First EnVIE Conference on Indoor Air Quality and Health for EU Policy

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Proceedings

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EnVIE scope

EnVIE is a co-ordination action on indoor air quality and health effects supported by the sixth framework R&D Programme of the European Union. It involves as partners 18 European Institutions from 11 countries covering a wide spectrum of scientific themes related to health and the built environment, as well as, with proven ability to disseminate and interface the outcome of the project with policy making.

Modern European citizens spend in excess of 90% of their time in indoor environments. Up to now national and European air quality policies have devoted most of their efforts towards policies aiming at the limitation of outdoor environmental concentrations for some specific pollutants, particularly, some as a result from industrial activities and automobile traffic. However, a growing awareness of the critical role of the indoor environment on public health has been leading to the recognition that indoor exposures to air pollution must be given higher attention in policy making in order 1) to better characterise and understand and assess the contribution of indoor spaces to environmental diseases; and 2) to set up coherent policies as regards the reduction of exposures.

It is therefore the objective of this project to increase the understanding of health impacts of indoor air quality. It will especially focus on the assessment of policy relevance of research into the health effects of isolated agents and mixtures, and will consider the implications for thresholds and safety margins for the general population and for people at work. It will address in particular how indoor air quality contributes to the observed rise in asthma and respiratory allergy in Europe as well as in other acute and chronic health impacts.

To respond to the objectives, EnVIE has identified three major and complementary issues as key questions regarding indoor air, estimated manageable in terms of scientific and technical proximity: health effects, exposure and characterisation of spaces and sources. Since the aim of EnVIE is to aggregate building blocks, interfacing sciences and policy making, EnVIE will specifically consider the issue of integration and policy interface. It will also pay significant attention to the dissemination, in particular, through the organisation of the EnVIE conferences and production of reports.

EnVIE will identify the most widespread and significant indoor causes and sources for these health impacts, and evaluate the existing and optional building and housing related policies for controlling them. It will address in particular how indoor air quality might contribute to the observed rise in asthma and respiratory allergy, together with acute and chronic health impacts.

The formal outcome of EnVIE, besides all fora and interactions with the scientific and policy making actors will be expressed in two sets of proceedings corresponding to two major Conferences, one held in June 2007 and the other being planned for the second semester of 2008.

The present publication contains the proceedings of the 1st Conference held in Helsinki in June 12-14.2007. The proceedings reflect the results of an exercise of interdisciplinarity focussed on the characterization of the relationship health effects – exposures – sources, following that order, in what represents a new methodology, referred as 'EnVIE approach' or 'EnVIE concept' as explained in Session 1B.

From the evaluation of the overall impact of poor indoor air quality in Europe, knowing the relative contributions of indoor environments to exposure and health effects, it is possible to make progress on the effective risk management and resources allocation to inform policy makers about evidence – based preventive policies. This will be the scope of the 2nd EnVIE Conference.

The final report will be ready in October 2008.

Porto, August 30, 2007

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08.30-09.00	Registration
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10.15-10.45	**Session 1B: Introduction of EnVIE concept and overview of EnVIE project** *Chair: A. Bartonova* *Rapporteur: P. Harrison* *Speakers: M. Jantunen and P. Carrer*
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11.00-12.30	**Session 2: Health topic - SBS, irritation, odours** *Chair: P. Carrer* *Rapporteur: D. Crump* *Speaker: L. Mølhave* Panel presentations and discussion Key exposure agent: VOCs - *H. Prejzner* Key sources: Building materials, consumer products - *H. Gustafsson*
12.30-13.45	Lunch
13.45-15.15	**Session 3: Health topic - Asthma and allergy** *Chair: M. Endrody* *Rapporteur: A. Bartonova* *Speaker: J. Sundell* Panel presentations and discussion Key exposure agent: Bio-aerosols - *A. Nevalainen* Key source: Moisture - *U. Haverinen-Shaugnessy*
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17.00-18.30*	**EnVIE project overview* –** *E. de Oliveira Fernandes*

*** To be attended only by ENVIE Partners.**

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Time	Topic
8.30-10.45	**Session 5: Health topic - Cancer, acute cardiovascular effects and COPD** *Chair: D. Kotzias* *Rapporteur: L. Mølhave* *Speakers: I. Annesi-Maesano and P. Carrer* Panel presentations Key exposure agent: Combustion products - *M. Jantunen* Key sources: Outdoor air, indoor combustion processes - *D. Crump* **In Focus: CO and acute intoxication** – *P. Carrer* **In Focus: Radon and lung cancer** - *J. McLaughlin* Panel discussion
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14.30-16.00*	**Three (3) Parallel Workshops** (WP 1, 2 and 3)*
16.00-17.00*	**Plenary session: Reporting from the workshops, Wrap-up.*** *Chair: E. de Oliveira Fernandes* *Speakers:* WP1 leader WP2 leader WP3 leader

*** To be attended only by ENVIE Partners.**

Session 1A Overview of European projects on indoor air quality and health

Development of WHO Guidelines for Indoor Air Quality (IAQ)

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ABSTRACT

The working group of the Global Update of WHO Guidelines for Air Quality recommended development of guidelines specific to indoor air, accounting for the global burden of disease associated especially with unvented indoor combustion of solid fuels and other factors that are not covered sufficiently by the general air quality guidelines. First phase to follow up consisted of a planning meeting convened in Bonn, Germany, in October 2006 that outlined a structure for the IAQ guidelines and identified exposure factors to be included into the IAQ guidelines. The factors include - next to traditional pollutant specific approaches - also biological agents and indoor combustion. In the second phase, the guideline development process is started from the subgroup of biological agents, followed up by addressing policy implications of actions to reduce health risks due to indoor air pollution with biological agents in the third phase. Further work on air pollutants and indoor combustion is to be developed in the near future.

PHASE 1: FOCUSING ON INDOOR AIR

The WHO Air Quality Guidelines (AQG) are designed to offer guidance in reducing adverse health impacts of air pollution based on expert evaluation of current scientific evidence. Especially, various problems in indoor air quality are recognized as important risk factors for human health in both developing as well as developed countries. The basic right for, and importance of, healthy indoor air has been emphasized also by the World Health Organization (WHO, 2000a).

Importance of indoor air is magnified by the substantial fraction of time populations spend within buildings. Indoor combustion is a source of pollution causing severe burden to health, especially for children and women in developing countries. In residences, day-care centres, elderly people homes and other special environments, indoor air pollution affects population groups that are especially susceptible due to

their health status or age. There is a substantial body of research on health effects of indoor exposures, listing many potentially hazardous compounds released indoors due to combustion, emissions from building materials, household equipment and consumer products. Microbial pollution comes from hundreds of species of bacteria, fungi, and moulds growing indoors. Indoor air quality management is made difficult not only by the large number and variation of indoor spaces but also the complex relations of indoor air quality and the building design, materials, operation and maintenance, ventilation and behaviour of the building users.

The recent update of the WHO AQG (WHO 2005, 2006a) for particulate matter, ozone, nitrogen dioxide, and sulphur dioxide specified that the AQG applied in all non-occupational environments, including indoors in households, schools, vehicles, etc. Although such traditional AQG in the form of concentrations for specific pollutants based on scientific review and assessment of health effects have been widely used in outdoor air quality management, they have had relatively little impact on management of indoor air quality in most countries.
Recognizing that management of air quality indoors requires different approaches to those applicable to outdoor exposures, the recent AQG update recommended that WHO explore development of AQG specifically designed to facilitate management of indoor air quality around the world.

SYSTEMATIC REVIEW APPROACH

The development of the WHO Indoor Air Quality Guidelines (IAQG) will be based on systematic review of the scientific evidence on health relevance of various pollutants in the indoor air and factors affecting indoor air quality. The principles of such a systematic review for health impact assessment are presented in the WHO guideline document (WHO, 2000b).

WHO guidelines are recommendations based on scientific evidence on health effects of certain exposures. Formulation of guidelines as exposure levels gives an objective measure of health risk that can be used as a reference point for design and maintenance of safe indoor environments. The use of the guidelines to create national and international legislation and standards needs to consider feasibility of various approaches besides direct control of indoor concentrations, including taxation of fuels and products, product and equipment use, maintenance, product composition, labelling, building construction, ventilation and education of professionals and the public to account for the importance of the indoor air quality for public health. However, as some exposure factors cannot be fully characterized using a concentration-based approach in the guideline formulation that has been used in the previous WHO guidelines (WHO, 1987, 2000c, 2005, 2006a), the indoor air guideline work needs to consider also qualitative indicators of health hazards and address them using guidance based on indicators of the hazards. Specifically, the guidelines should

- address various level of economic development,
- cover all relevant population groups and
- enable feasible approaches to reduce health risks from indoor air pollutants.

WORKING GROUP MEETING FOR START-UP OF THE WORK

Existing national and international experiences in indoor air quality regulation and conclusions of completed international projects provided the required background information for the WHO Working Group that convened in October 23-24, 2006, in Bonn with 38 experts, as well as representatives and observers from national and international institutions (WHO, 2006b).

In a series of plenary discussions and small drafting group sessions, the Working Group reviewed the general approach to the guidelines' formulation, discussed their scope and format, and agreed on the general contents of the background material. The final recommendations concerning the guideline development were made in plenary by consensus.

EVIDENCE REVIEW AND PRIORITIZATION

Due to the diversity of exposure conditions in indoor settings, three different areas requiring complementary approaches were identified:

A – Air pollutant specific guidelines
B – Biological agents
C – Combustion of solid fuels and kerosene

The working group divided into three parallel sessions to consider these areas separately, to assess the topics global importance for health, to summarize the existing scientific evidence, and give recommendations on the factors relevant for inclusion in the guideline development process. Conclusions of each subgroup are discussed shortly separately below.

Group A: Air pollutant specific guidelines

The task of the sub group was to identify those specific pollutants and agents that would require specific attention due to the characteristics of indoor environments.

The group identified a number of relevant systematic reviews and risk assessments of pollutants present in indoor environments. These reviews should be used as inputs to the development of the indoor air quality guidelines. One of the issues to be considered is the use of information from epidemiological studies based on ambient pollutant levels as exposure indicator.

The group also concluded that the WHO guidelines for environmental tobacco smoke (ETS) published in WHO Air Quality Guidelines for Europe, 2nd Edition (WHO, 2000c) and stating that there is no evidence for safe exposure level are clear and still valid. Therefore it was recommended not to include ETS in the current work. Plenary discussion concluded that the guidelines for other pollutants should be developed based on the assumption that ETS - a well recognized health hazard - is eliminated from the indoor spaces.

Group B: Biological agents

Exposures to biological agents in indoor environments were identified as a significant health hazard causing a wide range of health effects. There is vast evidence on hazards of several biological agents such as viruses, bacteria, damp, fungi, mites, pollens and allergens. However, the group concluded unanimously that in most cases it is not possible to identify individual species of the microbes or other specific biological agents responsible for the health effects. The exceptions are some common allergies, which can be attributed to specific agents or exposures, such as house dust mites or pets.

Biological agents in the indoor environment are attributable to dampness and inadequate ventilation. Excess moisture on any material leads to growth of microbes such as moulds, fungi and bacteria, which subsequently emit spores, cells, fragments and volatile organic compounds into the indoor air. Moreover, dampness initiates chemical and/or biological degradation of materials which also causes pollution of the indoor air. Dampness has been therefore suggested to be the strongest and most consistent indicator of risk for asthma and respiratory symptoms (e.g. cough and wheeze).

Inadequate ventilation is strongly associated with adverse health (sick building syndrome, inflammation, infections, asthma, sick leave, etc.) and reduces work performance in office buildings and learning capacities of students in schools. Proper ventilation is an important control for humidity, and prevention of condensation.

It was pointed out that ventilation systems may act as a source of health hazard e.g. in case of microbial growth and VOC emissions from accumulated sediments in the ventilation systems, but that overall ventilation should be seen as the solution for most indoor air quality problems, including those associated with biological agents.

Group C: Combustion of solid fuels and kerosene

Particulate matter (PM_{10} and $PM_{2.5}$) as well as carbon monoxide (CO) are good indicators of a large group of pollutants emitted in combustion of solid fuels. The group recognized that the current air quality guidelines for PM provide targets which are also valid for indoor environments. However the current guidelines for carbon monoxide (WHO, 2000c) need to be updated to reflect new information on chronic effects and to assure sufficient protection against acute poisoning in solid-fuel using households.
There are big differences in exposure levels to pollutants from solid fuel use between developing and developed countries. The group recognized that measurements of indoor air quality are difficult to be performed especially in developing countries, and that substantial improvements in reducing exposures can be achieved merely using indicators like “use of solid fuels indoors without proper ventilation / chimney” for identification of the need for action and formulation of corresponding technological recommendations regarding fuels, stove types, venting etc.
However, when available, measurements of indoor air quality provide quantitative information on the exposures, and in case of interventions allow assessing the

efficiency of the selected actions. There is much room left for technological solutions in exposure reduction (particularly for cooking) as relatively few resources have yet been applied.
The group set the focus of the development of indoor air quality guidelines on exposures to emissions from household use of solid fuels and kerosene, not just for heating and cooking but including also e.g., lighting and small commercial activities in households.

The group deliberated on plausible technical solutions to effectively control the sources and exposure pathways and listed the processes/solutions to be considered in the guidelines for indoor air quality:

PHASE 1: CONCLUSIONS

The recommendations for pollutants, agents, and factors to be included in the WHO IAQG were discussed in detail in the sub groups for agent specific guidelines, biological agents, and combustion of solid fuels. The recommendations of the small groups were presented in the plenary, discussed and finally agreed on. The pollutants and factors to be included in the guidelines are presented in the table 1.

In addition, the Working Group agreed on the role of indoor air quality as a significant determinant of population health. The development of WHO guidelines specific for indoor air quality is recommended for several reasons:

- Wide range of sources of air pollution specific to indoor spaces;
- Specificity of some exposures in indoor spaces in terms of pollution composition and exposure levels;
- Large fraction of time spent indoors affects population exposures;
- Separation of indoor and outdoor spaces which modify the exposures to a number of pollutants.

Besides the health-based recommendations for concentration levels not to be exceeded, the guidelines may formulate recommendations concerning indoor air quality problems using qualitative indicators, such as existence of dampness in the building structures leading to microbial growth or use of solid fuels in indoor spaces.

Table 1. Summary of factors to be included in the Guidelines on IAQ (WHO, 2006b)

Group A **Pollutants**	Group B **Biological agents**	Group C **Indoor combustion**
Formaldehyde Benzene Naphthalene Nitrogen dioxide (NO_2) Carbon monoxide (CO) Radon (Rn) Particulate matter[1] Halogenated compounds PAH[2], especially BaP[3]	**Dampness and mould** **Ventilation** - natural - forced / mechanical **Allergens** - from house dust mites - from pets	**Stove venting** - flues - hoods **Ventilation** - natural - forced **Combustion quality** **Fuels** - solid - processed solid - liquid - gas - electricity

[1] $PM_{2.5}$ and PM_{10}, [2] Polycyclic aromatic hydrocarbons, [3] Benzo[a] pyrene

DEFINITION OF INDOOR SPACES TO BE INCLUDED

The working group discussed how to define the indoor spaces that should be covered by the guidelines. It was concluded that the WHO guidelines for indoor air quality should cover indoor settings in which the general population or especially susceptible population groups like children, elderly, asthmatics etc. are potentially exposed to indoor air pollution. These include homes, schools, day care centres, public places as libraries or institutionalized settings like nursing homes. However, conditions that are specific to exposures in industrial settings, agriculture, mining and in other occupational settings where the exposure is related to the occupational activity of the occupants cannot be adequately addressed by the general guidelines for indoor air quality. Such settings are typically covered by work safety legislation or guidance.

As a first step in the development of IAQG, WHO decided to initiate a project to establish first recommendation on guidelines for biological agents, focusing on damp, mould and ventilation.

PHASE 2: TOWARDS WHO GUIDELINES ON INDOOR AIR QUALITY FOR MOULD, DAMPNESS, AND VENTILATION

As the first step in implementation of the recommendations of the WHO Working Group on IAQ guidelines, WHO established the steering group, which will supervise the WHO process and will advice WHO on all relevant scientific issues related to the Guidelines development and will assure consistency of the IAQ Guidelines development with the general approaches adopted for WHO AQG. The list of Steering Group members is available on the website

http://www.euro.who.int/air/activities/20070510_1.

WHO has initiated the development of Guidelines on dampness, mould and ventilation in spring 2007. It will be based on the review of health issues affected by dampness and mould in indoor spaces as well as the relation of health to ventilation system (both as a source of health hazard and as a part of solution to IAQ problems). These Guidelines should cover indoor settings in which the general population or especially susceptible population groups like children, elderly, asthmatics etc. are potentially exposed to indoor air pollution.

A group of experts with experience in epidemiology, toxicology and clinical aspects of health effects of exposure to biological agents in indoor air, experts in conditions affecting presence of biological contaminants in indoor air was invited to review the scientific literature and prepare background material according to the following structure:

1. General description
2. Effects of dampness on sources of indoor air pollutants and resulting exposure
3. Ventilation in relation to mould and dampness
4. Health effects associated with mould and dampness
5. Evaluation of human health risks

The WHO Working Group meeting will be convened in October 2007. It will formulate the Guidelines and will agree on detailed recommendations concerning finalization of background material to be contributed as the chapters of the WHO Guidelines for IAQ. The meeting report will be published on WHO web page. It will contain the summary of the workshop discussion and its conclusions, including the recommended text of the Guidelines sections for both chapters.

PHASE 3: ADDRESSING POLICY DEVELOPMENT FOR REDUCTION OF HEALTH RISKS DUE TO INDOOR AIR POLLUTION ASSOCIATED WITH BIOLOGICAL AGENTS

WHO recommendations for IAQG for damp, mould and ventilation are planned to be available by the end of 2007. However, the implementation of specific actions to achieve such Guidelines is still a difficult area for public health due to the great variety of indoor spaces, fragmentation of responsibilities and, in case of homes, limited mandate of public authorities for interventions.
Therefore a separate WHO project (a part of the project co-sponsored by EC DG Sanco, grant agreement 2005156) on policy implications of IAQG for damp, mould and ventilation will accumulate evidence on the actions implemented in various countries to address health hazards associated with dampness and mould and with inadequate ventilation, and will assess their effectiveness and suitability for reaching conditions as defined by the IAQ guidelines. This review will consider practical constraints of various risk management approaches, as well as their feasibility and costs, and will formulate recommendations for public policy aiming at the reduction of health impacts of biological contaminants of indoor air.

