, researchers compared the use of low intensity artificial light defined as 0.5 hours of artificial light at 2,800 lux with 1 hour of outdoor light. Even on overcast winter days the outdoor light in Switzerland reaches a level of >1,000 lux early in the morning and remains that high for at least 6 hours. While the study concluded that outdoor light was more effective than artificial light with outdoor light causing a 50% reduction in depressive symptoms, there also was a statistically significant reduction of 25% in depressive symptoms as measured by doctor administered Hamilton Depressive Rating Score in the group receiving the low dose artificial light although self reports of depressive symptoms did not improve for the group with the low dose artificial light exposure. In another study, depressed patients reportedly were exposed to 40% less moderate light, 100-1,000 lux per day compared to non-depressed controls.

Light therapy results in a rapid decrease in depressive symptoms. Most studies, including those cited above, have demonstrated positive results in as short a time as one week and, in most cases, a return of depressive symptoms once the intervention is stopped. Few researchers have followed participants over long periods of time. However in the few studies

that have followed patients for longer than 1 week, response rates increase with duration of the light intervention.

#### 4) Limitations

The causal pathway for depression is undoubtedly complex as indicated by the independent effects of light and standard antidepressant pharmacotherapy and, in the Haynes study, the mediation of the relationship between light and depression by whether the subjects' daily behavior followed a predictable pattern. To establish the independent effect of inadequate light, housing factors including such things as satisfaction with the dwelling, feelings of safety, comfort and control would have to be statistically controlled. Given the complex causal web, we would expect interaction or mediation between the variables.

#### 5) Suggested exposure rate in the European population

In 1996 unipolar depression was identified as the fourth leading cause of burden from all diseases accounting for some 3.6% of the world's total disability adjusted life years (DALYs) in 1990. Recently these estimates have been revised. It is estimated that by 2000, unipolar depression accounted for approximately 4.5% of DALYs worldwide and in high income countries a much greater burden with approximately 8.9% of DALYs attributed to depression.

#### Final Recommendation

Inadequate light may only account for a small fraction of the disease burden. However, given the magnitude of the problem and the inexpensive nature of the intervention, it bears further investigation

## Part II: Exposure situation

#### 6) Data sources used

Author(s)	Year	Title	Journal / Book	Location
Murray CJL and Lopez A.	1996		Global health statistics: A compendium of incident, prevalence and mortality estimates for over 2000 conditions.	Cambridge: Harvard School of Public Health.
Ustun TB, Ayuso-Mateos JL, Chatterji S, Mathers C and Murray CJL	2004	Global burden of depressive disorders in the year 2000	British Journal of Psychiatry	Vol. 184 Pg. 386-392

#### 7) General Evidence

The effect of inadequate interior light on the risk for doctor diagnosed depression has not been studied but given these intriguing findings it seems worthy of further investigation.

This research would bring together several lines of reasoning. First that light administered as a therapy has been demonstrated to improve depressive symptoms by about 25%. Second that the effects of light on reducing depression increase over time, at least in the few studies that have followed participants over time. Third, that when given as adjunct therapy, the benefits of light therapy and pharmacotherapy were independent.

#### 8) Specific Evidence

In the World Health Organization Eight City study of health and housing the participants with physician depression were more likely to be female, be poor, have only public health insurance, abstain from alcohol, be dissatisfied with their housing, take over the counter medications and were less well educated. They were also more likely to describe their general health as good or very good. There was no difference in the percent of depressed participants who were married compared to those who were not depressed. (See Table 1)

In this study, participants with physician diagnosed depression were also more likely to miss daylight and more likely to turn on the lights during daylight hours. (See Table 2)

#### 9) Limitations

These are very preliminary findings. The results cited above are based on only 1 observational study and require further investigation and corroboration. At this time we have examined the relationship of only one health outcome and adequacy of residential light. It seems likely that there are other health outcomes, notably falls that also are directly related to the adequacy of interior light. (See Table 3)

#### 10) Suggested exposure rate in the European population

In the World Health Organization Eight City study of health and housing the percent of participants with physician diagnosed depression is comparable with the findings of Uston et al (6% versus 8.9% in developed countries). The difference between the percent of depressed participants who were satisfied with interior light compared to non-depressed participants who were satisfied with interior light was 9%. The other depression variables showed a relationship of similar magnitude. In addition, there may be many adverse health effects associated with inadequate light. Only when these are delineated and quantified will we be able to assess the full impact of lighting on health.

There is not sufficient and reliable evidence for making a valid assessment at this time. Further study is warranted.

Table 1: Characteristics of Participants with Doctor Diagnosed Depression Compared to Participants without Depression

Characteristic	Depressed (%)	Not Depressed (%)	'p' Value
Health Insurance Status:			
Public Only	354 (71)	4,646 (59)	<0.0001
Other	145 (29)	3,247 (41)	
Marital Status			
Married	273 (55)	4,152 (53)	0.37
Separated/Not Married	226 (45)	3,741 (47)	
Education			
None/Primary	208 (42)	2,510 (32)	<0.0001
More than Primary	291 (58)	5,383 (68)	
Employment Status			
Full/Part time	168 (34)	3,535 (45)	<0.0001
Other	331 (66)	4,358 (55)	
Alcohol			
Abstain	247 (50)	3,035 (39)	<0.0001
Not Abstain	242 (50)	4,858 (62)	
Health Status			
Very Good/Good	103 (21)	5,136 (65)	<0.0001
Fair or Worse	396 (79)	2,757 (35)	
Satisfaction with Dwelling			
Dissatisfied/			<0.0001
Very Dissatisfied	109 (22)	854 (11)	
Not Dissatisfied	390 (78)	7,039 (89)	
Taking Over the Counter Medication:			
Pain	52 (24)	753 (28)	<0.0001
Cold/Flu	77 (35)	1,091 (40)	
Allergy	22 (10)	193 (7)	
Gastric	15 (7)	123 (5)	
Vitamins	12 (6)	387 (14)	
Sleep	29 (13)	80 (3)	
Other	11 (5)	87 (3)	
Gender			
Male	130 (26)	2,531 (32)	0.006
Female	365 (74)	5,322 (68)	
Socio-Economic Status			
Lowest (1)	130 (26)	2,531 (32)	0.006
Middle (2-4)	365 (74)	5,322 (68)	
Upper (5)	82 (16)	2,348 (30)	
Total	499 (6)	7,893 (94)	

Table 2: Depression and Adequacy of Residential Light

Evidence of Depression	Turns Lights on During Daytime	Satisfied with Light
Have you been depressed in last year?		
Yes	390 (39)**	243 (31)*
No	2,470 (32)	1,797 (24)
Was your depression diagnosed by a physician?		
Yes	166 (33)*	207 (42)*
No	1,877 (24)	2,572 (33)
Have you taken a prescription medicine for depression?		
Yes	199 (42)*	157 (33)*
No	2,580(33)	1,883 (24)

\*p<0.0001

\*\*p=0.0001

Table 3: Falls and Adequacy of Residential Light

Fall in the last 12 months	Turns Lights on During Daytime	Satisfied with Light
Yes	326 (38)*	241 (28)*
No	2,453 (33)	1,799 (24)

\*p <0.01

**Summary of main element of evidence between:**

**Cause/ Housing Factor: Radon**  
**Health Effects: lung cancer mortality**

Contributor: Olivier Catelinois, France

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**Part I: Health effects / Attributable risk / possible exposure-response relationship**

**Data sources used**

Priority should be given to international and/or European-based peer-reviewed and published studies

Author(s)	Year	Title	Journal / Book	Location
BEIR committee	1999	Health Effects of exposure to radon. BEIR VI. National Research Council, Committee on Biological Effects of Ionizing Radiations.	Book	International
Darby, S. et al.	2005	Radon in homes and risk of lung cancer: collaborative analysis of individual data from 13 European case-control studies.	BMJ	European community
Lubin, J. H.	2003	Studies of radon and lung cancer in North America and China.	Radiation Protection Dosimetry	International
Krewski, D. et al.	2005	Residential radon and risk of lung cancer: a combined analysis of 7 North American case-control studies.	Epidemiology	International
EPA	2003	Environmental Protection Agency. EPA assessment of risks from radon in homes.	Report	International
Tirmarche, M. et al.	2003	Quantification of lung cancer risk after low radon exposure and low exposure rate: synthesis from epidemiological and experimental data.	Final scientific report, European Commission DG XII	European community
Darby, S. Hill, D. Doll, R.	2001	Radon: a likely carcinogen at all exposures.	Anal Oncology	European community
Catelinois, O.	2003	Évaluation des risques associés aux rayonnements ionisants.	Thesis	European community

**General evidence** (*what effects and relationships are identified in literature?*)

Indoor radon exposure has adverse effects on health. The main hazard is lung cancer. Nowadays, epidemiological studies are performed to research other hazards such as leukemia.

**Specific evidence** (*how strong are the effects, which quantification has been identified?*)

In 1988, the International Agency for Research on Cancer (IARC) declared radon to be carcinogenic for humans (lung cancer): radon is classed in the group 1 (IARC 1988). This classification was obtained thanks to numerous results which come from either experimental animal or epidemiological studies, in particular among uranium miners.

Significant excess lung cancer risks were observed after exposure to quite high levels of measured radon among miners. These cohort studies allow the adjustment of risk model considering the diminution of the risk according to several time variables such as the time since exposure or the duration of exposure.

During the two last decades, case-controls studies were performed in the general population in order to quantify the risk associated to indoor radon exposure. The exposure-response relationship appears linear and significant even among those with quite low levels of measured radon (that is below 200 Bq.m<sup>-3</sup>). Unlike cohort studies, nowadays, none of case-controls studies allows the adjustment of risk model which consider the diminution of the risk according to several time variables.

**Limitations** (*are there any constraints and limitations associated with the data?*)

To perform lung cancer risk assessment attributable to indoor radon, several data must be combined, in particular the exposure-response relationship between radon exposure and the risk of lung cancer and the percentage of smokers.

Results available in 1988 were related to high exposure levels and the extrapolation and the transposition of this risk to the general population exposed to lower levels in a domestic environment raised numerous debates. Recent joint analysis of case-controls studies provides new results which permit to determine the lung cancer risk associated with exposure at home to the radioactive disintegration products of naturally occurring radon gas.

Because of the importance of the lung cancer risk induced by active smoking, the quantification of the possible interaction between tobacco and radon is essential. The main uncertainty associated with the exposure-response relationship is the quantification of the interaction between radon and tobacco. In 1999, the BEIR committee derived analysis from the only 5 miners' cohorts which allowed the analysis of this interaction. This analysis showed that lung cancer risk due to radon exposure differed according to the smoking status. Nowadays, research project which is on going at a European level aims to quantify more precisely this interaction.

To perform lung cancer risk assessment due to indoor radon considering the interaction between tobacco and radon, data on tobacco consumption are necessary. Unfortunately, such data are sparse in most countries, in particular in France.

**Suggested OR / incidence / attributable fraction for exposed population** (*what seems to be the most reliable and evidence-based estimation on the likelihood that the health effect is expressed?*)

Most of lung cancer risk assessment which were performed used exposure response relationship adjusted among miners. Nowadays, we own new exposure-response adjusted among general population thanks to joint analysis of case-control studies.

For EBD study, it would be reasonable to use different sources of exposure-response relationship to perform an uncertainty analysis according to the model.

According to the excess risk of lung cancer issued from the joint analysis of case-controls studies published by S. Darby et al. (about 16% per 100 Bq.m<sup>-3</sup>), the radon in homes would currently accounts for about 9% of the deaths from lung cancer and hence 2% of all cancer deaths in Europe.

Final recommendation

**There is some / partially sufficient and reliable evidence for making a valid assessment**

## Part II: Exposure situation

### Data sources used

Priority should be given to international and/or European-based studies in peer-reviewed literature

Author(s)	Year	Title	Journal / Book	Location
BEIR committee	1999	Health Effects of exposure to radon. BEIR VI. National Research Council, Committee on Biological Effects of Ionizing Radiations.	Book	International
EPA	2003	Environmental Protection Agency. EPA assessment of risks from radon in homes.	Report	International
Catelinois, O.	2003	Évaluation des risques associés aux rayonnements ionisants.	Thesis	European community

### General evidence *(is a relevant exposure identified in the literature?)*

Radon is a chemically inert radioactive gas of natural origin. It is produced by the disintegration of uranium and radium located in the earth's crust. Radon exposure is omnipresent for the general public, but at variable levels, because radon mainly comes from granitic and volcanic subsoils as well as from certain construction materials. Inhalation of radon is the main source of exposure to radioactivity in the general population of most countries. Most inhaled radon is rapidly exhaled, but the inhaled decay products readily deposit in the lung epithelium, where they irradiate sensitive cells in the airways, thereby enhancing the risk of lung cancer.

### Specific evidence *(how large is the exposure, which quantification has been identified?)*



Inhalation of radon is the main source of exposure to radioactivity in the general population of most countries. Indoor radon exposure is relatively well known in most parts of Europe.

**Limitations** *(are there any constraints and limitations associated with the data? Are there large variation within countries or by country or regions?)*

Radon levels of most parts of Europe are relatively well known and patterns of the radon levels have been described by means of maps. However, radon exposure in homes varies widely with area and season. More measurements are required to assess these variations. Moreover, the data is representative for country level but it is to be discussed to what extent it could be used on regional level.

**Suggested exposure rate in the (European) population** *(what seems to be the most reliable and evidence-based estimation on the general exposure within the population?)*

Several campaigns of indoor radon measurements have been performed all over the world. Due to quite high variability of indoor radon concentration within the same country and the same region, the mean of exposure is not useful to perform a risk assessment. Thus, it would be reasonable to perform risk assessment at the adequate level and considering both the variability of indoor radon concentration within the same region and uncertainties related to the knowledge of indoor radon concentration. This can be done in two steps:

Definition of indoor radon concentration categories within a region;

Consideration of the entire indoor radon concentration distribution in each concentration categories weighted by the proportion of population concerned.

Final recommendation

**There is some / partially sufficient and reliable evidence for making a valid assessment**

## Summary of main element of evidence between:

**Cause/ Housing Factor: House dust mites**

**Health Effects: Asthma / Allergies**

Contributor: David Crowther, UK

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### Part I: Health effects / Attributable risk / possible exposure-response relationship

*This contribution is concerned with the exposure situation only.*

### Part II: Exposure situation

#### Data sources used

Priority should be given to international and/or European-based studies in peer-reviewed literature

Author(s)	Year	Title	Journal / Book	Location
Bronswijk, J. E. van	1981	<i>House Dust Biology for Allergists, Acarologists and Mycologists</i>	NIB Publishers, Zoelmond, Netherlands	EU
Boer R de, Kuller, K. and Kahl, O.	1998	Water balance of <i>Dermatophagoides pteronyssinus</i> (Acari: Pyroglyphidae) maintained by brief daily spells of elevated air humidity	Journal of Medical Entomology, 35/6: 905-910	EU
Pretlove, S. et al.	2001	A combined transient hygrothermal and population model of house dust mites in beds	Proceedings of IAQ 2001: Moisture, Microbes and Health Effects: Indoor Air Quality and Moisture in Buildings, ASHRAE, San Francisco, USA, November 4 – 7	EU
van Strein, R.T. et al.	2004	The influence of air conditioning, humidity, temperature and other household characteristics on mite allergen concentrations in the northeastern United States	Allergy, 59: 645-652	USA

#### General evidence (is a relevant exposure identified in the literature?)

House dust mites (HDMs) have been found, in varying numbers, in many countries throughout the world. Less than a millimetre in size, they feed off human skin scale and live where a) skin scales accumulate, in such habitats as bedding, carpets, upholstered furniture and soft toys, and b) where conditions of temperature and relative humidity are conducive to population growth. Although HDMs are found in offices and other workplace environments, their predominant habitats are in dwellings. Their faecal pellets are friable and easily become

airborne, and the inhaled fragments are just right size to travel deep into the lung. These fragments contain several powerful allergens, the most often measured being Der f 1 and Der p 1, relating to the two most predominant species, *Dermatophagoides farinae* (most common in North America) and *D. pteronyssinus* (most common in Europe).

**Specific evidence** (*how large is the exposure, which quantification has been identified?*)

HDMs need a particular combination of temperature and relative humidity in order for rapid population growth to occur (assuming food is plentiful). On the other hand, they can survive unfavourable conditions for long periods and population growth will still occur provided that favourable conditions are experienced for at least 2 hours a day. However, such hygrothermal conditions are by no means universally found in dwellings. It is often stated that HDMs are ubiquitous and that there is little that can be done to reduce exposure, but this is a myth - indeed a dangerous myth. Several studies have found marked differences in mite numbers and/or Der p 1 concentrations between dwellings, even in the same locality, ranging from high to non-existent. Outside the field of building science, it is not generally appreciated quite how much hygrothermal conditions can vary between dwellings, variations that can make all the difference between whether HDMs survive or not.

A further common misconception is that hygrothermal conditions in mite habitats, such as beds or carpets, are mostly independent of room conditions, so that the latter are largely irrelevant. In beds, for example, it is assumed that human occupants provide independent and long lasting sources of warmth and moisture that do not dissipate significantly when they get out of bed. However, experiments using volunteers sleeping in fully instrumented beds in the laboratory have shown that this is not correct. Before the sleeper gets into bed, hygrothermal conditions in the mattress are the same as room conditions. These then change relatively quickly when the sleeper gets into bed and almost as quickly when the sleeper gets out of bed eight hours later. This return to room conditions is delayed only slightly by leaving bed coverings in place. In other words, for almost all of the 16 hours that the bed is unoccupied, hygrothermal conditions within the bed are the same as room conditions. Moreover, the location in the bed most favourable for mite population growth is not immediately next to the sleeper, which tends to be too dry – the effect of body heat tending to outweigh the effect of body moisture). Rather it is at intermediate positions nearer the edge of the mattress, which are affected by room hygrothermal conditions even when the bed is occupied. For both reasons, therefore, room conditions do indeed play a major role in determining whether mite populations grow or decline.

The three factors that primarily determine hygrothermal conditions in dwellings are:

Climate (i.e. hygrothermal conditions outside the home)

Building construction (air tightness, level of insulation, heating provision, etc.)

Occupant behaviour (moisture production from washing and drying, window opening habits, etc.)

**Climate** alone can account for some (but not all) of the observed difference in asthma prevalence between countries and regions. For example, asthma tends to be less prevalent at high altitudes and in colder climates. The reason for this relates to the fact that cold air cannot contain as much moisture as warm air. During winter, warm moist air from inside a dwelling is continually being exchanged (assuming adequate ventilation) for cooler drier air,

which is then heated up, and the relative humidity inside the dwelling thus falls. The extent to which it falls depends on how cold the outside air is, the level of ventilation and the temperature to which the thermostat is set: the colder the outside air and the more the ventilation and the warmer the indoor temperature, the lower the relative humidity. And once relative humidity falls below 45-50%, HDM populations start to dwindle.

In summer, outdoor temperatures rise, thereby limiting the scope for reducing mite populations in this way. As a result, marked seasonal variations are typically observed in mite populations, with peaks in late summer and autumn, when indoor conditions are most favourable, and troughs in late winter and spring, when conditions are least favourable. It has also been observed that if the troughs are low enough, too few mites survive to take advantage of the favourable conditions later in the seasonal cycle, leading to permanent reductions in mite populations (low enough to cause only minimal health problems) and even eradication. However, this can only be achieved if both a) winters are cold enough and b) standards of ventilation and heating are sufficiently high. Sub-tropical climatic regions without cold winters, such as much of Australia, cannot take advantage of this natural seasonal culling of mites, which partly explains their high asthma prevalence. Differences between temperate and continental climates can also be used to explain differences in asthma prevalence, although the picture is more complicated. Moreover, even in regions with cold winters, the seasonal culling of mites can be overridden if ventilation is inadequate, complicating the pattern further.

This brings us to the second factor: **building construction**. Since WW2, there has been a general and well- documented increase in air tightness in building construction and, especially after the oil crises of the mid-1970s, householders have become ever more energy conscious. As a result, in a deliberate attempt to conserve energy, ventilation standards have fallen. Since water vapour is continuously produced as the result of human occupation, indoor relative humidity levels have consequently tended to rise. At the same time, standards of thermal comfort have risen, with more reliance being placed on heating systems to provide warmth in winter, rather than clothing as before. There are thus now several reasons why householders are increasingly reluctant to lose expensively heated air for the purpose of ventilation. Furthermore, adequate ventilation in many modern homes can only be provided by the conscious act of opening a vent or window, which, given the above, tends not to happen, especially in the crucial winter months when the need for it, in terms of culling mites, is greatest.

In older, "leakier" types of construction, by contrast, adequate background ventilation is provided involuntarily by various means, such as via the multiple cracks around loose-fitting doors and windows, and open chimneys. However, there is far less tolerance than before of the "draughts" that such leaky construction tends to give rise to and while newly built housing has become ever more airtight, older housing is also being gradually rehabilitated. This is significant because, in terms of quantity, older housing constitutes by far the larger fraction of the housing stock. The rate at which rehabilitation is occurring varies greatly from region to region and country to country, but by a mixture of private enterprise, Do-It-Yourself and government sponsored programmes, the insulation and air tightness standards of the older housing stock is gradually being improved throughout the developed world. It is unlikely that older housing can ever be made as airtight as newly built housing, but it is

nevertheless becoming urgent that the need for adequate background ventilation should be recognized for all types of construction, both newly built and rehabilitated.

The health benefits of adequate ventilation are obvious. They relate to reducing exposure not only to mite allergens (in those climatic regions that can take advantage of seasonal culling), but also to other indoor pollutants, such as toxins from fungal growth, tobacco smoke and chemical outgassing from materials and household products. The health benefits of higher standards of insulation and the provision of affordable warmth are also obvious, for example for reducing the number of Excess Winter Deaths. However, the effect on mite populations is not so clearcut. HDM egg-to-adult development time increases rapidly as room temperature falls below 23°C, thereby significantly slowing population growth even when room relative humidity is high. Raising room temperature thus tends to shorten egg-to-adult development time and to favour mite population growth. On the other hand, raising room temperature has the simultaneous effect of lowering room relative humidity, which is unfavourable for population growth. The two effects thus tend to cancel each other out. Indeed modelling studies have demonstrated that the favourable effect of the rise in room temperature that results from improved insulation and heating provision tends to be outweighed by the unfavourable effect of the fall in relative humidity. This is to be welcomed, since it means that mite populations can potentially be controlled by modifying the hygrothermal environment without sacrificing the health benefits of providing affordable warmth. The key is the provision of adequate ventilation. Although this necessarily involves some loss of energy, this can be lessened in some cases by technological means, such as using heat exchangers. However, even without such active interventions, studies have shown that ventilation heat loss can be relatively modest, usually much less, in terms of carbon emissions, than the energy used by the average household on electrical appliances. Adequate ventilation is thus not incompatible with energy efficiency.

The third factor is **occupant behaviour** – how householders use their homes. This factor is again far more significant than is generally realised. Hygrothermal conditions can vary greatly between different households, even when living in identically constructed and located dwellings. To begin with, household moisture production can vary from 3 to 15 litres per day according to:

The number of occupants and how much of the day they spend at home

Their moisture producing activity, mainly washing, cooking and bathing (many UK householders still hang wet washing up to dry indoors).

Hygrothermal conditions are then affected by:

The extent to which windows are kept tight shut in winter to conserve heat

Whether internal doors (especially to kitchen and bathroom) are kept open or shut.

The temperature at which the thermostat is set and the number of hours the heating is on.

The fact that cold indoor temperatures inhibit mite population growth may explain why, in the UK for example, asthma is not as prevalent as one might expect in low income households, where indoor relative humidities tend to be high (e.g. due to drying clothes over radiators), but where indoor temperatures at the same time tend to be low, due to the lack of affordable warmth. With rising living standards and wider access to affordable warmth, indoor temperatures in low income housing can be expected to rise, so that inhibiting effect

of low temperatures will diminish. There are grounds for concern that, unless moisture production in such households is simultaneously curtailed, near ideal conditions for mite proliferation could be created.

**Limitations** (*are there any constraints and limitations associated with the data? Are there large variation within countries or by country or regions?*)

It can be seen that the net indoor temperature and relative humidity in a dwelling are the result of many different and interacting factors. It is therefore not surprising that hygrothermal conditions in dwellings can vary so much both from region to region and from dwelling to dwelling within a region. This level of complexity presents obvious problems when investigating links between housing characteristics and levels of HDM allergen. An approach adopted in several studies is to measure a range of variables in a large sample of dwellings and to use statistical techniques to determine associations with the number of mites or allergen levels found in each case. While interesting and useful results have been obtained, the percentage of variation explained by the factors examined is often low (e.g. 21% or less in the case of van Strien et al. 2004). In addition, although temperature and relative humidity are usually included, they are often simple spot measurements taken at one arbitrary moment in time. Given that both continuously vary, often over a wide range, from hour to hour, day to day and season to season, spot measurements are seriously inadequate for determining a possible association between these two factors and the mite population or allergen levels found.

A different approach is to make use of the fact that several validated computer models have now been developed by building physicists for simulating hygrothermal conditions in dwellings. Some are relatively simple, dealing with monthly average conditions, but others deal with hourly values and the full range of factors that contribute to the net result. It is worth emphasizing that these models are based not on statistical correlations but on the laws of physics and cause and effect relationships. A further recent development is the introduction of small, cheap sensors and data loggers for recording hygrothermal conditions in multiple locations within mite habitats. Exploiting these, our multi-disciplinary research group\* is currently engaged in a) extending existing models to specifically include mite habitats (currently just the bed, the most relevant for the UK) and then b) coupling them to newly developed mite population models that simulate the effect of changing hygrothermal conditions on mite population growth (Pretlove et al. 2001). Again, these are not statistical models, but are based on the observed physiological behaviour of mites under varying hygrothermal conditions.