The major objectives of this project are to increase national and international capacities to

1. develop and apply national policies addressing health risks due to biological contaminants of indoor air, and in particular those risks generated by dampness and mould as well as inadequate ventilation.
2. advocate for action on the indoor air quality across sectorial policies and facilitation of implementation of WHO Guidelines on IAQ.
3. mitigate exposure to biological contaminants of indoor air within the housing stock and / or schools as a means to protect the more vulnerable parts of the population (sick, elderly, children) that spend most time within these indoor spaces.

For providing adequate mechanisms, tools and technical solutions, the project will aim to develop:

- a variety of good practice examples from European countries, showing innovative, adequate, realistic and successful approaches towards improving IAQ
- policy briefs summarizing the most effective actions and approaches facilitating practical application of the WHO Guidelines on IAQ for dampness and mould, and for ventilation.

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Initiatives at the European Respiratory Society on indoor air quality

Isabella Annesi-Maesano[1,2] and Francesco Forastiere[3] on behalf of the Environmental and Health Committee of the European Respiratory Society (ERS)

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The European Respiratory Society (ERS) www.ersnet.org is a not-for profit, international medical organisation with over 8,000 members from 100 countries. It is the largest society in Europe promoting respiratory health and lung research in Europe. These objectives are accomplished by promoting basic epidemiological and clinical respiratory research, collecting and disseminating scientific information, organising congresses and conferences, producing scientific publications, supporting training and continuous education in respiratory medicine and collaborating with organisations representing patients. Its sister organisation the European Lung Foundation (ELF) www.european-lung-foundation.org was created by the ERS in 2000 with the mission of making its expertise in respiratory medicine and respiratory health more accessible to the European scientific community and the European public. The ELF is the only pan-European foundation dedicated to advancing lung health in all its aspects.

Indoor air quality is important because people spend more than 90% of their time indoors. Many biological and non-biological agents contaminate indoor air and several scientific studies have identified a number of specific air pollutants as the cause of medical problems among children and adults. Buildings tend to be constructed to be more energy efficient. To reduce the cost of heating or cooling, this energy efficiency translates into greater recirculation of air with indoor pollutants and decreased fresh air intake. A proper ventilation is critical since energy costs are driving the population into very tight unvented occupancy which leads to accumulation of pollutants. Indoor air quality has been associated with respiratory diseases such as asthma and allergies, chronic obstructive pulmonary disease, lower respiratory infections and lung cancer.

The link between indoor factors and respiratory diseases has been well elucidated and several documents provide background information of the scientific evidence. In particular, WHO documents on indoor air quality are available (http://www.who.int/mediacentre/factsheets/fs292/en/index.html). A comprehensive report of the European Federation of Allergy and Airways Diseases Patients'

Associations "Towards Healthy Air in Dwellings in Europe (the THADE Report) indicates the major issues in the field and presents several options for a European programme on indoor air quality
(http://www.efanet.org/activities/documents/THADEReport.pdf). In a position paper to be published soon, the Environment and Health Committee of the ERS highlights the sources and substances of concern for possible health effects, indicate priorities for intervention, and propose issues for policy building at EU level.

SOURCES OF INDOOR AIR POLLUTION AND SUBSTANCES OF CONCERN

The major sources of indoor air pollution are:

- Tobacco smoke, wood and coal combustion, and unvented kerosene heaters
- Building materials and furnishings
- Products for household cleaning and repair
- Central heating, cooling, air conditioning and humidifying systems
- Domestic flora and fauna, including all types of furry pets and birds

The most important non-biological substances include: respirable particles (from environmental tobacco smoke (ETS), fireplaces, wood and coal stoves, unvented kerosene heaters are the main source of respirable particles... which have been shown to be significantly associated with acute and chronic respiratory illnesses and with lung cancer (ETS). Exposure is higher in unventilated places. Large number of people are exposed to ETS and the potential harm is considerable for both children and adults. Almost 20.000 deaths per year have been estimated among non-smokers in Europe as result of passive smoking exposure at home or at work. A recent evaluation from IARC has considered emissions from coal combustion to be carcinogenic to humans (group 1) and emissions from combustion of biomass fuel (mostly wood) to be probably carcinogenic (group 2A). Gases (CO, NOx, NO_2) (from gas stoves, pilot lights on any gas appliance, kerosene stoves and heaters, and environmental tobacco smoke) among which NO_2 is associated with increased respiratory symptoms and asthma aggravation. Formaldehyde (a volatile organic compound present in urea-formaldehyde foam insulation, glues, adhesives, fiberboard, pressed board, plywood, particle board, carpet backing, and fabrics), which in high concentration causes irritation of the eyes and throat, nausea, and difficulty breathing. There is some evidence that chronic exposures to formaldehyde are associated with lung and nasopharyngeal cancer. Volatile and semivolatile organic compounds (from household products such as paints, paint strippers, aerosol sprays, and art supplies, pesticides, fungicides, herbicides, and the combustion of wood, tobacco, and kerosene) related to irritation symptoms; and radon (derived from the radioactive decay of radium, a ubiquitous element found in rock and soil but also found in most homes at very low levels). Long term exposure may pose a substantial risk of lung cancer. The health hazards from radon are among the best-characterized of any housing-related health hazard. They have been extensively reviewed by several national and international committees and have been the focus of a European collaborative analysis of case-control studies (Darby et al, 2005). The risk estimates

suggest that around one in 20 to one in ten cases of lung cancer can be attributed to residential radon exposure. Ventilation can reduce also radon concentration indoors.
The most prevalent biological agents found in indoor air include moulds, dust mites, viruses, bacteria, fungi, pollen grains, dust mites, insects and human and animal danders. Mould and dampness which encourages the growth of fungal spores may cause true allergy reactions, and mycotoxins released from moulds are thought to have independent toxicity. Dampness and mould have been linked to asthma. There is evidence for both onset of new asthma cases and increased asthma symptoms on previously sensitized individuals. Mould growth occurs when the ventilation is poor and the humidity levels are high. These conditions are not uncommon across Europe. Intervention studies have shown that increasing ventilation and reducing humidity can decrease mould. Dust mites are responsible for asthma-like symptoms and allergies both among children and adults. Endotoxins from gram negative bacteria have been found to contaminate ventilation systems and air-conditioning systems causing upper respiratory inflammation or pneumonitis. Pets including birds, dogs and cats have feathers and dander; excrete proteins in their saliva, urine, and feces that can be allergenic; and release other biologic organisms with respiration.

RESEARCH NEEDS AND PRIORITY SETTING

There is no formal assessment of priorities for intervention in EU as inventory of the indoor environment has not been completed, data on prevalence of the exposures are not uniformly collected and the overall health impact has not been estimated. However, it is proposed that ETS, indoor biomass combustion, dampness and moulds, and radon are the most critical exposures requiring public health attention. The health effects of ETS have been well elucidated in a document produced by the European Respiratory Society (ERS, Lifting the smokescreen, 2006, www.ersnet.org).

PROPOSAL FOR POLICY BUILDING AT EU LEVEL

The THADE Report referenced above has suggested several actions and measures at EU level for a better indoor quality.

The following actions have been recommended:

- Improve ventilation.
- Improve cleaning methods and housing hygiene.
- Avoid wall-to-wall carpeting.
- Moisture control to prevent accumulation of mould.
- Control the sources of pollution, e.g. tobacco smoke and emissions from building and consumer products.

And the following measures have been recommended to implement these actions:

- Prohibition and avoidance of smoking indoors.

- Labelling systems to control emissions from building and consumer products.
- Better building codes and guidelines for ventilation and moisture control.
- Education and information campaigns.

Following these suggestions, the Environnemental and Health Committee of the ERS has stated that some areas deserve priorities.

Environmental Tobacco Smoke

The simplest and most effective measure to reduce indoor air pollution would be to implement comprehensive smoking bans in work and public places. There is mounting evidence that smoking bans are effective in reducing ETS exposure, chronic respiratory symptoms, and cardiovascular disease. Although these trends are encouraging, it should be recognized that the children, as they are most vulnerable to the effects of ETS exposure, are not being adequately protected by current legislative efforts. Children are primarily exposed to tobacco smoke in the home, where legal restrictions do not apply. All the current measures to implement cessation programs and towards prohibiting smoking in public places are therefore important priorities. However, the emerging issue is now to consider the individual homes as an area for active preventive efforts.

Gas heaters and cooking

Open-flame unvented combustion indoors is a health risk, and should be avoided. Policy and technology to promote the use of non-polluting appliances and energy sources should be developed. Develop programmes to make healthier cooking and heating systems and safer fuel available to households, providing the population with access to improved stoves and cleaner fuels (kerosene, liquid petroleum gas).

Dampness and mould

Technical standards and guidelines to control moisture in residential and non-residential buildings from the health standpoint (dust mites, mould and other harmful effects of excess moisture) should be developed. This requires co-operation on several levels. Various working groups of the World Health Organisation and the International Council for Building Research Studies and Documentation (CIB) have done groundbreaking work, but their recommendations should be implemented through European directives and standards, changes in national building codes, and guidelines developed by professional societies that include training of professionals.

Control of harmful emissions from building materials and consumer products

European action should be taken to develop guidelines and procedures to measure emissions from building materials and consumer products. Guidelines should include the criteria for low polluting materials and products, and a labeling system. The Construction Products Directive ‘Hygiene, health and the environment’ (89/195/EEC; Official Journal of the European Commission 11 February 1989) states that ‘The construction work must be designed and built in such a way that it will not be a threat to the hygiene or health of the occupants or neighbors.’ The control of harmful

emissions from building materials would be a step towards implementation of the principles of the directive.
The control of exposure to radon gas relies on monitoring programs in radon-prone areas, coupled with education/information initiatives. Exposure to radon in dwellings is also the subject of Commission Recommendation of 21 February 1990 on the protection of the public against indoor exposure to radon (90/143/Euratom). However, there is currently no regulation at the European level. Important public health objectives would therefore be to establish radon levels guidelines and actions which could be coupled with national housing stock objectives and/or specific public health goals.
Energy efficiency is one of the driving forces of indoor air quality. To reduce the cost of heating or cooling, this energy efficiency translates into greater recirculation of air with indoor pollutants and decreased fresh air intake. Proper ventilation is critical since energy costs are driving the population into very tight unvented occupancy which leads to accumulation of pollutants. Health equity in indoor policy is a crucial issue.

RESEARCH NEEDS

Research constitutes a key aspect that is required in working towards the reduction of the health burden from indoor air pollution. Inventory of the indoor environment has to be completed, data on prevalence of the exposures have to be uniformly collected and the overall health impact has to be estimated. There is a need to investigate which interventions are effective and how they can be implemented in a successful and sustainable way. COPD has to be considered too. Lastly, evaluation of socioeconomic differences in health effects should be added.

SUMMARY OF ERS INITIATIVES ON INDOOR AIR AT THE EUROPEAN LEVEL

<u>DG Sanco</u>

1. Expert working group on indoor air
2. Working party on Environment and Health
3. Responses to the following Commission documents:

a) Public consultation on the SCHER preliminary report on Risk assessment on indoor air quality
b) Green Paper on smoke free environments

<u>DG Environment</u>

1. Consultative Forum on environment and Health: set up to monitor and advise on the implementation of the Action Plan on Environment and Health.
2. Vienna Intergovernmental Conference to agree a set of recommendations and draft a declaration.

ECDC

1. ECDC workshop on infectious diseases and environmental change, 28-30 March.

DG Research

1. After two years of campaigning by the ERS, chronic respiratory diseases were finally included in the FP7 Health theme (Cooperation Programme) under Translational Health - Major Diseases - and "Other Chronic Diseases" (final decision - 18 December 2006)
2. First FP7 calls were published 22 December 2006. The ERS organised a call for "expressions of interest" to its members with the intention to offer broad support, e.g. for activities related to dissemination and training.
3. ERS responded to the EC public consultation (1 May) on the Green Paper on the European Research Area.
   http://ec.europa.eu/research/era/questionnaire_en.html

JRC's Activities on Emerging Environmental Health Issues: Indoor Air Quality

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SUMMARY

The focus of the JRC strategy for the E&H area is on how to optimally integrate environment and health information on a common platform (the European Environment and Health Information System) and to develop methodologies to analyse and unveil causal relationships between environmental risk factors and human health outcomes. This includes the development and validation of methods and methodologies for monitoring, for exposure assessment and for evaluation and quantification of health effects due to environmental stressors. The JRC will co-operate with the EEA, WHO and other international and national organisations to provide the best support to the building of the platform for integrating the information on environmental quality, health and research to support the E&H policy-making process. In order to cover the whole chain from pollutant to disease, the JRC activities in E&H will be organized within three interrelated areas covering information integration, exposure assessment and health effects.
In the area of indoor air quality, JRC on the basis of competencies developed over the last two decades and on existing collaborations with known European experts is carrying out research providing support to Commission services for the implementation of health related Directives and regulations.
In line with the Commission's Environment and Health Strategy and Action Plan, launched in June 2004, JRC has provided support for the formulation and execution of projects on indoor air quality (INDEX project), which are dealing with the assessment of existing knowledge worldwide on:

- Type and levels of chemicals in indoor air and
- Available toxicological information to allow the assessment of risk to health and comfort.

The main outcome of the INDEX project was the prioritization of chemical compounds and suggestions for the establishment of indoor exposure limits in the EU.
Following the recommendations of the Workshop on "Urban Air, Indoor Environment and Human Exposure" in Thessaloniki/Greece in April 2000 (The Thessaloniki statement), "that future clean air policies should take into account the total human air exposure of European citizens, which will necessarily include exposures to pollutants from both outdoor and indoor sources", in order to fill in the existing gaps in information on levels and distribution of air pollutants indoors, JRC is carrying out with the support of partners from the Member States field studies at European level, to

evaluate indoor/outdoor relationships and personal exposure concentrations for priority air pollutants and for different confined environments (AIRMEX project).
Preliminary evidence indicates that:

- In reference to the EU ambient air limit value for benzene of 5 μg/m^3 (annual mean) to be introduced by the year 2010, about 28% of the measured outdoor concentrations, 30% of the indoor concentrations, and 40.5% of the personal exposure concentrations exceeded this limit value.
- In Southern European cities indoor/outdoor as well as personal exposure concentrations are higher than in cities of Central Europe. In Athens and Catania in buildings located in the city centre there is almost no difference between indoor and outdoor pollutant levels.
- Concentrations in schools and kindergartens are generally lower than in public buildings and offices with public access.
- True personal exposures cannot be determined directly from measurements pertaining from fixed ambient background monitoring stations. In order to evaluate possible health effects associated with the presence of pollutants indoors and outdoors the best way for this will be to carry out measurements of personal exposure concentrations taking into account micro-environmental activity patterns and personal behaviour.

EC Actions on Indoor Air Quality

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BACKGROUND

In the Sixth Environment Action Programme the European Commission's commitment to provide "an environment where the level of pollution does not give rise to harmful effects on human health and the environment", is clearly stated.

The European Commission, with strong support from the Member States and the European Parliament, has put forward the ***European Environment and Health Strategy*** (the Strategy) in June 2003 (1). This integrated Strategy for Environment and Health, known as the "SCALE initiative", had the ultimate objectives to reduce the disease burden caused by environmental factors in the EU, to identify and to prevent new health threats caused by environmental factors and to strengthen EU capacity for policymaking in this area. SCALE meant to be incremental in scope and to be implemented in consecutive phases. It is based on **S**cientific evidence, focused on **C**hildren and other vulnerable population groups, meant to raise **A**wareness, improve the situation by use of Legal instruments and ensure a continual **E**valuation of the progress made.

The European Environment and Health Strategy was jointly issued by: the Directorates-General of Health (DG SANCO), Environment (DG ENV), Research (DG RTD) and the Joint Research Centre (DG JRC). It has a long-term vision seeking to address the links between poor health and environmental problems.

The European Environment and Health Strategy will be implemented in cycles, initially focusing on four priority diseases:

- childhood respiratory diseases, asthma, allergies
- neuro-developmental disorders
- childhood cancer
- endocrine disrupting effects

An Action Plan for 2004-2010 has been acknowledged for the implementation of this strategy during the first cycle. The preparation of this Action Plan was based on full stakeholder involvement. More then 300 experts were involved. Nine technical Working Groups (TWGs) and a Consultative Group were set up, consisting of

environment experts and health experts from Member States (Ministries of Health and Environment), academia, industry and major stakeholder organisations.

On 9 June 2004, the Commission adopted the ***EU Environment & Health Action Plan 2004-2010 (EHAP)*** (2). The action plan, which is based on the general orientations, set out in the Commission's June 2003 Communication, also served as the Commission's input to the Budapest European Environment and Health Ministerial conference (23-25 June 2004).

This work was lead by the Commissioners for Health, the Environment and Research and is being taken forward by the relevant Directorates General in the Commission (Health and consumer protection, Environment, Research and the Join Research Centre). The Action Plan has been developed in close cooperation with the World Health Organization (WHO) and is in line with their work on environment and health issues.

After the action plan was issued (June 2004) this was followed by a European Parliament report and resolution (rapporteur: Ms F. Ries, France MEP), which has been adopted early 2005.

With regard to indoor air quality the resolution:

- Welcomes the Commission's willingness to continue to act to put an end to smoking in enclosed spaces and encourages it to designate environmental tobacco smoke a class 1 carcinogen;
- Calls for research into the impact of new construction materials on health;
- Invite the Commission in cooperation with the Member States, to introduce a system for labelling the environmental and health effects of construction products and materials;
- Stresses that the quality of air inside buildings cannot be improved without a wide-ranging approach that takes into account the many sources of pollution: combustion apparatus, equipment and furniture and human activity, and **calls on the Commission to draft a Green Paper dealing specifically with domestic pollution.**

A conference organised by the Dutch Presidency and the Commission took place on the environment and health area in December 2004 at the Egmond aan Zee (The Netherlands). The conference had a session dedicated to the indoor air quality.

The main conclusions of the indoor air session concern:

- A European initiative to address indoor air pollution, starting with emphasis on the improvement of building products and ventilation system and further development and harmonisation of testing and labelling for building construction products enable people to identify low-emitting products;
- Smoking bans and other policies across Europe;
- Adequate elimination of combustion products generated indoor.

Another conference on Environment and Health has been organised and run in June 2005 by the Luxembourg Presidency. The conclusions were similar to the NL

conference with an added focus on environment and health ambulance. It was stressed that it is necessary to identify guidelines that favour the principle of precaution.

The EU Action Plan is an operational document setting out 13 key actions for the period until 2010, grouped around three broad categories:

- Improving the **information** chain by developing integrated environment and health information (actions 1-4). Measures include development of relevant health indicators, integrated monitoring of the environment and identification of different routes through which people are exposed, and bio-monitoring of humans.
- Filling the knowledge gap by strengthening **research** (actions 5-8). Actions focus on strengthening European research activities. This process is being implemented though the EU Research Framework Programmes.
- **Response**: review policies and improve communication (actions 9-13) concern the conclusions from the improved information and action. This will be done by, raising awareness, communicating better on risks, and training and educational activities.

The integrated environment and health information system aims to determine exposure to the main pollutants, the health impacts of that exposure, and the sources of the exposure**.** Current work tries to identify how far existing information can answer these questions, and/or what modifications to information requirements are cost-effective and feasible. A number of initiatives support this analysis, including the ENHIS projects launched under the Public Health Programme and coordinated by the WHO in addition to and Environment & Health work within JRC. In the framework of actions 1 to 4 of the Action Plan, after a broad stakeholders consultation, the Commission has undertaken an extensive **review of current environment and health information and monitoring systems** in 2006 (3). The review makes concrete recommendations for increasing linkage and integration between existing systems, enhancing efforts on research and human bio-monitoring, and improving data collection procedures, which are formulated in 14 concrete tasks. Implementation of the tasks mentioned in the Environment & Health Information Review and Implementation Plan has started.

To implement Task 5 on Indoor Air, DG ENV funded a study on "*Ranking of indoor air health problems using health impact assessment*", starting in January 2007. This study will be carried out by VITO (Belgium) over 10 months (see Chapter 6 below).

The European Commission in close cooperation with Member States also succeeded in concentrating ***research*** funding on priority actions highlighted in the Action Plan such as research on priority diseases and on environment and health interactions in the Sixth Framework Programme of Research (FP6) (2002-2006) (4). Both Council and Parliament supported the need for further efforts in this area under the Seventh Framework Programme of Research (FP7) (2007-2013) (5), such as human bio-monitoring, **indoor air quality** and long-term health impacts of early exposures to environmental stressors. The Commission will continue to devote efforts to exploit the outcomes of the projects and their usefulness for possible policy action. Translating these results into policy action is a long-term priority and will increase in prominence during the implementation of the Action Plan.

The concrete actions in terms of "response" will be further defined and developed as our understanding improves. Response will be more effective after full understanding on how environmental factors are responsible for health problems. This involves completing the knowledge and information chain. Work is, however, being carried out on three main areas:

- **Indoor air quality**
- Training of professionals
- Electromagnetic fields

EC ACTIONS ON INDOOR AIR QUALITY

Improving indoor air quality was done by several activities under Action 12 of the EU Environment and Health Action Plan 2004-2010. This action contains 2 key elements:

- Addressing environmental tobacco smoke (ETS) and,
- Developing networks and guidelines on other factors affecting indoor air quality (dampness, mould, building material, indoor effects of outdoor emission and their health implication) by using research and exchange of best practice.

Concerning indoor air quality, there has been considerable expectation for actions at Community level. While activities on ETS are now taken forward both on the Community and Member States level, the more general issue is how to best deal with the indoor environment as a whole.

Concerning ETS, in January 2007, the Commission adopted the Green paper "Towards a Europe free from tobacco smoke: policy options at EU level" (6) and launched a broad consultation process, on the best way to tackle passive smoking in the EU. Currently, the Commission is preparing a follow-up initiative on smoke-free environments, due to be adopted in 2008. The Commission is also preparing a report on the implementation of the Council Recommendation 2003/54/EC (7) on the prevention of smoking and on initiatives to improve tobacco control including a detailed analysis of national anti-smoking policies and regulations. At international level, the Commission contributed to the development of guidelines on the protection from exposure to tobacco smoke in the context of the WHO Framework Convention for Tobacco Control (8). The document will be adopted at the second Conference of the Parties to the Convention in July 2007.

DG SANCO: co-ordinated action on Indoor Air Quality

In May 2005 DG SANCO mandated the Scientific Committee on Health and environmental Risks (SCHER) to deliver an opinion on a possible risk assessment strategy to support policy on the indoor air issue, to identify potential areas of concern in relation to the different pollutants and to consider risks associated with the use of air fresheners. The SCHER issued a separate opinion (9) on air fresheners on 27

January 2006. With regard "Risk Assessment on Indoor Air Quality", the Committee issued an opinion for public consultation on January 2007 (10). This latter opinion is under adoption.

In order to co-ordinate possible actions, DG SANCO established in October 2006 an expert working group to follow up the opinions of the Scientific Committee and to fulfil the expectations from the political side, Member States and other stakeholders who asked the Commission to use a wide approach and take concrete actions on a number of pollutants/areas (Parliament Resolution (11), Dutch and Luxembourg Presidency conferences in December 2004 and June 2005). The expert group (composed by external independent experts, and representative of the EU Member States, NGOs and Industry) has been mandated to provide a forum for the exchange of best practice and information, to advise the Commission on EU programmes and policies related to indoor air quality and to give opinions on actions aimed at reducing relevant pollutant concentrations. The working group will have to follow main recommendations of relevant projects (such as the DG JRC INDEX report) and the future opinion of the SCHER committee.

The first meeting held on 27 October 2006 in Luxembourg, where a tentative work plan for the group has been discussed. Discussion has been focussed on three main themes:

- Information and education to the public on practice to improve indoor air quality;
- Guidance for EU MS and exchange on best practices to lower/reduce pollutants concentration of priority compounds;
- Linking different EU strategies relevant to the field of indoor air quality.

In the second meeting held on 25 May 2007 in Luxembourg, a draft work plan for the experts group was discussed. It consists of four actions on: (a) Information and education to the public; (b) working together with the MS on issues related to priority pollutants; (c) working with manufacturers and constructors and (d) co-ordinate European Commission activities. The list of topics suggested to be included in each action along with a detailed work plan should be finalised by September/October 2007 and the actions should be implemented till 2010.

A number of projects in the field of indoor air quality have been funded under the DG SANCO public health programme. The main projects funded are:

- The INDEX project ("*Critical appraisal of setting and implementation of indoor exposure limits in EU*") (http://ec.europa.eu/health/ph_projects/2002/pollution/fp_pollution_2002_exs_02.pdf), co-ordinated by DG JRC (2002-2004), identified a list of "priority compounds" on the basis of health impact criteria. Five compounds (formaldehyde, carbon monoxide, nitrogen dioxide, benzene and naphthalene) have been identified as high priority and suggestions and recommendations on potential exposure limits and actions have been formulated.

- The THADE project ("*Towards Health Air in Dwellings in Europe*") (http://www.efanet.org/activities/documents/THADEReport.pdf), co-ordinated by

the European federation of Asthma and Allergy (2001-2003), investigated the association among indoor air pollutants and respiratory diseases. Several recommendations have been formulated for international, national and local level to improve air quality in dwellings. The results of this project confirmed that air pollution in dwellings is a relevant health problem. It is a complex problem that must be addressed at European and international levels, and it involves the medical profession, scientific societies, patients' organizations, lawmakers, architects and the building industry.

- The HESE project ("*Health Effects of Schools Environment"*), co-ordinated by the University of Siena (2002-2005) (http://www.hese.info), highlighted the high presence of particulate, moulds, and allergens related to poor ventilation, which appears to be extremely common in European classrooms. Poor ventilation is likely to increase airway inflammation and the risk of asthma in allergic children and could even increase the risk of sensitisation in healthy schoolchildren.

- The BUMA project ("*Prioritization of BUilding MAterials as indoor pollution sources"*), co-ordinated by the University of Western Macedonia and the State General Laboratories of Cyprus (2006-2009), (http://www.buma-project.eu). The project main objectives are: (i) the formation of a comprehensive database containing up-to-date quantified emitted compounds by construction products and other building materials; (ii) the classification and prioritisation of building materials from the developed database with respect to hazardous compounds emission factors and the relevant exposure levels; (iii) the creation of an Indoor exposure expert modelling system linked to the above mentioned data base; (iv) the production of relevant guidelines for policy-making actions.

- The HealthyAIR project, ("*Network of actions and activities that address the effect of construction products on Indoor Air*"), co-ordinated by the TNO Build Environment and Geosciences (The Netherlands) (2006-2009) aims at defining, initiating and developing activities that improve indoor air quality and reduce exposure to indoor air pollution sources, in particular of construction products.

DG Enterprise & Industry: Indoor air and construction materials

According to Annex I of the Construction Products Directive (CPD), construction products must satisfy specified essential requirements, where the works are subject to notified regulations containing such requirements. To comply with Essential Requirement no 3 (hygiene, health and the environment - ER3), the construction works must be designed and built in such a way that they will not be a threat to the hygiene or health of the occupants or neighbours. Therefore, standardisation work under the CPD provides harmonised test methods with regard to the performance of a construction product.

Based on mandate M/366 developed by the Commission's Expert Group on Dangerous Substances (EGDS), CEN/TC 351 has started its work on horizontal test methods early 2006. The first technical reports are expected to be due for the CEN voting procedure by the end of 2007.

In the second half of 2007 the expert group will assess a first set of product standards for construction products under the Construction Products Directive (CPD) and the mandates for these products for possible amendments to include relevant requirements for ER3 wherever necessary. Based on the amended mandates, references to (harmonised) test/measurement methods for relevant dangerous substances covered by EU or notified national regulations will be added to the harmonised product standards (whenever necessary). It is expected that the first mandates for construction products assessed by the expert group and including all relevant requirements for ER3 will be sent to CEN by the end of 2008.

General comment: The current chemical policy through dangerous substances, dangerous preparation and limitation directives allow to deal with dangerous substances from the legislative side. In addition, industry's current own activities under the Responsible Care and Product Stewardship Programmes are recognised and provide a starting point for other detailed industrial activities.

DG JRC funded indoor air quality related projects

- The European Collaborative Action (ECA) "*Urban Air, Indoor Environment and Human Exposure*" (formerly "Indoor Air Quality & its impact on Man" is running since 1986 by the DG JRC (http://www.jrc.cec.eu.int/pce/pce-sa-expotools07-eca.html). The focus of this activity is urban & indoor air pollution exposure assessment, seen as part of environmental risk assessment in support of urban and indoor air quality management. Work within ECA addresses urban outdoor and indoor sources of pollution, interaction between outdoor and indoor air quality of buildings, and exposure to pollutants from outdoor and indoor sources in relation to health and comfort. This can be a basis for integrated urban air quality management to minimise exposures to air pollutants. So far 25 state-of-the-art reports have been published. ECA recent activities include:

    - In 2005, ECA issued the report no. 24 on a "*Harmonisation of existing indoor material emissions labelling systems in the EU: inventory of existing schemes*". This report critically reviews and discusses recent developments concerning the indoor material labelling schemes at European level.
    - In 2006, ECA issued the milestone report no. 25 on "*Strategies to determine and control the contributions of indoor air pollution to total inhalation exposure (STRATEX)*". This report collates the respective information and describes the strategies to determine population exposure to indoor air pollutants. Its major goal is to emphasise the importance of the contribution of indoor air to total air exposure. Taking this contribution into account is a prerequisite for sound risk assessment of air pollution. The strategies described in this report should be considered as a framework that may have to be adapted to specific situations by policy makers, risk assessors and risk managers.
    - In May 2007, a new ECA WG 26 started on "*Ozone-Initiated Chemistry and Its impact on Indoor Air Quality and Human Health*". This WG will summarise the current state-of-the-art concerning indoor air pollution and

health due to chemical reactions occurring indoors and to prioritise research goals for the future.

- The AIRMEX project ("*European Indoor **Air** **M**onitoring and **Ex**posure Assessment Project*"), (http://www.jrc.ec.europa.eu/project/airmex/index.htm), funded by the DG JRC (2003-) aims at: (i) identifying and quantifying the main air pollutants in public buildings, including schools and kindergartens; (ii) identifying the main sources of these pollutants; and (iii) estimating people's exposure and evaluating possible health effects due to chronic exposure to these pollutants, especially for children.). In the frame of the AIRMEX project measuring campaigns in Catania (I), Athens (GR), Arnhem and Nijmegen (NL), Brussels (B), Milan (I), Thessaloniki (GR), Nicosia (CY) and Leipzig (D) were carried out to estimate indoor/outdoor relationships and personal exposure concentrations for selected volatile organic compounds (aromatics, carbonyls, terpenes). To complete the picture in characterising the factors affecting well-being and health, measurements for biological pollution indoors focusing on allergenic bacteria and fungi and on inflammatory response have started in 2006.

DG TREN, Energy and Transport, Directorate D: "New and Renewable Energy Sources, Energy Efficiency and Innovation"

The Annex of the Energy Performance of Buildings Directive (EPBD) (12) lists indoor climatic conditions, including outdoor climate as one of the issues that should be taken into account when calculating the energy performance of a building. Air-tightness is also mentioned in the annex. CEN (European Committee for Standardisation) has been mandated by the Commission to develop a set of standards to calculate the energy performance of a building. One of these standards specifically deals with the criteria for the indoor environment. A paper prepared by a CEN author explains the role of indoor climate and the Directive, and concludes:

The new directive for energy performance of buildings requires considerations of the indoor environment. It must therefore not be possible to fulfil requirements for the energy performance by decreasing the indoor environmental quality.

Indoor air quality is a fairly important component of Intelligent Energy Europe-supported projects, such as SAVE, Altener, STEER, etc. There is a pre-requisite that air quality is given in all Eco-buildings projects and all Concerto projects (together with comfort, health, well-being, acceptance of inhabitants, etc). The Construction Products Directive is under amendment and it is envisaged to include energy efficiency aspects in it.

The site for accessing the new searchable database for these projects is IntellEbase (http://europa.eu.int/comm/energy/iebase/introduction.cfm). IntellEbase is the European Commission's public dissemination database for non-technological projects supported by the Community in the field of energy efficiency (SAVE programme) and renewable energy sources (ALTENER programme). Here detailed and summarised information on most of the 700 SAVE and ALTENER projects from 1996 and onwards can be found. An information source on EPBD and related

European Activities can be found on the EPBD Buildings Platform (http://www.buildingsplatform.eu/).

Several energy-related building demonstration projects in the Eco-Buildings and Sustainable Development programmes in the 6th RTD Framework Program deal with indoor air quality and indoor climate. These can be found on the CORDIS project database: http://www.cordis.lu/fp6/projects.htm#search.

DG RTD: Funding schemes for Indoor Air Quality in FP7 + past projects funded in previous FPs related to Indoor Air Quality

Research on "Environment and Health" was given greater prominence in the **5th EU Research and Technological Development Framework Programme (FP5)** with the development of a specific 'key action' within the Quality of Life and Management of Living Resources programme, effectively building on limited research actions undertaken in FP4 and earlier. The remit of the 'Environment and Health' key action was broad with its' aims being to:

- determine how environmental factors contribute to health problems such as allergies, respiratory diseases, neurodegenerative diseases, and cancer, with a view to reducing harmful effects;
- develop and improve methods for assessing the health risk due to environmental hazards; and
- inform the public on links between environment and health, and to provide a scientific basis for legislation on environmental hazards.

A total of more than 20 EU supported research project under this key action considered the health impact of air quality, primarily with respect to ambient air pollution but some also taking into account issues related to indoor air quality.

- AIRALLERG (*Effects of outdoor and indoor air pollution on the development of allergic disease in children* - http://ec.europa.eu/research/quality-of-life/ka4/pdf/report_airallerg_en.pdf);
- ECHRS II (*European Community Respiratory Health Survey*) (http://www.ecrhs.org);
- HELIOS (*Biomarkers for the non-invasive assessment of acute and chronic effects of air pollutants on the respiratory epithelium*) (http://ec.europa.eu/research/quality-of-life/ka4/pdf/report_helios_en.pdf);
- *MOCALEX (Measurement* of occupational allergen exposure) (http://ec.europa.eu/research/quality-of-life/ka4/pdf/report_mocalex_en.pdf);
- PATY (*Combined analyses of cross-sectional studies on respiratory health of children and air pollution*) (http://www.lshtm.ac.uk/pehru/paty);
- RUPIOH ("*Relationship between ultrafine and fine particulate matter in indoor and outdoor air and respiratory health*" - http://ec.europa.eu/research/quality-of-life/ka4/pdf/report_rupioh_en.pdf);
- CHILDRENGENONETWORK (*European Network on children's susceptibility and exposure to environmental genotoxicants)* (http://ec.europa.eu/research/quality-of-life/ka4/pdf/ report_childrengenonetwork_en.pdf);

- GEN-AIR (*Molecular changes and genetic susceptibility in relation to air pollution and environmental tobacco smoke: a case-control study in non-smokers nested in the epic investigation*) (http://ec.europa.eu/research/quality-of-life/ka4/pdf/report_gen-air_en.pdf); AIRNET (*A thematic network on air pollution and health*) (http://ec.europa.eu/research/ quality-of-life/ka4/pdf/report_airnet_en.pdf);
- E21-4AYC ("*Environmental influences and infection as aetiological agencies in atopy and asthma in young children*" - http://ec.europa.eu/research/quality-of-life/ka4/pdf/report_e21-4yc_en.pdf);
- PINCHE ("*Policy interpretation network on children's health and environment*" - (http://www.pinche.hvdgm.nl)

Research topics included: development of biomarkers and predictive toxicology for selected air pollutants; chemical and biological characterisation of particulate mater air pollution (primarily from car exhaust); cohort and epidemiological studies and air pollution; impact on vulnerable groups such as children; comparison of indoor and outdoor air quality and respiratory health, etc. A further 13 projects were supported on environmental influence on the development of asthma and allergies, many including air quality/airborne allergens within the scope of the investigations.