The advantage of this approach is that we will potentially be able, for any given climate zone, dwelling type and occupation behaviour pattern, to predict the likely level of mite infestation. First of all, a combined hygrothermal/population model of this kind could become part of a mite allergen exposure model that could be relevant for any area for which climate, dwelling and occupant behaviour data sets are either available or could be derived using reasonable simplifying assumptions. Secondly, where exposure is found to be high, the combined model could be used to explore alternative ways to reduce it, such as by improving insulation and/or ventilation standards. It should be noted, however, that although results to date have been promising, the models being developed have not yet been fully validated and

the percentage of variation explained has still to be determined. Until then, our hypothesis that the latter will be superior to statistical models remains unproven.

Although it is attractive to see how far one can go in explaining observed variations in mite numbers and allergen simply in terms of hygrothermal conditions, it can be argued that other factors are likely also to play a role. In effect, the physics/biology modelling approach provides a basis for determining the inherent likelihood of mite infestation for a particular dwelling or type of dwelling, given climatic region and occupant characteristics. Further research may indeed uncover other factors that affect mite and allergen levels, but hopefully these can also be modelled and integrated with previous models.

A further limitation to be noted relates to mite sampling techniques. Various methods have been tried and the most common is currently to use a vacuum machine. However, HDMs have hooks at the end of their 8 limbs that enable them to grip strongly onto the fibrous material surrounding them. Unless the vacuum machine is of unusually high power, sampling tends to remove primarily the weakest members of the population and those that happen to be nearest to the surface. It is almost impossible to estimate what percentage of the total population in the habitat has been sampled and, if repeat sampling is required to measure population change over time, one cannot be sure that the sampling has not affected the observed change. More research is needed to resolve these issues.

**Suggested exposure rate in the (European) population** (*what seems to be the most reliable and evidence-based estimation on the general exposure within the population?*)

The worldwide ISAAC (Phase 2) and European ECRHS studies are providing data on the mite numbers and allergen levels measured in the dwellings surveyed (see <http://isaac.auckland.ac.nz> and <http://www.ecrhs.org> ). It may be possible to use these data sets to estimate general exposure.

\* funded by the UK Engineering and Physical Sciences Research Council

Final recommendation

**There is some / partially sufficient and reliable evidence for making a valid assessment**

## **Summary of main element of evidence between:**

### **Cause/ Housing Factor: Social factors and crowding Health Effects: General health status**

Contributor: James Dunn, Canada

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This short report briefly presents a framework for understanding the role that social factors of housing have on health, with a specific emphasis on 'crowding and general health status'. In this attempt to outline a method for estimating the burden of ill-health from substandard housing, it is necessary to situate individual dimensions of housing within a broader framework, for at least two reasons. First, housing has many dimensions, including, social, bio-physical, design, etc. and to isolate a specific factor is somewhat of an artificial exercise, partly because there is a high degree of synergy and interdependency between individual factors. Moreover, social factors of housing interact significantly with bio-physical-chemical factors and design factors as well (something that is seldom considered in most studies).

The primary mechanism linking social factors of housing and health, both general and mental health, is through the stress process. Arguably, the most important stressors that can arise from housing are the following:

- (un)affordability
- insecure tenure
- crowding and residential density
- disrepair
- geographical isolation from...
- services
- social contacts
- employment opportunities
- transportation

and their effects on health have been demonstrated in the following outcomes:

- general health (e.g., self-rated health status)
- mental health
- generalized distress
- depressive symptomatology



## Exposure of the population

### Data sources used

Author(s)	Year	Title	Journal / Book	Location
Healy, J.D.	2002 (1994-1997)	Housing Conditions and Self-Reported Health: A Cross-European Analysis	Dept. Environmental Studies, Univ. College Dublin	14 EU countries

### General evidence

European Community Household Panel (ECHP) reports the findings of a survey of 14 European countries on a number of housing conditions including the burden of housing costs and household crowding. The measure of household crowding is self-reported perception of crowding, specifically, whether the household has enough space to meet their needs or is otherwise overcrowded.

### Specific evidence

The prevalence of self-reported overcrowding varies between 9.8% (Netherlands) and 27.9% (Portugal).

### Limitations

The self-report measure is inadequate. Undoubtedly surveys with objective measures of housing (for example, persons per room or persons per sleeping room) are available in population surveys, perhaps for instance, in LARES.

Suggested OR / incidence / attributable risk fraction for exposed population  
=> unknown, but probably available in LARES or other surveys

Final recommendation:

Strong potential, worthy of further research

## Health outcome(s)

### Data sources used

Author(s)	Year	Title	Journal / Book	Location
Bierman, et al.	1999	How Well Does a Single Question about Health Predict the Financial Health of Medicare Managed Care Plans?	<i>Effective Clinical Practice</i> March/April	USA
van Doorslaer, et al.	1997	Income-related inequalities in health: Some international comparisons	<i>Journal of Health Economics</i> 16: 93-112	international
Miilunpalo S, et al.	1997	Self-rated health status as a health measure: The	<i>J Clin Epidemiol</i> 50:517-528	USA

		predictive value of self-reported health status on the use of physician services and on mortality in the working-age population		
Gold, et al.	1996	Assessing the health of a nation: The predictive validity of a preference-based measure and self-rated health	<i>Med Care</i> 34:163-177	review
Weich	1997	Prevention of the common mental disorders: A public health perspective	<i>Psychological Medicine</i> 27: 757-764	UK

#### General evidence

In general, fair or poor self-rated health status is a measure that is used widely (van Doorslaer, et al. 1997) and has been shown to be highly correlated with other 'harder', physical measures of health (mortality, diagnosed morbidity, symptom reporting) (Miilunpalo S, et al. 1997), health care utilization (Bierman, et al. 1999) and also functional status (Gold, et al. 1996). It is also highly graded by socio-economic status and negatively associated with stress.

The common mental disorders, anxiety and depression, are highly disabling even at sub-clinical levels. They too are highly graded by socio-economic status and strongly associated with most measures of stressful life circumstances.

#### Specific evidence

##### General Self-Rated Health

Bierman, et al. use data from the 1992 Medicare Current Beneficiary Survey (MCBS), a nationally representative probability sample of Medicare beneficiaries (n=8775). Total Medicare expenditures were higher with lower levels of self-rated health. Annual age- and sex-adjusted Medicare expenditures in the year after health assessment were \$8743 for beneficiaries reporting poor health and \$1656 for those reporting excellent health, for a fivefold difference ( $P < 0.001$ ; adjusted R<sup>2</sup> for a model predicting expenditure with age, sex, and global health = 0.044). These differences in expenditure largely reflected the higher hospitalization rates seen among beneficiaries reporting poorer health. Age- and sex-adjusted hospitalization rates per 1000 beneficiaries were 675 among those reporting poor health, 437 among those reporting fair health, 321 among those reporting good health, 200 among those reporting very good health, and 136 among those reporting excellent health, for a fivefold difference ( $P < 0.001$ ; adjusted R<sup>2</sup> for model predicting hospitalizations = 0.042).

For other health status measures (disability level, social functioning, and number of comorbid conditions), worse health status was again related to higher total Medicare expenditures in the year after health assessment. For these measures, an approximate threefold difference in expenditures was seen between the worst health state and the best health state.

### Common Mental Disorders

According to Weich, the common mental disorders, depression and anxiety, are significant sources of illness and disability, even at a sub-clinical level. They have a combined community prevalence rate between 15% and 30%, and account for 1/3 of work days lost to illness and 1/5 of general practice consultations in the United Kingdom. The common mental disorders “are associated with impairments in physical and social functioning at least as severe as those associated with physical illness.” (p.757). Even in mild form, they are highly disabling; one study shows that low levels of depression result in 51% more days lost from work than major depression.

### Limitations

Self-reported data, but despite this, both measures are incredibly robust. One difficulty with measures of the common mental disorders is that they are developed as screening tools and tend not to be validated for use as continuous measures, rather, they establish a threshold based on the greatest sensitivity and specificity for predicting the likelihood that a person has a clinically diagnosable mental illness. In other words, the thresholds tend to miss ill-health that is significantly disabling.

Suggested OR / incidence / attributable risk fraction for exposed population

Incidence will likely be over 20% for common mental disorders and slightly less than that for fair/poor self-rated health. Odds ratios could be as high as 5.

## Dose-response relationship between exposure and outcomes

Data sources used

Author(s)	Year	Title	Journal / Book	Location
Dunn, et al.	2004	Gender, socio-economic attributes of housing, and self-rated health.,	<i>Reviews on Environmental Health</i> 19(3-4): 1-19	Canada
Regeoczi	2003	When context matters: a multilevel analysis of household and neighbourhood crowding on aggression and withdrawal	<i>J. Environmental Psychology</i> 23: 451-464	Canada
Lepore, et al.	1996	Role of control and social support in explaining the stress of hassles of crowding	<i>Environment &amp; Behavior</i> 24(6): 795-811	USA
Evans, et al.	1996	The role of interior design elements in human responses to crowding	<i>Journal of Personality &amp; Social Psychology</i> 20(1): 41-46	USA

### General evidence

Large amount of evidence showing a relationship between residential crowding and a variety of measures, including mental health, aggression and withdrawal, psychological distress, general health status. The relationships are generally quite strong, but there is little consistency in measures, which may be an obstacle to BoD calculations.

### Specific evidence

Dunn, et al. (2004), in a survey of Vancouver households showed that women living in more crowded households (measured by persons per bedroom) were at greater risk for psychological distress (OR=2.27), but men living more crowded households were less likely (OR=4.0) to suffer from psychological distress. In a survey of households in Toronto neighbourhoods, Regeoczi (2003) shows that neighbourhood density conditions the relationship between household density and aggression and withdrawal. Lepore, et al. (1996), in one of the few prospective studies of this topic, showed that the relationship between hassles and psychological distress was stronger in more crowded households than less crowded households. Evans, et al. (1996) found that the relationship between crowding & psychological distress is significantly reduced after controlling for 'architectural depth'.

### Limitations

Mostly cross-sectional studies, so subject to possible bias due to self-selection (i.e., people with mental health problems either choose or are forced to live in very crowded or very isolated housing circumstances). That said, one study (Lepore, et al. 1996) was prospective and showed expected relationship, meaning that the direction of causation (i.e., crowding =>

mental health) is plausible. There is little consistency in both exposure and outcome measures across studies, so calculation of a summary OR may be difficult.

Suggested OR / incidence / attributable risk fraction for exposed population

Differences between crowding measures makes it difficult to summarize this. Re-analysis of existing data may enable a common metric.

## Summary of main element of evidence between:

### **Cause/ Housing Factor: Housing conditions Health Effects: Mental Health**

Contributor: Gary W. Evans, USA

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#### Multi-family housing and mental health.

##### Part I. Health effects

###### 1) **Data sources used**

Fanning, D.M. 1967. Families in flats. *British Medical Journal*, 4, 382-386. UK

Wilcox, B.L. & Holahan, C.J. 1976. Social ecology of the megadorm in university student housing. *Journal of Educational Psychology*, 68, 453-458. US

McCarthy, D. & Saegert, S. 1976. Residential density, social overload, and social withdrawal. *Human Ecology*, 6, 253-272. US

Saegert, S. 1982. Environment and children's mental health: Residential density and low income children. In A. Baum & J.E. Singer (Eds.), *Handbook of psychology and health, vol. II* (pp. 247-271). Hillsdale, NJ: Erlbaum. US

Evans, G.W., Wells, N.M. & 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. Review of international work.

Gifford, R. in press. Satisfaction, health, security, and social relations in high rise buildings. In A. Seidel & T. Heath (Ed.), *Social effects of the built environment*. London: E & FN Spon. Review of international work.

###### 2) **General evidence**

Large, multi family housing is associated with mental health problems.

###### 3) **Specific evidence**

Four studies, all with random assignment of individuals to multi family dwellings vs. detached housing or smaller units found poor mental health. Evidence included visits to the doctor in one study and standardized assessments of social interaction and influence/control in two and standardized measures of children's psychological problems. Incidence rates for GP visits for psychoneurotic disorders were 36.1 and 17.9 per 1000 among wives of serviceman in multi-family units vs. detached single family residences (Fanning). This difference was especially large for women with young children. Mc Carthy & Saegert found that 14 story high-rise (2.43) vs. 3 story, low rise (4.77) low-income public housing residents felt more crowded (1-6 point rating scales throughout, lack of control (4.97; 3.00), less social activity with other residents (3.48; 2.19) and 3.3% and 24.1% of residents of high- and low-rise residents, respectively felt they 'belonged' to their housing project. Wilcox and Holahan uncovered differences in social support in low-rise (6.4) compared to high-rise (4.4) and in social involvement with dorm residents (6.6 vs. 4.2) (1-7 point rating scales). Low-income elementary school aged boys but not girls who were residents of high-rise public housing had

higher levels of psychological symptoms (20.17) compared to boys living in low rise buildings (13.99) (Saegert).

#### **4) Limitations**

Only the Fanning study is large (n=1500) and all have specialized samples, wives of military personnel (Fanning), low-income public housing residents (McCarthy & Saegert; Saegert) and college undergraduates in student housing (Wilcox & Holahan), respectively.

#### **5) Suggested incidence**

The OR for high rise housing and mental health problems is on the order of 2.0 for adults and probably larger for women with young children. Effects may exist on children as well but the size of the effect cannot be estimated at this time.

#### **Final Recommendation**

There is some partially sufficient and reliable evidence for making a valid assessment. A much larger number of studies with weaker research designs converge with those summarized here (Evans et al.; Gifford).

### **Part II: Exposure Situation**

#### **6) Data sources used**

US Census Supplemental Survey. 2001. Table 4. Profile of Selected Housing Characteristics. Washington, DC. USA.

National Board of Housing, Building and Planning. Sweden Ministry of Regional Development. 2004. Table 3.1 Dwelling Stock by type of building. Housing Statistics in the European Union.

#### **7) General evidence**

Large numbers of people live in large, multi family dwellings throughout Europe and North America. This is especially true for urban centers and for lower and middle class individuals.

#### **8) Specific evidence**

In America approximately 22% of the total 118,000,000 housing units consist of three or more units. Of the European Union countries reporting housing units separately for Total and Multi-family, the percentages range from 39% multi-family (Denmark) to 72% multifamily (Germany). The average across the 12 EU countries is 56% multi-family housing.

#### **9) Limitations**

Note Belgium, Estonia, Greece, Hungary, Ireland, Italy, Lithuania, Luxembourg, Malta, Netherlands, Poland, Portugal, and the UK are not included in these statistics because total housing units are not broken out by single and multi-family. The UK and Italy are among the largest in total housing units in the EU.

#### **10) Suggested exposure**

Unless the proportion of multi-family units is vastly different in Italy and the UK compared to the rest of the EU, the rate of 50% is a reasonable estimate for EU. Within the US, the number is less than half that figure, reflecting the larger proportion of Americans who live in single family, detached homes.

**Final recommendation**

There is sufficient and reliable evidence for making a valid assessment.

**Living on upper floors of high-rise housing and mental health.**

**Part I. Health effects**

**1) Data sources used**

Fanning *Ibid.*

**2) General evidence**

Individuals living on higher floors evidence more mental health problems.

**3) Specific evidence**

Random assignment of families to higher floors in housing associated with more MD visits for psychological symptoms (63.0 vs. 127.3 per thousand for ground vs. third floor) among 1500 wives of British and Canadian servicemen living in Germany.

**4) Limitations**

The Fanning study was focused on military wives living on base outside their home country. There is only one study examining floor level and mental health with a strong research design. It is worth noting that many other studies with weaker research designs find similar trends (Evans et al.; Gifford).

**5) Suggested incidence**

The estimated OR is on the order of 2.0.

**Final Recommendation**

There is some partially sufficient and reliable evidence for making a valid assessment.

**Part II: Exposure Situation**

**6) Data sources used**

US Census Bureau American Housing Survey. 2001. Table 1A-2 Height and Condition of Building. Washington, DC, USA.

National Board of Housing, Building and Planning. Sweden Ministry of Regional Development. 2004. Table 2.5 Dwellings in high rise buildings. Housing Statistics in the European Union.

**7) General evidence**



Large numbers of people live on the upper floors of high rise buildings throughout Europe and North America. This is especially true for urban centers and for lower and middle class individuals.

#### 8) **Specific evidence**

In America of the 118,000,000 housing units, 26% are 3 or more stories high and 7% are four stories or higher. 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% (UK) to 39% (Poland).

#### 9) **Limitations**

Data are missing for Cyprus, Estonia, Finland, Ireland, Latvia, Lithuania, Malta, and Sweden for EU countries.

#### 10) **Suggested exposure**

For the US, the estimate of exposure to buildings above 3 stories is approximately 7% and for the EU 15%.

#### **Final recommendation**

There is sufficient and reliable evidence for making a valid assessment.

### **Housing Quality and Mental Health**

#### **Part I. Health effects**

##### 1) **Data sources used**

Wilner, D.M., Walkley, R.P., Pinkerton, T.C. & Tayback, M. 1962. *The housing environment and family life*. Baltimore: Johns Hopkins Press. US

Davie, R., Bulter, N. & Goldstein, H. 1972. *From birth to seven*. London: Longman. UK

Duvall, D. & Booth, A. 1978. The housing environment and women's health. *Journal of Health and Social Behavior*, 19, 410-417. Canada

Elton, P.J. & Packer, J.M. 1986. A prospective randomised trial of the value of rehousing on ground of mental illness. *Journal of Chronic Diseases*, 39, 221-227. UK

Halpern, D. 1995. *Mental health and the built environment*. London: Taylor & Francis. UK

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. UK

Evans, G.W., Wells, N.M., Chan, E., & Saltzman, H. 2000. *Journal of Consulting and Clinical Psychology*, 68, 526-530. US

Gifford, R. & LaCombe, C. in press. Children's socioemotional health and housing quality. *Journal of Housing and Built Environment*. Canada

##### 2) **General evidence**

Poor quality housing (structural quality, maintenance, climate, hazards) is consistently associated with poor quality mental health.

### **3) Specific evidence**

Eight well conducted studies including some with longitudinal designs that took advantage of natural experiments in housing improvements converge on evidence that substandard housing quality is related to poorer mental health. Wilner et al studied 600 African American female residents of public housing, half of whom moved to improved quality housing. Adults who moved to better housing 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; 59% vs. 49% life satisfaction]. Groups were equivalent pre-relocation. Children of families who moved to better housing also did better in school. Davie et al. in a national study of 10,000 children from birth to age 7 in the UK found that the presence of basic housing amenities (hot water, own bath, indoor bath) was associated with a about a 30% difference in teacher ratings of the child's adjustment to school. Significant differences were also found for reading and mathematics performance. Among married women with at least one child in Canada, structural deficiencies were correlated .08 with use of tranquilizers and .15 with psychiatric impairment (Duvall & Booth). Elton and Packer found that low income residents of council housing in the UK who moved to better housing had less depression and anxiety (2.5, 1.2) immediately after the move compared to those who did not move (7.6, 6.5). Using a criterion of 50% reduction in symptoms of anxiety and depression combined, 82% of those who received better housing improved compared to 29% of those who remained. Moreover these improvements in mental health persisted one year later. Halpern in a similar design of council housing improvements found diminished anxiety and depression among those whose homes improved with remodeling. For example 89% and 32% had clinically significant levels of anxiety and depression, respectively pre-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. Evans et al found cross-sectional ( $R^2 = .13$ ) and prospective, longitudinal improvements ( $R^2 = .13$ ) in psychological health among women with better housing. Welch and Lewis in a cross sectional study of 10,000 UK adults found an 1.4 increased odds for common mental disorders among those in housing with structural problems. Finally Gifford and Lacombe found that housing quality was significantly correlated ( $r = .39$ ) to mental health among 9-12 year olds.

### **4) Limitations**

Few limitations. The data are robust across countries and include several longitudinal studies with mental health improving in concert with improved housing quality. More work is needed to understand the underlying psychological processes that link housing quality to mental health.

### **5) Suggested incidence**

Housing quality clearly impacts mental health among adults and children. The order of magnitude of the effect is between 2 and 4 OR.

### **Final Recommendation**

There is sufficient and reliable evidence for making a valid assessment.

## **Part II: Exposure Situation**

### **6) Data sources used**

US Census Bureau American Housing Survey. 2001. Table 2-7 Additional Indicators of Housing Quality. Washington, DC, USA.

European Community Household Panel. Lack of amenities by household and income group. <http://epp.eurostat.cec.eu.int/extraction/retrieve/en/theme3/housing/prholds/quality/amen>

### **7) General evidence**

Large numbers of people live in substandard housing throughout Europe and North America. This is especially true for lower class individuals.

### **8) Specific evidence**

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<sup>1</sup> and another 4% as moderate physical problems<sup>2</sup>. In 15 EU countries, 17% are missing one or more of the following amenities: hot running water, bath or shower, or a no flush toilet. Sixteen percent are missing central heating.

### **9) Limitations**

Only limited aspects of housing quality are assessed and EU data is missing several countries (see above Limitation).

### **10) Suggested exposure**

In the US approximately 6% of all housing units are experiencing poor quality housing. In the EU this figure is higher, on the order of 15%.

### **Final recommendation**

There is sufficient and reliable evidence for making a valid assessment.

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#### Footnotes

<sup>1</sup> Severe physical problems is defined as a housing unit with any one of the following problems: lack of indoor plumbing, or flush toilet, or bathtub or shower. Having been uncomfortably cold for 24 hours or heating broke down at least three times prior winter. No electricity or all three of the following: exposed wiring, room with no working outlet, three blown fuses/tripped circuit breakers in last 90 days. In public hallways no working light fixtures and loose or missing steps and loose/missing railings and no working elevator. Having any five of the following: water leaks from outside, leaks from inside of structure, holes in floors, holes/cracks in walls or ceilings, more than 8 x 11 inches of peeling paint or plaster, signs of rodents in last 90 days.

<sup>2</sup> Moderate physical problems have none of the severe problems above and any of the following: toilet broken for six hours on at least three occasions during last 3 months; unvented heater as primary heat source; lacking kitchen sink, refrigerator or cooking equipment for exclusive use of household; any three of the public hallway problems above; any three of the structural/upkeep problems listed above.

**Summary of main element of evidence between:**

**Cause/ Housing Factor: Cold-related mortality  
(cardiovascular/respiratory)**

**Health Effects: Excess winter mortality / seasonality**

Contributor: Jonathan Healy, Ireland

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**Part I: Health effects / Attributable risk / possible exposure-response relationship**

**Data sources used**

Priority should be given to international and/or European-based peer-reviewed and published studies

<b>Author(s)</b>	<b>Year</b>	<b>Title</b>	<b>Journal / Book</b>	<b>Location</b>
Clinch, JP and Healy, JD	2000	Housing standards and excess winter mortality	<i>Journal of Epidemiology and Community Health</i> ; 54: 719-20	Ireland, Norway
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	<i>Lancet</i> ; 349: 1341-6	EU
Healy, JD	2003	Excess winter mortality in Europe: a cross-country analysis identifying key risk factors	<i>Journal of Epidemiology and Community Health</i> ; 57: 784-9	EU
Healy, JD	2004	Housing, fuel poverty and health: a pan-European Analysis	Book published by Ashgate: Aldershot	EU
Howden-Chapman, P	2004	Housing standards: a glossary of housing and health	<i>Journal of Epidemiology and Community Health</i> ; 58: 162-8	International

**General evidence** (*what effects and relationships are identified in literature?*)

Cold exposure/stress is a statistically significant factor in all-cause excess winter mortality, especially mortality from cardiovascular and respiratory diseases.

The main causes of excess mortality in the winter season are: ischaemic heart disease and cerebrovascular disease; these two causes of death account for approximately 85% of all mortality cases. The commonly-held assertion that pneumonia is a major cause of excess winter mortality is erroneous, with less than 5% of excess winter deaths caused by same.

Cold housing is also linked with a number of cardiovascular and respiratory morbidity outcomes, as well as increases in psychological conditions such as depression.

**Specific evidence** (*how strong are the effects, which quantification has been identified?*)

Cold stress from both indoors and outdoors are equally important factors in explaining seasonal mortality.

Pan-European research indicates a strong inverse climatic gradient. Multivariate regression analysis has indicated that the gradient for levels of excess winter mortality is at its highest in countries with the mildest winters and lowest in the coldest countries. The thermal efficiency of housing has been attributed formally as a causal factor in the multivariate research.

There are several other statistically significant factors related to excess winter mortality, including macroeconomic and socio-economic factors (e.g. per-capita GDP, income inequality) and healthcare provision.

There is a very strong relationship between cold indoor temperature, cold stress and mortality effects in the over-65 age cohort, with up to 90% of excess winter deaths occurring in the over-65 population.

*Ceterus paribus*, the proportion of cold-related excess deaths resulting from indoor, rather than outdoor, cold stress has been put at between 44% and 50%. However, there is little corroboration of these findings and limited scientific backing.

There is a relationship between cold indoor temperature and a number of morbidity effects, though it is difficult to disentangle due to the ecological nature of studies. Irish research indicated that households enduring cold (or 'fuel-poor' households) were over three times as likely to report respiratory conditions and almost three times as likely to self-perceive ill-health caused by cold housing.

**Limitations** (*are there any constraints and limitations associated with the data?*)

There are several limitations related mainly to the ecological design of such studies (see overleaf).

The macro-level datasets employed in many of the studies in this field make it difficult to disentangle effects, and there is probably a high degree of multicollinearity.

**Suggested OR / incidence / attributable fraction for exposed population** (*what seems to be the most reliable and evidence-based estimation on the likelihood that the health effect is expressed?*)

Cold stress from indoors is *probably* accountable for between a third and a half of all excess winter deaths.