Under the **6th RTD Framework Programme**, a co-ordination action on "*Indoor Air Quality and Health Effects (EnVIE)"*, co-ordinated by IDMEC (Portugal) (2004-2008), has been supported under the 'Scientific Support to Policies' programme. EnVIE is designed to interface science and policy making in the field of indoor air quality and will collect and interpret scientific knowledge from on-going research, in particular from EU funded projects and the Joint Research Centre's activities. The output of the coordination action will be to elaborate policy relevant recommendations based on a better understanding of the health impacts of indoor air quality. More specifically, it will: (a) increase the understanding of the Europe-wide public health impacts of indoor air quality; (b) identify the most widespread and significant indoor causes for these health impacts; (c) evaluate the existing and optional building and housing related policies for controlling them; and (d) address in particular how indoor air quality might contribute to the observed rise in asthma and respiratory allergy, together other acute and chronic health impacts. Further information concerning this project can be found at: http://www.envie-iaq.eu/.

- The PRONET project ("*Pollution reduction options network*" - http://www.proneteurope.eu), aims at facilitating the exchange and evaluation of interventions on environment and health exposure reduction measures on a regional level, and promote implementation of successful initiatives in other regions of Europe. The focus on the exchange of useful practices in two areas: (i) the reduction of traffic-related health hazards; and (ii) improvement of indoor air quality.

- The CAIR4HEALTH Specific Support Action ("*Clean air for health - research needs for sustainable development policies*" - website under construction) will, among others, aim at strengthening and exploitation of research results obtained by European and other projects related to air quality and health impacts. Its main goal is to provide research support for several air quality-related policies in the EU. The

focus will be on ambient air issues although indoor air quality will be touched upon.

- The GA2LEN network of excellence ("*Global allergy and asthma European network*" - http://www.ga2len.com) has established an international network of European centres of excellence conducting specific integrated multidisciplinary research programmes on issues relating to environment (including outdoor and indoor pollution), nutrition, lifestyle (including occupation), infections and genetic susceptibility.

- The GABRIEL integrated project ("*A multidisciplinary study to identify the genetic and environmental causes of asthma in the European Community*" - http://www.gabriel-fp6.org) aims at the identification of how genes and the environment cause the development of asthma and of both risk and protective factors, with the long-term aim of preventing the illness. The environmental stressors considered include indoor air pollution.

- The ECNIS network of excellence ("*Environmental cancer risk, nutrition and individual susceptibility*" – http://www.ecnis.org), is focused on environmental causes of cancer, which include pollutants in indoor air.

- The EUROLYMPH (Collaborative European action into environmental, nutritional and genetic factors in non-Hodgkin's lymphoma aetiology - no website) also considers indoor pollutants as risk factors for lymphoma.

- The NOMIRACLE integrated project ("*Novel methods for integrated risk assessment of cumulative stressors in Europe*"- http://viso.jrc.it/nomiracle) is developing a research framework for the description and interpretation of cumulative exposures and effects. It includes the development of methods for explicit modelling of exposure and risk in space and time (specifically, a temporal model for the indoor air pollution with volatile organic compounds).

- The INTARESE project ("*Integrated assessment of health risks of environmental stressors in Europe*" – http://www.intarese.org) is an ambitious project aiming at producing a new integrated risk assessment framework, based on the full chain approach (causal chain spanning sources of pollution, releases into various media, dispersion and transport, exposure medium inhalation/dermal contact/ingestion, intake, uptake, dose, health effects and impacts), based on three existing frameworks with differing approaches and aims. One policy area of concern included is housing: includes the effects both separately and in combination of environmental tobacco smoke, indoor air pollution (e.g., from cooking and heating, moulds, furnishings etc), noise and indoor climate (including temperature and dampness) on acute and chronic health (respiratory illness, cardiovascular illness, winter mortality and infant mortality).

- The ENVIRISK project ("*Assessing the risks of environmental stressors: Contribution to the development of integrating methodology*" - http://envirisk.nilu.no) will attempt at integrating the modelling of environmental releases, dispersion and human activity into an exposure modelling framework, and to provide the necessary interfaces for its integration with health effects modelling

into an integrated environmental health risk modelling framework. Indoor air pollution is included.

- The HEIMTSA integrated project ("*Health and environment integrated methodology and toolbox for scenario assessment*" - website under construction) will consider human exposures (e.g., outdoor and indoor air pollution, water, noise, odour, metals, dioxins) by multiple routes, using new methods (exposure scenarios and probabilistic modelling), including consumer exposure to facilitate applications of the full-chain approach.

- 2-FUN ("*Full-chain and uncertainty approaches for assessing health risks in future environmental scenarios*" - http://www.2-fun.org) includes a case study in Poland the aim of which is an environmental sources assessment of children exposed to toxic metals and verification of the intermediate results through the use of a monitoring dataset in the environment (already existing). Indoor sources of pollutants will be considered.

- DROPS ("*Development of macro and sectoral economic models aiming to evaluate the role of public health externalities on society*" - http://www.nilu.no/DROPS) has as the overall objective a full-chain analysis related to impact of health protection measures related to priority pollutants as identified by the European Environment and Health Action Plan, in order to support the development of cost-effective policy measures against pollution-related diseases and their wider impacts. Extension of existing methodologies and models to provide an impact-pathway-based model for evaluation of the role of public health externalities on society, made operational for the selected compounds will be carried out, and the focus will be on ozone, heavy metals (mercury, cadmium, arsenic, nickel, lead), polychlorinated biphenyls (PCBs), dioxins and indoor air pollution.

Under the **7th RTD Framework Programme**, projects related to health impacts of exposure to indoor air pollutants will be funded in the first place by the Environment and Health activity under the Environment theme of the Cooperation programme. The Environment theme has two topics open in the first call for proposals related to indoor air quality: Performance indicators for health, comfort and safety of the indoor built environment, and Indoor air pollution in Europe: an emerging environmental issue. Fifteen proposals were received in this area and are under evaluation.

DG ENV: Ranking of indoor air health problems using health impact assessment

In 2007 DG ENV launched a project on "ranking of indoor air health problems using health impact assessment". The project is carried out by VITO (Belgium) who organised a Workshop on this issue on 29-30 March 2007 in Brussels. In the Workshop, an attempt was made to discuss with the policy makers and experts from the EU MS about indoor air quality in order to provide:

(a) recommendations for future (EU-wide) policies;
(b) recommendations on strategies to control and monitor indoor air quality;
(c) a focused list of priorities for further research.

Three cornerstones for an indoor air policy could be: (1) *indoor air quality guidelines*, (2) *indoor air monitoring programmes* and (3) *sanitation plans*. Although a complete uniform policy is not the way to go, standardisation should be attempted where possible. The emissions from a particular building material for instance, should be regulated to the same level in all MS. A standardized EU labelling system could be the best way forward to enhance indoor air quality. However, labelling alone might be insufficient and not reach people who do not understand the labels. At an EU- level, monitoring programs exist on a project basis, awaiting implementation of monitoring programs by the member states. There are no standardised methodologies available. An example of this is the AIRMEX project (DG JRC). Standardised methodologies should be developed to conduct uniform Europe wide monitoring programs.

An overview of explicit (INDEX, SCHER opinion, DG SANCO Indoor Air expert working group) or implicit (THADE) prioritisation of indoor air chemicals performed in former EU projects (see chapter 1) or working groups was performed. The WHO work on development of indoor air quality guidelines is also considered. Taking into account the common prioritisation from SCHER, INDEX, THADE, WHO and the DG SANCO Indoor Air expert working group for *ETS, formaldehyde, CO, particles, NO_2, benzene, naphthalene, moulds, mites, dampness/moisture and CO_2 (as proxy for ventilation)*, a recommendation was made to focus the indoor air policy in the EU on these compounds at first stage.

Other EU projects

- WHO project on "*Development of WHO guidelines for Indoor Air Quality*" (http://www.euro.who.int/Document/AIQ/IAQ_mtgrep_Bonn_Oct06.pdf). In October 23-24, 2006 the corresponding WHO WG met in Bonn and reviewed the general approach to the guidelines' formulation, discussed their scope and format, and agreed on the general contents of the background material. Initial recommendations were proposed addressing guidelines for specific agents/substances, biological agents and combustion of fuels indoor.

- EXPOLIS-INDEX ("*Human Exposure Patterns for Health Risk Assessment: Indoor Determinants of Personal Exposures in the European EXPOLIS Population in Athens, Basel, Grenoble, Milan, Helsinki, Oxford, and Prague*"), funded by CEFIC/LRI (2002-2004), (http://www.ktl.fi/expolis/EXPOLIS-INDEX2004/EXPOLIS-INDEX_FINAL_REPORT.pdf). The EXPOLIS-INDEX study investigated the factors which determine human exposures to air pollutants in indoor environments. This information is crucial to assess the health risks related to indoor exposures and to propose mitigation strategies for harmful indoor contaminants. To achieve these goals, measurements of pollutant concentrations on person and air pollution levels in homes, at work and outdoors must be combined with information on time spent in indoor and outdoor locations.

A detailed record of current and past projects in indoor air quality can be found in the IERIE database that contains over 200 different projects related to indoor air pollution (13).

The Commission will continue supporting research activities on indoor air quality through the Commission funding programmes. Future actions will focus on information to the public and professionals, exchange of best practices at national and local level and on coordination of ongoing policies/strategies linked to the indoor air quality. The Commission will also take into consideration the WHO recommended guidelines and consider if specific action is necessary in order to avoid potential hazardous exposures, particularly in schools or places where children spend time.

DISCUSSION, CONCLUSIONS & RECOMMENDATIONS

In the period 2007-2010, the Commission in close cooperation with Member States will continue to implement the various actions foreseen in the Environment and Health Action Plan 2004-2010. To this end, the Commission will maintain its focus on the integration of environment and health concerns into other policy areas as well as on the integration of the many actors involved. A ***strengthened cooperation*** between environment policy, health policy and the corresponding research fields is one of the major achievements over the last 3 years. This is leading to the development of an integrated environment and health policy field, which must be taken up by a range of policy areas such as transport, energy, chemicals, employment. In order to strengthen EU capacity for policymaking in the area of environment and health, the Commission will gradually step up its effort to exploit the outcomes of research projects and other information gathering efforts and their translation into policy action, in particular for issues such as indoor and outdoor air as well as climate change and health, where integration is deemed essential. To achieve this goal, an integrated approach is needed within the framework of the Community's Sustainable Development Strategy.

In contrast to the well-elaborated and implemented EU ambient air policies (under the form of the Air Quality framework directive 1996), an integrated EU policy on indoor air quality is not yet available. Currently, indoor air quality is fragmentally tackled in sector oriented policies, but, an overall, integrated indoor air policy at the EU level is still missing. However, as with ambient air, envisaging such a similar framework for indoor air might be useful as guidance for both research and future action plans. The main EU directives including explicitly an indoor air quality aspect, or indirectly regulate indoor air quality are:

- the construction products directive 89/106/EEC Essential Requirement N°3 "Hygiene, Health and the Environment"
- the energy performance of buildings directive 2002/91/EC
- the gas appliances directive 1990/396/EEC
- the heating appliances directive 1992/42/CEE
- the eco-design directive 2005/32/EC
- the dangerous substance directive 1976/646/EEC
- the general product safety directive 2001/95/EC

The REACH regulation (2006/121/EEC) is also expected to influence indoor air quality. Other EU instruments contributing to good indoor air quality are the eco-labels.

In the VITO's Workshop it was discussed that the establishment of an integrated and optimised/harmonised framework on indoor air might be a future objective for the EC that should:

- Improve the coherence between the existing legislation. The focus of this framework should be on major, essential components, to protect the people for common pollutants.
- Take into account all related EU directives dealing with indoor air, ambient air and energy (as not only influence each other but also contribute to the total human exposure in indoor environments). Harmonisation of these EU directives is necessary. A consistent review of existing legislation is needed to optimise/harmonize the current directives.
- Include an information platform for increasing the public awareness of indoor air and for stimulating the improvement of human behaviour in private indoor spaces.
- Form a common basis for EU regulations, however, the best measures to be undertaken and its optimal implementation should not be uniform across the EU MS to account for differences in cultural habits and climate conditions.

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Session 1B Introduction of EnVIE concept and overview of EnVIE project

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BACKGROUND

European citizens want to live longer, healthier, in an environment of low involuntary risks, and at an affordable cost. Urban environmental policies should, therefore, manage the determinants of health as far upstream as possible and improve the citizens' quality of life. People are exposed to a multitude of chemical, physical and biological stressors in their environment, some of which are apparently harmless, others of low health significance and some incur significant risks to health, at least for vulnerable individuals. Human exposure to environmental contaminants occurs via various pathways (air, water, food...) and routes of entry (inhalation, ingestion and dermal). Exposure via air occurs outdoors and in different indoor µenvironments; e.g. home, workplace, transit. Indoor air pollution from different sources may cause or aggravate illnesses, increase mortality, and have major economic and social impacts.

Knowing the relative contributions of these µenvironments to exposure and health effects is essential for effective risk management and resources allocation.

HEALTH EFFECTS OF INDOOR AIR

Different pathways from indoor sources lead to a broad variety of health outcomes that are attributable to the indoor environment (See Figure 1, Jantunen et al. 2000).

Building-related illness (BRI) is a term referring to illness brought on by exposure to the building air, where a defined illness, directly linked to agents in the indoor environment, is diagnosed. Legionaire's disease and hypersensitivity pneumonitis are examples of BRI that can have serious, even life-threatening consequences. Some indoor pollutants are known or suspected carcinogens (radon, environmental tobacco smoke (ETS), asbestos, benzene) and add to the underlying cancer risk for the European populations. Lung cancer, in particular, has been clearly associated with indoor radon, ETS, and asbestos.

Also hypersensitivity initiation and allergies are sometimes associated with contaminated indoor air; various reports have linked indoor allergens with one or more of the following allergic manifestations among the occupants:

- Rhinitis, with 'hay fever' symptoms such as nasal congestion, runny, nose, sneezing, conjunctivitis..
- Asthma, with symptoms such as wheeze, tightness of the chest and shortness of breath.
- Extrinsic allergic alveolitis (hypersensitivity pneumonitis), with acute pneumonia-like fever, cough, tightness of the chest and lung infiltration, or chronic development of cough, shortness of breath and infiltration of the lungs.
- Humidifier fever, with symptoms including fever, chills, muscle ache and malaise, but no obvious respiratory effects.

The term sick building syndrome (SBS) is used to describe cases in which building occupants experience acute symptoms and discomfort that are apparently linked to the time they spend in the building, but for which no specific illness or cause can be assigned. The complaints may be localised in a particular room or zone or may be widespread throughout the building. Many different symptoms have been associated with SBS, including respiratory complaints, irritation, and fatigue. Sensory perception of odours and mucous irritation lead to perception of poor air quality and possible risks thereof, and consequently to stress or behavioural responses (opening a window, leaving the building). The symptoms of stress vary, depending on the host, not on the cause of the stress. Other environmental stressors such as noise, vibration, crowding, ergonomic stressors and inadequate lighting can produce symptoms that are similar to those of poor air quality.

Finally discomforting information about exposures and health risks, and job-related psychosocial problems may cause stress symptoms that are undistinguishable from those of sensory perceptions.

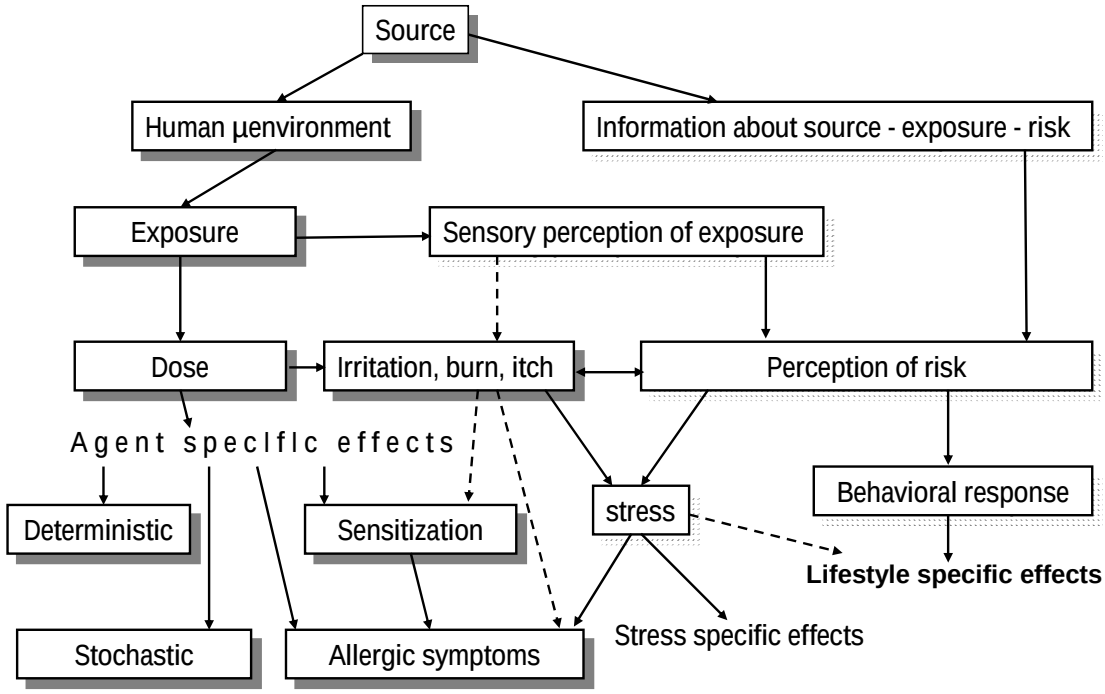


Figure 1. The entangled pathways from source to the different agent, stress and lifestyle related health outcomes.

REVIEWS OF MAIN IMPORTANT INDOOR AIR RELATED HEALTH EFFECTS

INDEX project

The INDEX project "Critical appraisal of the setting and implementation of indoor exposure limits in the EU (2002–2004)" was funded by the European Commission´ DG SANCO and JRC was given the assignment to identify priorities and to assess the needs for a Community strategy and action plan in the area of indoor air pollution by: setting up a list of compounds to be regulated in indoor environments with priority on the basis of health impact criteria; providing suggestions and recommendations on potential exposure limits for these compounds; and providing information on links with existing knowledge, ongoing studies, legislation etc. at world scale (Kotzias et al. 2005).

On the basis of the available information 14 compounds (out of initial 41 candidate compounds) were selected for a detailed assessment i.e.: acetaldehyde, alpha-pinene, benzene, carbon monoxide, d-limonene, formaldehyde, meta-, orto- and para-xylene, naphthalene, ammonia, nitrogen dioxide, styrene and toluene.

Finally a list of compounds, consisting of 5 chemicals, with potential of high indoor concentrations, uncontested health impacts, and options for effective risk management were selected to be regulated with priority.

Formaldehyde

Predominant symptoms of formaldehyde exposure in humans are irritation of the eyes, nose and throat, together with concentration-dependent discomfort, lachrymation, sneezing, coughing, nausea and dyspnoea. Because of its high chemical reactivity, formaldehyde is the most important sensory irritant among the chemicals assessed in the present report. Due to being ubiquitous pollutant in indoor environments and to the increasing evidence indicating that children may be more sensitive to formaldehyde respiratory toxicity than adults. IARC announced there was sufficient evidence that formaldehyde causes nasopharyngeal cancer in humans and re-classified it as a Group 1, known human carcinogen (previous classification: Group 2A). IARC also reported there was limited evidence that formaldehyde exposure causes nasal cavity and paranasal cavity cancer and "strong but not sufficient"evidence linking formaldehyde exposure to leukemia.

Carbon monoxide

Carbon monoxide (CO) is a tasteless, non-irritating, odourless and colourless toxic gas which can cause acute and lethal poisonings (a large number of deaths occur annually in fires, workplaces and in indoor environments). Currently available evidence suggests that individuals with heart disease (including stable exercise-induced angina, coronary artery disease, and ischaemic heart disease), in fact with any condition that may compromise oxygen sufficiency, are under the greatest risk from exposure to CO. In addition, population groups with either increased probability or increased severity of health effects include fetuses, pregnant women, and young

infants, individuals with anemia or respiratory disease, the elderly, children, and persons with peripheral vascular disease and chronic obstructive lung disease.

Nitrogen dioxide

Exposure at NO_2 indoor levels could generate effects in the pulmonary function of asthmatics, considered to be the subjects most susceptible to acute NO_2 exposure. For long-term exposures, increased respiratory symptoms and lung function decreases in children have been documented to be the most sensitive effect in the general population. On the other hand, indoors, as well as outdoors, NO_2 is strongly associated with other combustion products, most notably fine particulate matter (fPM), and it is difficult to separate its effects from those of these co-pollutants. The independent effects of NO_2 have been only observed at much higher concentrations than normally found in indoor air.

Benzene

It is a genotoxic carcinogen (IARC class 1). Results from nine monitoring surveys indicate that the European population is experiencing in their homes an increased risk in contracting leukaemia induced by benzene from the indoor air.

Naphthalene

In relation to carcinogenicity, naphthalene is not genotoxic in vivo and thus tumour development, observed in rodents, is considered to arise via a non-genotoxic mechanism. Also, the underlying mechanism for the development of nasal tumours in the rat is considered to be the chronic inflammatory damage seen at this site. Two to 20% of individuals in defined Mediterranean subpopulations exhibit an inherited deficiency in glucose-6-phosphate dehydrogenase (G6PD). They may develop haemolytic anemia and its sequel at low naphthalene exposures than are harmless to most. Infants and neonates form the other subpopulation, which is sensitive to naphthalene toxicity.

THADE project

The European Federation of Allergy and Airways Diseases Patients Associations (EFA) carried out the EU-funded project entitled “Towards Healthy Air in Dwellings in Europe – THADE” (Franchi et al. 2006). This study was endorsed by the European Academy of Allergology and Clinical Immunology (EAACI), European Respiratory Society (ERS), European Federation of Building Services Engineers (REHVA), Global Initiative on Asthma (GINA), International Society on Indoor Air Quality and Climate (ISAIQ); sixteen associations affiliated to the EFA network took part in the project.
The major health exposures affecting occupant health in dwellings were found to be tobacco smoke, dust mites, pet allergens, cockroaches, mould, pollen, nitrogen oxide, formaldehyde, VOCs, indoor-generated particulate matter, man-made mineral fibres, radon, CO and CO_2.

Four main conditions have been related to indoor air quality:

1. Building-related illnesses are conditions that are directly attributable to environmental agents present in the air of a building (e.g. CO intoxication).
2. Several allergic diseases have been associated with indoor air pollution, namely rhinitis with hay fever symptoms, asthma with wheezing, tightness of the chest and shortness of breath. In addition, extrinsic allergic alveolitis with acute pneumonia-like bouts of fever, cough and lung infiltration can result from poor air quality.
3. Sick building syndrome refers to buildings in which most of the building occupants experience acute health and comfort effects that seem to be linked to the time they spend in the building, but in which no specific illness or cause can be identified.

In particular, indoor pollutants can affect the respiratory system in various ways. In fact, they can cause or exacerbate acute and chronic respiratory diseases and they can also cause a decline in respiratory functions and sensitization to common aeroallergens.
Subjects particularly susceptible to indoor air contaminants include people with allergy or asthma; people with chronic respiratory disease; people with a suppressed immune system; and contact lens wearers. Other subjects may be vulnerable to certain pollutants. For example, people with heart disease may be more affected by exposure to lower levels of CO than healthy individuals.

US National Occupational Research Agenda on indoor work environments

The US National Institute for Occupational Safety and Health (NIOSH) and diverse partners within the US occupational health community developed the National Occupational Research Agenda (NORA) that identified 21 priority areas in which new research could most effectively reduce work-related illnesses, injuries, and deaths in the coming decade. For the priority area “Indoor work environment” the team identified 3 types of heath effects as priorities for increased research (Mendell et al. 2002):

1. building-influenced communicable respiratory infections, due to occupant sources (e.g. influenza, common cold, tubercolosis) or building sources (Legionnaires’ disease, Pontiac fever, fungal infections);
2. building-related asthma, hypersensitivity pneumonitis and allergic diseases;
3. non-specific building-related symptoms (including so-called sick building syndrome)

The implementation of preventive measures has been estimated to prevent adverse health effects among millions of indoor workers and provide annual economic benefits in the order of a billion dollars.

The NORA team has not proposed research on all adverse health effects potentially related to indoor work environments. For the prevention of such exposures as environmental tobacco smoke, radon, asbestos and carbon monoxide and their respective health effects, strategies were considered to be well understood. On the

contrary, for some exposures, such as those that might influence cancer, neurotoxic effects, reproductive effects or "multiple chemical sensitivity" insufficient evidence was available to specify the responsible exposures and to estimate health or economic effects in indoor work environments.

WHO guidelines for indoor air

The working group of the recently published Global Update of the WHO Guidelines for Air Quality (WHO, 2006), recommended development of WHO guidelines for indoor air quality. Based on this recommendation World Health Organization convened a working group meeting for the development of indoor air quality guidelines representing scientific expertise in epidemiology, toxicology, exposure assessment, developing country issues, indoor combustion, biological agents, building construction, ventilation and indoor air quality management. The working group outlined three tasks required for the guideline development in 2007-2009 (WHO, 2007):

1. To list the specific chemicals for which numerical guidelines can be prepared;
2. To assess the biological contamination of indoor air
3. To assess the effluents of indoor combustion of solid fuels

1. List of chemicals

The group identified two groups of pollutants.
- Group 1: Pollutants for which the development of guideline is recommended, including Formaldehyde, Benzene, Naphthalene, Nitrogen dioxide (NO_2), Carbon monoxide (CO), Radon (Rn), Particulate matter ($PM_{2.5}$ and PM_{10}), Halogenated compounds (tetrachloroethylene, tricholoethylene), Polycyclic aromatic hydrocarbons (PAH), especially Benzo-a-pyrene (BaP).
- Group 2: Pollutants for which the current evidence is uncertain or not sufficient for guidelines, including Toluene, Styrene, Xylenes, Acetaldehyde, Hexane, Nitric oxide (NO), Ozone (O_3), Phthalates, Biocides, Pesticides, Flame retardants, Glycol ethers, Asbestos, Carbon dioxide (CO_2), Limonene, Pinene, TVOCs.

2. Biological agents

Exposures to biological agents in indoor environments were identified as a significant health hazard causing a wide range of health effects. It is usually impossible to identify individual species of the microbes or other specific biological agents responsible for the health effects; the exceptions are some common allergies, which can be attributed to specific agents or exposures, such as house dust mites or pets.

3. Combustion of solid fuels and kerosene

The group set the focus of the development of indoor air quality guidelines on exposures to emissions from household use of solid fuels and kerosene, not just for heating and cooking but including also e.g., lighting and small commercial activities in households. The group also recognized that the health relevant exposure to combustion products is affected also by the outdoor air quality. While moving

pollution emitted indoors to the outdoor air with venting radically improves indoor air quality, it still causes harmful exposure to people both outdoors as well as via outdoor pollution infiltrating back indoors. Therefore the reduction of the emission should be an objective of the health relevant actions. In some areas processed solid fuels, e.g., charcoal and "clean coal" may be part of the solution. Control of household use of coals contaminated with S, F, As, Pb, Hg needs to be considered as particularly important.

EU Scientific Committee on Health and Environmental risks (SCHER): Preliminary report on risk assessment on indoor air quality

To provide a scientific basis for the assessment of risks to human health from indoor air quality and for the development and implementation of policies, the SCHER was asked to identify a Risk Assessment Strategy to support policy on indoor air quality (SHER, 2007).

A number of factors in the indoor environment can affect well-being and health. The main factors are: Chemicals for intended use or unintentional emissions from different sources, radon, particles, microbes, humidity, pets and pests.

Indoor environment contains a large number of different chemical compounds. Availability of data on exposures to specific chemicals, their toxicity and associated health risks are highly variable. Therefore, a priority ranking of chemicals and exposures which cause concern is difficult and uncertain. However, the SCHER considered that formaldehyde, carbon monoxide, nitrogen dioxide, benzene, naphthalene, environmental tobacco smoke (ETS), radon, lead and organophosphate pesticides are agents of concern in indoor environment.

For most other pollutants the data available are yet too limited for risk assessment as indoor air pollutants. Consumer products, one source of chemicals in indoor environment, emit mostly volatile organic compounds. Lack of data on true exposure for emissions from consumer products has hampered the evaluation of the associations with possible health effects most of which are also caused by other factors. The recent data suggest that some of the emitted products may react further in air and on surfaces producing secondary products, including ultrafine particles. The health effects of those reaction products are poorly known.

The association of adverse health effects with dampness and water damage in buildings is repeatedly demonstrated in epidemiological studies, but the causative agents, mechanisms and all health consequences are not known. This is potentially a serious indoor air problem in EU. More research is needed to understand the associations between exposure indicators and the magnitude of the health effects in the EU countries.

Combined and mixture effects of indoor air pollutants can so far only rarely be assessed. Neither the databases nor methodologies exist to evaluate the breadth and depth of such combined effects. The SCHER recommends the production of data in order to make the evaluation of combined effects of indoor air pollutants feasible. In addition, the SCHER recommends taking also into account routes of exposure other

than inhalation (dermal, ingestion) in risk assessment and contribution of indoor exposure to the total exposure from other sources in all environments.

The risk assessment should be transparent to allow the evaluation of its strengths and weaknesses.

ENVIE: THE MOST IMPORTANT INDOOR AIR RELATED HEALTH EFFECTS

Aim of the EnVIE project is to evaluate the overall impact of poor indoor air quality on health in Europe and to inform policy makers about evidence-based preventive policies.

The following diseases (not in rank order) have been prioritised as being caused or aggravated by poor indoor air quality:

- Asthma & asthma-like symptoms
- Allergies: rhinitis, conjunctivitis, atopic dermatitis
- Lung cancer
- Chronic obstructive pulmonary disease (COPD)
- Respiratory infections
- Acute cardiovascular effects
- Acute/chronic CO poisoning
- SBS symptoms

One way of providing information for evidence-based policy is "health impact assessment", i.e., the quantification of the health effects of the exposure to indoor pollution. The health impact assessment of indoor pollution needs (i) epidemiology, to provide data on the relative risks at real life exposure settings and levels, (ii) toxicology to assess the mechanisms of action of and thus to provide biological plausibility for the health effects of indoor air contaminants, and (iii)frequencies, distributions and determinants of the exposures within the population.

Causal relationships between some indoor exposures and diseases have been well established and some quantitative assessments of the relative risks or of the dose response function are already available from published meta-analyses. For example: ETS has been well recognized to be causally associated with lung cancer, other respiratory and cardiovascular diseases; indoor air CO is an unquestioned cause of thousands of deaths from acute poisoning annually in Europe; indoor radon has been classified by IARC as a carcinogen and the results of a large European collaborative study on radon and lung cancer is about to be published. There are other exposures that have been causally associated with diseases, but for which comprehensive meta-analyses have not yet been published. Finally, in many cases no casual associations between contaminants and certain outcomes have been confirmed in spite of suggestive evidence in the literature.

It has to be underlined that in a recent review about indoor air quality it has been stated that the improvement of indoor air quality by a factor of 2-7 compared with

existing standards not only decreases the risk of health effects (like allergic symptoms and asthma in the homes), but also increases the productivity in offices and learning in schools (Fanger, 2006; Seppänen et al, 2006).

ROLES OF BUILDINGS, INDOOR AIR AND VENTILATION IN AIR POLLUTION EXPOSURES AND RISKS

Modern European citizens spend – in average – over 90% of their time indoors. Indoor air originates from outdoors, carrying outdoor air contaminants indoors with varying degrees of penetration: some are effectively transferred indoors (e.g. $PM_{2.5}$, P = 50–90%), others are adsorbed on building structures and indoor surfaces (e.g. H_2S), some readily react with indoor air co-pollutants (e.g. O_3). Also indoor environments contain sources of contaminants, which, due to the low indoor air exchange rates compared to outdoor environments, may lead to quite high levels. These have been widely studied for a range of chemicals, particles and biological contaminants, and in the presence of indoor sources, indoor contaminant concentrations are higher, sometimes 10 or 20 times higher (e.g.: formaldehyde) than the respective outdoor air levels. The combination of the generally higher indoor concentrations and the overwhelming fraction of time spent indoors results in the overall domination of indoor air in air pollution exposures – and their respective health consequences - regardless of whether the sources are indoors or outdoors.

Radon is a natural and carcinogenic air contaminants, which is produced in the soil as the only gas phase radioactive decay product of natural uranium, and drawn into indoor air by the air pressure gradient caused by the normally lower indoor relative to outdoor air pressure. The indoor air radon concentrations often exceed outdoor air levels by 1...2, even 3 orders of magnitude.

Indoor exposures to allergens from outdoor sources – e.g. pollen - affect sensitive individuals. The sources of other sensitising agents, e.g. mould spores and particles, are often found indoors. While the building – in particular persistent moisture in its structures - is often the cause of the latter, it may, at the same time, provide significant protection against the former exposures, and this protection can be further enhanced by the ventilation equipment, indoor space cleaning and occupant behaviour.

The function of ventilation is to ensure the delivery of fresh air and extraction of the contaminants from the indoor sources (e.g. human metabolism, occupant activities, consumer products, furnishing, building equipment and materials). Yet, ventilation may also draw in polluted outdoor air, the ventilation system may itself become contaminated and, thus, a source of pollution. Insufficient ventilation may cause moisture to accumulate in the building, unbalanced ventilation may result in radon buildup, uncontrolled air leaks, and moisture condensation in hidden building structures, and excessive ventilation with no heat (or cool) recovery wastes energy.

AIR QUALITY POLICIES & INDOOR AIR

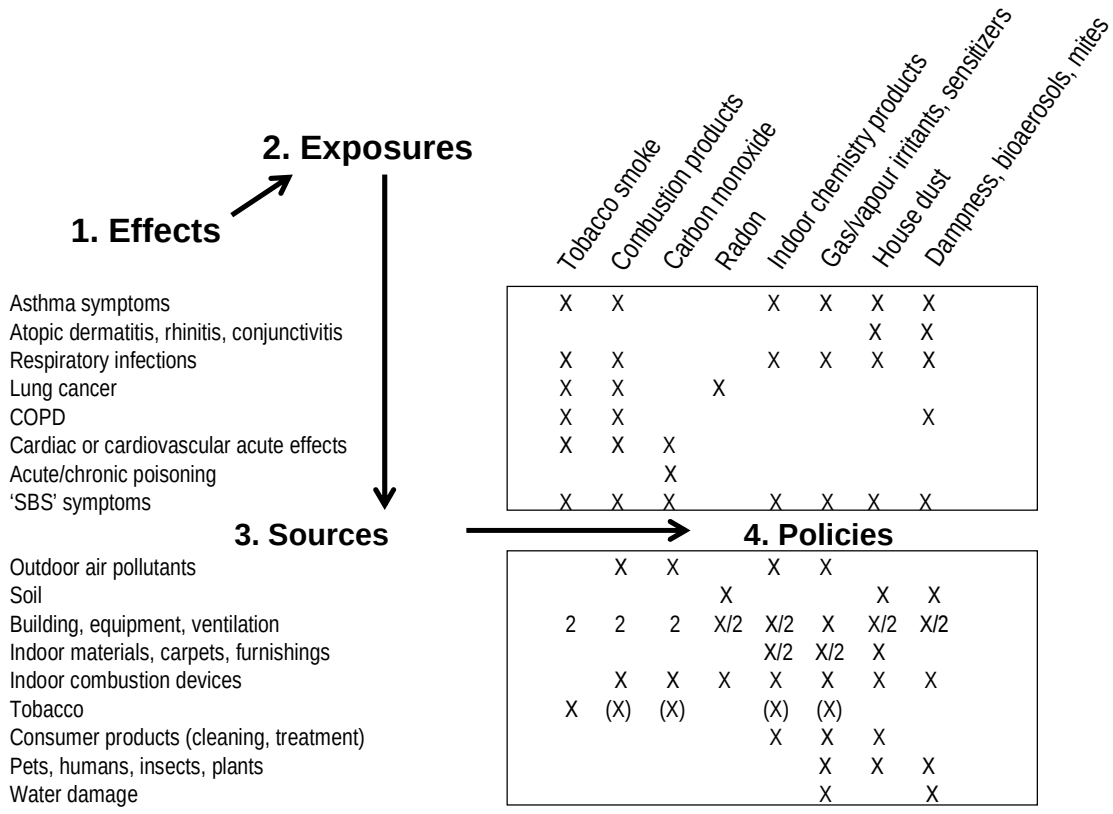
European air quality policies have devoted most of their efforts to control the urban outdoor air concentrations of a short list of regulated air contaminants from heat and power generation, industrial processes and traffic. Although there is no reason to relax the society's preoccupation towards these issues, it is now recognized that new policies should be focussed on indoor exposures to identify, control and eliminate the indoor sources of pollution, and to also reduce the exposures to air pollution of outdoor origin. In the case of most VOCs, the focus should be on the indoor sources, building materials, consumer products and occupant activities. In the case of NO_2 and fine PM, both indoor source control and filtration of the outdoor air contaminants may be considered. Exceedances of the European and National outdoor air CO standards are becoming rare. Yet toxic CO exposures are not vanishing, they claim thousands of lives annually and are currently caused almost exclusively by indoor combustion sources, which could be regulated more easily than many other sources in the buildings.