Final recommendation

**There is some / partially sufficient and reliable evidence for making a valid assessment**

**Part II: Exposure situation**

**Data sources used**

Priority should be given to international and/or European-based studies in peer-reviewed literature

Author(s)	Year	Title	Journal / Book	Location
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	<i>Lancet</i> ; 349: 1341-6	EU
Gemmell, I <i>et al.</i>	2000	Seasonal variation in mortality in Scotland	<i>International Journal of Epidemiology</i> ; 29: 274-9	Scotland
Laake, K and Sverre, JM	1996	Winter excess mortality: a comparison between Norway and England plus Wales	<i>Age and Ageing</i> ; 25: 343-348	Norway, England, Wales
McKee, M, <i>et al.</i>	1998	Seasonal variation in mortality in Moscow	<i>Journal of Public Health Medicine</i> ; 20: 268-74	Russia
Wilkinson, P <i>et al.</i>	2001	Cold comfort: the social and environmental determinants of excess winter deaths in England, 1986-1996	Book published by the Policy Press, London	England

**General evidence** (*is a relevant exposure identified in the literature?*)

Yes, there are some dose-response functions identified and they are generally expressed as the number of extra deaths or variations in mortality risk per one degree Celsius fall in ambient temperature.

The dose-response rate is dependent on mean winter environmental temperature, and thus, there is a high degree of clinical heterogeneity.

**Specific evidence** (*how large is the exposure, which quantification has been identified?*)

A recent Russian study demonstrated that the gradient of deaths from cerebrovascular and ischaemic heart disease (combined) against temperature is of the magnitude of 0.7% per 1°C fall in temperature, (McKee *et al.*).

All-cause mortality was found to rise by around 2% for each degree Celsius fall in outdoor temperature below 19° Celsius in the UK (Wilkinson *et al.*).

Increases in all-cause mortality per 1°C fall in temperature were calculated to be: 0.3% in Finland, 0.6% in Germany and the Netherlands respectively, 1.4% in the UK, 1.5% in southern Italy and 2.2% in Greece (Eurowinter).

Scottish data indicate a 1% rise in all-cause deaths resulting from a 1°C fall in mean temperature with a one-week time lag between dose and response (Gemmell *et al.*).

The risk of death relative to the summer minimum was about 1.5:1 in the coldest homes and about 1.3:1 in the warmest homes (Wilkinson).

English and Welsh data from the early 1990s reported an increase of approximately 3,500 deaths in England and Wales (approximately 1/1,000 in the population aged 45 years and over) per 1 degree Celsius reduction in winter temperature, after adjustment for age and influenza. This amounts to an increase of 2.1% increase in excess winter mortality per 1°C fall in environmental temperature (Lakke and Sverre).

**Limitations** (*are there any constraints and limitations associated with the data? Are there large variations within countries or by country or regions?*)

There are several limitations related mainly to the ecological design of such studies. There is a some degree of *statistical heterogeneity* (differences in the reported effects), *methodological heterogeneity* (differences in study design) and *clinical heterogeneity* (differences between studies in key characteristics of the participants, interventions or outcome measures) which makes interpretation of findings complex, and the identification of a dose-response function challenging.

**Suggested exposure rate in the (European) population** (*what seems to be the most reliable and evidence-based estimation on the general exposure within the population?*)

The exposure depends on geography/climate. A 1-2% rise in all-cause mortality per 1°C fall in temperature is a reliable dose-response function for 'median' climates in Europe. Very cold climates demonstrate lower dose-response rates, typically less than 1% per 1°C fall in temperature. Milder, Mediterranean climates demonstrate higher exposure rates of 2% or more.

The dose-response rates found in the epidemiology literature corroborate the inverse/'paradoxical' climate gradient identified in the health effects literature.

Final recommendation

**There is sufficient and reliable evidence for making a valid assessment**



## Summary of main element of evidence between:

**Cause/ Housing Factor: Cats, dogs, mites**  
**Health Effects: Respiratory and allergic effects**

Contributor: Joachim Heinrich, Germany

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### Part I: Health effects / Attributable risk / possible exposure-response relationship

#### Data sources used

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- Bornehag CG, Sundell J, Hagerhed L, Janson S; DBH Study Group. Pet-keeping in early childhood and airway, nose and skin symptoms later in life. *Allergy*. 2003 Sep;58(9):939-44.
- Braback L, Kjellman NI, Sandin A, Björkstén B. Atopy among schoolchildren in northern and southern Sweden in relation to pet ownership and early life events. *Pediatr Allergy Immunol* 2001; 12: 4-10.
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- de Meer G, Toelle BG, Ng K, Tovey E, Marks GB. Presence and timing of cat ownership by age 18 and the effect on atopy and asthma at age 28. *J Allergy Clin Immunol*. 2004 Mar;113(3):433-8.
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**General evidence** (*what effects and relationships are identified in literature?*)

Early exposure to dog(s) and cat(s) are mostly associated with decreased risks for the development of allergic sensitization against common aeroallergens, of atopic dermatitis, hay fever, and asthma. Studies on early exposure to cat and their association with cat specific IgE showed conflicting results. Cohort studies showed that increased early exposure to mite (and

cat allergen) was associated with increased risk of specific IgE to mite and cat allergens in school-aged children. No strong and consistent effect was found for development of asthma. Exacerbation of asthma might be affected by exposure to mite and cat allergens.

**Specific evidence** (*how strong are the effects, which quantification has been identified?*)

The protective effect estimates for early cat and dog exposure are quite strong, the increased risk for specific allergic sensitization with increasing mite and cat allergen exposure as well.

**Limitations** (*are there any constraints and limitations associated with the data?*)

The major concerns with regard the apparently protective cat and dog exposure effects come from bias by potential reverse causation. Families with a cat or a dog have no (or less) pet allergy and consequently show a protective effect.

**Suggested OR / incidence / attributable fraction for exposed population** (*what seems to be the most reliable and evidence-based estimation on the likelihood that the health effect is expressed?*)

Final recommendation

**There is some / partially sufficient and reliable evidence for making a valid assessment**

## **Part II: Exposure situation**

### **Data sources used**

See papers referenced in Part I.

Zock JP et al. 2006. Distribution and determinants of house dust mite allergens in Europe.

Heinrich J et al. 2006. Cat allergen levels in mattress dust and its predictors across Europe.

### **General evidence** (*is a relevant exposure identified in the literature?*)

Pet ownership is very common in Europe, but varied between countries. Cat allergen levels in Europe reflect cat ownership and housing conditions (in non-cat owners). Mite allergen high frequencies of levels range from high frequencies of non detectable concentrations (Iceland, Nordic countries) and high levels in UK. Besides climatic conditions housing also affects mite allergen levels in particular Der p 1.

### **Specific evidence** (*how large is the exposure, which quantification has been identified?*)

The effects of housing factors on cat and mite allergen levels are small. Other factors such as cat ownership (cat allergen) and climatic factors (Der p 1 allergen) have much stronger influences on allergen levels indoors.

**Limitations** (*are there any constraints and limitations associated with the data? Are there large variation within countries or by country or regions?*)

Major limitations comparing allergen levels between countries come from non-standardized methods in dust collection, dust extraction and assay between each single study. The ECRHS II is the only study on a European level which used identical methods for each country-specific location. Within the ISAAC also indoor dust was standardized collected and is assayed for allergens.

**Suggested exposure rate in the (European) population** (*what seems to be the most reliable and evidence-based estimation on the general exposure within the population?*)

Final recommendation

**There is some / partially sufficient and reliable evidence for making a valid assessment**

## Summary of main element of evidence between:

**Cause/ Housing Factor: Poverty and housing deprivation**  
**Health Effects: General health effects**

Contributor: Philippa Howden-Chapman, New Zealand

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### Introduction

Inadequate housing is one of the key mediating pathways between low income and poor health. In this short paper, I have tried to avoid the conceptually incorrect tendency of viewing marginal groups such as ‘the poor’ as “problem groups, different and separate from the rest of society” (Rose 1992) and considered the available evidence that there is a continuum of inadequate housing.

In general, there is moderately strong evidence that inadequate housing is related to poorer health, but the concept of *inadequate* housing is often conflated with *unaffordable* housing and many studies fail to control for more than one measure of socio-economic status known to be associated with poorer health, e.g. income, education or employment status (Graham 2001). Most studies also fail to adjust for demographic status, which is clearly associated with health status, e.g. age, ethnicity, gender and type of household or individual risk behaviour such as smoking. All these characteristics are likely to interact and in many cases restrict housing choice. For example, people from ethnic minorities with disability are more likely to suffer double discrimination (Harrison and Davis 2001).

Furthermore, most studies are cross-sectional rather than longitudinal and there are very few experimental studies available (Thomson, Petticrew et al. 2001). There are also few multi-level studies or community-based studies, that include data on inadequate housing, which control for national or regional policies, household and individual effects. Inadequate housing is strongly associated with socio-economic deprivation at a neighbourhood level, but here I will exclude the compounding effect of location and neighbourhood.

### Promising research areas

The following areas, which are strongly associated with poverty, have been reasonably well researched and moreover provide important levers for policy action will be briefly reviewed: the quality of the housing stock and multiple housing exposure; re-housing, housing tenure patterns and levels of social housing; energy efficiency; fuel poverty and household crowding.

### **The quality of the housing stock<sup>1</sup>**

The 1996 English House Condition Survey (EHCS) showed that that 1,522,000 UK dwellings did not meet the required fitness standards and only 14% of the total had satisfactory energy efficiency (SAP) ratings (Department of the Environment Transport and

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<sup>1</sup> I have excluded the extensive literature on lead poisoning as this is being covered in a separate paper.

the Regions 2000). Although, except for the presence of mould, selected housing variables from the EHCS have shown little agreement with census-deprived variables at electoral ward level (Aylin, Morris et al. 2001). Recently, the quality of the housing stock has been measured in a cross-sectional survey of 14 EU countries using the European Community Household Panel and, while generally improving, was found to be particularly problematic in Portugal, Spain and Greece for a substantial number of households (Healy 2004).

A broader concept of *multiple housing deprivation* refers to the experience of poor housing over the course of a life-time. Poor housing conditions in infancy, childhood and adulthood such as overcrowding and damp, cold dwellings, have a cumulative detrimental effect on physical and mental health and appears to pose health risks of similar proportions to smoking and, on average, greater than that posed by excessive alcohol consumption (Marsh, Gordon et al. 1999). Children appear to be particularly vulnerable to prolonged exposure to poor housing. Associations between poorer housing conditions in childhood and mortality from common diseases in adulthood are not strong, but are distinguishable from other aspects of social and economic deprivation (Dedman, Gunnell et al. 2001) (Wilner, Price-Walkley et al. 1960; Dedman, Gunnell et al. 2001).

Fires are a major cause of mortality, particularly among poorer people who because of their social and economic circumstances are more likely to smoke. (Runyan, Bangdiwala et al. 1992; Roberts 1995; DiGiuseppi, Roberts et al. 1998; Istre, McCoy et al. 2001).

#### Rehousing

While it is impossible to specify the nature and the size of the health gain, several before and after UK studies have shown that the mental health of tenants has improved after housing renewal or community regeneration, (Wilner, Walkley et al. 1960; Hopton and Hunt 1996; Smith, Alexander et al. 1997; Ambrose 2000) although some have reported adverse effects on general health after rehousing. (Smith, Smith et al. 1993; Blackman and Harvey 2001) In the USA, preliminary results of experimental relocation of families from areas of deprivation to improved housing in middle-income areas have shown social and health gains (Katz, Kling et al. 2001) and in another study, when the shift was to private housing, an improvement in mental health. (Leventhal and Brooks-Gunn 2003)

#### **Housing tenure and levels of social housing**

People on low incomes are less likely to own their own homes. Households' tenure choices –whether to rent or to buy- are affected by a number of financial institutions, private and governmental, as well as the available levels of social housing.

Robust cross-country evidence shows that people who rent rather than own their own houses report poorer health status, although the mechanism is still unclear. Several British studies have shown more variation in mortality between owners and tenants within occupational social class groups than between social classes within tenure classes. These relationships are also related to self-perceived health, rates of long-term illness and general practitioner consultation rates (Macintyre, Ellaway et al. 1998; Macintyre, Hiscock et al. 2001).

There seems to be a dearth of comparative case literature on the impact of the large variations in social housing in Europe and how this impacts on housing costs for those on

low and middle incomes. Similarly, arrangements for paying for heating vary, for example, unlike the UK, heating is usually included in the rent in Norway (Laake and Sverre 1996) and the Netherlands.

### **Energy efficiency**

People on low incomes are more likely to live in older houses so that disentangling the effect of income and housing is problematic. Older homes are less likely to be thermally efficient and therefore are less likely to afford protection against the cold and are less likely to have central heating. English evidence suggests that when housing tenure, being on state benefits, being in an energy-efficient house were adjusted for, these factors were *not* associated with indoor temperature, although individually they were each important determinants of indoor temperature (Wilkinson, Landon et al. 2001).

Insulating existing homes, providing effective safe heaters, and where necessary subsidised power, has been shown theoretically (Levy, Nishioka et al. 2003) and in practice, to increase older people's health and well-being (Keatinge 1986; Keatinge, Coleshaw et al. 1989; Heckman and Smith 1995; Hopton and Hunt 1996; Keatinge, Donaldson et al. 1997; Thomson, Petticrew et al. 2001; Thomson, Pettigrew et al. 2003) and the health of children with asthma (Somerville, Mackenzie et al. 2000). In some cases the intervention prevented only further deterioration in health (Hopton and Hunt 1996).

The New Zealand Housing, Insulation and Health Study showed in a community trial that retrofitting insulation to older houses showed a small, but significant effect of an increase in indoor temperature on objective and subjective measures of temperature and comfort, self-perceived health, days off school and work and a trend to reducing hospitalisation for respiratory disease. Although some energy rebound occurs (benefits taken as lower fuel bills) energy usage was also significantly reduced (Howden-Chapman, Crane et al. 2005; Howden-Chapman, Matheson et al. 2005). Excluding any excess winter mortality, this study showed a two-to-one benefit to cost ratio.

Damp houses are harder to heat and are more likely to have mould.<sup>2</sup> A New Zealand survey found that a third of houses reported mould and in a multiple regression analysis, both the condition of the house ( $p < 0.01$ ) and the usual number of residents ( $p < 0.01$ ) were significantly associated with mould, after controlling for tenure, insulation and owning a dehumidifier (Howden-Chapman, Saville-Smith et al. 2005).

### **Fuel poverty and excess winter morbidity and mortality**

Fuel poverty is the inability to afford inadequate warmth depends on the building structure and heating system as well as household income (Boardman 1991). It has been estimated that about 16% of UK households are suffering from fuel poverty (Department of the Environment Food and Rural Affairs and the Department of Trade and Industry 2001) and the highest incidence is found among the long-term ill and disabled.

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<sup>2</sup> I have also excluded the extensive literature on mould as this is being covered in a separate paper.

While the poorest individuals tend, in Ireland at least, spend three times more than the average on energy relative to income (Clinch and Healy 1999), overall there is only limited evidence directly linking health outcomes with fuel poverty and most of the evidence is indirect and related to excess winter mortality and morbidity. Excess winter mortality is about a fifth higher in temperate countries with comparatively mild winters such as Britain (Curwen 1990/91), England (Wilkinson, Landon et al. 2001), Portugal (Healy 2004) and New Zealand (Isaacs and Donn 1993; Davie 2004) than in continental Europe and Scandinavia. Inadequate housing, in particular, cold indoor temperatures due to lack of central heating (as well as outdoor clothing (Donaldson, Ermakov et al. 1998)) has been suggested as an intervening variable, although there is contrary evidence related to differential exposure to outdoor cold stress (Keatinge 1986; Keatinge, Coleshaw et al. 1989; Donaldson and Keatinge 2003). Indeed, others have concluded that, rather than the introduction of central heating, socioeconomic progress has a more important role to play (Kunst, Looman et al. 1991).

Nonetheless, the Eurowinter Group found independent associations with home heating and outdoor cold stress (Eurowinter Group 1997). The inverse relationship between excess winter mortality with outdoor temperature and central heating has also been shown for older people in Britain (Aylin, Morris et al. 2001) where there was a 1.5% higher odds of dying in winter for every 1°C decrease in the 24-h mean winter temperature below the 10-year national average, and for all ages in England (Wilkinson, Landon et al. 2001) where the coldest homes had a risk around 20% greater than the warmest homes and mortality was found to rise by around 2% for each degree Celsius fall in outdoor temperature below 19°C. A US study has also showed a strong association of the temperature-mortality relation with latitude, with a greater effect of cold temperatures on mortality risk in more southern cities and of warmer temperatures in more northern cities. The percentage of air conditioners in the south and heaters in the north, which is used as an indicator of SES, also predicted weather-related mortality (Curriero, Heiner et al. 2002).

However, at an ecological level, there is little evidence of a social gradient on excess winter mortality (Gemmell 2001), whether measured by deprivation (Shah and Peacock 1999) or rurality (Lawlor, Maxwell et al. 2002) although excess winter deaths rise sharply with age. A Scottish study measuring multiple deprivations did show an effect (Stirling, Howieson et al. 2005). Population-based studies have also failed to find a socioeconomic gradient in excess winter mortality (Wilkinson, Pattenden et al. 2004). It may be, as most of this work has been done in the UK that the relatively high standards of social housing are confounding these relationships.

There has been less work on vulnerability to excess winter morbidity, but recent research has identified a 1.7 higher risk in some years among older people at risk of cold homes in London (Rudge and Glilchrist 2005) .

### **Household crowding**

People who are tenants on low incomes face financial pressures to lower the rent per person, so that crowding is the 'rational' economic decision. (The corollary under-occupancy, people living in houses that are too large for their needs and may be difficult to heat is also a problem).



Crowding has long been known to increase the risk of infectious diseases, such as tuberculosis (Coetzee, Yach et al. 1988) and hepatitis B (Milne, Allwood et al. 1987). A case control study of meningococcal disease in New Zealand children under 8 years (1997-99: 202 cases and 313 controls) identified crowding (as defined by the Canadian National Occupancy Standard) as the key risk factor with odds ratio of 10.7, 95% CI 3.9 to 29.5 (Baker, McNicholas et al. 2000). Crowding also has an impact on mental health (Entner Wright, Caspi et al. 1998) and seems to be a contributor at an ecological level to premature mortality (Kellett 1993). A number of adult diseases, such as *Helicobacter pylori*, have been traced back to exposure to crowding in childhood. (Mendall, Goggin et al. 1992)

### **Effect size**

The strongest evidence for the effects of inadequate housing on the health of people on low income are in relation to cold indoor temperatures, damp and mould and crowding. The Housing, Crowding and Health Study of new tenants in social housing in New Zealand showed crowding decreased compared to those still on the waiting lists in 60% of households, showed no change in 30% and increased in crowding in 8% of households (Baker and Zhang 2005).

However, the nature of most of these studies means that temperature and presence of mould are reported subjectively and can therefore be misclassified. This is likely to underestimate the influence of home heating on the temperature-mortality relationship.

### **Exposure to the indoor domestic environment**

Cross-sectional surveys have found that people in the OECD spend most of their time inside (between 75% and 90%). Information about indoor exposure comes from time-use surveys, but most do not differentiate between settings, e.g. time spent in homes, offices or cars. An exception is (Healy 2004) where he reports that length of time spent shivering, an indication of thermal stress, is associated with inadequate housing.

The exposure of population groups to different indoor environments has rarely been assessed. Inferences are usually made indirectly from types of household and demographic characteristics. For example, sole mothers, the unemployed, the disabled and older people, all of whom are likely to be on lower incomes, are assumed to stay inside more than other groups. Therefore, inadequate housing is likely to have a more marked impact on these low-income groups.

### **Policy conclusions**

Adequate income is the main determinant of health, but safe, affordable housing for those on low incomes is in short supply in countries where there is little social housing. The private rental market often fails to supply adequate housing because it is capital gains rather than rental incomes that generate a landlord's profits. Landlords can be unresponsive to quality and health concerns in low-income housing. For example, making a capital investment like insulating the house and install sustainable heating, which can reduce mould and improve fuel poverty, only pays dividends for the landlord over a long-term number of years. This is a clear case for arguing that, where there is market failure in inadequate housing, national or local governments should intervene.

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## **Summary of main element of evidence between:**

**Cause/ Housing Factor: Environmental tobacco smoke**  
**Health Effects: Respiratory diseases and pregnancy outcomes**

Contributor: Marittaa Jaakkola, Finland / UK

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### **Part I: Health effects / Attributable risk / possible exposure-response relationship**

#### **Respiratory effects in children and effects on pregnancy outcomes**

From the health effect point of view it is meaningful to discuss separately children's prenatal and postnatal exposure to environmental tobacco smoke (ETS) and to divide exposure during pregnancy (prenatal exposure) further into mother's active smoking and non-smoking mother's ETS exposure during pregnancy. It has been shown that tobacco smoke constituents are transferred from the circulation of mother to the fetus through placenta as a consequence of both mother's active and passive smoking. The effects of all types of childhood exposures have been reviewed recently by Jaakkola and Jaakkola (Scan J Work Environ Health 2002; 28 Suppl 2: 71-83) and Lindbohm *et al.* (Scan J Work Environ Health 2002; 28 Suppl 2: 84-96).

#### ***Mother's active smoking during pregnancy***

Mother's active smoking has been associated with low birth weight (defined as <2500 grams), preterm delivery (<37 weeks) and spontaneous abortion. The birth weight of a newborn of smoking mother is estimated to be on average 150-200 grams lower compared to a newborn of non-smoking mother, and the risk of low birth weight is estimated to double in relation to mother's smoking. Recent cohort studies have also shown that mother's active smoking during pregnancy is a significant determinant of childhood asthma, even after adjusting for potential confounders including low birth weight. Some studies have also linked mother's active smoking to significantly reduced lung function in newborns and in school-age children.

#### ***Mother's ETS exposure during pregnancy***

Mother's passive smoking during pregnancy has been linked to both low birth weight and to preterm delivery, the evidence being stronger for low birth weight. Two meta-analyses assessed based on 11 studies that the birth weight of newborns of ETS exposed mothers is significantly lower compared to newborns of unexposed mothers, the estimated effect being -31 grams (-44 to -19 grams). The risk of low birth weight (LBW) or small for gestational age (SGA) was estimated as 1.19 (1.08-1.32). A recent study of approximately 400 nonsmoking women from Finland assessed their ETS exposure during pregnancy based on hair nicotine concentration (in µg/g) (Jaakkola *et al.* Environ Health Perspect 2001; 109: 557-61). The risk of both LBW and SGA was increased in relation to mother's ETS exposure at home and at work. When grading the exposure into no or low (<0.75), moderate (0.75 to <4.00) and high ( $\geq 4$ ) categories according to hair nicotine, the following dose-response relations with adjusted OR were observed:

<b>Exposure</b>	<b>OR for LBW</b>	<b>OR for SGA</b>	<b>OR for preterm delivery</b>
No/low	1	1	1
Moderate	1.28 (0.59-2.60)	1.05 (0.44-2.49)	1.30 (0.30-5.58)
High	1.55 (0.55-4.43)	1.18 (0.34-4.19)	6.12 (1.31-28.7)

Only a few studies have addressed the role of mother's passive smoking for respiratory diseases in children. There is some evidence that mother's ETS exposure during pregnancy is related to reduced lung function in newborns and perhaps also later in childhood and increased risk of asthma in school-age children. In addition, low birth weight that has been associated with mother's ETS exposure is a risk factor for development of asthma in children.

### ***Children's postnatal ETS exposure***

Since the first reports of the effect of parents' smoking on children's respiratory symptoms and infections were published in 1970s, abundant evidence has accumulated on the adverse health effects related to children's exposure to ETS. Tens of studies from different parts of world have been published and rather recently several meta-analyses have been carried out. These effects were reviewed recently by Jaakkola and Jaakkola (Scan J Work Environ Health 2002; 28 Suppl 2: 71-83).

### **Respiratory symptoms**

There is convincing evidence that parents' smoking increases the risk of all chronic respiratory symptoms in children. The estimated ORs related to either parent (mother or father) smoking were in a recent meta-analysis: 1.24 (1.17-1.31) for wheezing, 1.40 (1.27-1.53) for cough, 1.35 (1.13-1.62) for phlegm production, and 1.31 (1.08-1.59) for breathlessness (Cook *et al.* Thorax 1997; 52: 1081-94). There is evidence that the risk of symptoms increase with increasing number of household smokers and with increasing number of cigarettes smoked inside home.

### **Asthma**

More than 40 studies have addressed the role of ETS exposure for development of asthma in children and several meta-analyses on these have been published (California Environmental Protection Agency 1997; Strachan *et al.* Thorax 1998; 53: 204-12.). The increased risk of asthma related to household smoking seems to be strongest in youngest children, but remains statistically significant also in older children and in teenagers. Studies from Europe, USA, and Australia also provide strong evidence that ETS exposure is related to bronchial hyperresponsiveness in children. The risk estimates from recent meta-analyses according to study design are:

<b>Study design</b>	<b>Age group</b>	<b>OR 95% CI</b>
Cross-sectional	School age	1.361.20-1.55
Case-control	1-18 years	1.591.27-1.99
Longitudinal	<3 years	2.081.59-2.71
	5-7 years	1.311.22-1.41
	1-17 years	1.131.04-1.22
Bronchial hyper-responsiveness	School age	1.291.10-1.50

Longitudinal studies published after these meta-analyses have confirmed a significant relation between ETS exposure at home and asthma in children (Nafstad *et al.* *Epidemiology* 1997; 8: 293-7; Gold *et al.* *Am J Respir Crit Care Med* 1999; 160:227-36). Based on the evidence it seems reasonable to conclude that ETS exposure is causally link to asthma in children.