National air quality policies on indoor air have until recently consisted of scattered regulations on building materials and equipment, HVAC equipment in particular, ventilation rates and concentration guidelines on a few chemicals (mainly formaldehyde) and radon. The indoor air issues in Europe have since 1987 been broadly covered in the 27 reports of the European Concerted Action on Indoor Air Quality and its Impact on Man (Since 1999, Indoor Air, Human Exposure and Urban Environment). DG Sanco funded the JRC/IHCP Coordinated IndEx project (2002-2005), which for the first time in Europe evaluated the indoor air chemicals for which exposure and health data was available in Europe, and recommended general IAQ policies concerning ventilation, combustion devices, source control, occupant behaviour, building maintenance, etc. and regulations for a shortlist of chemicals consisting of Formaldehyde, Benzene, NO_2, CO and Naphthalene. Biological contaminants, radon and ETS were not included, because they were not chemicals. In 2006 WHO/Europe initiated the preparation for Indoor Air Quality Guidelines, expected for 2008-9.

EnVIE OBJECTIVES AND APPROACH

It is therefore the objective of the EnVIE project to increase the understanding of the Europe-wide public health impacts of indoor air quality. The project will identify the most widespread and significant indoor causes and sources for these health impacts, and evaluate the existing and optional building and housing related policies for controlling them. It will address in particular how indoor air quality might contribute to the observed rise in asthma and respiratory allergy, together with other acute and chronic health impacts. The project does not intend to conduct new experimental or field research, but rather to build on the broad scientific experience, as well as the literature of reports and articles which have accumulated in the domestic and international indoor air research projects as well as the EU, WHO, ISIAQ and CIB committees and expert groups during the past 20 years.

Many previous indoor air quality and policy assessments have taken specified contaminants or indoor sources as the starting points. The logic behind is the flow of molecules from sources via environment to exposure, dose and the consequent health outcome. EnVIE follows an opposite logic, starting from the most pronounced indoor air related health outcomes (which have often also other sources and causes), then identifying the most widespread indoor air exposures which are likely to cause these health outcomes, and the most common sources which dominate the indoor air exposures – see Figure 1. The 1st objective of this approach is to focus from the start on those indoor air quality issues of the highest Europe-wide health relevance. Having defined a shortlist of such indoor health-exposure-source chains, the project will evaluate the policy alternatives for minimising the unwanted health consequences in terms of achievable public health benefits, invasiveness, as well as political, legal, technological, economical and social feasibility. The 2nd objective is to identify and describe a set of beneficial and feasible indoor air quality policy alternatives for Europe. Europe wide applicability brings, aside of the health benefits, also the economical benefits of enhanced competition in a broader marketplace.



'X, X, X' denote different levels of impact. '2' denotes secondary influences.

Figure 2. Flowchart of the EnVIE project highlighting the health, exposure, and source issues selected in the first stage of the project.

ENVIE ACTIVITIES AND PRODUCTS

The EnVIE project will centre around two EnVIE Conferences. The 1st EnVIE Conference will cover the selected issues from health outcomes, via exposures to sources. The 2nd EnVIE Conference will focus on alternative source control policies, evaluating the achievable exposure and health impact gains, as well as the other requirements and consequences of these alternatives. In EnVIE Workshops the WP:s will on one hand create the detailed programmes for the EnVIE Conferences, and on the other hand critically evaluate and edit their results. Through (i) careful planning by the EnVIE steering committee and (ii) well instructed preparation by the conference presentation authors, the presen tation texts, (iii) critically reviewed and edited by the EnVIE WP:s and complemented with discussion summaries, will form core chapters of the EnVIE project report.

The EnVIE Final Report will consist of the edited EnVIE Conference proceedings complemented with a general introduction and an executive summary, which will also include the general recommendations.

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Session 2 SBS, Irritation, Odours

Perceptions, subjective symptoms and syndromes related to IAQ and their use in guideline settings

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INTRODUCTION

Definitions

Health is a state of complete physical, mental and social well-being and not merely the absence of decease or infirmity (WHO 1948). The effects of interest indoors therefore include both adverse effects and changes of well-being. Building-Related Illness (BRI) is a group of known causalities between symptoms and indoor exposures to air pollutants. Generally, the causalities have a uniform clinical picture and a specific cause of the complaints. Many BRIs´ are low exposure levels manifestations of adverse effects known from high exposures e.g. occupational exposures. The symptomatology is important for diagnoses of adverse effects at low exposure levels indoors. The prevalence of building-related symptoms (BRI) is commonly used to characterize the indoor air quality (IAQ) in office buildings (Niemela et al 2006). An association may exist between BRI and productivity or sick leave (Niemela et al 2006).

Objective health effects are quantifiable changes or signs observed by an independent observer (not the exposed). In contrast, symptoms and perceptions are personal experiences or judgements made by the exposed occupant. Symptoms are unspecific i.e. many exposures may cause each of them. Therefore they do not alone identify the exposure cause. For each symptom multiple response modifiers and multiple biases are possible and different persons may have different spectrum and intensity of symptoms. Also, most indoor exposures may cause a number of different signs and symptoms. Therefore, objective measurements of effects are preferred and subjective ratings should be substituted by objective measurements where possible but few are available. On the other hand, objective measurements are expensive and time consuming, a fact which in many cases prevent their use and in the absence of instrumentation for chemical detection of small amounts of some air pollutants, the senses remain the most sensitive indicator system (Berglund et al 1992). Added to this is that discomfort is subjective by nature and cannot be measured without subjective evaluations. Many symptoms are therefore important *per se*, and cannot be substituted by objective measurements.

Aims

This presentation is aimed at both an update on the biological background for known symptoms and perceptions in IAQ science and practice as well as presenting some of the newest literature in the field. In addition recommendations are given on how to use symptom's and perception's reports in field investigations and IAQ sciences. The paper discusses how substitution of subjective evaluations can be made with objective measurements, and if IAQ guidelines can be defined for signs and subjective symptoms. Finally, recommendations are given on guideline settings for IAQ.

This review includes literature younger than a review made by Berglund et al (1992). It does not pretend to be complete but merely summarizes uses of symptoms and perceptions during the last 5-7 years in IAQ research and managements of buildings. The focus is on symptoms and subjective ratings, not on objective health effects.

BRIEF DESCRIPTION OF SYMPTOMS AND PERCEPTIONS AND THEIR CAUSES

Indoor airborne exposure of humans to indoor pollutants may either affect sensory systems or result in tissue changes. Table 1 summarises some of the biological reactions or processes which may be active in human responses to poor IAQ.

The chemical sense

The chemical senses include specialised receptors for odours and irritants in eyes, facial skin and nose cavity. These senses incorporate n. Olfactorius (odours) and n.Trigeminus (irritants). Most odorous compounds are also irritants and visa versa and the chemical sense acts as a warning system (Berglund et al 1992). Mixtures of pollutants may interact and one odorant may mask other odorants (Pan et al 2000). The time course of effects may show adaptation or accumulation of effects which create problems for the interpretation of causality.

Unspecific pain and irritative receptors

Unspecific pain and irritative receptors in the skin or mucosa of eye, nose, throat, and air ways are other sensory systems active in response to indoor air pollution. These senses detect the status of the tissues including presence of absorbed irritants or initiate release of irritating signalling compounds or reflexes in the exposed tissue.

Visual observations

Visual observations are involved in observations of skin rashes, smog, or dust in the air and thus influence the evaluation of the effects of poor IAQ.

Immunological responses

Immunological responses to IAP include diseases such as allergic asthma and extrinsic allergic alveolitis (hypersensitivity pneumonitis) which are the two most serious allergic diseases caused by allergens in indoor air. Allergic rhinoconjunctivitis and humidifier fever are other important diseases. The biological mechanisms include

immunological specific IgE sensitisation to an airborne allergen. The type of symptoms observed in allergic asthma is characterised by reversible narrowing of the lower airways leading to difficulties in breathing, tightness of breath, respiratory sounds etc. Other symptoms are itching of the eye and/or the nose, sneezing, watery nasal secretion and some stuffiness of the nose. Pulmonary function during an attack shows an obstructive pattern in serious cases together with reduced respiratory ventilation capacity. Many objective measurements are available but may not apply at low indoor exposure levels where the symptomatology becomes more important.

Inflammation

Two types of sensory irritation appear in the literature to be related to indoor climate and air quality: a primary sensory irritation caused by direct stimulation of sensory cells by environmental exposures and a secondary irritation following changes in the skin, mucous membranes, or other tissues (Berglund et al 1992). Each of these may subsequently lead to the other. Often inflammation is a direct effect of chemicals on the tissue cells leading to cell damages. Through release of mediating compounds, the cells may signal the need of activation of defensive responses. Inflammation is characterised by a sensation of heat ("calor'), redness ("rubor"), swelling ("tumor"), pain ("dolor") and a certain loss of function in the tissues affected. Non allergic asthma type of responses may be related to inflammatory responses. Irritative effects on tissues can be a considerable annoyance either in terms of severity of effects on an individual or in terms of the number of persons affected. Irritative effects causing tissue changes in the skin and mucous membranes have been reported in many forms, although they have seldom been seen in an adverse form to follow exposure to normal indoor air (Berglund et al 1992).

The body's signalling systems

Body signalling systems may be activated by biomarkers or neural activity. These reactions follow both from immune and inflammatory type of responses and include biomarker or mediator compounds released in the tissues and neural activity in the form of reflexes. Both are signaling the status of the body and initiate defensive responses where needed immune responses or weak irritative reactions may lead to release of signalling compounds or biomarkers such as histamine or cytokines. These compounds may by themselves be irritative in the tissue and may thus accelerate the irritative effects. These responses may be observable as rashes, skin reddening etc. Neural reflexes are often defensive reactions. The symptoms are related to watering eyes, secretion of mucosa or tears, increased blood flow in the exposed tissues, bronchial constriction, or cilia movements in the upper airways, or cough. The effects may often but not always appear at the site of contact on the exposed skin or mucosa.

Central nervous system (CNS)

Symptoms or perceptions are reported as processed evaluations incorporating many symptoms or perceptions such as perceived comfort or sensation of air quality. It is not known how this is done in the Central Nervous System (CNS). They are typically reported by the occupant as prevalence or intensity of symptoms or perceptions. It is, however, known that the reports are strongly affected by personal or external biases, and that adaptation and sensitization frequently appear.

THE TWO DIMENSIONS OF SYMPTOMS AND REPORTS

Indoor air pollutants may each activate a multitude of biological mechanisms and subjects are often experiencing many exposures at the same time. Because of this and the complex nature of the resulting subjective reports as described above, no consistent and general agreed classification exists of reports of symptoms or evaluations of IAQ. Here a suggested classification is found in table 2 which shows three groups of perceptions, symptoms, well-being and other subjective health effects in relation to IAQ. The table is a modification of a classification suggested by (Berglund et al 1992). The three groups of symptoms and perceptions are here called "Perceptions of body functions", "Environmental perceptions", and "Processed reports or evaluations". These classes are defined with consideration to whether they can be replaced by objective measurements or not or if the target value for their prevalence in guideline setting is zero or not.

Perceptions of body functioning

Perceptions of body functioning are reported as symptoms of mal-functioning body systems, inside the body or on the body surface. These may be caused by immune or inflammatory reactions. Focus of the occupants' reports are on the type of organs affected such as eyes, nose, mouth, or throat (exposed mucosa), skin, respiratory malfunction, allergic asthma responses, non-immune responses, bronchial constriction, CNS changes (e.g. reaction time and errors), and increased responsiveness (e.g. hyper reactivity, allergy). Many of the biological mechanisms mentioned above may be involved in each reported symptom. Typical symptoms are dryness, increased secretion, perceived irritation, soreness, cough, tightness of breath, headache, rashes, stinging, itching, burning. Although biologically different, subjective reports of irritation of mucous membranes in eyes, nose, and throat caused by inflammatory or immunological responses cannot be separated from responses of the chemical sense n.Trigeminus. In principle, objective measurements can be used for most if not for and objective measurements exist for many of the physiological effects reported by these body perceptions but not for all. These body perceptions are characterized by a target value for guideline settings and recommendations of zero prevalence.

Environmental perceptions

Environmental perceptions include perception of the environment including the presence of any air pollutants. Typical reports include odours as response to odorants in the air (n. Olfactorius) and irritation (n. Trigeminus and the chemical sense) in mucous membranes, nose and eyes, and facial skin (unspecific censors in the skin may be included). Other important IAQ senses relate to air temperature, humidity, and vision. The environmental senses also include hearing, taste, noise, draught, and illumination. These will not be dealt with in this summary. Adverse perceptions are unwanted changes of life quality and thus full value health effects (Berglund et al 1992). However some level of perceptions is required to allow persons to follow changes in the status of their environment. It follows that these perceptions may have a D-R relation of U-form i.e. in guideline settings the prevalence target is non-zero.

The detection of ocular and nasal sensory irritation increases as a function of vapour concentration at much higher rate than that for the detection of odour. However the odour intensity of mixtures of odorants cannot presently be predicted (Cometto-Muniz et al 2004). Although biologically different, subjective reports of activation of the chemical sense and n.Trigeminus cannot be separated from responses caused by irritation of mucous membranes in eyes, nose, and throat following inflammatory or immunological reactions in the tissues. Recent work in this area is therefore summarized above.

Processed evaluations and syndromes

The processed evaluations are based on multiple symptoms or perceptions. They are interpersonal dynamic interactions expressing the person's emotional content of body and environmental perceptions.
Processed evaluations are important indicators of IAQ. By definition they are based on psychological processes and thus cannot be documented without using subjective reports. However, they are difficult to use in scientific research and in investigation of buildings with poor IAQ.

They include many symptoms´ complexes such as syndromes (a spectrum of related symptoms) and overall evaluations of many symptoms combined into one evaluation. An example is the "Sick Building Syndrome" (SBS) in which the affected workers report non-specific symptoms only during the time at work, most often with no known cause (Berglund et al 1992). Symptoms reported in SBS have typically included mucous membrane and eye irritation, cough, chest tightness, fatigue, headache, and malaise. The criteria for the definition of SBS are summarised in Table 3. More details on the SBS can be found in a monograph prepared by a group of experts for the Committee of the COST 613, and the reader is referred to that document for further information (EU 1989, Berglund et al 1992). The use of SBS should be discouraged and replaced by multi-symptom questionnaires such as MM 40 (Lahtinen et al 2004).

Other examples are "Perceived air quality" which is a mixture of odour, irritation, stuffiness, feeling of heavy head, stuffy or stale air resulting from stimulation of both the nerves Trigeminus and Olfactorius, "Comfort" or "Well-being" which seem to be a mixture of body symptoms or body perceptions. Discomfort and general well-being are in many investigations used as independent evaluations. "General Well-being" or "General Symptoms" in many publications seem to be a mixture of body symptoms or body perceptions, etc. Finally, productivity and absenteeism report a mixture of the perceptions, or body reports seem to be processed evaluations and a mixture of the perceptions or reports mentioned above.

Productivity and learning capacity are also integrating CNS changes. Results from a preliminary study yield a significant association between classroom-level ventilation rate and test results student performances on standardized aptitude tests that are administered to students on a yearly basis (Shaughnessy et al 2006).A review of 23 studies suggests that a linkage exists between typical BRIs and productivity indicators such as task or work performance or absence from work. Quantitative associations between BRS and productivity were demonstrated in two office environments (Niemela et al 2006).The existing literature indicates that ventilation has a significant

impact on several important human outcomes including task performance and productivity among occupants or sensory panels (Seppanen et Fisk 2004). It has now been shown that poor indoor air quality in buildings can in addition to causing visitors to express dissatisfaction. The size of the decrease of productivity in most aspects of office work performance appears to be as high as 6-9%, the higher value being obtained in field validation studies (Wyon 2004).In an intervention study the performance of four simulated office tasks improved monotonically with increasing ventilation rates, and the effect reached formal significance in the case of text-typing. For each two-fold increase in ventilation rate, performance improved on average by 1.7% (Wargocki et al 2000).Another intervention study indicated that the indoor air quality improved productivity by 11%, compared with a 4% reduction of productivity among the control group of workers (Menzies et al 1997).Recent studies show that improvement of IAQ by a factor of 2-7 compared with existing standards increases office productivity and school learning significantly, while decreasing the risk of allergic symptoms and asthma in homes (Fanger 2006).

IDENTIFICATION OF THE MAIN INDOOR AIR POLLUTANTS AND RELATED SOURCES CAUSING THE DISEASE

Symptoms are unspecific i.e. many exposures may cause each of them. Therefore they do not alone identify the exposure cause. The relevant IAPs are those which can stimulate our senses or may cause tissue changes i.e. all known indoor airborne chemicals at some level (maybe excluding radon and CO). The pollutants may be gasses, vapours, viable or non-viable aerosols or particulate matter, allergens, etc. The risk factors also include technical causes such as ventilation, humidity and temperature. The sources of IAP are found indoors and outdoors and include humans, their activities, processes, maintenance, furniture, etc.

Perceptions of body functioning

In recent investigations symptoms related to mucous membranes in eyes, nose, mouth, and throat are symptoms frequently related to poor IAQ (Skyberg et al 2003, Peitersen et al 2006). These symptoms are reported from office buildings (Reijula et al 2004, Wolkoff et al 2006). or buildings with low ventilation (Wargochi et al 2000). Symptoms have been related to house or office dust exposures (Pan et al 2000; Skulberg et al 2004, Chao et al 2003), chemical contaminants from the sewer system and damp construction materials (Putus et al 2004), and with mould exposure (Ebbehøj et al 2005; Hirvonen et al 1999; Park et al 2006). Pharyngeal dryness increased when temperatures rose and was alleviated with a rise in relative humidity (Reinikainen et al 2003). Symptoms related to skin areas are frequently reported from field surveys (Skyberg et al 2003). Recently reported or suggested causes are exposures to mould (Ebbehøj et al 2005), storing of organic waste in the home (Herr et al 2004a,b), and house,or office dust (Skulberg et al 2004). Examples of respiratory symptoms are cough, tightness of breast, asthmatic symptoms, phlegm, wheeze, chest tightness, attacks of shortness of breath, and attacks of cough. These symptoms are reported from buildings with low ventilation (Wargochi et al 2000). Chemical contaminants from sewer system and damp construction materials (Putus et al 2004) exposures to house or office dust (Pan et al 2000; Herr et al 2004b), mould exposures

(Hirvonen et al 1999; Putus et al 2004; Chao et al 2003) have been suggested as cause. Significantly increased lower respiratory symptoms were associated with Endotoxin in floor dust (Park et al 2006). In field surveys, symptoms related to CNS and performance are frequent (Skyberg et al 2003). Examples are difficulty in thinking clearly, concentration difficulty, headache, feeling of fatigue, heavy-headedness, sluggishness, sleepiness, nausea, etc. These symptoms are reported from buildings with poor ventilation (Wargochi et al 2000). The symptoms are often work-related (Reijula et al 2004). Occupants in open-plan offices more frequently complain about CNS symptoms than occupants in multi-person and cellular offices (Peitersen et al 2006). Some reported or suggested causes are house or office dust (Pan et al 2000), and moulds in the indoor environment (Hirvonen et al 1999; Ebbehøj et al 2005).

Environmental perceptions

Dampness in dwellings, with emissions of odorous compounds, is associated with an increase in symptoms (Engvall et al 2002). The indoor climatic conditions seem to influence the perception of odours. Any kind of humidity seems to increase odour sensation (Reinikainen et al 1997, 2003). A combination of odours and signs of high humidity in buildings was related to an increased occurrence of all symptoms (Engvall et al 2002). Increasing ventilation decreased the percentage of subjects' odour reports, and increased the perceived freshness of air (Wargochi et al 2000). N.Trigeminus and the chemical sense for irritation are found in mucous membranes of nose and eyes, and facial skin.

Processed evaluations and syndromes

Occupants in open-plan offices are more likely to perceive poor air quality than occupants in multi-person and cellular offices (Peitersen et al 2006, Reijula et al 2004, Wargochi et al 2000). The recently reported or suggested causes are chemical contaminants from the sewer system and damp construction materials (Putus et al 2004), with mould exposure (Ebbehøj et al 2005), lack of office cleanliness, and low job satisfaction (Chao et al 2003). Processed ratings such as perceived "Air Quality" may be significantly correlated with other responses (Pan et al 2000). Recently several groups have discussed a prioritising of the most IAQ relevant compounds (WHO 2006, 2007, Cochet et al 2006, Kotzias et al 2005, Anonymous 2006).

EPIDEMIOLOGY: INCIDENCE/PREVALENCE OF THE DISEASE, RISK ATTRIBUTABLE TO IAQ, TIME TREND

The most frequent effects related to indoor air quality (IAQ) seem to be acute physiological or sensory reactions, psychological reactions, and subacute changes in sensitivity to environmental exposures (Berglund et al 1992). Objective, adverse health effects of poor IAQ are well known but rare compared to the prevalence of unwanted symptoms and perceptions (Berglund et al 1992). Because of the unclear and subjective nature of evaluations and complaints no clear definitions exist for the unacceptable prevalence and no reference values or golden standards exist on which conclusions can be based. In the literature, the levels of prevalence, which have been called abnormal range from 10% to 100% depending on the symptom or perception in

question. There is good and substantial evidence for the relation between Indoor Air Pollutants (IAP) and symptoms and perceptions (Berglund et al 1992).

IDENTIFICATION OF SUSCEPTIBLE POPULATION SUBGROUPS

It is well documented that risk groups exist and many response modifying factors affect the occupants' responses. Examples of known risk factors are health status (atopy, sick persons, skin temperature), demographic data (age groups incl. children, occupation, job function, gender), life style (smoking), psycho-social loads (low social support or satisfactions, psychosocial and personal biases), exposure scenario (previous exposures, competing sensory stimulation, interactions between concurrent exposures, adaptation, accumulation, duration of exposure). Interactions between concurrent exposures and adaptation processes are characteristic of the sensory systems involved in the perception of odour and mucosal irritation, further the duration of exposure influences the perception (Berglund et al 1992).

Women report symptoms more often than men (Ebbehøj et al 2005; Reijula et al 2004; Skyberg et al 2003; Bullinger et al 1999; Runeson et al 2003, 2006). This may be an effect of less favourable working conditions under which women are employed (Bullinger et al 1999). Responding women may have a lower sense of coherence (SOC) value, a psychological measurement of a life attitude (Runeson et al 2003). Individuals experimentally given a harmful bias reported significantly more health symptoms following exposure indicating induction of a strong personal bias (Dalton 1999). Therefore psychosocial and personal reasons may dominate general symptoms (Ebbehøj et al 2005). Sick Building Syndrome (SBS) may be more common in younger subjects (Runeson et al 2003). Atopic disposition is a possible risk factor for skin irritation (Herr et al 2004a,b, Chao et al 2003, Runeson et al 2003, 2006, Reijula et al 2004, Skyberg et al 2003). Lifestyle including passive smoking and psychosocial load are also predictors of symptoms (Skyberg et al 2003). Also occupation, job functioning, low social support or satisfactions are risk factors (Skyberg et al 2003, Chao et al 2003, Runeson et al 2006).

GUIDELINE SETTINGS

Challenges of the use of symptoms in IAQ science and practice

From the previous chapters it appears that indoor air pollutants cause unspecific effects and that these do not unambiguously identify the exposure. A multitude of biological mechanisms are involved in the responses to multiple exposures indoors and only few objective measurements are available. Some types of responses can not be replaced by objective measurements and often the effects and exposure cannot be quantified. Added to this, the resulting subjective reports are affected by bias and response modifiers. It follows that traditional toxicological procedures to the establishment of guidelines seem impossible to use for these subjective responses and evaluations.
In IAQ guideline settings three types of DR relations must be considered. These are perceptions and symptoms with known causality, based on quantifiable effects and exposures, unspecific symptoms with unknown causality, and hypothetical causalities waiting for further investigations.

Guidelines for perceptions and symptoms with known causality

As described in the introduction, a BRI is characterized by a known causality between health and a certain exposure. At low exposure levels only unspecific symptoms may be present and often symptoms are the most sensitive effect of IAP. It follows that most IAQ guidelines for BRI will be defined from such symptoms. At low exposure levels the presence of these unspecific symptoms does not by themselves identify the causal exposure. This must be identified by other means e.g. measurements. Formaldehyde is an example for which a threshold for irritation/odour could be defined in the lab. Another example is asthma or COPD caused by many types of air pollutants. For these diseases the symptomatology is important for the diagnoses.

Because of the known causality behind a BRI, thresholds and guideline values can be defined following traditional procedures using symptoms in controlled lab settings and quantifiable exposures. In this way thresholds, NOEL, and LOEL can be defined where interactions from other types of exposures can be excluded.

For most of the health effects for which objective measurements are available D-R relations and thresholds are not available and few of the thousands of relevant chemicals have been examined at low exposure levels. Despite this some progress has been seen. Recently several groups have discussed guideline settings for the most IAQ relevant compounds (WHO 2006, 2007, Cochet et al 2006, Kotzias et al 2005, Anonymous 2006). Several procedures for prioritizing are available by which the most important pollutants can be identified. However, no consensus exists. While we are waiting for missing data, substitute measures might be helpful. At low IAQ exposure range a lowest concentrations of interest (LCI) type of estimates may be useful. Recommended low and a higher action levels may also apply (Bluyssen et al 1997). Again no consensus exists for such procedures. Under all circumstances an ALARA principle should be followed.

In IAQ guideline settings apportionments between allowable contributions from different sources must be discussed. We do not know how to deal with it. A special case of this is how indoor/outdoor fractions are coordinated in I/O guidelines.

Guidelines for unspecific symptoms with unknown causality

Assuming that all BRI with known traditional mono-factorial causality are dealt with as described above, it can also be assumed that all causalities which in higher exposure ranges might cause more adverse and irreversible health effects in occupants are under control. However, a group of causalities remain to be dealt with. These typically include effects with multifactor relationships following mixtures of exposures (cocktail effects). For the reasons mentioned above, many such symptoms and perceptions in mixed real life exposures do not qualify for traditional guideline settings. A broad spectrum of causes is possibly contributing to the prevalence of individual symptoms or perceptions in any particular building and to SBS. Because of the ill-defined causality, lack of quantifiable effects and exposure measurements etc. no strict traditional guidelines can be established. However, the importance of such complaints is well documented and guidance, recommendations, labelling systems, and emission control become the tool of prevention. These less strict guidelines are acceptable only for discomfort and SBS etc. and only if averse health effects can be excluded e.g. because all relevant exposures are under guideline regulation as mentioned above. Also the combined effects of cocktail exposures are unsolved both scientifically and administrative. Some additive procedures may be taken over from occupational guideline settings.

It follows that a set of good practices for construction, maintenance, and building usage should be developed which covers all relevant risk factors (a healthy building's regulations). The risk factors include technical causes such as ventilation, humidity and temperature, IAP sources, maintenance etc. In any case an ALARA principle should be followed.

An example of the complex nature of such guidelines is Endotoxin in building dust which may indicate dampness and possible microbial growth and thus increased risk of building-related symptoms including building-related asthma, respiratory, and systemic symptoms (Park et al 2006). Building type especially open-plan offices may be a risk factor for adverse environmental perceptions and symptoms (Peitersen et al 2006). Indoor air temperature and humidity may be important for the perceived air quality and SBS symptoms (Fang et al 2004). Perceived indoor environments, non-specific symptoms, and their associations are associated with the season (Mizoue et al 2004).

Guidelines for unknown or hypothetical causalities

An example of an unknown or hypothetical causality is multiple chemical sensitivity (MCS). This is presently hypothetical and more research is needed before guidelines can be defined. In any case an ALARA principle should be followed.

DISCUSSIONS AND CONCLUSIONS

Use of symptoms and perceptions as indicators of indoor air quality

This review shows that not much has changed since the report of Berglund et al. (1992). The poorly defined symptoms remain poorly understood. The disability associated with IAQ symptoms and syndromes still generates controversy (Hodgson 2002).

Three types of subjective evaluations or reports related to IAQ are identified. They are "Perceived Body Functions", "Environmental Perceptions", and "Processed Reports or Evaluations". "Perceived Body Functions" describes changes in body functioning and are focussed on individual organs or tissues. "Environmental Perceptions" addresses exposure factors in the environment. Subjective evaluations are essential for these two last types of evaluations and they can probably not be replaced by objective measurements. The science of psychophysics offers a variety of sensory models for studying indoor air quality effects and for indoor air quality characterization (bio-assays).

No simple causal D-R relation can be expected for subjective symptoms and perceptions and unknown biases make it difficult to use occupants' reports in science and investigations as their personal biases can be strong. In real life situations, the symptoms or subjective reports prevalence's should not be used as exposure measurements and subjective reports from buildings may only qualify as screening tools. It is concluded that the use of SBS should be discouraged and replaced by

multi-symptom questionnaires. Personality and personal vulnerability such as gender, age, atopy, and asthma, as well as indoor exposures, should be considered in both indoor environmental epidemiology and in practical handling of buildings with suspected indoor problem, especially when the technical investigations fail to identify any obvious technical malfunction (Runeson et al 2003, 2004, 2006). It is important to combine technical measurements or inspections with a longitudinal evaluation of occupant reactions (Engvall et al 2005) and indoor air temperature and humidity may be important for the perceived air quality and SBS symptoms (Fang et al 2004).

A WHO expert group has recommended that odours can be measured through the immediate response of the non-adapted olfactory system (visitor situations). It should be noted that odour intensity measured by visitors does not necessarily correlate with the perceptions of the occupants (WHO 1987, Berglund et al 1992). Therefore occupants' reports are also needed. Regulatory agencies now require sensitivity, validity, reliability, and biological meaningfulness of sensory methods applied for indoor air quality control (Berglund et al 1992). Therefore, investigators should use a strong quality assurance policy in IAQ evaluations based on subjective reports. However, to reduce bias a trained external panel may have to be included in IAQ investigations. Control groups and norm values in reference groups are difficult or even impossible to use in relation to IAQ. Taking this into consideration, the search for norm values or a framework seems to be of limited value (Neuner & Seidel 2006).

Objective methods may only apply for body perceptions and some environmental perceptions, and suggested indicator of activated defence mechanisms include indicators of inflammation and immune system responses, changed biomarker values in lavages, condensed exhalation, blood, and tear liquid (e.g. cytokines, cells), reddening eyes and skin, skin irritation, and rashes. Recent indications of new biomarkers for changed body functions caused by poor IAQ have appeared. Inflammatory markers may predict high prevalence of respiratory symptoms (Hirvonen et al 1999). Lu et al indicated that the urinary 8-hydroxydeoxyguanosine (8-OHdG) level was significantly associated with SBS complaints (Lu et al 2007). This is also that case for matrix metalloproteinase 9 (MMP9), leptin, and alpha melanocyte which may stimulate hormone (MSH), vascular endothelial growth factor (VEGF), immunoglobulin E (IgE), and pulmonary function (Shoemaker & House 2006). Neurological functioning may in the future be monitored objectively through *v*isual contrast sensitivity (VCS), an indicator of neurological function, which was abnormally low in SBS patients (Shoemaker & House 2006) and performance measurements may be used as processed measurements of CNS function. Examples are errors made while typing, number of calls made in call centres, and absence from work. Physiological changes may in the future be registered objectively through blinking frequency (Nøjgaard et al 2005). Peak flow and respiratory measurements are available for respiratory effects and allergy-asthmatic changes. An interesting observation is that Shoemarker et al indicated that cholestyramine (CSM) therapy may be an effective therapy against SBS (Shoemaker & House 2006). The indicated objective methods can only be used for body perceptions, but many are themselves not real health effects but merely biomarkers which in addition also are strongly influenced by biases.

Guideline settings based on symptoms reports and perceptions in IAQ

Several working groups have shown that principles for setting of IAQ guidelines can be defined based on combinations of existing procedures. The WHO has initiated a working group to define such guidelines and recommendations. The future guidelines may include both traditional guidelines for single compounds and a set of guidance and recommendations for healthy buildings covering situations with only minor adverse health changes or discomfort.

Many suggested objective measurements (e.g. mediators) which are in progress to be used in guideline setting are not real health effects but merely biomarkers of ongoing changes, and are strongly influenced by biases. It is often questionable if they can be used as substitute measurements. In IAQ guideline settings three types of DR relations must be considered. These are perceptions and symptoms with known causality, based on quantifiable effects and exposures (BRI), unspecific symptoms with unknown causality, and hypothetical causalities waiting for further investigations. It is concluded that future guidelines for ventilation rate based on comfort and health should no longer be independent of indoor air temperature and humidity.
Because of the known causality behind a BRI, thresholds and guideline values can be defined following traditional procedures using symptoms in controlled lab settings and quantifiable exposures. In this way thresholds, NOEL, and LOEL can be defined where interactions from other types of exposures can be excluded. Some progress has been seen recently in approaching guidelines for IAQ. Several procedures for prioritizing are available by which the most important pollutants can be identified. In guideline settings apportionments between allowable contributions from different sources must be discussed. A special case of this is how indoor/outdoor fractions are coordinated in I/O guidelines.

The combined effects of cocktail exposures most be dealt with both scientifically and administratively. Some additive procedures may be taken over from occupational guideline settings. Symptoms and perceptions in such mixed real life exposures do not qualify for traditional guideline settings and guidance; instead recommendations, labelling systems, and emission control become the tool of prevention. These less strict guidelines are acceptable only for discomfort and SBS etc and only if adverse health effects can be excluded e.g. because all relevant exposures are under guideline regulation as mentioned above.

Guidelines for undocumented or hypothetical causalities are presently hypothetical and more research is needed. No rational guidance except an ALARA principle can be recommended.

OPEN QUESTIONS AND RESEARCH NEEDS

There is a strong need for research on:

- How humans report symptoms and perceptions.
- On biological mechanisms involved in human responses to IAQ.
- Replacement of some of symptoms and perceptions with objective measurements.
- A quality assurance policy in IAQ evaluations based on subjective reports.
- Toxicological data for IAQ relevant compounds.
- The interactions between multiple exposures (cocktail problems).

A consensus is required on:

- Procedures for prioritizing among the most important IAQ pollutants.
- Interim procedures for estimation of substitute data until more accurate toxicological data become available.
- A set of good practices for construction, maintenance and building usage should be developed for all non industrial building types which cover all IAQ relevant risk factors (a healthy building's regulations).
- Apportionments and coordination of I/O guidelines.