### **Respiratory infections**

A large number of studies have investigated the relations between childhood ETS exposure and lower respiratory tract infections, including acute bronchitis, bronchiolitis and pneumonia. Sometimes also respiratory symptoms such as wheezing and cough have been included in this outcome. There is consistent evidence that ETS exposure increases significantly the risk of lower respiratory infections, the risk being highest in early childhood. A meta-analysis based on 24 community-based studies in gave a summary OR of 1.57 (1.42-1.74) for either parent smoking, and 1.72 (1.55-1.91) for maternal smoking (Strachan *et al.* *Thorax* 1997; 52: 905-14). Several studies have found a dose-response relation between the risk of infections and increasing exposure, measured as increasing number of smokers or increasing number of cigarettes smoked at home. More recent cohort studies have confirmed these findings (Nafstad *et al.* *Eur Respir J* 1996; 2623-9). In addition, there is increasing evidence suggesting that childhood ETS exposure is related to increased risk of middle ear infections, estimated OR from meta-analyses being 1.66 (1.33-2.06) for acute otitis media and 1.41 (1.19-1.66) for recurrent otitis media.

### **Lung function**

More than 40 cross-sectional studies have investigated the effect of parents' smoking on lung function levels in children, while less than 10 studies have evaluated the effects longitudinally. Systematic quantitative reviews have indicated that childhood ETS exposure is related to small significant deficit in children's spirometric lung function. A meta-analysis including 21 studies estimated that forced expiratory volume in 1 second (FEV1) of exposed children is 1.4% (1.0-1.9%) lower than that of unexposed children (calculated as difference in lung function between exposed and unexposed expressed as % of lung function in unexposed) (Cook *et al.* *Thorax* 1998; 53: 884-93). The corresponding effect estimates for FVC and MEF were 0.4% (0.0-0.8%) and 5.0% (3.3-6.6%), respectively. In many studies, the effect of maternal smoking was stronger than that of other household smokers. The presence of two smokers at home had a stronger effect than mother's smoking alone. In the largest longitudinal study including more than 8000 children from six U.S. cities, the effect estimates related to maternal smoking were -3.8 ml/year (-6.4 - -1.1) for FEV1, -2.8 ml/year (-5.45 - 0.0) for FVC, and -14.3 ml/s /year (-29.0 - 0.3) for FEF25-75 (Wang *et al.* *Am J Respir Crit Care Med* 1994; 149: 1420-5).

### **Childhood exposure and adult respiratory diseases**

More recently, studies have also addressed the question, whether childhood exposure to ETS is related to respiratory diseases in adulthood. In the case of lung cancer, the studies have provided somewhat contradictory results, but a recent meta-analysis performed by IARC in 2004 (IARC Monograph 83, 2004) estimated that in women the OR of lung cancer related to mother's smoking in childhood was 1.50 (1.04-2.14) based on 9 studies and that related to

father's smoking 1.25 (0.94-1.60) based on 10 studies. Some cross-sectional studies have investigated the effect of childhood exposure on asthma and COPD later in life. These have provided inconsistent results. A Norwegian 11-year follow-up study of almost 3000 subjects 15-70 years of age at baseline estimated for adult asthma an OR of 2.9 (1.6-5.5) in relation to mother's smoking during pregnancy, an OR of 1.9 (1.1-3.2) in relation to mother's smoking postnatally in childhood, an OR of 3.5 (1.8-6.8) for combined exposure, and an OR of 1.2 (0.7- 2.0) in relation to other household smokers (Skorge *et al.* Am J Respir Crit Care Med 2005; 172: 61-66).

### **Respiratory effects in adults**

The first reports that linked spouse's smoking to lung cancer in women were published in 1981. After that an increasing number of studies on ETS exposure and lung cancer have been published and these were reviewed recently by IARC (IARC Monograph 83, 2004). Fewer studies have been published on ETS and other respiratory diseases in adult populations, but more recently the interest in these has increased. These studies were reviewed recently by Jaakkola and Jaakkola (Scan J Work Environ Health 2002; 28 Suppl 2: 52-70).

### **Lung cancer**

To date 8 cohort studies and about 50 case-control studies from different parts of world have investigated the relation between ETS exposure and lung cancer. These have assessed ETS exposure at home and/or at work, these two being those environments where adults spend most of their time. Several meta-analyses have been performed, the most recent ones by Boffetta (Scan J Work Environ Health 2002; 28 Suppl 2: 30-40) and by IARC (IARC Monograph 83, 2004). The results show a small, but significantly increased risk related to ETS exposure, as summarized here:

<b>Type of exposure</b>	<b>Gender</b>	<b>OR (95% CI) by Boffetta</b>	<b>OR (95% CI) by IARC</b>
ETS at home (Spouse smoking)	Women	1.25 (1.14-1.38)	1.24 (1.14-1.34)
	Men	1.25 (0.95-1.65)	1.37 (1.02-1.83)
	Combined	1.34 (0.72-2.49)	
Work ETS	Women	1.17 (1.02-1.33)	1.19 {1.09-1.30}
	Men	1.23 (0.78-1.94)	1.12 (0.80-1.56)
	Combined	1.17 (1.04-1.32)	

Studies have shown evidence of dose-response relation between increasing lung cancer risk and increasing ETS exposure, measured as number of household smokers or smoking co-workers, duration of exposure, or cumulative exposure. Recent studies have adjusted extensively for confounders, including diet and other lifestyle factors and socioeconomic status, and some studies have adjusted for potential misclassification of ETS exposure. These adjustments have not altered the effect estimates substantively. In summary, the evidence is consistent with ETS exposure being a cause of lung cancer.



### **Asthma**

There are fewer studies on ETS exposure and asthma in adult populations than in children, but recently there has been an increasing interest in this topic. To date one longitudinal, one incident case-control, two prevalent case-control and about five cross-sectional studies have addressed this question in adults. All of the studies reported increased risk of asthma in relation to ETS exposure, although this was not statistically significant in all of them. The ORs from these studies have been between 1.1 and 4.7. The 10-year longitudinal study from California reported an OR of 1.45 (1.21-1.80) for workplace exposure, while home exposure was not related to a significantly increased risk (Greer *et al.* J Occup Med 1993; 35: 909-15). The Finnish population-based case-control study with clinically confirmed, new adult-onset cases of asthma reported an adjusted OR of 2.16 (1.26-3.72) for workplace ETS exposure and 4.77 (1.29-17.7) for home exposure in the last 12 months (Jaakkola *et al.* 2003; 93:2055-60). Adjusted OR for combined exposure was 1.97 (1.19-3.25). There was evidence of dose-response relation between cumulative exposure at home/ combined cumulative exposure and the risk of asthma.

### **COPD**

To date approximately 8 studies have addressed the role of ETS exposure for chronic obstructive pulmonary disease (COPD), half of them being longitudinal, 3 case-control and 1 cross-sectional study. The ORs from the studies have varied between 1.3 and 5.5. The most recent report was from a population-based sample of 2,113 U.S. adults 55-75 years of age, which defined COPD as doctor-diagnosed chronic bronchitis, emphysema or COPD (Eisner *et al.* Environ Health 2005; 4: 7-14). The adjusted OR for the highest quartile of lifetime home ETS was 1.55 (1.09-2.21) and for the highest quartile of lifetime work exposure 1.36 (1.002-1.84).

### **Respiratory symptoms**

The relations between ETS exposure and chronic respiratory symptoms have been addressed in approximately 16 cross-sectional and 2 longitudinal studies of adult populations. These have provided consistent evidence that both ETS exposure at home and at work are significantly related to increased risk of all chronic respiratory symptoms, including cough, phlegm production, wheezing and dyspnoea. In a large cross-sectional study from Switzerland, the adjusted OR for any ETS exposure was 1.69 (1.23-2.31) for phlegm production, 1.99 (1.41-2.82) for wheezing and 1.44 (1.18-1.75) for dyspnea (Leuenberger *et al.* Am J Respir Crit Care Med 1994; 150: 1222-8). An 8-year longitudinal study of 117 Canadian young adults reported the following adjusted ORs related to combined home and work ETS exposure: 1.55 (0.61-3.90) for cough, 1.15 (0.64-2.06) for wheezing, and 2.37 (1.25-4.51) for dyspnoea (Jaakkola *et al.* J Clin Epidemiol 1996; 49: 581-6).

### **Lung function**

To date approximately 20 cross-sectional, 1 case-control and 4 longitudinal studies have investigated the relation between ETS exposure and lung function in adults. In a recent meta-analysis based on 9 cross-sectional studies ETS exposure was found to be related to a small, but significant reduction in FEV1 (Carey *et al.* Epidemiology 1999; 10: 319-26). The estimated effect of ETS on FEV1 was -2.7% (-4.1 - -1.2%) (defined as the difference in FEV1 between the exposed and unexposed, expressed as a % of the level in the unexposed). The few longitudinal studies published on this topic have not found any significant effect of

ETS exposure on development of spirometric lung function in adults, but this may be explained by the relatively low ETS concentrations in these studies.

### **Pneumonia**

To date only one published study has investigated the relation between ETS and pneumonia in adults. This was a population-based case-control study from USA evaluating the relation between tobacco smoke exposure and invasive pneumococcal infections (Nuorti *et al.* N Engl J Med 2000; 342: 681-9). The adjusted OR in relation to ETS exposure was 2.5 (1.2-5.1).

Final recommendation

**There is sufficient and reliable evidence for making a valid assessment for asthma, respiratory infections, respiratory symptoms and low birth weight in children, and for lung cancer, asthma and respiratory symptoms in adults.**

**There is some / partially sufficient and reliable evidence for making a valid assessment for lung function impairment in children and for preterm delivery, and for lung function impairment, COPD and pneumonia in adults.**

### **Part II: Exposure situation**

Environmental tobacco smoke (ETS) exposure is defined as exposure of a non-smoker to tobacco combustion products from smoking by others (Jaakkola and Jaakkola, Eur Respir J 1997; 10: 2384-97). Commonly used synonyms for this are passive smoking, involuntary smoking and exposure to secondhand smoke (SHS). ETS is a combination of sidestream smoke (SS), defined as smoke released directly into environment from the burning end of cigarette between puffs, and exhaled mainstream smoke (MS). MS is smoke inhaled by the smoker during puff drawing. SS forms about 80% of ETS. 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.

Exposure to ETS can be measured directly by measuring personal exposure to tobacco smoke constituents, such as nicotine and respirable suspended particles (RSP), with personal monitors, or indirectly by questionnaires and interviews, by measuring tobacco smoke constituents in different microenvironments, or by measuring biomarkers. Biomarkers are proxies for dose rather than for exposure. The most commonly used biomarkers are cotinine in body fluids and hair nicotine. Cotinine is a major metabolite of nicotine and has a half-life of about 20 hours in non-smokers. Hair nicotine measures exposure over the last 1-2 months.

Health effect studies have most commonly used questionnaire-based assessment of ETS exposure, since questionnaires enable assessment of past exposures which are usually relevant for health effects, and in addition, questionnaires are a cheap way for assessing exposure. Some studies have combined this with use of biomarkers.

Population-based studies in Europe and in USA have measured cotinine in non-smokers and have found that some degree of exposure to ETS is very common among non-smoking populations, since more 80% have had detectable levels of serum cotinine (Riboli *et al.* Cancer Causes Control 1990; 1: 243-52; Pirkle *et al.* JAMA 1996; 275: 1233-40).

The European Community Respiratory Health Survey (ECRHS) provides information on ETS exposure at home and at work in several European countries based on questionnaire assessment (Janson *et al.* Lancet 2001; 358: 2103-9). It shows that the proportion of exposed adults is very variable between European countries. The proportions of adults with any ETS exposure are the highest in Spain (56-76%), Italy (55-62%), and Netherlands (56-59%), while the lowest proportions are observed in Sweden (20-22%). When looking at exposure at home Spain again has the highest proportion of exposed adults (42-51%) and Sweden the lowest (9-15%). Similar pattern applies for workplace exposure. Finland was not part of ECRHS, but in a 15-year follow-up study of a population sample, ETS exposure in Finland declined among non-smoking women from 23% in 1985 to 13% in 2000, and among non-smoking men from 27% in 1985 to 14% in 2000 (Jousilahti and Helakorpi, Scand J Work Environ Health 2002; 28 suppl 2: 16-20).

For child populations I was not able to find a similar study covering a wide range of European countries, but based on separate publications, the following prevalences of ETS exposure have been reported for children: 42% in England and 60% in Scotland (Somerville *et al.* J Epidemiol Community Health 1988; 42: 105-10); 66% in the Netherlands (Dijkstra *et al.* Am Rev Respir Dis 1990; 142: 1172-8); and 7% in Finland, 15% in Sweden, 32% in Norway, 46% in Iceland and 47% in Denmark (Lund *et al.* Scand J Soc Med 1998; 26: 115-20).

ETS exposure in child and adult populations has been reviewed recently by IARC (IARC Monograph 83, 2004).

Final recommendation

**There is sufficient and reliable evidence for making a valid assessment for adults, for children a further literature search should be made. The prevalence of exposure is very variable across European countries.**

My overall conclusion is that ETS exposure is still in many European countries the most important preventable indoor pollutant.

**Summary of main element of evidence between:**

**Cause/ Housing Factor: Lead**  
**Health Effects: Adverse health effects**

Contributor: David E. Jacobs, USA / Mary Jean Brown, USA

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**Part I: Health Effects / Attributable Risk / Exposure-Response Relationship**

Health Effects considered: Reduced IQ, Cognitive Deficits, Adverse Neurobehaviorial Effects, Increased Hypertension, Probable Human Carcinogen, Criminality and others

Data Sources Used

Authors	Year	Title	Journal/Book	Location
Fewtrell L, Kaufman, R, Pruss-Ustun A.	2003	Lead: Assessing the environmental burden of disease at national and local levels	WHO Environmental Burden of Disease, Series 2	Global
Fewtrell, L, Pruss-Ustun A, Landrigan P, Ayuso-Mateos J.	2004	Estimating the global burden of disease of mild mental retardation and cardiovascular disease from environmental lead exposure	Env Research Feb;94(2): 120-33	Global
Lanphear et al.	2005	Low-level environmental lead exposure and children's intellectual function: An international pooled analysis	Env Health Perspectives 113:894-899 (2005)	Boston, Cincinnati, Cleveland, Mexico City, Port Pirie (Australia), Rochester NY, Yugoslavia
Jacobs DE	1995	Lead-Based Paint as a Major Source of Childhood Lead Poisoning: A Review of the Evidence (Book Chapter)	<u>Lead In Paint, Soil and Dust: Health Risks, Exposure Studies, Control Measures and Quality Assurance</u> , ASTM, Philadelphia, p. 175-187, 1995.	Global
Nevin R	2000	How lead exposure relates to temporal changes in IQ, violent crime, and unwed pregnancy,	Environmental Research A83, 1-22.	
National Academy of Sciences	1993	Measuring Lead Exposure in Infants, Children, and Other Sensitive Populations	Book	Global
ATSDR	Sept 2005	Toxicological Profile for Lead (Draft Update)	Government Publication <a href="http://www.atsdr.cdc.gov/toxprofiles/tp13.html">http://www.atsdr.cdc.gov/toxprofiles/tp13.html</a>	Global
Dietrich et al.	2001	Early Exposure to Lead and Juvenile Delinquency	Neurotoxicology and Teratology 23, 511-518	Cincinnati (USA)
Bellinger DC and Needleman HL.	2003	Intellectual impairment and blood lead levels.	N Engl J Med 2003;349:500	Boston

## General Evidence

The literature on lead toxicology is large and the evidence of adverse health effects from lead exposure is substantial for both children and adults. In previous impressive work, WHO estimated global blood lead levels for all WHO regions and estimated 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 16<sup>th</sup> position in leading risk factors at the global level. In children, the neurological, cognitive and developmental problems are of principal concern, although numerous other harmful effects have also been documented. Cognitive deficits have also been associated with blood lead levels in the elderly. Other adverse health effects include anemia, decreased renal function, gastrointestinal effects, adverse reproductive health and at higher exposure levels, reduced stature, hearing loss, encephalopathy, seizures, coma and death. While fatalities are now rare, several cases have been associated with exposure to lead-based paint hazards in housing in Great Britain, France and the U.S. over the past few decades. Neurobehavioral problems include reduced IQ, attention deficits, hyperactivity, reduced organizational skills, and aggression and other anti-social behavior. Several studies have linked childhood lead exposure with juvenile delinquency and criminal behavior in later life. One longitudinal study showed that childhood blood lead and self-reported delinquent behavior had an  $r$  square value of 0.055, after controlling for a large number of confounding variables (i.e., 6% of the variation in delinquent behavior can be explained by blood lead level in childhood). Another study demonstrates a secular trend in lead exposure and crime rates over many decades. Finally, inorganic lead has been classified as a probable human carcinogen by both IARC and U.S. EPA.

## Specific Evidence

The earlier WHO analysis used a linear relationship of 2.6 IQ points lost per 10  $\mu\text{g}/\text{dL}$  blood lead interval for blood lead levels between 5 and 20  $\mu\text{g}/\text{dL}$  and a loss of 3.5 IQ points for blood lead levels above 20  $\mu\text{g}/\text{dL}$ , based on a meta analysis from 1994. However, a more recent international pooled analysis published in 2005 shows that the relationship is *not* linear and that IQ decrements are far higher in the first 10  $\mu\text{g}/\text{dL}$  increment, where exposures for the bulk of the world's population is. Using a log-linear model, the new pooled analysis found an IQ decrement of 3.9 points for an increase in blood lead level from 2.5  $\mu\text{g}/\text{dL}$  to 10  $\mu\text{g}/\text{dL}$  and progressively smaller decrements at higher blood lead levels. At the lower blood lead ranges, this represents an approximate increase of 66% ( $2.6/3.9 = .67$ ) in the IQ/blood lead inverse relationship. In short, the previous WHO estimate should be updated to reflect the log-linear model and the higher IQ/blood lead slope estimate. This would be expected to increase the global burden of disease due to lead exposure and also make the exposure range of interest more consistent with prevailing exposures in Europe and the U.S.

The earlier WHO estimate did not include the portion of violence due to childhood lead exposure, although the 2002 WHO World Health report did include an estimate of over 20,000 DALYs (14% of the total) due to injuries caused by intentional violence. Some percentage, perhaps as much as 5%, of these injuries could be related to lead exposure.

While lead contamination has been documented in many environmental media, food and in hundreds of consumer products, 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. Airborne lead particulate levels are now quite low in Europe and the U.S., due to the successful phase out of lead in gasoline and control of industrial emissions. Lead in food has also been greatly reduced due to elimination of cans with lead solder. But historic deposition into residential soils and housedust from previous lead gasoline use and lead paint in older housing, particularly substandard low-income housing, remains a significant source.

With regard to dose/response, 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  $\mu\text{g}/\text{ft}^2$  to 100  $\mu\text{g}/\text{ft}^2$  increases geometric mean blood lead levels in children from 2.8  $\mu\text{g}/\text{dL}$  to 7.3  $\mu\text{g}/\text{dL}$ . Holding interior floor dust lead constant at 5  $\mu\text{g}/\text{ft}^2$ , an increase in soil and exterior dust lead from 10 ppm to 1,000 ppm increases blood lead levels from 3.2 to 5.3  $\mu\text{g}/\text{dL}$ .

A study in Italy estimated that 4.4% of DALYs could be attributed to childhood lead poisoning (need full article to see if they reported housing-based lead exposure).

In Germany, 3.1% of water samples were found to exceed WHO guidelines.

#### Limitations

The earlier WHO analysis relied mainly on IQ and increased blood pressure and did not include the other well-documented health effects of lead exposure. It assumed that relatively small IQ decrements caused "disease" only for the fraction of the population approaching mild mental retardation (IQ=70) and increased blood pressure. Under this definition, for most people lead exposure does not constitute a "disease," in the classic medical sense. Therefore, one issue worthy of consideration for this project is whether the "disease burden" or the "health burden" of housing-related lead exposure should be assessed. Limiting the analysis to disease burden alone is likely to underestimate the overall health impact of lead in housing.

Indeed, for many contaminants found in housing, the medical model simply does not work well. In the case of lead, medical treatment, such as chelation, is limited in effectiveness, particularly if the source of exposure is not controlled. Indeed, one study showed that while chelation was effective in lowering blood lead level, it did not increase cognitive function. In short, the issue of whether we are estimating the global burden of housing-related disease or the global health burden of inadequate housing for this project is worth discussion. This should be decided in light of the WHO definition of health: "Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity."

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 socio-economic status, parental IQ, nutrition and others, all of which could confound the blood lead/IQ relationship. However, it is noteworthy that most studies show an inverse relationship between blood lead and IQ and those that do not did so due to lack of 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 consider the reported effect size of blood levels less than 10 µg/dL to be improbably large. The reasons that the effect could be overestimated include 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 (for more on this see Bellinger DC and Needleman HL. Intellectual impairment and blood lead levels. N Engl J Med 2003;349:500)

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. Some investigators are using bone lead as a more integrated exposure measure, but data for this metric are limited.

The relationship between blood lead and criminal or anti-social behavior also has many confounding influences. Finally, different countries may have different types of lead exposures, with differing degrees of bioavailability and uptake rates.

Likelihood that health effect is expressed

IQ decrement: For blood lead levels 2.4 – 10 µg/dL, 10 – 20 µg/dL, and 20 – 30 µg/dL, IQ decrements are 3.9, 1.9, and 1.1 points, respectively (this is from the updated international analysis)

Cardiovascular disease: For men, a 1.25 mmHg increase is associated with each 5 µg/dL increase in blood lead level between 5 and 20 µg/dL, and an increase of 3.75 mm Hg above 20 µg/dL in blood lead level; for women, the estimates are 0.8 mm Hg and 2.4 mm Hg for the respective blood lead ranges.

Criminal Behavior: 5% of criminal behavior is associated with childhood lead exposure

Anemia: Expressed at blood lead levels above 20 µg/dL.

Final Recommendation

**There is sufficient and reliable evidence for making a valid assessment.**

## **Part II: Exposure Situation**

Data Sources Used

Authors	Year	Title	Journal/Book	Location
Fewtrell L, Kaufman, R, Pruss-Ustun A.	2003	Lead: Assessing the environmental burden of disease at national and local levels	WHO Environmental Burden of Disease, Series 2	Global
Fewtrell, L, Pruss-Ustun A, Landrigan P, Ayuso-Mateos J.	2003	Estimating the global burden of disease of mild mental retardation and cardiovascular disease from environmental lead exposure	Env Research (2003)	Global
Jacobs et al.	2002	The Prevalence of Lead-Based Paint Hazards in U.S. Housing	Environ Health Perspect 110:A599-A606, Sept 13, 2002	U.S. (nationwide)
Lanphear BP, TD Matte, J Rogers, RP Clickner, B Dietz, RL Bornschein, P Succop, KR Mahaffey, S Dixon, W Galke, M Rabinowitz, M Farfel, C Rohde, J Schwartz, P Ashley, DE Jacobs	1998	The Contribution of Lead-Contaminated House Dust and Residential Soil to Children's Blood Lead Levels: A Pooled Analysis of 12 Epidemiologic Studies	Env. Research, 79:51-68, 1998	12 cities (all U.S.)
OECD/OCDE	1999	Phasing Lead Out of Gasoline	United Nations Environment Program	Global
European Union	2005	Age of European Housing ( <a href="http://mrw.wallonie.be/dgatlp/HousingStats">http://mrw.wallonie.be/dgatlp/HousingStats</a> )	Website	Europe
Claeys F, Sykes C, Limbos C, Ducoffre	2003	Childhood lead poisoning in Brussels. Prevalence study and etiological factors	J. Phys. IV France 107	Brussels
Duggan MJ and Inskip MJ	1985	Childhood exposure to lead in surface dust and soil	Public Health Review 13:1-54	Numerous British, European and US studies
Valent F, Little D, Bertollini R, Nemer LE, Barbone F, Tamburlini G	2004	Burden of disease attributable to selected environmental factors and injury among children and adolescents in Europe	Lancet June 19 363(9426) 2032-9	Europe
Rubin et al.	2002	Lead poisoning among young children in Russia	Env Health Persp June 110(6) 559-62	Russia
Mayan ON, Henriques AT, Calheiros JM	2001	Childhood lead exposure in Oporto, Portugal	Int J Occup Env Health Jul-Sept 7(3) 209-16	Portugal
Zietz B et al.	2001	Lead contamination in tap water of households with children in Lower Saxony, Germany	Sci Total Env Jul 25 (275(1-3): 19-26	Germany
Elliot et al	1999	Clinical lead poisoning in England	Occup Environ Med Dec 56(12) 820-4	England
Sinnaeve O, et al.	1999	Chronic lead poisoning in children today	Arch Ped Jul 6(7) 762-7	France
Bouman HD, Kleinjans JC	1998	Lead in drinking water	Sci Total Env Jan 19; 209(2-3) 255-71	Netherlands
Zejda JE	1995	Blood lead levels in urban	Central Eur J	Poland



		children of Katowice Voivodship, Poland	Public Health Jun 5(2) 60-4	
Millstone E, Russel J	1995	Lead toxicity and public health policy	J R Soc Health Dec 115(6) 347-50	England
Cambra K, Alonso E	1995	Blood lead levels in 2- to 3-year-old children in the Greater Bilbao Area (Basque Country, Spain)	Arch Env Health 1995 Sept – Oct 50(5) 362-6	Spain
Nedellec V et al.	1995	Evaluation of decontamination interventions in 59 homes of children with lead poisoning	Rev Epidmiol Sante Publique 43(5):485-93	France
Wilson J et al.	2004	Evaluation of the HUD Lead-Based Paint Hazard Control Grant Program	Government Report <a href="http://www.cenrforhealthyhousing.org/">http://www.cenrforhealthyhousing.org/</a>	U.S. (14 cities)

### General Evidence

Fewtrell et al. estimated population blood lead levels for all the WHO regions using the best available data in 2003. Mean blood lead levels in urban children in EurA and AmerA were reported to be 3.5 and 2.2 µg/dL, respectively. The WHO 2003 report on lead stated that “In developed countries where leaded gasoline has been phased out, the highest environmental exposures to lead generally affect children of lower-income families living in degraded housing.” Studies on lead-contaminated dust, soil, paint and water in housing have been found for France, Brussels, Italy, Portugal, Poland, England, Spain and the U.S. Lead in paint, dust and soil is regarded by the U.S. Centers for Disease Control and Prevention as the principal source of exposure for most U.S. children today.

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. It is not known whether the ban was enforced in each country. There is evidence that the U.S. ban in 1978 was effective, because the prevalence of lead paint in post-1978 housing is under 3%. One article also suggests that Great Britain issued regulations in the early 1970s to limit lead in paint. An article from France indicates lead paint is prevalent in houses built before 1948. Another from Spain shows higher blood lead levels in children living in housing built before 1950.

Despite national differences, age and condition of housing has consistently been highly correlated with prevalence of deteriorated lead-based paint, lead contaminated dust and soil and lead in drinking water. There are reliable data on age of housing in both the US and Europe (see attachment). Because different countries banned the use of lead paint in housing at different times, older dilapidated housing could be a reasonable surrogate for prevalence of lead hazards in housing if national studies of lead-contaminated housing are unavailable for European countries. For example, such an estimate for each country could link the year the ban was implemented, the number of existing housing units built before that time that are still in use today and what percentage of those units are substandard.

Lead in soil and settled dust comes from two principal sources—historical use of lead in gasoline and deteriorating paint in housing. Lead in drinking water is primarily housing-based, due to the contribution from leaded pipes and brass fixtures. While there are a few reports of lead contaminated food (e.g. paprika in the Czech Republic), the literature does not indicate this is a significant source in Europe and the U.S. Based on a United Nations estimate, 58 countries have phased out lead gasoline as of 2001. Romania, Poland and the Confederation of Independent States are the only European nations not to have eliminated leaded gasoline as of 2001. Industrial emissions of lead and glazed pottery in Western Europe are not thought to be major contributors to lead exposure today.

### Specific Evidence

With regard to dose-response, a pooled analysis of 12 studies of dust, soil and children's blood lead levels showed that an increase in floor dust lead from 1 to 100  $\mu\text{g}/\text{ft}^2$  more than doubled the geometric mean children's blood lead level (from 2.3 to 5.9  $\mu\text{g}/\text{dL}$ ), holding all other variables at their average levels in the U.S.. A soil/ exterior dust lead increase from 10 to 4,000 ppm also doubled the geometric mean blood lead level from 2.3 to 4.4  $\mu\text{g}/\text{dL}$ .

A major British review of lead in settled dust from 1985 concluded that for each increment of 1,000  $\mu\text{g}/\text{g}$  in settled dust lead concentration, there is an average increase of about 5  $\mu\text{g}/\text{dL}$  in blood lead level in young children. That review stated that European rural exterior dust lead levels were between 35 to 150  $\mu\text{g}/\text{g}$  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 U.S. had average interior dust lead levels ranging from 5-1,000  $\mu\text{g}/\text{ft}^2$ . That review agrees with the pooled analysis from the U.S. that settled dust lead measurements expressed in loading ( $\mu\text{g}/\text{ft}^2$ ) are better correlated with children's blood lead level than do measurements expressed in concentration ( $\mu\text{g}/\text{g}$ ). While this review was completed in 1985 when lead gasoline was still in use, it stated that "lead in dust levels prior to the introduction of leaded petrol were not very different from those found today..." although the review also stated such estimates were uncertain due to sample size limitations.

In the U.S. the average contribution to blood lead level from lead in drinking water is estimated to be 0.5  $\mu\text{g}/\text{dL}$ .

In Brussels, Claeys et al. reported an odds ratio of 4.4 for blood lead level and pre-1940 housing; this increased to 7.2 for buildings undergoing renovation.

In Paris, paint and dust samples were collected in 137 buildings and 74% presented high dust and/or paint lead content. Blood samples were collected from 145 out of a total of 189 children residing in these buildings and blood lead levels were higher than or equal to 10 micrograms/dl for 65% of these children.

In England, blood lead analyses for 4424 people (estimated at about 5% of such analyses in England over 7 years) found that among 547 children aged 0-4, 45 (8.2%) there was a blood lead concentration in excess of 25  $\mu\text{g}/\text{dL}$ , the action level in the United Kingdom for investigation, or removal of environmental sources of lead at the time of the study. Another

study in Birmingham England found dusts and soils with a geometric mean value for lead in surface (0-5 cm) garden soils of 266  $\mu\text{g/g}$  and in housedusts of 561  $\mu\text{g/g}$  (excluding old mining areas). A subsequent detailed survey of 97 householders in Birmingham with 2-year-old children showed dust lead loading in the home environment to be an important predictor of blood lead concentrations in young children, when both variables fell within the normal range for the U.K.

A study in Poland identified “apartment quality” as a significant predictor of blood lead level in children.

In Basque, Spain, blood lead levels were higher among children who lived in houses constructed prior to 1950. The geometric averages of lead in house dust, park soil, and park dust were 595, 299, and 136  $\mu\text{g/g}$ , respectively. A statistically significant linear correlation was found between blood lead level and lead content in park dust, a finding that explained 9% of the variation in blood lead level; a subgroup of these children was also found to have a strong linear association between blood lead and lead content in house dust.

In the U.S., 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.

In a study of 3,000 housing units in the U.S. 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. A French lead paint abatement study also showed significant decreases in children’s blood lead and dust lead levels.

#### Limitations

There are likely to be significant differences across countries in the portion of the population blood lead level that can be assigned to housing conditions, because each nation likely has a different regulatory history with regard to lead paint and lead gasoline. Comprehensive nationwide surveys of the prevalence of lead in paint, house dust, soil in yards and drinking water may not be available for each country.

#### Final Recommendation

**There is sufficient and reliable evidence for making a valid assessment.**

## Summary of main element of evidence between:

**Cause/ Housing Factor: VOC and chemical emissions in dwellings**  
**Health Effects: Respiratory and allergic effects**

Contributor: Bernhard Link, Germany

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### Part I: Health effects / Attributable risk / possible exposure-response relationship

#### Data sources used

*Priority should be given to international and/or European-based peer-reviewed and published studies*

Author(s)	Year	Title	Journal / Book	Location
Molhave, L.	1991	Indoor climate, air pollution, and human comfort	J Expo Anal Environ Epidemiol	1(1), 63-81
Pappas GP et al.	2000	The respiratory effects of volatile organic compounds	Int H Occup Environ Health	6(1), 1-8
Takigawa T. et al.	2004	Were volatile organic compounds the inducing factors for subjective symptoms of employees working in newly constructed hospitals?	Environ. Toxicol.,	19(4), 280-290
Wieslander G. et al.	1997	Asthma and the indoor environment: the significance of emission of formaldehyde and volatile organic compounds from newly painted indoor surfaces	Int Arch Occup Environ Health	69, 115-124
Diez, U. et al.	2003	Redecoration of apartments promotes obstructive bronchitis in atopy risk infants--results of the LARS Study	Int. J. Hyg. Environ. Health	206(3), 173-179
Rumchev K. et al.	2004	Association of domestic exposure to volatile organic compounds with asthma in young children	Thorax	59, 746-751

#### General evidence (*what effects and relationships are identified in literature?*)

Evidence for health effects comes from experimental studies with defined VOC-mixtures (Molhave et al., 1991; Pappas et al., 2002), from studies in connection with investigations of the "Sick Building Syndrome" (Tagigawa et al., 2004) and from studies of health effects after redecoration measures (Wieslander et al., 1997; Diez et al., 2003; Rumchev et al., 2004)

#### Respiratory effects:

Reduced lung function (FEE<sub>25-75</sub>) at 50 mg/m<sup>3</sup> VOCs only in atopic individuals.

Short term exposure (2 hours) to high VOC concentrations (> 8 mg/m<sup>3</sup>) leads to irritation of mucous membranes in eyes, nose and throat.

Frequency of complaints, discomfort, and irritation at lower concentrations (0.2 – 3 mg) are increasing proportional to VOC-concentrations (sick building syndrome)

Increased frequencies of pulmonary infections and obstructive bronchitis in children in the first two years of life after redecoration of the apartments.

Allergic effects:

Increase of asthmatic effects (wheezing, sensitization) in children during the first three years of life after redecoration of the apartments.

Also in adults, an association was found between indoor painting and frequency of allergic symptoms in several studies.

**Specific evidence** (*how strong are the effects, which quantification has been identified?*)

Respiratory effects:

Severe objective effects in adults only at very high VOC concentrations ( $> 25 \text{ mg/m}^3$ ). Effects are reproducible in exposure chambers with defined mixtures of 21 different VOC.

At lower concentrations the subjective sensibility (discomfort, symptoms) varies in a wide range

Allergic effects:

Health effects were often asked by self-recorded questionnaires. Symptom definition and exposure measurements were different in the studies. Allergies in young children can lead to asthma.

**Limitations** (*are there any constraints and limitations associated with the data?*)

VOCs in the indoor air are mixtures of many substances with different toxic properties. The composition of the measured VOC varies in different dwellings according to different sources for the single VOCs. Therefore TVOC-values are sum parameters with only limited toxicological significance. Toxic effects of individual VOCs are not considered.

VOC concentrations in the dwellings are not constant over time. Measurements are not standardized. Methods differ in different studies (numbers of VOCs analyzed, quantification, time and duration of sampling). In many cases, only qualitative data (redecoration during the last year) from self-reported questionnaires are available.

In the German LARS-study, children with a higher risk for allergies were recruited. The evaluated odds ratios are not transferable to the normal population.

**Suggested OR / incidence / attributable fraction for exposed population** (*what seems to be the most reliable and evidence-based estimation on the likelihood that the health effect is expressed?*)

For respiratory effects of high VOC concentrations (SBS), OR are not transferable to other situations.

For health effects after redecoration, the following OR were reported:

Adults:

**At least one symptom related to asthma:** OR=1.43(1.01-2.36) for newly painted dwellings during the last 12 months (Wieslander et al., 1997). TVOCs differed significant in dwellings with newly painted surfaces (average TVOC:  $413 \text{ } \mu\text{g/m}^3$  versus  $302 \text{ } \mu\text{g/m}^3$  in living rooms)

Children:

**Obstructiv bronchitis after redecoration of apartments:** OR=4.1 (1.4-11.9) for the first and OR=4.2 (1.4-12.9) in the second year in **atopy risk infants** (Diez et al., 2003)

**Risk of asthma for children between 0.5 and 3 years:** OR=1.27 (1.18-1.37) for 10 µg/m<sup>3</sup> increase of TVOC exposure (TVOC = sum of 10 defined VOC). Median TVOC was 55 µg/m<sup>3</sup> for all children (88 cases and 104 controls), 79 µg/m<sup>3</sup> for cases, and 36 µg/m<sup>3</sup> for controls

Final recommendation

**There is some evidence for making a valid assessment**

## Part II: Exposure situation

### Data sources used

*Priority should be given to international and/or European-based studies in peer-reviewed literature*

Author(s)	Year	Title	Journal / Book	Location
Rehwagen, M. et al.	2003	Seasonal cycle of VOCs in apartments	Indoor Air	13(3), 283-91
Oppl, R. et al.	2000	Innenraumluft und TVOC : Messung, Referenz- und Zielwerte, Bewertung	Bundesgesundheitsbl.	43(7), 513-518
Raw GJ et al.	2004	Exposure to air pollutants in English homes	J Expo Anal Environ Epidemiol	14 Suppl 1 S85-94
AGÖF	2005	AGÖF-Orientierungswerte für Inhaltsstoffe von Raumluft und Hausstaub	www.agoef.de	

### General evidence (is a relevant exposure identified in the literature?)

VOCs are emitted from numerous indoor sources (especially organic solvents in paints, adhesives, new carpets, environmental tobacco smoke) and generally exceed outdoor concentrations by a factor of about 10..

### Specific evidence (how large is the exposure, which quantification has been identified?)

Most cases of SBS are associated with elevated VOC concentrations; but high VOC levels are not generally combined with complaints. High concentrations were found after redecoration measures and in apartments with bad ventilations. New buildings show higher VOC levels than older houses.

During the last years, a decrease of VOCs is reported (mean value 1994: 280 µg/m<sup>3</sup>; 2001: 150 µg/m<sup>3</sup>; Rehwagen et al.,2003).

### Limitations (are there any constraints and limitations associated with the data? Are there large variation within countries or by country or regions?)

Exposure to VOCs depends on different influencing factors like ventilation, temperature, humidity, season, time after redecoration. Sampling methods and analyses differ in different studies (active - passive sampling, quantization of single/all VOCs). In many cases,

measurements were performed after complaints of the inhabitants (Oppl et al., 2000; AGÖF, 2005). Only limited data from representative studies are available (Rehwagen et al., 2003; Raw et al., 2004).

No information is available about exposure to VOCs caused by country specific sources

**Suggested exposure rate in the (European) population** (*what seems to be the most reliable and evidence-based estimation on the general exposure within the population?*)

Mean concentrations are in the range from 140 to 500 µg/m<sup>3</sup> in different studies; 95th percentile in the range from 500 to 1700 µg/m<sup>3</sup>. Painting measures are associated with an increase of some hundred µg/m<sup>3</sup>, depending on the time after redecoration. The following table shows a comparison of several studies:

Author	Description	Median	95 <sup>th</sup> percentile	Method used
Oppl et al., 2000	indoor air measurements after complaints of the users; buildings without replacements during the last 3 months (n=95)	530 µg/m <sup>3</sup>	1300 µg/m <sup>3</sup>	active sampling on tenax, no ventilation during sampling
AGÖF 2005	indoor air measurements, in most cases after complaints of the users (n > 2000)	300 µg/m <sup>3</sup>	1000 µg/m <sup>3</sup>	active sampling on tenax/charcoal, no ventilation during sampling
Oppl et al., 2000	indoor air measurements in buildings with replacements during the last 3 months (n=72)	1600 µg/m <sup>3</sup>	6200 µg/m <sup>3</sup>	active sampling on tenax, no ventilation during sampling
Rehwagen et al., 2003	randomly selected apartments at Leipzig	138 µg/m <sup>3</sup>	538 µg/m <sup>3</sup>	passive sampling over a period of 4 weeks, only 30 individual VOCs evaluated
Raw et al.,	randomly selected apartments in England	202 µg/m <sup>3</sup>	1010 µg/m <sup>3</sup>	passive sampling over a period of 4 weeks, all VOCs above 0.1 µg/m <sup>3</sup> evaluated

Final recommendation

**There is partially sufficient and reliable evidence for making a valid assessment**

## Summary of main element of evidence between:

### **Cause/ Housing Factor: Ventilation in the dwelling** **Health Effects: Respiratory and allergic effects**

Contributor: Ian Matthews, UK

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#### **Part I: Health effects / Attributable risk / possible exposure-response relationship**

##### **Data sources used**

Priority should be given to international and/or European-based peer-reviewed and published studies

<b>Author(s)</b>	<b>Year</b>	<b>Title</b>	<b>Journal / Book</b>	<b>Location</b>
Godish T & Spengler JD	1996	Relationships between ventilation and indoor air quality: a review	Indoor Air	
Menzies D & Bourbeau J	1997	Building-related illnesses	New Eng J Med	
Øie L et al	1999	Ventilation in homes and bronchial obstruction in young children	Epidemiology	Oslo
Wargocki P et al	2002	Ventilation and health in non-industrial indoor environments: report from a European Multidisciplinary Scientific Consensus Meeting (EUROVEN)	Indoor Air	
Emenius G et al	2004	Building characteristics, indoor air quality and recurrent wheezing in very young children (BAMSE)	Indoor Air	Stockholm
Bornehag CG et al	2005	Association between ventilation rates in 390 Swedish homes and allergic symptoms in children	Indoor Air	Sweden

**General evidence** (*what effects and relationships are identified in literature?*)

#### **Ventilation in the dwelling: respiratory and allergic effects**

Reviews of the effects of ventilation on indoor air quality (Godish T et al, 1996) and on health symptoms (Menzies & Bourbeau 1997) have concentrated on non-residential buildings. The European Multidisciplinary Scientific Network on Indoor Environment and Health (EUROVEN) carried out a systematic literature review to up-date the evidence base and extend it to residential dwellings (Wargocki P et al, 2002). Thirty papers were judged as suitable for detailed review but of these only five related to homes. Of these five only one considered a health outcome (Øie L et al 1999) and the other four related to effects of ventilation upon indoor air quality (Harving et al, 1993; Norback et al 1995; Sundell et al 1994; Warner et al 2000).



To provide an updated review, a literature search was conducted, in the context of residences only, for the period 1990 – 2005 using SCOPUS ®. SCOPUS ® covers scientific, technical, medical and social sciences literature. This database contains 27 million records covering 14,200 peer reviewed titles and has 100% medline coverage. Using this navigation tool several keyword searches were carried out as follows: Ventilation and Asthma and Home (111 refs); Ventilation and Allergy and Home (20 refs); Indoor Air and Home and Ventilation and Health (62 refs); Ventilation and the Home Environment (156 refs). A number of these references were judged to provide background information relevant to the scope but of those falling within the remit of providing scientific evidence only 3 related to ventilation and health and 13 to ventilation and air quality and these are considered below.

A nested case-control study was conducted within a prospective birth cohort study (BAMSE-children born between February 1994 and late 1996) in Stockholm to assess the influence of building characteristics and ventilation rate on recurrent wheezing in children up to two years of age (Emenius G, 2004). The definition of recurrent wheezing was three or more reported episodes of wheezing after three months of age, combined with the use of inhaled steroids or symptoms of bronchial hyper reactivity except when the child had a common cold. Two hundred and ninety four children were recruited at the age of 1 and 246 at the age of 2 yielding 181 cases and 359 controls. Both cases (recurrent wheezing) and controls had to reside in the same dwelling as when they were born. Most children (75%) lived in apartment houses and the rest in single family homes (detached, semi-detached etc). Four-week measurements of air change rate per hour (ACH) were made in all rooms of each home using a passive tracer gas technique. The mean of all ACH measurements was 0.68 (s.d = 0.32) and ACH exceeded 0.5 (minimum recommended in Swedish guidelines) in 69% of homes. The Odds Ratio (adjusted for gender, heredity of allergic disease, maternal smoking, breast feeding and building age) for recurrent wheezing in children up to 2 years of age in relation to installed ventilation system and Air Change rate were:

<b>Exposure</b>	<b>Number of cases (%)</b>	<b>Number of controls (%)</b>	<b>Adjusted OR</b>	<b>95% CI</b>
Exhaust ventilation (vs. natural ventilation)	85 (47.0)	151 (42.1)	1.1	0.6-2.0
Balanced ventilation (vs. natural ventilation)	43 (23.8)	78 (21.8)	0.8	0.4-1.7
Ventilation rate $\leq$ 0.5 ACH vs. lower	130 (71.8)	240 (67.0)	1.3*	0.8-2.0

\* Also adjusted for outdoor temperature.

Neither the type of ventilation system in use nor the air change rate calculated both as ACH and litres per second and per person were directly associated with recurrent wheezing.

Øie et al aimed to assess the effect of residential ventilation on bronchial obstruction as well as the potentially modifying effect that ventilation may have on effects of indoor exposures. A matched pair case-control study was carried out based on the Oslo Birth Cohort (children born in 1992-93). Questionnaire information was collected at birth, 6, 12, 18 and 24 months.

Cases were all children with two or more episodes with symptoms and signs of bronchial obstruction or one episode lasting more than 1 month. Air change rates were measured with a passive tracer gas method for 172 pairs and building characteristics and type of ventilation was recorded. The type of ventilation system did not produce an odds ratio significantly different from 1; OR = 1.11 (C.I. 0.71-1.76). Neither was the odds of developing bronchial obstruction associated with residential total air change rate; OR = 0.7 (0.39 – 1.25) comparing  $ACH < 0.5h^{-1}$  with  $ACH \geq 0.5h^{-1}$ .

The adjusted odds ratios for bronchial obstruction during the first 2 years of life according to indoor exposures were:

Total Air change Rate	ETS-Exposure		Dampness Problem(s) (verified)		Plasticizers Exposure Index	
	aOR	95% CI	aOR	95% CI	aOR	95% CI
Low	1.8	0.35-9.66	9.6	1.05-87.45	12.6	1.00-159
All	1.6	0.93-2.56	2.4	1.25-4.44	2.9	1.54-5.49
High	1.5	0.74-3.20	2.3	0.83-6.39	2.6	1.02-6.58

The authors inferred that the risk of bronchial obstruction and the exposures was systematically stronger in the low air change group, indicating that air change is a determinant of health in dwellings with indoor emissions.

The Dampness in Buildings and Health (DBH) study in Sweden was a cross-sectional questionnaire investigation involving 14,077 children 1-6 years of age in Varmland. A nested case control study selected 198 symptomatic controls (Bornehag CG, 2005). The selection criteria for cases was that, in the initial questionnaire and in a follow-up questionnaire 1.5 years later, they had to report at least two symptoms of ‘wheezing during the last 12 months without a cold’, ‘rhinitis during last 12 months without a cold’ and ‘eczema during the last 12 months’. The inclusion criteria for controls was no such symptoms in either questionnaire. Eighty three per cent of the children lived in single family houses most of which had natural ventilation and a kitchen fan. Multi-family houses were mostly ventilated with mechanical systems. Between October 2001 and April 2002 ventilation rates of the entire home and of the bedroom of the index-child were measured during 1 week with a passive tracer gas method in 390 homes. In single-family houses the mean ACH’s in total building and child’s bedroom were 0.34 and 0.32 for cases and 0.38 and 0.37 for controls ( $p = 0.025$  and  $0.020$ ).

**The association between ventilation rate (ach) indoor and case status in single-family houses was:**

	Odds Ratio (95% CI)			
	Total building		Child’s bedroom	
Quartile for ventilation rate	Min-Max (ach)	Adjusted Odds Ratio**	Min-Max (ach)	Adjusted Odds Ratio**
Fourth quartile (ref)	0.44-1.43	1.0	0.42-1.79	1.0
Third quartile	0.33-0.43	1.17 (0.57-2.42)	0.31-0.41	0.94 (0.46-1.95)

Second quartile	0.24-0.33	1.35 (0.66-2.74)	0.21-0.31	1.56 (0.77-3.12)
First quartile	0.05-0.24	1.95 (0.94-4.04)	0.02-0.21	1.77 (0.87-3.65)

\*\* Adjusted for sex, smoking in family, observed moisture problems in the dwelling  
In single-family houses there was an indication of a dose-response relationship between ventilation rate and the risk of being a ‘case-child’ but the results did not reach significance. Case children with doctor-diagnosed rhinitis and eczema living in single family houses had a lower ventilation rate in the child’s bedroom compared with controls but no association was found between ventilation rate and doctor-diagnosed asthma:

**Ventilation rate in single-family houses for case children with a doctor-diagnosed disease compared with controls**

Diagnosed disease	Cases with disease		Controls		P-value t-test
	N	Mean ventilation (ach)	N	Mean ventilation (ach)	
Total building ventilation					
Asthma	101	0.36	169	0.38	0.253
Rhinitis	82	0.35	169	0.38	0.233
Eczema	107	0.34	169	0.38	0.032
Children’s bedroom ventilation					
Asthma	99	0.34	166	0.37	0.207
Rhinitis	79	0.32	166	0.37	0.073
Eczema	106	0.31	166	0.37	0.024

Thus the results of this study suggest that associations between ventilation rates and wheeze, rhinitis and eczema may occur only when the air change rate is below 0.5ACH.

**Specific evidence** (*how strong are the effects, which quantification has been identified?*)

The three studies reviewed were of good design but no statistically significant effect of ventilation was seen for either recurrent wheezing or bronchial obstruction in children up to 2 years old. There was some evidence that doctor diagnosed rhinitis and eczema was affected by ventilation rate despite the difference in mean ventilation rate between cases and controls being relatively small (i.e. approximately 15%).

There are indications that ventilation may act as an effect modifier when ventilation is low and indoor exposures are high. However this effect was markedly greater for the damp and plasticizer exposure than for ETS which is perhaps surprising.

**Limitations** (*are there any constraints and limitations associated with the data?*)

The consistency of results is affected by the variation in house type and ventilation rate between studies.

In the study reported by Bornehag about 80% of the single-family houses and 60% of the multi-family houses had ventilation rates less than 0.5 ACH. In the studies reported above by Emenius and Oie the ventilation rates observed were higher than in this study and they also had a greater proportion of multi-family houses. In the Norwegian study 63% of the homes had a ventilation rate above 0.5ACH and 52% of the buildings were multi-family houses. In the Emenius study the mean ventilation rate was 0.68 ACH and about 75% of the buildings were multi-family houses.

The concentration of any indoor pollutant (which may be a causal factor for respiratory and allergic effects) is affected directly by emission and inversely by air change rate. The evidence reviewed relates to environments which were not pollutant free at baseline and in which pollutant sources and concentrations will have varied.

The data only refers to houses and environments in Sweden and Norway.

**Suggested OR / incidence / attributable fraction for exposed population** (*what seems to be the most reliable and evidence-based estimation on the likelihood that the health effect is expressed?*)

None of the odds ratios measured for recurrent wheezing or bronchial obstruction were statistically significant. The possible effect size of ventilation as measured by the confidence intervals on the point estimate of odds ratio were:

Ventilation rate  $\geq 0.5$ ACH compared to  $< 0.5$ ACH, Confidence Interval for odds ratio (0.8 – 2.0).

Ventilation rate in fourth quartile compared to ventilation rate in first quartile, Confidence Interval for odds ratio (0.87-3.65).

Final recommendation

**There is some / partially sufficient and reliable evidence for making a valid assessment**

## Part II: Exposure situation

### Data sources used

Priority should be given to international and/or European-based studies in peer-reviewed literature

Author(s)	Year	Title	Journal / Book	Location
Harving H et al	1993	House-dust mites and associated environmental conditions in Danish	Allergy: European Journal of Allergy and Clinical	Denmark

		homes	Immunology	
Harving H	1994	House-dust mite exposure reduction in specially designed, mechanically ventilated "health" homes.	Allergy	Denmark
Sundell J et al	1995	Ventilation in homes infested by house-dust mites	Allergy	Stockholm
Fletcher AM et al	1996	Reduction in humidity as a method of controlling mites and mite allergens: the use of mechanical ventilation in British domestic dwellings	Clinical and Experimental Allergy	UK
Stephen FR et al	1997	Ventilation and house air tightness: Effect on indoor temperature and humidity in Southampton, UK	Building Services Engineering Research and Technology	UK
Øie L et al	1998	The ventilation rate of 344 Oslo residences	Indoor Air	Oslo
Garrett MH et al	1998	Indoor airborne fungal spores, house dampness and associations with environmental factors and respiratory health in children	Clinical and Experimental Allergy	Australia
Emenius G et al	1998	Mechanical ventilation protects one-storey single-dwelling houses against increased air humidity, domestic mite allergens and indoor pollutants in a cold climatic region	Clinical and Experimental Allergy	Sweden
Niven R et al	1999	Attempting to control mite allergens with mechanical ventilation and dehumidification in British houses	Journal of Allergy and Clinical Immunology	UK
Warner JA et al	2000	Mechanical ventilation and high-efficiency vacuum cleaning. A combined strategy of mite and mite allergen reduction in the control of mite-sensitive asthma	Journal of Allergy and Clinical Immunology	UK
Stephen RK	2000	Positive input ventilation in dwellings	BRE Environmental Engineering Centre	UK
Emenius G et al	2000	Window pane condensation and high indoor vapour contribution – markers of an unhealthy indoor climate?	Clinical and Experimental Allergy	Sweden
Howieson SG et al	2003	Domestic ventilation rates, indoor humidity and dust mite allergens: are our homes causing the asthma pandemic?	Building Services Engineering Research Technology	UK

**General evidence** (*is a relevant exposure identified in the literature?*)

In 344 Norwegian residences 36% had air change rates  $<0.5h^{-1}$ . (Oie L 1998) In thirty single family homes in Stockholm, single storey homes had higher concentrations of house dust mite (HDM) and a tendency to lower ACH. (Sundell J. 1995).

In UK, ten houses were fitted with mechanical ventilation heat pump recovery units with additional dehumidification and compared with ten control homes. The units failed to confer mite allergen reduction compared to controls. (Niven R 1999). In Southampton, UK, 20

homes with MVHR units were compared with 20 controls. During the winter water vapor values in the intervention homes ( $6.75\text{gkg}^{-1}$ ) were lower than control homes ( $7.53\text{gkg}^{-1}$ ) ( $p < .001$ ) (Stephen F.R. 1997). In North Lancashire, UK, MHRV units were fitted to 32 homes and compared with 36 control homes. There was a 12% reduction in absolute humidity comparing intervention with control homes. (Howieson SG. 2003). In Stockholm 59 houses were investigated and the proportion of homes with indoor water concentrations  $\geq 3\text{gm}^{-3}$  was significantly greater in homes with ACH  $< 0.5$  compared to those with ACH  $> 0.5$ . ( $p < .001$ ) (Emenius et al 2000).

In Southampton, 40 homes were randomized to mechanical ventilation or control. Homes with mechanical ventilation achieved significantly lower humidity levels and Der p1 concentration than those without ( $p < .001$ ). (Warner J 2000) In the UK in a test house and 15 field-monitored houses an input ventilation fan was installed in the loft space. There was a 10% reduction in relative humidity in the test house but in the field-monitored houses input ventilation was not consistently effective in reducing relative humidity. (Stephen RK 2000)

In 22 of 59 homes in Stockholm mechanical supply and exhaust ventilation were installed. Mechanical ventilation increased the possibility of reaching an ACH  $\geq 0.5$ . The proportion of houses with an absolute indoor humidity of  $7\text{g kg}^{-1}$  was significantly higher ( $p = 0.01$ ) in homes with ACH  $< 0.5$ . (Emenius G. 1998)

In the North West of England 9 homes with MVHR units and 9 control houses were monitored for RH and Der p1 concentrations. The MVHR unit did not reduce indoor humidity to levels capable of retarding mite population growth. (Fletcher AM 1996)

Eighty households in Victoria, Australia were investigated with respect to fungal spores in indoor air and building characteristics. Multiple regression indicated that lack of regular ventilation through open windows for much of the year was a significant risk factor for indoor spore concentration. (Garrett MH 1998).

**Specific evidence** (*how large is the exposure, which quantification has been identified?*)

Most studies report that mechanical ventilation achieves significant reduction in indoor humidity levels. The effect on reduction in mites and Der p1 is less certain.

**Limitations** (*are there any constraints and limitations associated with the data? Are there large variation within countries or by country or regions?*)

Studies used only a small number of homes and different ventilation systems were used. The effect of outdoor humidity in different climates and seasons, as well as occupant generated water vapour, were not sufficiently considered.

**Suggested exposure rate in the (European) population** (*what seems to be the most reliable and evidence-based estimation on the general exposure within the population?*)

Final recommendation

**There is some / partially sufficient and reliable evidence for making a valid assessment**

## Summary of main element of evidence between:

**Cause/ Housing Factor: Dampness and mould**  
**Health Effects: Respiratory symptoms, asthma**

Contributor: Aino Nevalainen, Finland

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### Part I: Health effects / Attributable risk / possible exposure-response relationship

#### Data sources used

Priority should be given to international and/or European-based peer-reviewed and published studies

Author(s)	Year	Title	Journal / Book	Location
NAS/IOM	2004	Damp Indoor Spaces and Health	Book (Committee Report)	
Bornehag CG et al	2001	Dampness in Buildings and Health	Indoor Air	Vol. 11:72-86
Patovirta R et al.	2004	The remediation of mold damaged school – a three-year follow-up study on teachers' health	Central Eur J Public Health	12:36-42
Dales RE et al.	1991	Adverse health effects among adults exposed to home dampness and molds	Am Rev Resp Dis	143:505-509
Rudblad et al.	2002	Slowly decreasing mucosal hyperreactivity years after working in a school with moisture problems	Indoor Air	12:138-144
Dales RE et al.	1991	Respiratory health effects of home dampness and molds among Canadian children.	Am J Epidemiol	134:196-203
Jaakkola MS et al.	2002	Indoor dampness and molds and development of adult-onset asthma: a population-based incident case-control study.	Environ Health Persp	110:543-547
Kilpeläinen M et al.	2001	Home dampness, current allergic diseases, and respiratory infections among young adults	Thorax	56:46-467
Pirhonen I et al.	1996	Home dampness, molds and their influence on respiratory infections and symptoms in adults in Finland	Eur Resp J	9:2618-2622
Brunekreef B et al.	1989	Home dampness and respiratory morbidity in children	Am Rev Resp Dis	140:1363-1367



**General evidence** (*what effects and relationships are identified in literature?*)

There is quite an extensive documentation on the association between building dampness and/or mould and adverse health effects. There are approximately 100 population studies on this subject and the results are mostly similar: there is an association, but the calculated risks vary since there is a lot of variation in the definitions of the exposure, and the health outcomes are diverse. Many symptoms that are reported are unspecific for their nature.

The health outcomes may be divided into three main categories:

Irritation symptoms of the eyes and upper airways (e.g., cough, wheeze, asthma symptoms, running nose, sore throat, hoarseness)

Symptoms in lower airways (e.g., dyspnea, development of asthma, lower respiratory illness in otherwise healthy children), respiratory infections

Other health outcomes: e.g., COPD, fever, fatigue, headache, neurological symptoms, skin symptoms, rheumatologic diseases, allergic alveolitis, sarcoidosis

Adverse health outcomes have been reported among both children and adults. Most studies have been cross-sectional but also studies with a case control design have been published. Furthermore, most studies have dealt with domestic exposures to dampness and mold. Also a few intervention studies have been published indicating a positive effect of remediation on the occupants' health. Most of these intervention studies have been carried out in schools, reporting the symptoms of schoolchildren and teachers.

Although the association is well documented, the causal connections between the exposing agents and the health outcomes are not well known (see Part II below). It is not well understood why building-related mold causes health effects. Furthermore, the pathophysiological mechanisms are not well known. Only a minor part of the effects can be explained by IgE-mediated allergy. There is evidence about inflammatory reactions among the exposed. There are also indications on toxic reactions.

**Specific evidence** (*how strong are the effects, which quantification has been identified?*)

Perhaps the strongest evidence exists on the association of dampness with cough, wheeze and on asthma; there is evidence on both onset of new asthma cases and increased asthma symptoms on previously sensitized individuals. The ORs vary between 1.4-2.2. For other health outcomes, the ORs vary remarkably from study to study and from symptom to symptom; approximately between 1.1-4.6.

The literature has been carefully reviewed by several working groups during the recent years. Their conclusions are very similar: the evidence of the findings on cough, wheeze and asthma is strong. However, it is generally agreed that the evidence on many other health outcomes (see above) is still suggestive or insufficient. This is partly due to the fact that they have not been specifically attributed in the studies.

**Limitations** (*are there any constraints and limitations associated with the data?*)

There are enough well-done epidemiological studies with large enough populations and in different countries that the main body of the data is reliable. The main limitations are

associated with the assessment of exposure (see below). Since there is no commonly accepted metrics for dampness and/or microbial exposure, the estimations on the exposed populations are not quite precise.

**Suggested OR / incidence / attributable fraction for exposed population** (*what seems to be the most reliable and evidence-based estimation on the likelihood that the health effect is expressed?*)

As indicated above, the risk for cough, wheeze and asthma vary between OR 1.4-2.2.

Final recommendation

**There is sufficient and reliable evidence for making a valid assessment**

## Part II: Exposure situation

### Data sources used

Priority should be given to international and/or European-based studies in peer-reviewed literature

Author(s)	Year	Title	Journal / Book	Location
Nevalainen et al.	1998	Prevalence of moisture problems in Finnish houses.	Indoor Air	4: (Suppl), 45-49
Bornehag CG et al	2005	Dampness at home and its association with airway, nose and skin symptoms among 10,851 preschool children in Sweden: a cross-sectional study	Indoor Air	15 (Suppl 10):48-55
Zock et al	2002	European Community Respiratory Health Survey. Housing characteristics, reported mold exposure, and asthma in the European Community Respiratory Health Survey.	J Allergy Clin Immunol	110:285-292
Dales et al	1999	Testing the association between residential fungus and health using ergosterol measures and cough recordings.	Mycopathologia	147:21-27

**General evidence** (*is a relevant exposure identified in the literature?*)

The answer is yes and no. If the relevant exposure is defined as “dampness” or “signs of moisture”, it can be said that the relevant exposure has been sufficiently identified. Although the terms used in the different studies vary (e.g., dampness, moisture, condensation, damp spots), they mean the same thing, i.e., undesired dampness or moisture in the indoor

environment. The exposure is usually assessed as visible signs of dampness, such as discoloration of wood, detaching of materials or paint, signs of leakage and as visible condensation. Odor of mold or earth is also a commonly used indication of dampness. The signs of dampness may vary since also the mechanisms how water can enter the indoor environment or the building structure. The main types of water intrusion are leakages, condensation or capillary rise of water from the ground.

It is clear that dampness or moisture as themselves are surrogates of the actual exposures, i.e., the agents in the indoor environment that actually cause the health effects. The agent-specific exposures are not well understood. There is quite good evidence that the microbial profile (fungi and bacteria) of a damp building differs from that of a normal building both qualitatively and quantitatively, but the links between e.g. concentrations of airborne fungi and health effects are not clear. House dust mites only explain part of the phenomenon. The problems with the exposure assessment of damp building are mainly attributed to methodological issues. Methods that adequately quantify microbial material are only now being validated. Determination of microbial toxins from indoor air is still difficult due to low concentrations and a large variety of toxins.

**Specific evidence** (*how large is the exposure, which quantification has been identified?*)

The prevalence of “dampness” in buildings varies largely between studies, probably mainly due to different metrics used. Some studies have used trained inspectors, but most have relied on self-reporting. The assessment whether the observations are perceived as “problematic” causes also variation from study to study. Prevalence of dampness and/or mold varies roughly between 5-50%. It is possible to make a more precise estimate once the exposure is defined in a uniform way (for example, using the same terms and same memory period).

**Limitations** (*are there any constraints and limitations associated with the data? Are there large variation within countries or by country or regions?*)

The problems have already been addressed above. It is clear that the exposure data is not precisely uniform. However, the terms used in the dampness metrics often mean the same in spite of different wording.

**Suggested exposure rate in the (European) population** (*what seems to be the most reliable and evidence-based estimation on the general exposure within the population?*)

As mentioned above, the prevalence depends on how the exposure is defined. Once this has been agreed upon, it is possible to make an evidence-based estimation on the exposure.

Final recommendation

**There is some / partially sufficient and reliable evidence for making a valid assessment**

## Summary of main element of evidence between:

**Cause/ Housing Factor: Neighbourhood noise**  
**Health Effects: unspecific health effects**

Contributor: Hildegard Niemann, Germany

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### Part I: Health effects / Attributable risk / possible exposure-response relationship

Non-auditory health effects of noise have been studied in humans for a couple of decades using laboratory and empirical methods. Biological plausibility has been derived, based on the general stress concept. Classical biological risk factors have been shown to be elevated in subjects that were exposed to high levels of noise. From this, the hypothesis emerged that persistent noise stress increases the risk of cardiovascular disorders. The health effects of permanent noise stress can reveal themselves 10-15 years later in different functional systems [Graff et al. 1968].

Noise is not only a physical stimulus, but also an individually experienced noise-event with a corresponding emotional reaction [Matsui et al. 2001; Matsui et al. 2004]. An insufficient ability to cope with the noise can therefore lead to emotional stress. The noise annoyance has to be classified as an insufficient ability to cope with noise.

#### Data sources used

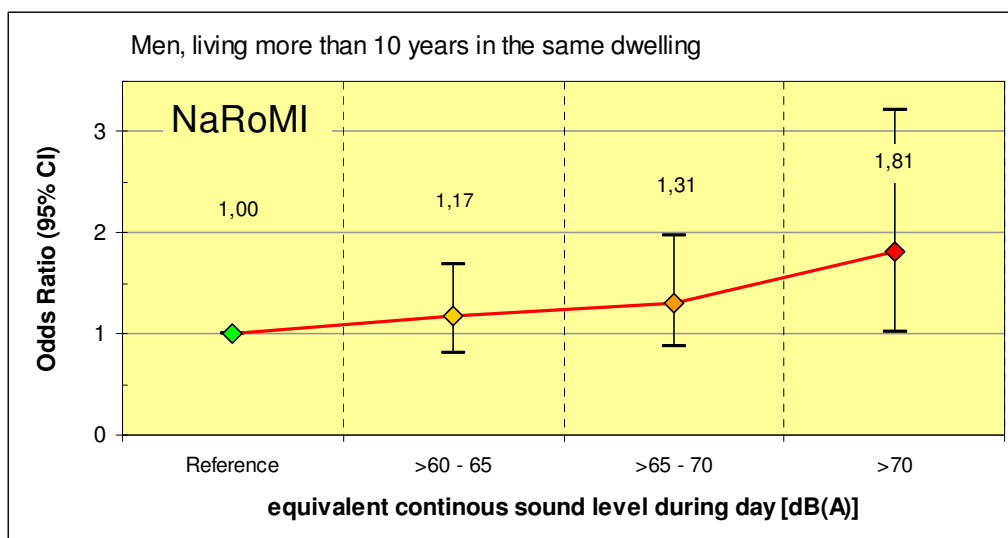
Author(s)	Year	Title	Journal / Book	Location
Babisch, W.; Beule, B.; Schust, M.; Stark, H.; Wende, H.; Ising, H.	2005	Traffic noise and Myocardial Infarction - results from the NaRoMi-study	Epidemiology 2005;16:33-40.	Germany
Babisch W.	2005	Updated review of the relationship between transportation noise and cardiovascular risk. Dose-effect curve and risk estimation	Quantifying Disease From Environmental Noise; WHO-Technical-Meeting, Stuttgart 23.-24.06.2005	Meta analysis
Miedema, Henk M.E.	2002	Relationships between exposure to single or multiple transportation noise sources and noise annoyance	Paper 5038933-2002/5 WHO - Technical meeting on exposure-response relationships of noise on health	Meta analysis
Niemann H, Maschke C, Hecht K	2005	Lärmbedingte Belästigung und Erkrankungsrisiko – Ergebnisse des paneuropäischen LARES-Survey	Bundesgesundheitsblatt, 2005 (48), 315-328	

#### General evidence

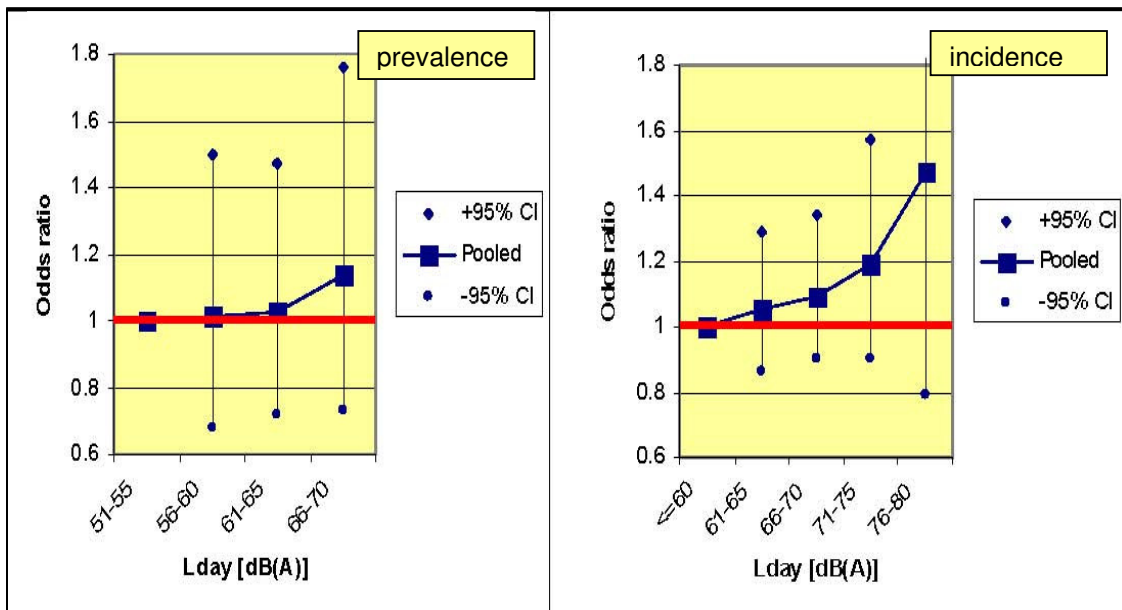
Systematical and quantitative reviews have been published in the past, summarizing the results of studies that have been carried out up to the end of the last century, and assessing the evidence of the relationship between community noise and cardiovascular disease outcomes.

The highest degree of evidence was for the association between community noise and cardiovascular endpoints. Further large-scale epidemiological studies were carried out in the last 5 years. The new judgement about the epidemiological studies today was made by Babisch [2005] with respect to the identification of good quality studies. All the studies were evaluated with respect to the following criteria for the inclusion or exclusion in the evaluation process. Necessary criteria were: (1) peer-reviewed in the international literature, (2) reasonable control of possible confounding factors (stratification, model adjustment (regression), matching), (3) objective assessment of exposure and (4) objective assessment of outcome. Additional criteria for the evaluation were: (5) type of study and (6) dose-response assessment. Details will be given elsewhere [WHO; 30. march 2005].

All six criteria were fulfilled by the two prospective cohort studies carried out in Caerphilly and Speedwell [Babisch et al. 1999, Babisch et al. 2003], the two prospective case-control studies carried out in the western part of Berlin ("Berlin I" and "Berlin II") [Babisch et al. 1994], and the new prospective case-control study carried out in entire Berlin ("NaRoMI" = "Berlin III") [Babisch et al. 2005]. The studies refer to road traffic noise and the incidents of myocardial infarction. For higher noise categories higher risks were relatively consistently found amongst the studies. The dose-response curve for men in the high ranking NaRoMI-study is shown in figure 1 and the pooled effect of all of the selected studies in figure 2.



**Figure 1: Association between Traffic noise equivalent continuous sound level and MI-Incidence for men (living more than 10 years in the same dwelling) [Babisch 2004].**



**Figure 2: Pooled effect estimates (meta analysis) of descriptive and analytic noise studies of the association between road traffic noise level and the prevalence (left graph) and incidents (right graph), respectively, of myocardial infarction (odds ratio  $\pm 95\%$  confidence interval) [Babisch 2005]**

The results of the evaluation confirmed on an epidemiologic level an increased health risk from elevated chronic transportation noise exposure. The evidence is classified as sufficient. On the other hand an increasing chronic noise exposure is clearly coupled with increasing noise annoyance. The dose response relationship of equivalent continuous sound level and annoyance has been calculated by Miedema for different transportation sources in a huge meta analysis (e.g. [Miedema 2002]). The evidence of the curve could be classified as sufficient.

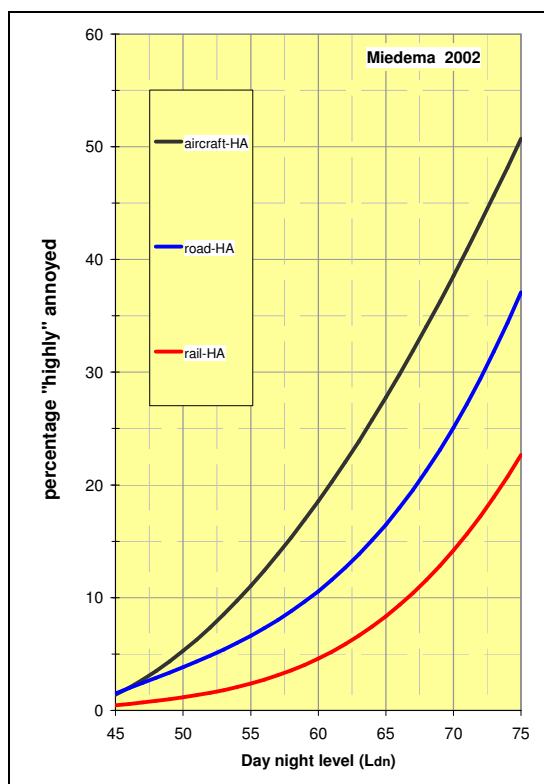


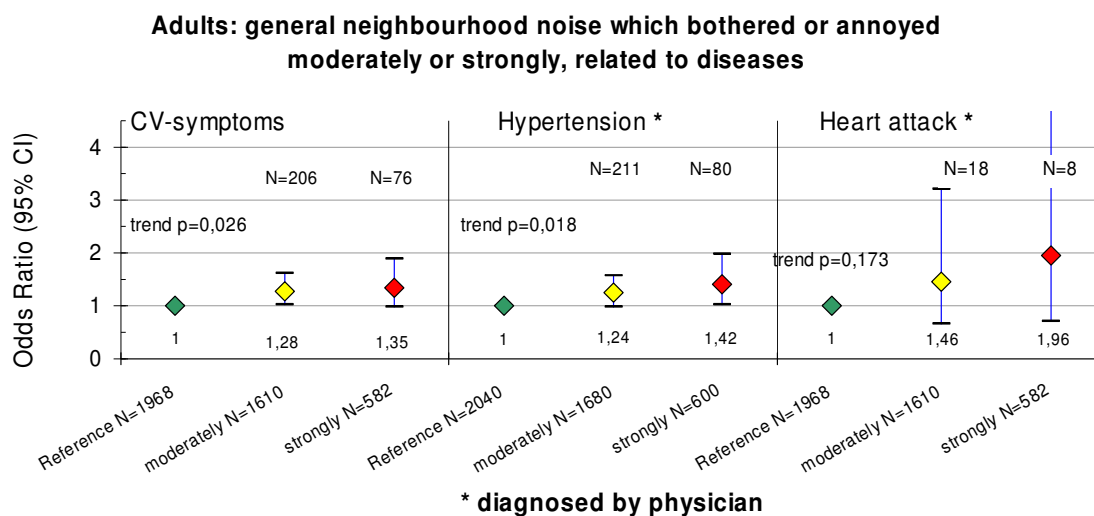
Figure 3: Dose effect relationship of equivalent continuous sound level and annoyance from rail, road and air traffic.

Both results together establish general evidence that emotional noise stress caused by chronic annoyance can trigger or contribute to the development of cardio-vascular diseases.

### Specific evidence

Epidemiological studies on the health effects of chronic annoyance by neighbourhood noise were rare up until recently. Due to experimental studies we presumed today, that the health effects of chronic annoyance are dependent on the intensity to the annoyance, but only insignificantly dependent on the source of the annoyance. That is confirmed by the LARES-study

The LARES-Study shows comparable health impacts by chronically strong annoyance due to traffic noise as well as due to neighbourhood noise. The result of the LARES-study regarding the effect of neighbourhood noise induced annoyance on hypertension as well as CV-symptoms is shown in the following figure.



**Figure 3: Relative disease risks for adults (18-59 years) who indicated noise induced annoyance by neighbourhood noise within the last 12 months, in comparison with adults without neighbourhood noise induced annoyance (comparison group). Block wise adjusted (Information regarding the control variables in). CV-symptoms: Sum score for cardiovascular symptoms. [Niemann, 2005]**

In adults, chronic annoyance by neighbourhood noise was related to increased risks for the cardiovascular system, as was the case for traffic noise. Significantly increased risks appeared for cardiovascular symptoms and hypertension in conjunction with strong chronic annoyance by neighbourhood noise. The trend over severity classes was likewise significant.

### Limitations

The LARES-study is a cross-sectional study which cannot prove the temporal sequence of annoyance and illness. That the illnesses triggered the annoyance causally in the LARES-study, could not be excluded as a possibility, but it is over all not probable. For clarification of this a longitudinal study would be necessary.

### Suggested OR / incidence / attributable fraction for exposed population

Based on the results of the LARES-study it is to be assumed, that the relative hypertension risk for adults (18-59 years) is increased by chronically strong annoyance by approximately 40 % in the European cities. If it is possible to keep away chronically strong annoyance by neighborhood noise 5 % of all hypertension treatments could be avoided. The attributable fraction for the exposed population is 30 %.

Final recommendation

**There is some/partially sufficient and reliable evidence for making a valid assessment.**



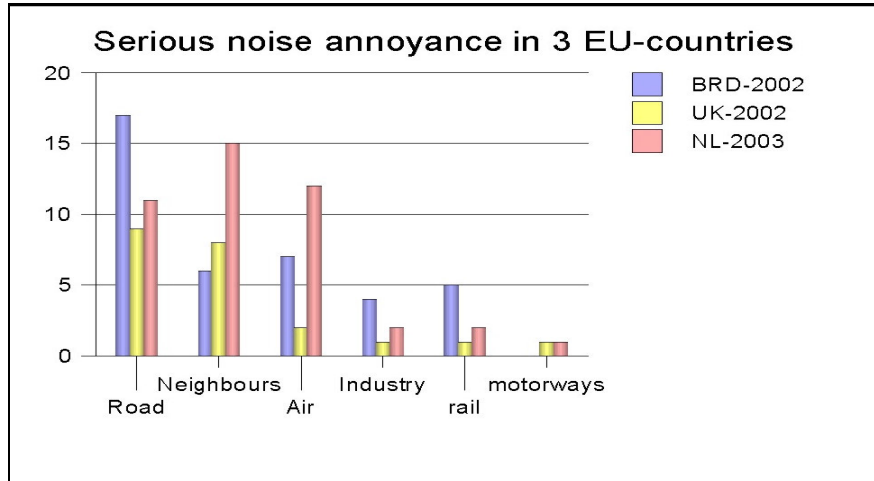
## Part II: Exposure situation

### Data sources used

Data for the exposure situation regarding neighbourhood noise annoyance is mostly given in reports of the departments of environment, health or statistic. This statistical data is published more rarely in peer-reviewed literature and is collected mainly nationally.

Author(s)	Year	Title	Journal / Book	Location
Kuckartz, U., Grunenberg, H.	2002	Umweltbewusstsein in Deutschland 2002. Ergebnisse einer repräsentativen Bevölkerungsumfrage.	Berlin: Bundes Ministerium für Umweltschutz (BMU)	Germany
EAM Franssen, JEF van Dongen1, JMH Ruysbroek, H Vos1, RK Stellato	2004	Hinder door milieufactoren en de beoordeling van de leefomgeving in Nederland Inventarisatie verstoringen	RIVM rapport 815120001 / 2004; TNO rapport, Bilthoven, 2004- 34	Netherland
Van den Berg, M.	2004	Neighbour Noise: A rational Approach	Proceedings of the 2 <sup>nd</sup> WHO International Housing and Health Symposium 29.09- 01.10.2004 Vilnius, Lithuania	Netherland
Department for Environment, Food and Rural Affairs	2002	Neighbour and Neighbourhood Noise - A Review of European Legislation and Practices:	Research Contract EPG 1/2/36 March 2002	United Kingdom

## General evidence



**Figure 4: Relative shares of sources of serious noise annoyance in UK, Germany and the Netherlands. A direct comparison between countries cannot be made with too much confidence due to somewhat different ways of interviewing, but it is clear that neighbour(hood) noise ranks in the top 3 as main causes of annoyance.**

For reasons of comparability, with regard to neighbourhood noise the total of inside and outside noises are presented. Apart from annoyance, also a fair amount of sleep disturbance is caused by neighbourhood noise. In the UK 18% of the people interviewed stated that rest and sleep were disrupted by neighbour noise, which is the same percentage as for road traffic noise. In the Netherlands 6% of the population is seriously disturbed in their sleep by neighbour(hood) noise (12% by road traffic and 2% by aviation noise).

[Martin van Berg (2004) Neighbour Noise: A rational Approach, Proceedings of the 2nd WHO International Housing and Health Symposium 29.09-01.10.2004 Vilnius, Lithuania]

## Specific evidence

The noise situation by neighbourhood noise cannot be described sufficient by equivalent continuous sound levels or maximum sound levels. Usually, neighbourhood noise is a sound with high information content such as language, music or even the noise of footsteps. It is in the nature of human hearing to have ones attention drawn to such informative sounds, even if the sound level is relatively low. An adequate indicator for neighbourhood noise is therefore the annoyance. Annoyance by neighbourhood noise can already appear at low noise levels, as soon as the sound exceeds the hearing threshold. The annoyance is frequently influenced by psychological and situation-related components.

Therefore it is necessary to collect the incidents of annoyance by standardised questions (see ICBEN – standard).

The predominant source of noise annoyance in living quarters is traffic. The second, sometimes the third, largest source of noise annoyance in the living quarters is the neighbourhood. The neighbourhood noise annoyance ranks mostly in front of airplane and railway noise annoyance. The annoyance potential by neighbourhood noise is therefore relatively high but is often not taken into consideration.

In general it can be concluded, that the asked noise annoyance represents a reliable indicator of the dismay of a section of the population due to sources of noise.

### **Limitations**

Data for all European countries for annoyance by neighbours or neighbourhood noise are not available (e.g. from the new EU countries). The available information is often difficult to compare because the annoyance scales are different, because the standard ICBEN scale is not in use in all European countries.

### **Suggested exposure rate in the (European) population**

Based on the results of the LARES-study it is to be assumed, that strong noise annoyance (strong and extremely from the ICBEN-scales) by neighbourhood noise, is detectable in approximately 8% of the inhabitants of European cities.

Final recommendation

### **There is sufficient and reliable evidence for making a valid assessment**

#### LITERATURE

Babisch W, Ising H, Gallacher JEJ. Health status as a potential effect modifier of the relation between noise annoyance and incidence of ischaemic heart disease. *Occupational and Environmental Medicine* 2003;60:739-745.

Babisch W, Ising H, Gallacher JEJ, Sweetnam PM, Elwood PC. Traffic noise and cardiovascular risk: The Caerphilly and Speedwell studies, third phase - 10 years follow-up. *Archives of Environmental Health* 1999;54:210-216.

Babisch W, Ising H, Kruppa B, Wiens D. The incidence of myocardial infarction and its relation to road traffic noise - the Berlin case-control studies. *Environment International* 1994;20:469-474.

Graff C, Bockmühl F, Tietze V (1968): Lärmbelastung und arterielle (essentielle) Hypertoniekrankheit beim Menschen. In: Nitschkoff S, Kriwitzkaja G (Hrsg) Lärmbelastung, akustischer Reiz und neurovegetative Störungen. Georg Thieme Verlag Stuttgart 112-126

Matsui T, Uehara T, Miyakita T, Hiramatsu K, Osada Y, Yamamoto T. Association between blood pressure and aircraft noise exposure around Kadena airfield in Okinawa. In: Boone R, ed. *Internoise 2001. Proceedings of the 2001 International Congress and Exhibition on Noise Control Engineering*, The Hague, Vol. 3. Maastricht: Nederlands Akoestisch Genootschap, 2001:1577-1582.

Matsui T, Uehara T, Miyakita T, Hiramatsu K, Osada Y, Yamamoto T. The Okinawa study: effects of chronic aircraft noise on blood pressure and some other physiological indices. *Journal of Sound and Vibration* 2004;277:469-470.

WHO European Centre for Environment and Health - Bonn Office. Night noise guidelines. [http://www.euro.who.int/Noise/activities/20040721\\_1](http://www.euro.who.int/Noise/activities/20040721_1) (accessed 30 March 2005) 2005.

## Summary of main element of evidence between:

**Cause/ Housing Factor: Home design**  
**Health Effects: Accidents and injuries**

Contributor: Mathilde Sengölge, Austria

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### Home design and stairs and Falls

#### Part I: Health effects / Attributable risk / possible exposure-response relationship

##### Data sources used

Priority should be given to international and/or European-based peer-reviewed and published studies

Author(s)	Year	Title	Journal / Book	Location
Moore R, Ormandy D.	2004	Home safety in the U.K.: review of the influence of human and housing factors.	Rev Environ Health. 2004 Jul-Dec;19(3-4):253-70.	United Kingdom
<a href="#">Startzell JK,</a> <a href="#">Owens DA,</a> <a href="#">Mulfinger LM,</a> <a href="#">Cavanagh PR.</a>	2000	Stair negotiation in older people, a review.	J Am Geriatric Soc 2000; May 48(5):567-80.	United States
Roys, MS	2001	Serious stair injuries can be prevented by improved stair design.	Applied Ergonomics 2001;31(2):135-9.	United Kingdom

##### General evidence (what effects and relationships are identified in literature?)

- Serious stair injuries can be prevented by improved stair design
- The number of steps or risers is an important design parameter that affects fall risk on stairs. Several researchers have found that a disproportionate number of falls occur on stairways that have a small rise.
- Even a 5mm irregularity, particularly at the top or bottom of a stairway, can disrupt foot movements to cause a fall

##### Specific evidence (how strong are the effects, which quantification has been identified?)

- In the UK there were 4000 deaths from home accidents and 2.5 million injuries; and due to stairs, 497 deaths and 230.000 injuries
- Because many users, especially older persons, may not have sufficient physical control to negotiate stairs with reduced goings, they have a high risk of a fall from stairs with smaller goings. The building industry has resisted to increasing the size of goings claiming that it takes extra space for the staircase and, hence, increases cost.

- Stairs cause 10% of all fatal fall injuries

**Limitations** (*are there any constraints and limitations associated with the data?*)

No EU incidence or prevalence for stair injuries, for home injuries in total

**Suggested OR / incidence / attributable fraction for exposed population** (*what seems to be the most reliable and evidence-based estimation on the likelihood that the health effect is expressed?*)

Final recommendation

**There is sufficient and reliable evidence for making a valid assessment**

## Part II for Stairs and Falls: Exposure situation

### Data sources used

Priority should be given to international and/or European-based studies in peer-reviewed literature

Author(s)	Year	Title	Journal / Book	Location
Roys, MS	2001	Serious stair injuries can be prevented by improved stair design.	Applied Ergonomics 2001;31(2):135-9.	United Kingdom

**General evidence** (*is a relevant exposure identified in the literature?*)

When exposure is taken into account, stairs are one of the most hazardous locations in buildings

**Specific evidence** (*how large is the exposure, which quantification has been identified?*)

**Limitations** (*are there any constraints and limitations associated with the data? Are there large variation within countries or by country or regions?*)

**Suggested exposure rate in the (European) population** (*what seems to be the most reliable and evidence-based estimation on the general exposure within the population?*)

Final recommendation

**There is some / partially sufficient and reliable evidence for making a valid assessment**

## **Windows, Balconies and Falls**

### **Part I: Health effects / Attributable risk / possible exposure-response relationship**

#### **Data sources used**

Priority should be given to international and/or European-based peer-reviewed and published studies

<b>Author(s)</b>	<b>Year</b>	<b>Title</b>	<b>Journal / Book</b>	<b>Location</b>
Istre GR, McCoy MA, Stowe M, Davies K, Zane D, Anderson RJ, Wiebe R	2003	Childhood injuries due to falls from apartment balconies and windows.	Inj Prev. 2003 Dec;9(4):349-52.	Texas, United States
Vish NL, Powell EC, Wiltsek D, Sheehan KM	2005	Pediatric window falls: not just a problem for children in high rises	Inj Prev 2005;11:300-303.	Chicago, United States
Weill, Florence CSC	2005	Falls from windows in Paris, France	To be submitted	Paris, France
Pressley JC, Barlow B	2005	Child and adolescent injury as a result of falls from buildings and structures	Inj Prev 2005;11:267-273	United States

#### **General evidence** (*what effects and relationships are identified in literature?*)

- Most common injuries are head trauma and extremity fracture, median age 2 years, majority from third floor or lower.
- Current building codes do not apply to older apartments, where most of these falls occurred. Nevertheless, these factors may be amenable to environmental modifications that may prevent most of these falls.
- Best practice building codes are those that require window bars or child resistant safety catches that limit the opening to less than 100mm on all second story or higher windows

#### **Specific evidence** (*how strong are the effects, which quantification has been identified?*)

- For more than two thirds of balcony related falls, the child fell from between the balcony rails, all of which were spaced more than 4 inches (10 cm) apart. On-site measurement showed the rails were an average of 7.5 inches (19 cm) apart; all of these apartments were built before 1984. For more than two thirds of window related falls, the window was situated within 2 feet (61 cm) of the floor.
- Very young minority is nearly twice that of whites
- Window guard law, with annual enforcement, has been effective in New York City where exposure to high rises associated with multifamily dwellings is higher than the national average

**Limitations** (*are there any constraints and limitations associated with the data?*)

Cases small: range from 90 to 100

**Suggested OR / incidence / attributable fraction for exposed population** (*what seems to be the most reliable and evidence-based estimation on the likelihood that the health effect is expressed?*)

- Vish et al.: Each year there are an estimated 15 window falls per 100 000 Chicago preschool children

- Pressley et al.: In the US the incidence for injury from falls (national average) from buildings is 2.81 per 100 000 people

Final recommendation

**There is sufficient and reliable evidence for making a valid assessment**

**Part II for windows and falls: Exposure situation**

**Data sources used**

Priority should be given to international and/or European-based studies in peer-reviewed literature

Author(s)	Year	Title	Journal / Book	Location

**General evidence** (*is a relevant exposure identified in the literature?*)

**Specific evidence** (*how large is the exposure, which quantification has been identified?*)

**Limitations** (*are there any constraints and limitations associated with the data? Are there large variation within countries or by country or regions?*)

**Suggested exposure rate in the (European) population** (*what seems to be the most reliable and evidence-based estimation on the general exposure within the population?*)

Final recommendation

**There is no sufficient and reliable evidence for making a valid assessment**

## **Low SES and home injuries**

### **Part I: Health effects / Attributable risk / possible exposure-response relationship**

#### **Data sources used**

Priority should be given to international and/or European-based peer-reviewed and published studies

<b>Author(s)</b>	<b>Year</b>	<b>Title</b>	<b>Journal / Book</b>	<b>Location</b>
Istre GR, McCoy M, Carlin DK, McClain J.	2002	Residential fire related deaths and injuries among children: fireplay, smoke alarms, and prevention.	Inj Prev. 2002 Jun;8(2):128-32.	United States
Hjern A, Ringback- Weitoft G, Andersson R.	2001	Socio-demographic risk factors for home-type injuries in Swedish infants and toddlers.	Acta Paediatr. 2001 Jan;90(1):61-8.	Sweden
<a href="#">DiGuseppi C</a> , <a href="#">Edwards P</a> , <a href="#">Godward C</a> , <a href="#">Roberts I</a> , <a href="#">Wade A</a>	2000	Urban residential fire and flame injuries: a population based study.	<a href="#">Inj Prev.</a> 2000 Dec;6(4):250-4.	United Kingdom
Delgado J, Ramirez- Cardich ME, Gilman RH et al.	2005	Risk factors for burns in children: crowding, poverty and poor maternal education	Inj Prev. 2002; 8;38-41	Peru

#### **General evidence** (*what effects and relationships are identified in literature?*)

- The highest rates for residential fires occurred in the youngest children (<5 years) and in census tracts with lowest income
- 77.5% of burns to children under 18 years admitted to the A&E units occurred in the home
- those at highest risk of urban residential fire and flame injuries are the elderly, young children, and *the poor*

**Specific evidence** (*how strong are the effects, which quantification has been identified?*)  
see above

#### **Limitations** (*are there any constraints and limitations associated with the data?*)

Inequalities for home injuries as a whole is not known, due to problems of linking home injuries with SES indicators

**Suggested OR / incidence / attributable fraction for exposed population** (*what seems to be the most reliable and evidence-based estimation on the likelihood that the health effect is expressed?*)



Low income (OR 2.8, 95% CI 2.0 to 0.9), overcrowding (OR 2.5, 95% CI 0.4 to 0.8) were associated with an increased risk for burns

Final recommendation

**There is sufficient and reliable evidence for making a valid assessment**

**Part II for low SES and home injuries: Exposure situation**

**Data sources used**

Priority should be given to international and/or European-based studies in peer-reviewed literature

Author(s)	Year	Title	Journal / Book	Location
<a href="#">DiGuseppi C</a> , <a href="#">Edwards P</a> , <a href="#">Godward C</a> , <a href="#">Roberts I</a> , <a href="#">Wade A</a>	2000	Urban residential fire and flame injuries: a population based study.	<a href="#">Inj Prev</a> . 2000 Dec;6(4):250-4.	United Kingdom

**General evidence** (*is a relevant exposure identified in the literature?*)

- 77.5% of burns to children under 18 years admitted to the A&E units occurred in the home

**Specific evidence** (*how large is the exposure, which quantification has been identified?*)

**Limitations** (*are there any constraints and limitations associated with the data? Are there large variation within countries or by country or regions?*)

Only known for the UK

**Suggested exposure rate in the (European) population** (*what seems to be the most reliable and evidence-based estimation on the general exposure within the population?*)

Final recommendation

**There is no sufficient and reliable evidence for making a valid assessment**

## Summary of main element of evidence between:

### **Cause/ Housing Factor: Housing and Neighbourhood conditions Health Effects: Fear of crime**

Contributor: Edmond D. Shenassa, USA

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#### **1. Bibliography (partial)**

- Alfonzo MA. 2005. To Walk or Not to Walk? The Hierarchy of Walking Needs. *Environment and Behavior*.37(6):808-836.
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#### **2. General evidence.**

##### 2a. Introduction.

Defensible space theory (Newman, 1972) has been proposed to explain a link between the physical environment and fear of crime. This concept may also be useful to further link the physical environment with its health consequences. These health consequences can be both physical (e.g., physical activity) and psychological (e.g., anxiety, depression). In this report, I focus on physical activity to provide a clear quantifiable example.

##### 2b. Defensible Space.

According to defensible space theory, design features that act as territorial displays, indicate occupancy, or provide natural surveillance opportunities can increase residents' territorial claims. According to this theory, these features influence whether a property is perceived as vulnerable to crime. Later works have expanded on this theory to include characteristics of the immediate space surrounding the dwelling as well as the larger neighborhood.

##### 2c. Physical activity.

Exercise is an important health behavior to examine in the context of place. Physical inactivity is second only to smoking with regard to negative health consequence. It is linked

with high blood pressure, back pain, atherosclerosis, coronary heart disease, and diabetes, many of which can be attributed to the link between physical inactivity and obesity. In Europe, the prevalence of obesity is steadily increasing, a trend that appears to be associated with an ever-more-prevalent sedentary lifestyle. In turn, obesity and a sedentary lifestyle are both linked with residence in areas that are not designed for residential use. In contrast, residence in proximity of parks and other aesthetically pleasing public spaces, predict an elevated likelihood of exercise and normal weight.

### Specific evidence.

#### 3a. – Characteristics of defensible spaces

##### Dwelling

Actual Barriers	Physical impediment to access - High Fence - Hedges
Symbolic Barriers	Physical characteristics suggesting owner's care, investment, and willingness to defend - Nameplates on doors and bells - Ornamentation - Personal planting - "Beware of Dogs" signs
Surveillability	Ease of ability to scan outside areas - Ability to view outside activities - Windows on both sides of building
Road Surveillability	Ability to see the property from the road or from neighbors dwellings - Windows visible from the road - Proximity of other homes
Traces of Occupancy	Environmental Cues that indicate vacancy or occupancy - Curtains in the window - Lights on after dark - Cars parked nearby
Control	Control of access to private and immediate public space
Physical Layout	Private entrances Hallways that share with only few apartment Small open spaces with observable boundaries. Relatively few units in the building Low rise building

##### Immediate surrounding area

Appropriation	Ability and willingness to alter areas around residences
Territorial	Block Watch Signs Care of common areas (i.e. picking up trash, grass maintenance) Monitoring of activities in near-home spaces

Social	<p>Informal social control (e.g., residents' willingness to intervene to stop inappropriate behaviors near home spaces).          Use of near-home space for social activities          Time spent relaxing with others          The prevalence of these behaviors are influenced by:              Floor level              Physical proximity to other units              Doorway orientation              Interaction nodes (e.g., mailboxes, recreational facilities)</p>
C. Physical	<p>Physical presence in the space.          Frequent use of near-home space</p>

Neighborhood

Physical Condition	<p>Well-maintained homes          Broad, well lit, streets</p>	- Green space
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3b. Partial list of studies of perceived safety and physical activity – European studies are in bold

Reference	Sample Characteristics	Sampling Process	Primary Exposure	Results
Li et al. 2005	56 neighborhoods in Portland, OR - N=577 - Age: 65-94 - 64% women	Stratified sample w/ over sampling of households below poverty.	Perception of <u>physical</u> Safety of neighborhood and sidewalks	Perceived safety predicted walking in the neighborhood.
<b>Foster et al. 2004</b>	<b>England</b> <b>N=4265</b> <b>- age: 16-74</b>	<b>Random sample (postcode address file)</b>	<b>Perception of neighborhood safety during the day</b>	<b>Perceived safety predicted walking among women.</b>
Humpel et al. 2004	Coastal Australian city and surrounding suburbs - N=399 - Age: > 40 - 57% women	Convenience Sample (health insurance organization through mail-out surveys)	Perception of neighborhood crime	Perceived safety independent of walking in the neighborhood.
Crank JP et al. 2003	Ada County, Idaho, USA N= 806 No age breakdown No mention of race	Random sample (area codes)	Perceptions of safety in neighborhood and downtown	Perceived safety predicted recreational activity outside the house

Reference	Sample Characteristics	Sampling Process	Primary Exposure	Results
<b>De Bourdeaudhui j, et al. 2003</b>	<b>Ghent, Belgium</b> - N=521 Age: 18-65, 48.3% women	<b>Random sample</b>	<b>Perception of safety from crime &amp; traffic</b>	<b>Perceived safety from crime or traffic independent of walking or physical activity</b>
Evenson et al. 2003	North Carolina, U.. - N=671 - Age: 25-37 - 100% women - Latina immigrants	Convenience sample (local community) locations	Fear of crime in the neighborhood	Perceived safety independent of Physical activity.
<b>Green et al. 2002</b>	<b>Liverpool, UK</b> N= 407 “Generally elderly;” race not reported No Gender breakdown	<b>Convenience Sample (residents in 21 tower blocks on 4 estates)</b>	<b>Perception of neighborhood crime</b>	<b>Perceived safety predicts relatively better physical and mental health.</b>

### Limitations

Existing studies have generally used small samples drawn from small geographic areas (relative to LARES).

Factors that mediate the association between perception of safety and physical activity have not been adequately examined.

### 5. Suggested associations – Results from analysis of LARES data

Compared to non-exercisers, occasional exercisers who perceived their area of residence to be safe were 28% more likely to engage in exercise than those who did not perceive their neighborhood to be safe (95% CI: 1.07, 1.52). Among men, there was no association between perceived safety and exercise among frequent exercisers. Among women, the OR for occasional exercisers is 1.22 (95% CI 1.00-1.53) and 1.42 (95% CI 1.04-1.93) for frequent exercisers, respectively.

### Concluding remarks

Existence of a steep socioeconomic (SES) gradient in health is by now well established. Efforts to redress these disparities, however, are nascent and can benefit from improved understanding of pathways between SES and Health. Thus far, these remedial efforts have been mostly informed by two influential lines of research, one focusing on the individual and the other on the community. The individual level approach has been generally focused on socioeconomic status and its accompanying levels of control and perceived or real stress. This line of work has built on seminal paradigms of “fight or flight”, “learned helplessness”,

and “perceived efficacy” put forth by Selye (1956), Seligman (1970), and Bandura (1986) respectively. The other line of research initiated by Durkheim (1951), Faris & Dunham, (1939) and expanded by Hollingshead & Redlich (1958), Weschler & Pugh (1967), Dohrenwend and Dohrenwend (1969), and others focused on social determinants of health and has led to the contemporary literature on trust, social cohesion, community efficacy, and other related constructs as “fundamental causes” of health. More recent works, greatly influenced by the work of Wilson (1996), have been consistent in demonstrating that both individual and social-level determinants exert an independent influence on health. These works, and the relatively recent introduction of hierarchical statistical models to public health studies, have allowed simultaneous consideration of both individual and social level determinants of health.

If one accepts the simultaneous importance of individual and social level determinants of health, it follows that housing is a likely mediator of the SES-health link. Recent evidence (e.g., Shenassa, et al., 2003; Jargowsky, 1997; Dunn, 2003) suggests that housing may be a fulcrum between individual and social-level determinants of health. Although individual opportunities for redressing social-disparities in health are at best few, and there is a paucity of political support for broad social programs to redistribute wealth, *existing* funds and policies for “home improvement” programs may provide effective strategies for improving individual and community health.

## **Summary of main element of evidence between:**

### **Cause/ Housing Factor: Particulate Matter and fine particles in indoor air Health Effects: Respiratory and allergic effects**

Contributor: Jan Sundell, Denmark / Sweden

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Each day we inhale 20,000 liters of air compared to just a few liters of food and drink. During a normal life (70 years) more than 70% of the total exposure (in mass) is air in homes, schools, offices etc. By far most important is air in the home and especially in the bedroom. For more vulnerable populations, e.g. children, elderly and sick people the air in homes is even more accentuated.

There is a vast scientific literature on outdoor particulate air pollution and health. It is well established that fine particles (PM<sub>2.5</sub>) are associated with mortality, and hospitalization for cardio-pulmonary disease (WHO 2003). It is estimated that annually about 100 000 deaths occurs in Europe (725 000 years of life lost) due to particulate matter in outdoor air (WHO 2003).

As people are mainly exposed to ambient particles indoors, a large majority of these deaths, besides the morbidity etc, should be attributed to indoor settings, mainly the home. A main problem is that indoor exposure, besides being dependant on outdoor air pollution, infiltration, and ventilation etc also is depending on indoor sources of particles. Such sources, like tobacco smoking, cooking etc varies greatly between, regions and buildings and will easily result in concentrations of particles that can be magnitudes higher than normally achieved in ambient air. Also ventilation practices and air tightness varies vastly between different regions. In some regions air is mainly taken directly from the ambient air (window opening etc), in other regions air is mostly recirculated (for cooling, or heating). Such different ventilation techniques means different exposures to ambient pollutants. Such factors are not thoroughly dealt with in epidemiological studies on ambient air and health. Studies presenting results from ambient, indoor and personal exposures, show that indoor exposures are most important for total exposure.

The scientific literature on indoor exposures to particles and health has recently been reviewed by a European interdisciplinary group (Schneider et al. *Indoor Air* 2003;13:38-48) The main conclusions were:

“The group stresses that no conclusions can be drawn from the present study regarding the relative contribution to health effects of particles per se, of particulate matter containing low levels of ubiquitous allergens, toxins or irritants, particles of outdoor origin, or of specific major components. It can only be concluded that there is insufficient evidence to support use of particulate matter measured indoors as mass or number concentration as a generally applicable risk indicator for health effects in the indoor, non-industrial environment. There is a fundamental lack of conclusive research on indoor airborne particulate matter and health effects, including the role of ultrafine particles. “

Thus, if ambient exposure to particles matters with regard to health, and the evidence is strong, then indoor exposure to particles should be a very important issue! However, studies of indoor exposures and health are almost non-existing with regard to particles.

To complicate matter, a large number of ultrafine particles are created indoors due to the reaction between constituents in common cleaning products (containing e.g. limonene) and normal concentrations of ozone from outdoor air.

### **Part I: Health effects / Attributable risk / possible exposure-response relationship**

Data sources used (WHO 2003, Sarnat et al. 2005, Schneider et al. 2003... )

A large number of health effects are suspected (mainly cardiovascular and cardiopulmonary) from studies of ambient air. As the main exposure to ambient particulate matter is indoor a major part of these health effects are possibly due to indoor exposure. However, as there is an almost total lack of studies on indoor exposures and health no attributable risk can be calculated, and no exposure-response relationships can be given.

Unless data from studies on ambient air and health could be used? In, that case, the recent result from a WHO meeting in Bonn can be used (to be discussed!).

General evidence (*what effects and relationships are identified in literature?*)

The evidence from epi studies are strong that there exists an association between ambient particle exposure and health (many health effects!). For indoor exposures, the scientific evidence is non-existing.

Specific evidence (*how strong are the effects, which quantification has been identified?*)

The effects and evidences are strong with regard to outdoor air exposures and health, but data on indoor exposures and health are missing.

Limitations (*are there any constraints and limitations associated with the data?*)

It is not easy to translate the epi data regarding ambient air to exposures indoor air and health.

Suggested OR / incidence / attributable fraction for exposed population (*what seems to be the most reliable and evidence-based estimation on the likelihood that the health effect is expressed?*)

If the assessments regarding exposures in ambient air, and in developing countries are accepted some risk assessments can be given! If relying on studies on indoor exposures in developed countries, no assessments can be made.

Final recommendation

**There is no sufficient and reliable evidence for making a valid assessment**



## Part II: Exposure situation

Data sources used

Priority should be given to international and/or European-based studies in peer-reviewed literature

Author(s)	Year	Title	Journal / Book	Location
Schneider et al.	2003	“EUROPART”. Airborne particles in the indoor environment. A European interdisciplinary review of scientific evidence on associations between exposure to particles in buildings and health effects.	Indoor Air	2003;13:38-48
WHO	2003	Health aspects of air pollution with particulate matter, ozone and nitrogen dioxide	WHO	

General evidence (*is a relevant exposure identified in the literature?*)

For indoor settings no relevant particle exposure is defined, mass, size, number...??

But there is evidence from studies on ambient air regarding mass.

Specific evidence (*how large is the exposure, which quantification has been identified?*). No good studies have been conducted regarding indoor air.

Limitations (*are there any constraints and limitations associated with the data? Are there large variation within countries or by country or regions?*)

There are certainly large variations between countries and regions, but not studied with regard to health consequences.

Suggested exposure rate in the (European) population (*what seems to be the most reliable and evidence-based estimation on the general exposure within the population?*)

Final recommendation (*please delete the answers that do not apply*)

**There is no sufficient and reliable evidence for making a valid assessment**

## Summary of main element of evidence between:

**Cause/ Housing Factor: Heat / indoor temperatures**  
**Health Effects: Cardiovascular effects/ excess mortality**

Contributor: Oliver Thommen, Switzerland

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### Part I: Health effects / Attributable risk / possible exposure-response relationship

#### Data sources used

Priority should be given to international and/or European-based peer-reviewed and published studies

Author(s)	Year	Title	Journal / Book	Location
Basu, R., Samet J.M.	2002	Relation between elevated ambient temperature and mortality: a review of the epidemiologic evidence	Epidemiol Rev 24(2): 190-202	Review (US)
Naughton, M. P. et al.	2002	Heat-related mortality during a 1999 heat wave in Chicago	Am J Prev Med 22(4): 221-227	Chicago
Kaiser, R. et al.	2001	Heat-related death and mental illness during the 1999 Cincinnati heat wave	Am J Forensic Med Pathol 22(3): 303-307	Cincinnati
Semenza, J. C. et al.	1996	Heat-related deaths during the July 1995 heat wave in Chicago	N Engl J Med 335(2): 84-90	Chicago
Kilbourne, E. M et al.	1982	Risk factors for heatstroke. A case-control study	Jama 247(24): 3332- 3336	St Louis and Kansas City

#### General evidence (*what effects and relationships are identified in literature?*)

Elevated ambient temperature has adverse effects on mortality.

#### Specific evidence (*how strong are the effects, which quantification has been identified?*)

##### Risk factors:

##### *Total mortality*

The strongest risk factors for heat-related death were living alone (OR ranged between 2.3 and 8.1) and not leaving home daily (OR ranged between 5.8 and 6.7).

The risk of heat-related death was increased for people with known medical problems who were confined to bed (OR ranged between 5.5 and 8.2) or who were unable to care for themselves (OR, 4.1). Also at increased risk were those who lived on the top floor of a building (OR, 4.7). Mental illness showed a strong positive association with heat-related death too (OR, 14.0).

### *Heat stroke*

Alcoholism, living on the higher floors of multistory buildings, and using major tranquilizers (phenothiazines, butyrophenones, or thioxanthenes) were factors associated with increased risk for fatal heat stroke.

### Protective factors:

#### *Total mortality*

The strongest protective factor was a working air conditioner (OR ranged between 0.03 and 0.3). The risk of death was reduced for people with access to transportation (OR, 0.3). Having social contacts such as group activities or friends in the area was protective.

### *Heat stroke*

Factors associated with decreased risk for fatal heat stroke were using home air conditioning, spending more time in air-conditioned places, and living in a residence well shaded by trees and shrubs. Being able to care for oneself, characteristically undertaking vigorous physical activity, but reducing such activity during the heat, and taking extra liquid were also associated with decreased risk.

### **Limitations** (*are there any constraints and limitations associated with the data?*)

#### Case control studies

Relatively younger age of case patients. The quality of surrogate data can be of questionable reliability.

The selection of controls are a potential source of bias, as people with mental illness may not answer the door or refuse to talk to the interviewer. Mental illness of controls is self-reported and therefore likely to be underreported. Mental illness of cases reported by surrogates and/or obtained through medical records is possibly overestimated. The use of information of surrogates lead to potential misclassification of risk factors.

Underestimation of the risk associated with social isolation, since people with few social contacts were excluded (no identifiable surrogate). A limitation inherent in the use of death certificates is the possible misclassification of causes of death.

**Suggested OR / incidence / attributable fraction for exposed population** (*what seems to be the most reliable and evidence-based estimation on the likelihood that the health effect is expressed?*)

#### Final recommendation

**There is some / partially sufficient and reliable evidence for making a valid assessment**

## Part II: Exposure situation

### Data sources used

Priority should be given to international and/or European-based studies in peer-reviewed literature

Author(s)	Year	Title	Journal / Book	Location
Grize, L. et al.	2005	Heat wave 2003 and mortality in Switzerland	Swiss Med Wkly 135: 200–205	Switzerland
Kosatsky, T.	2005	The 2003 European heat waves	Euro Surveill 10(7): 148-149	Europe
Beniston, M.	2004	The 2003 heat wave in Europe: A shape of things to come? An analysis based on Swiss climatological data and model simulations	Geophys Res Lett 31: 2002-2026	Europe
Luterbacher, J. et al.	2004	European seasonal and annual temperature variability, trends, and extremes since 1500	Science 303(5663): 1499-1503	Europe
Schär, C. et al.	2004	The role of increasing temperature variability in European summer heatwaves	Nature 427(6972): 332-6	Europe
Stott, P. A. et al.	2004	Human contribution to the European heatwave of 2003	Nature 432(7017): 610-614	Europe
Haines, A. Patz, J. A.	2004	Health effects of climate change	Jama 291(1): 99-103	
Meehl, G. A. Tebaldi, C.	2004	More intense, more frequent, and longer lasting heat waves in the 21st century	Science 305(5686): 994-997	Europe/ North America
Dorozynski, A.	2003	Heat wave triggers political conflict as French death rates rise	Bmj 327(7412): 411	France
Patz, J. A. Kovats, R. S.	2002	Hotspots in climate change and human health	Bmj 325(7372): 1094-1098	
IPCC	2001	Climate Change 2001: Impacts, Adaptation and Vulnerability. Contribution of Working Group II to the Third Assessment Report of the Intergovernmental Panel on Climate Change.	Cambridge, U.K.: Cambridge University Press., p. 1032	
Kovats, R. S. et al.	1999	Climate change and human health in Europe	Bmj 318(7199): 1682-1685	Europe

### General evidence (*is a relevant exposure identified in the literature?*)

New record extreme events occur every year somewhere in the globe, but in recent years the number of such extremes have been increasing. This corresponds to the forecasts of experts in the field of climate change.

Climate change is expected to increase average summer temperature and the frequency and intensity of hot days and heat waves. An increase in the duration, intensity and frequency of (summer) heat waves is expected to have an impact of all causes of death (and is associated with heat cramps, heat syncope (fainting), heat exhaustion, heat stroke, dehydration).

Under all emission scenarios proposed by the Intergovernmental Panel on Climate Change (IPCC), atmospheric concentrations of CO<sub>2</sub> rise markedly. Under such conditions extremes in temperature increase. Observations and models show that present-day heat waves over Europe and North America coincide with a specific atmospheric circulation pattern that is intensified by ongoing increases in greenhouse gases, indicating that it will produce more severe heat waves in those regions (e.g. France, Germany, the Balkans) in the future. For example, it is anticipated that the equivalent of the UK heatwave in summer 1976, which occurs once every 310 years under the current climate, may occur every 5 to 6 years by 2050.

#### Heat Wave 2003

It seems likely that past human influence has at least doubled the risk of heat waves such as 2003. The European summer climate might experience a pronounced increase in year-to-year variability in response to greenhouse-gas forcing.

The deaths that occurred during the heat wave 2003 were not simply a displacement of expected deaths for that year. A substantial loss of life-years occurred. The thousands who died in the European heat wave show current failings in dealing with this threat.

France was the European country most affected by the heat wave with an estimated excess mortality of 54%. Across 13 of the largest French cities, 14 800 excess deaths were estimated for the period between August 1 and August 20. The combination of elevated temperatures during the day and during the night showed the strongest association with mortality, alluding to the importance of elevated night temperature for the heat effects on health. Other European countries such as Switzerland, Italy, Spain, Portugal, the Netherlands, Germany, England/Wales and Belgium also reported excess mortality during summer 2003.

**Specific evidence** (*how large is the exposure, which quantification has been identified?*)

**Limitations** (*are there any constraints and limitations associated with the data? Are there large variation within countries or by country or regions?*)

The relationship between climate change and extreme weather events is not yet that well understood. Therefore there are uncertainties in forecasts of the future health effects of climate change.

(Climate change poses a range of challenges to health, but many of the linkages are complex and a range of other social, behavioral and environmental factors also affect the health outcomes in question).

**Suggested exposure rate in the (European) population** (*what seems to be the most reliable and evidence-based estimation on the general exposure within the population?*)

Final recommendation

**There is some / partially sufficient and reliable evidence for making a valid assessment**

## Summary of main element of evidence between:

**Cause/ Housing Factor: Housing improvement**  
**Health Effects: Physical and mental health**

Contributor: Hilary Thomson, UK

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### Part I: Health effects/Attributable risk/possible exposure-response relationship

#### Data sources used

Thomson, H., M. Petticrew, et al. (2001). Health effects of housing improvement: systematic review of intervention studies. *British Medical Journal* **323**: 187-90.  
<http://bmj.bmjournals.com/cgi/content/full/323/7306/187>

Ellaway, A., S. Macintyre, et al. (2005). Graffiti, greenery, and obesity in adults: secondary analysis of European cross sectional survey. *British Medical Journal* **331**: 611-612.  
<http://bmj.bmjournals.com/cgi/content/full/bmj;331/7517/611>

#### **General evidence** (*what effects and relationships are identified in literature?*)

A systematic review of world literature dating back to 1936 was carried out to investigate the possible health impacts of housing improvement. Nineteen studies (of any design, in any language) which had assessed the health of residents whose houses had been improved were identified. The data from these studies are summarized below and in the attached table:

- housing improvements can improve residents' health, in particular their mental health;
- housing improvements may sometimes have negative as well as positive health impacts
- housing improvements often lead to related changes, for example rent increases, which in turn may lead to negative health impacts linked to financial strain;
- the original residents may move to another area and not benefit from the improved housing.

#### **Specific evidence** (*how strong are effects, which quantification has been identified?*)

#### **What type of housing improvement is most likely to lead to different types of health improvement?**

The different types of housing improvement may have different types of health impacts, these are reviewed below.

#### **Housing led neighbourhood regeneration** (*New housing, major housing refurbishment accompanied by neighbourhood improvement or relocation to a new neighbourhood*)

Housing led neighbourhood regeneration is likely to lead to improved levels of mental health among residents but the potential for improved physical health or general wellbeing is unclear. Although some studies did find improved physical health, there were also studies which found that physical health or levels of general health were worse after the housing improvement.

One study reported a dose response effect; the degree of health improvement was directly related to the degree of housing improvement experienced. Those residents who experienced the greatest degree of housing improvement also experienced the greatest health improvement.

### **Energy efficiency improvements**

Energy efficiency improvements (e.g. installation of heating or insulation) may lead to a small improvement in levels of general health as well as a reduction in respiratory symptoms. There is no suggestion of impacts on measures of mental health. In one study, following installation of improved heating, the amount of school time lost due to asthma among school children fell significantly.

### **Rehousing to meet medical/mobility requirements of residents**

Rehousing residents with specific medical or mobility requirements to more suitable housing is likely to lead to improvements in health, in particular improvements in mental health.

### **Limitations** (*are there any constraints and limitations associated with the data?*)

#### **Lack of research evidence**

The main limitation is the small amount of research evidence about the impacts of housing improvement, as well as the poor quality of research in this area. This does *NOT* mean that housing improvement does not lead to health improvement: rather that little is known about the health impacts of housing improvement. Although the links between poor housing and poor health are well documented there is very little research evidence to confirm that investment to alleviate poor housing conditions will lead to improved health. Indeed, some of the available research evidence suggests that housing improvement does not lead to improved health and may even lead to negative health impacts.

There is insufficient evidence around specific housing improvements to know what type of housing improvement is most cost-effective in terms of health improvement.

### **Other changes associated with housing improvement which may affect health**

Housing improvement is accompanied by changes to the wider housing context. Some of these factors may also affect health, either negatively or positively. Impacts which have been reported to be part of housing improvement programmes are summarized below:

**Positive impacts:** improved perceptions of safety, greater community involvement and increased area satisfaction.

**Negative impacts:** increased housing costs, the displacement of original residents, social exclusion and community division (for those in neighbouring areas not benefiting from the improvements), disruption, uncertainty and lack of control with respect to moving.

Only some of these 'other' impacts have been linked to health impacts. Most notably increased housing costs can add financial strain and may indirectly affect health by reducing the money available to spend on adequate heat, food and other necessities.

Identifying changes commonly associated with housing improvement which may also affect health, either positively or negatively, may help minimize some adverse effects while maximizing the potential for health gain.

### **Housing improvement, neighbourhood change and health**

In addition to improvements to indoor housing conditions, much housing investment includes improvements to the surrounding neighbourhood, thus improving the outdoor housing environment.

Very little is known about the health impacts of new neighbourhood investment and health but cross-sectional data suggests that a neighbourhood may have a small health effect independent of individual socio-economic status. The appearance of the local neighbourhood, such as greenery, graffiti and other incivilities, has been linked to self-reported health, anxiety, depression, sexually transmitted disease, and physical activity.

Findings from the recent cross-European LARES study reported that those living in residential neighbourhoods with more greenery and less litter and graffiti were significantly more likely to be physically active and less likely to be overweight. This relationship is independent of age, sex, socio-economic status and city of residence. (Ellaway et al 2005)

**Suggested OR / incidence / attributable fraction for exposed population** (*what seems to be the most reliable and evidence-based estimation on the likelihood that the health effect is expressed?*)

### **Impact of housing improvement on mental health**

The strongest research evidence for the health impacts of housing improvement relates to positive impacts on mental health. Nine studies assessed impact on mental health and in each study positive impacts were reported. It is difficult to provide an estimate of effect size as the measures used vary across the studies. To illustrate the impacts on mental health an example of a study of rehousing from slum conditions (conducted in US, Wilner 1958\* & 1960\*) is presented here. Eighteen months after the move there was an increase in those reporting a 'positive mood' in both the intervention group and the control group but the increase in the intervention group was greater, +13.6% v +10.6% (intervention v control group).

With respect to impacts on physical health there was no consistent trend observed. An example comparing two recent studies of housing led regeneration in the UK is presented below:

### **Impact of housing improvement on respiratory health**

*1-4 years after improvement*

Ambrose\*            -11% asthmatic & bronchial symptoms  
                             +25% coughs and colds (recent flu epidemic)

*5 years after improvement*

Blackman\*        +4.2% acute respiratory conditions (adult)  
                             +12% chronic respiratory conditions (adult)  
                             -4.7% acute respiratory conditions (children)  
                             +2.3% chronic respiratory conditions (children)

\* from Thomson et al, BMJ, 2001.



Final recommendation

**There is some/partially sufficient and reliable evidence for making a valid assessment**  
**Part II: Exposure situation**

**Data sources used**

Bonnefoy, X. R., M. Braubach, et al. (2003). "Housing and Health in Europe: Preliminary Results of a Pan-European Study." American Journal of Public Health 93(9): 1559-1563.

Thomson H & Petticrew M. Is housing improvement a potential health improvement strategy? WHO Health Evidence Network 2005. (commissioned report for European policy makers)  
[http://www.euro.who.int/HEN/Syntheses/housinghealth/20050214\\_2](http://www.euro.who.int/HEN/Syntheses/housinghealth/20050214_2)

**General evidence** (*is a relevant exposure identified in the literature?*)

The amount and type of housing improvement required to generate health improvement will vary depending on:

**Baseline housing conditions:** the greatest potential for housing improvement to lead to health improvement will be where baseline housing conditions are very poor. Baseline housing conditions will often depend on a country's stage of economic development, history of housing investment and enforcement of building controls. In situations where housing is very poor, it is likely that the level of health improvement will be directly related to the extent of housing improvement experienced.

**Vulnerability to hazards of poor housing:** the greatest potential for health gain will be among those most vulnerable to the harmful effects of poor housing (i.e. those with poor health, the elderly and the very young).

**Levels of exposure to poor housing (individuals):** exposure to housing will vary between individuals depending on the amount of time spent indoors. Residents of poor housing and who spend the most time indoors will be most likely to benefit from housing improvement

**Relative importance of specific hazard within country/region:** the relative importance of some hazards may vary between countries or regions. This means that the potential for some specific types of housing improvement to lead to health improvement will also vary between countries. (*see section 9 below*)

**Specific evidence** (*how large is the exposure, which quantification identified?*)

**What is the extent of housing improvement and housing need in the European Region?**

Not known- locally relevant data required for each country. (*see section 9 below*)

**UK example:** In the UK there is a mix of publicly funded housing improvement; large-scale area based housing led regeneration, and programmes to deliver energy efficiency

improvements individual houses. For example, in Scotland since 1999, 129,760 homes (around 5% of population) have been insulated through Warm Deal programme leading to: increase in average energy rating on the NHER scale (+0.95) savings in average annual fuel bills of £95 for owner-occupiers and £62 for local authority tenants <sup>1</sup>

Levels of fuel poverty are falling. In Scotland, between 1996-2002 the proportion of fuel poor households fell from 35% of all households to 12%. <sup>2</sup> The amount of non-decent housing appears to be falling. In England, between 1996 and 2003 there was a significant reduction (-10%) in the proportion of households living in non-decent housing. There has also been a significant narrowing of the gap between absolute numbers of vulnerable households living in non-decent housing and all households living in non-decent housing. <sup>3</sup>

A national survey of housing conditions and health in Scotland was carried out in 2002; links between housing condition and health were statistically significant but small. <sup>4</sup> Fuel poverty, levels of disrepair and presence of central heating was not linked to self-reported health, respiratory health or mental health among children or adults. Housing factors which were linked to respiratory health were use of heating, and satisfaction with heating. Housing factors which were linked to poor self-reported health were the energy rating of the house, satisfaction with house and local neighbourhood, and tenure.

<sup>1</sup> <http://www.scotland.gov.uk/library3/housing/bfwd-00.asp#b8>

<sup>2</sup> <http://www.scotland.gov.uk/News/Releases/2003/11/4517>

<sup>3</sup> <http://www.dh.gov.uk/assetRoot/04/11/76/98/04117698.pdf> (page 48)

<sup>4</sup> [http://www.communitiesscotland.gov.uk/stellent/groups/public/documents/webpages/otcs\\_006018.pdf](http://www.communitiesscotland.gov.uk/stellent/groups/public/documents/webpages/otcs_006018.pdf)

**Limitations** (*are there any constraints and limitations associated with the data? Are there large variation within countries or by country or regions?*)

Locally relevant priorities for housing improvement are required.

Housing conditions and the amount of housing improvement ongoing across the European Region will vary widely and will be influenced heavily by local political, cultural, social, economic, and climate factors. For example, there have been significant social, political and economic changes in both eastern and western Europe that have influenced housing environments. The changes may have been the starkest in post-Communist countries, but ageing housing stock, decentralization of government responsibility, pressures on energy reserves and lifestyle shifts have had implications for housing needs across Europe. (Bonney et al) Sociopolitical and cultural context may also influence the meaning and value attached to housing conditions, size, design and ownership. Culture and climate present additional factors which will affect the amount of time spent in the home and related exposure to domestic hazards.

Moreover, while certain housing features are considered low or negligible health risks in some countries, due to variations in enforcement of building controls and other contextual factors, in other countries the same housing features may pose a significant risk to health. For example, in Turkey the second most common cause of accidental death is falling from a flat roof. Falls are most common there in summer, when people often sleep on the roof to keep cool.

**Scope of summary**

The research evidence presented here relates only to improvements to general physical housing condition and does not include housing improvements to reduce domestic accidents, infestation, or exposure to lead, radon, or allergen sources such as house dust mite.

Final recommendation

**There is some / partially sufficient and reliable evidence for making a valid assessment**

**Table 1: Evidence from controlled and uncontrolled intervention studies of specific health impacts of housing**

Effect on outcomes measured	General Health or wellbeing	Symptoms/ Illness & health service use	Respiratory	Mental health	Mortality	Social
<b>Rehousing/refurbishment plus relocation from slum area or community regeneration</b>						
	↔ Unclear effects on measures of general health +	↔ Unclear effects on symptoms or illness episodes ++  ↔ Unclear effects on health service use +	↔ Conflicting findings from 4 studies	↑↑ Consistent improvements in mental health ++	↓↓ Increased +	↑↑ Numbers of smokers reduced +  ↑↑ Increased community involvement, social support, sense of belonging and feeling of safety. Reduced fear of crime and sense of isolation +  ↓↓ Increased rents led to reduced income to buy adequate diet +
<b>Medical Priority Rehousing (MPR)</b>						
	↑↑ Improved objective measure and self-reported health +	↔ Unclear effects on health service use +		↑↑ Improvement in objective measure & self-reported mental health ++		
<b>Energy Efficiency Measures</b>						
	↑↑ Improved objective measure of health +	↔ Unclear effects on general symptoms +	↑↑ Reduction in respiratory symptoms +	↔ No significant difference in emotion and mental health +		↑↑ Less school time lost due to asthma, but not other symptoms +

**Direction of effect**  
 ↑↑ Improvements to health or reductions in illness  
 ↔ No clear effect on health or illness indicators  
 ↓↓ Reductions in health or increases in illness

**Strength of evidence**  
 +++ Strong association: evidence from prospective controlled studies with good levels of follow up  
 ++ Moderate association: evidence from at least one prospective controlled studies  
 + Weak association: evidence from uncontrolled studies

**Reproduced from:** Thomson, H., M. Petticrew, et al. (2003). "Health Impact Assessment of housing improvements: incorporating research evidence." Journal of Epidemiology and Community Health 57: 11-16.

*Annex 4 Causal web for housing burden of disease*