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Table 1. Biological processes involved in response to poor IAQ

- The chemical sense for odorants and irritants in face, eyes, and nose
- Unspecific pain/irritative receptors in skin
- Vision
- Immune responses
- Inflammatory responses
- Body signalling systems
- Mediators
- Neural reflexes
- Interpretation in CNS

Table 2. Perceptions, symptoms, well-being, and other subjective health effects in relation to IAQ

Perceptions of body functioning, symptoms of malfunction of body functioning

- Eyes, nose, mouth, throat
- Skin
- Indicators of respiratory malfunction, asthma, allergic responses, non immune based responses, bronchial constriction
- Indicators of CNS malfunction, performance, and productivity

Environmental perceptions

- Odours, n. Olfactorius, odour masking, adaptation.
- Irritation n. Trigeminus

Processed reports or evaluations

- General well being
- Indoor Air Quality
- Sick Building Syndrome (SBS)
- Productivity and learning

Table 3. The Sick Building Syndrome (Berglund et al 1992)

A high proportion of the occupants of the building must be reacting, and the symptoms, and reactions observed belong to the following groups:

A. Acute physiological or sensory reactions
- Sensory irritation of mucous membranes or skin
- General malaise, headache, and reduced performance
- Unspecific hypersensitivity reactions, dryness of skin
- Odour or taste complaints

B. Psychosocial reactions
- Decreased productivity, increased absenteeism
- Contacts to primary health care
- Initiatives to modify the indoor environment
- Sensory irritation in eyes, nose, and throat must be dominating
- Systemic symptoms (e.g. from stomach) must be infrequent
- No obvious causality can be identified e.g. in the form of high exposure to single agents.

Essential requirements on construction products and current European standardization of emission test methods

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INTRODUCTION

Since materials in building structures, and especially those applied to surfaces in large quantities, are permanently exposed to the indoor air, it is crucial to develop an understanding to what extent they contribute to indoor air pollution. Numerous laboratory investigations have been reported concerning releases of chemical substances from interior building materials. However, several of these substances are seldom associated with complaints or building related illness. It could therefore be worthwhile to give an overview of technically well-documented case studies where chemical substances have been positively identified in the indoor air in various types of buildings. This updated overview is limited to surface materials as primary emission sources and based on an international review of case studies (1) in which most of the cases not have been published earlier. The review is covering various materials containing more or less volatile functional chemicals or their degradation products.

The overview does not include radon or formaldehyde, as the sources of these substances can be regarded as well known. Since building materials can be considered as key emission sources, the current development of standardised emission test methods within the frame of the Construction Products Directive (2) are described.

INDOOR EXPOSURES TO VOCS AND GASEOUS CONTAMINANTS FROM BUILDING MATERIALS

The highest personal exposures to VOCs are often due to high residential indoor concentrations. Indoor sources for VOCs were identified in *source attribution analyses* on the *EXPOLIS*-Helsinki data. The most influential indoor sources identified were cleaning products (Acetone, Terpenes, Aldehydes, 1-Butanol, Hexanal), building products (Octanal, Formaldehyde, Terpenes, Acetaldehyde and Benzaldehyde) and air refresheners (Limonene) (3, 4).

Also Naphthalene has been associated with bitumen, which has been used in buildings for moisture sealing. In the *EXPOLIS* study, e.g., in the sub-sample of high Naphthalene exposures in Athens, all were due to high home indoor concentrations (100 - 1000 $\mu g/m^3$), and home - but not workplace - naphthalene concentrations were also the determining factors in the Naphthalene exposures of 2/3 of the other study subjects. (5) In most of these cases, however, the source of Naphthalene was mothballs, not building materials.

The simplest approach for assessing the relative contributions of indoor sources to indoor exposures, is to look at the indoor/outdoor (I/O) concentration ratios, which have been extensively reported since the late '70's. Ratios close to 1.0 indicate outdoor air as the main contributor to indoor exposure. High ratios up to 10 and above indicate that indoor sources dominate the indoor concentrations. This approach is, however, not reliable for compounds with high reactivity or affinity to indoor surfaces. In the *EXPOLIS* study in Helsinki, the mean I/O ratio was highest for acetone (145) from human metabolism followed by formaldehyde (6, 3).

Indoor sources contribute also to bioaerosols (Nevalainen 2007) and combustion products (Jantunen 2007), which are covered in these proceedings under session 3 (see page 90) and session 5 (see page 164) respectively.

CHEMICAL EMISSIONS FROM SURFACE MATERIALS - CASE STUDIES

Various types products have been reported as primary emission sources on-site in buildings, e.g. linoleum flooring (7), polysulfide sealant (8), alkyd paint for radiators (9, 10), water repellent (11) and preparations based on coal tar such as damp proof membranes (12), wood preservatives (1). A case example of xylamite as source of indoor air pollution is presented by Prejzner et al. 2007 on page 219 of these proceedings.

The following overview of technically well-documented case studies has been limited to polymeric surface materials in direct contact with the indoor air such as textile flooring, resilient flooring and latex paint. Polymeric materials may emit residual monomers, process solvents and other functional chemicals during the use-phase.

In most of the case studies complaints had been made of the indoor climate and the substances concerned had been released over periods of several years. Bearing in mind the fact that a chemical substance that has been proven to be present in indoor air may have various origins, the indicated source of emission have been identified by an unmistakable odour or by parallel investigations, e.g. in a climate chamber. If not otherwise stated the presented case studies are selected from European countries.

Cork tiles – phenol

Plastic-laminated cork tiles laid on the floor in an office had a very strong odour, even when being unpacked and laid. Chemical measurements in the newly-built part of the office a year later indicated the presence of **phenol** (15 µg/ m^3) in the air. The cork tiles consisted of a wearing layer of transparent vinyl plastic, and the layer of cork beneath it contained phenolic resin. It was shown that phenol was released from the material, with the result that it was decided as essential to replace the floor covering. The manufacturer of the material has subsequently modified the phenol-based resin.

Vinyl flooring – dodecylbenzene

Alkyl benzenes, among them being **dodecylbenzene**, are common process solvents for plasticizers in the manufacture of **vinyl floor coverings**. This type of process

solvent has been detected in several investigations of indoor air (100-200 µg/m^3) resulting in complaints about the air quality.

Vinyl flooring – dodecene

Shortly after moving into an office building several employees complained about the air quality. It can be estimated that the concentration of **dodecene** in the premises was 40 µg/ m^3. Dodecene is used as a solvent in the manufacture of **vinyl floor covering**, but in this particular case dodecene was a decomposition product produced during manufacture of the floor covering, and originating from dodecylbenzene.

Vinyl flooring – TXIB

In several public buildings "**TXIB**" have been present in the indoor air, in the range of 100 µg/ m^3. TXIB stands for 2,2,4-trimethyl-1,3-pentadioldiisobutyrate, which is used as a process solvent in the manufacture of certain **vinyl floor coverings**. Extraction of one type of vinyl floor covering showed that it contained 7% TXIB.

Textile carpet - 4PC

4-phenylcyclohexene (4-PC) has been detected in offices in which **wall-to-wall carpets** have been laid. Four weeks after laying, the concentrations where about 15 µg/ m^3. 4-PC can be formed by the manufacture of the layer of styrene-butadiene rubber used to bind the fibres to the carpet backing. This undesirable and odorous substance is formed of the reaction of styrene and 1,3 butadiene. The presence of the substance has aroused considerable attention in the USA, where 4-PC has been found in the EPA main building in Washington D.C. and elsewhere.

Textile carpet – styrene

Ever since it was built, a strong smell was noticeable in a school (Texas, USA). The average ventilation rate was 3 air changes/h. After eight years, measurements were made, and found up to 0.9 ppm of **styrene** in the air. The styrene was found to be coming from the underside of the **wall-to-wall carpet** that covered an area of 6500 m² where the underside consisted of foamed styrene-butadiene rubber.

Vinyl flooring – EHA

Chemical measurements in a school indicated the presence of **ethylhexyl acrylate** (about 30 µg/ m^3) in a corridor outside a classroom. Emissions from samples of **vinyl floor coverings** were also investigated, including those from a sample of unused floor covering that had been kept in the school store. More than five years after delivery, this floor covering was found to be releasing several different substances, including ethylhexyl acrylate. This was most probably coming from the surface varnish layer of the floor covering. This layer of varnish is intended to be cured by UV light, and ethylhexyl acrylate can remain as a residual monomer if the layer is incompletely cured.

Latex paint – butylphtalate

In over 60 both newly-built and older renovated residential buildings it was noticed that pot plants were not growing normally and that in many cases the new shoots were completely white. It was also noted that paint on the walls was not completely dry in the room where the affected plant were kept. The type of paint could be identified in several of the cases and in all of them a certain **latex paint** from one and the same manufacturer had been used.
Test plants were kept under glass covers, together with a sample of the latex paint. It was found that the white shoots occurred only in presence of those of the manufacturer's paints that contained **dibutyl phtalate** (DBP). The concentration of DBP in the room air where the discolouration first was observed was 40 $\mu g/m^3$.

Textile carpet - ethylhexanol etc

Two years after completion as a building in (Oregon, USA), complaints were received about a smell of **2-ethylhexanol** in concentrations ranging from 34 $\mu g/m^3$ to 138 $\mu g/m^3$. **Heptanol and nonanol** were also detected in the air. A laboratory investigation showed that the alcohols came from a PVC layer on the underside of the wall-to-wall carpet. The PVC layer contained the plasticizers (phthalate esters) diethylhexyl phthalate (DEHP) and heptylphthalate and nonylphthalate to make the carpet soft and easier to lay. Surprisingly, the PVC layer also contained a considerable amount of calcium oxide, resulting in a high pH in this layer and the phthalate esters were decomposed by alkaline hydrolysis. Complete hydrolysis of a phthalate plasticizer results in its irreversible decomposition to phthalic acid and a volatile and usually odorous alcohol. The ester hydrolysis is catalysed by hydroxyl ions.

MOISTURE INDUCED CHEMICAL DEGRADATION OF MATERIALS

Besides the release of substances to the indoor air due to primary emission, damp building materials give rise to volatile substances formed during secondary reactions. Moisture induced chemical degradation of e.g. vinyl flooring, floor adhesive and self-levelling flooring compound may give rise to the formation of odorous substances. In most of cases, the substances concerned have been released over a period of several years.

Flooring materials – ethylhexanol

The phthalate plasticizer content in resilient vinyl floor coverings can amount to 30% of the weight of the material. A large number of cases have been reported especially from the Nordic countries concerning a heavy, sickly smell from vinyl floor coverings as a result of hydrolysis of phthalate plasticizers (13). Until the last years the most common phthalate plasticizer in floor coverings has been diethylhexyl phthalate (DEHP) giving rise to 2-ethylhexanol, with reported indoor concentrations ranging from about of 10-30 $\mu g/ m^3$ in various premises.

The chemical degradation of the plasticizer is strongly accelerated by the presence of alkali.

All concrete is alkaline, and the dampness of the concrete is of considerable importance for the formation of odorous alcohols. Self-levelling flooring compounds based on Portlandcement, used to smooth and level the surface of concrete floors, do not differ significantly in chemical terms from concrete. This means that damp self-levelling compound ("smoothing compound"), too, can result in hydrolysis of the plasticizer in vinyl floor covering.

The types of adhesives most commonly used for laying floor coverings are **dispersion-based**. This type of adhesive is often based on acrylate copolymers of 2-ethylhexacrylate. **2-ethylhexanol** is a feedstock material (an estering alcohol) for the manufacture of 2-ethylhexylacrylate. In the same way as plasticizers, 2-ethylhexylacrylate can be hydrolysed by damp alkaline concrete, thus reforming the alcohol, which can be smelt.

Vinyl flooring – cresol

An unpleasant smell of horse stable was noticeable in newly-built extension to a hospital. Chemical measurements indicated the presence of **phenol and p-cresol**, both in concentrations around 10 $\mu g/m^3$. Laboratory investigation of the **vinyl floor covering** in the premises also indicated emission of these substances. p-cresol occurs naturally in horses' urine, which can explain the strange smell in the premises. The floor covering contained phosphorus plasticizer which degraded as a result of the effect of damp concrete. Cresol and phenol are decomposition products from this type of plasticizer. The same type of floor covering, lying on old and drier floor/ceiling structures, exhibited no noticeable release of phenol or cresol. Vinyl floor coverings containing phosphorus plasticizer are used in premises with particularly stringent fire safety requirements.

Self-levelling flooring compound – amines

Self-levelling flooring compounds based on Portlandcement and containing casein as a levelling agent may give rise to odorous substances when laid on a damp subfloor. In alkaline conditions, casein and other proteins break down as a result of hydrolysis to form volatile amines and other substances. A breakdown substance of casein, **ortho-aminoacetophenone**, has a very unpleasant odour that can be recognised at concentrations as low as of the order of nanogram/m^3. Ortho-aminoacetophenone, is formed as a result of breakdown of the amino acid tryptophane. Portland cement-based casein-containing self-levelling compound may also release ammonia, which is capable of discolouring oak parquet laid on top of it.

CHAMBER TESTING OF PRODUCTS AS A TOOL TO IDENTIFY CHEMICAL EMISSIONS

Building materials shall meet official requirements and market demands on e.g. mechanical strength, fire resistance and be resistant against humidity variations, abrasion and the influence of microbiological and chemical degradation. In order to maintain intended properties of the material various functional chemicals are used. For hygienic, health and environmental reasons and in order to maintain the properties of the material in the construction, it is important that the functional chemicals remain bound in the material during the use-phase and are not released into the ambient

environment. Therefore, laboratory studies are needed, where the potential for release of substances under the intended use conditions are estimated. Such studies should preferably be performed under experimental conditions in test chambers corresponding to relevant exposure scenarios.

Test chambers methods are used for:

- official approval procedures
- voluntary labeling
- development of low-emitting products

The use of standardised scenarios and emission test methods are important, especially when building materials are examined in relation to official approval procedures. Test chambers are mainly used for the determination of area specific emission rate, (µg/m2 x h) at constant temperature, relative humidity and air exchange rate. Emission test chambers are designed to permit the testing of samples of various types of materials and can range in size from mL to several m^3 (14). Portable emission cells, where the test material becomes a part of the cell itself, can be used for identifying emission sources on-site in buildings (15, 16).

EC CONSTRUCTION PRODUCTS DIRECTIVE AND HEALTH PROTECTION

The Construction Products Directive (2) has been established to examine construction product safety and to break down technical barriers to trade in the European Economic Area. Construction products are defined as all products permanently incorporated in construction works, including both buildings and civil engineering works.

In addition to the traditional essential requirements such as safety in case of fire, stability and mechanical resistance, the CPD also requires that hygiene, health and environmental protection shall be considered. All essential requirements concerning the use-phase of the construction products are covered.

Essential requirements (ER) defined in CPD:
ER 1 Mechanical resistance and stability
ER 2 Safety in case of fire
ER 3 Hygiene, health and the environment (e.g. substances in indoor air)
ER 4 Safety in use
ER 5 Protection against noise
ER 6 Energy economy and heat retention

The essential requirements are intended to be incorporated in several hundred harmonised technical specifications [hTS] such as European standards [EN] and technical approvals [ETA] for construction products. After a transitional period, construction products may only be placed on the market if they conform to the specifications and display the CE mark.

The aim of the environmental requirement (ER3) is to protect the health of occupants and neighbours and the immediate environment, which essentially comprises the e.g. indoor air. The implementation of the environmental criteria requires test methods especially designed for the determination of the release of dangerous substances from construction products. The European Commission has therefore given the task to CEN to develop standardised test methods for the release of dangerous substances (17).

CURRENT STANDARDISATION OF EMISSION TEST METHODS

Since 2006 a Technical Committee (18) at CEN is responsible for developing environmental test methods under the CPD. The end objective is the elaboration of European standards [EN] for environmental properties. These standards will be supporting standards which are referred to in the forthcoming revised technical specifications for construction products. The methods shall take into account the intended conditions of use of the products (release scenarios) and address emission to indoor air, release to soil, surface water and ground water.

The environmental test methods shall preferably be applicable to all or most product groups ("horizontal methods") which may release substances under a specific release scenario. For the release into indoor air horizontal test methods applicable to several surface materials have been developed (19).

Within the framework of the REACH Regulation (20) e.g. the release of chemical substances from articles and preparations has to be assessed. Construction products are not exempted from this obligation. The horizontal release methods developed within the framework of CPD will therefore support the intention of REACH with respect of construction products.

CONCLUSIONS

Emission from construction products to the indoor air have been reported for a wide range of substances including those formed during secondary reactions causing complaint of irritation and odour. Emission testing of construction products is therefore an important tool to identify chemical emissions that could cause problems with the indoor air quality.

The Construction Products Directive [89/106/EEC] is now addressing emission of dangerous substances including to indoor air and standardised test methods are under development by CEN. The REACH regulation [EC1907/2006] being implemented from June 2007 will also impact on the control of emissions from articles including construction and consumer products.

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Session 3 Asthma and allergy

Asthma and allergies: The role of the home environment

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BACKGROUND

The incidence of asthma and allergy has increased throughout the developed world over the past forty years (1). The incidence is much higher for children than adults. From being a relatively uncommon disease, a few decades ago, allergies today, in many regions, are affecting a large part of the population. The European Allergy White Paper (1997) noted that with the exception of AIDS, only few diseases, besides allergies, have increased two- or three-fold within a short time (2). Allergic diseases are supposed to be caused by a complex interaction between genetic and environmental exposures. The temporal trends in allergy prevalence, the differences in the risk of allergy between urban and rural populations of the same ethnicity and the short time period for which the prevalence of allergic diseases have increased, indicate that changes in environmental exposures rather than genetic factors are the most likely explanation for the increase (3, 4).

But, what changes in environmental exposures are important for the increase in allergies?

In the search of causative factors it's important to note that small children are particularly at risk. Thus the exposure during pregnancy and first years of life seems more important than exposure later in life. Children have a higher metabolism and faster respiratory rate compared to adults resulting in higher intake of food, drink and air per unit of body volume, i.e. higher dose which is further enforced by their hand-to-mouth behaviour. The exposure (in mass) during pregnancy is defined by the exposure of the mother, while the exposure of babies mainly consist of indoor air (around 80%), and food, mainly breastmilk. In developed countries more than 50 % of the total exposure (in mass), during a 70 year life consists of air in the home, while outdoor air, food and liquids, and industrial air stands for around 7% each. The rest of the exposure is air in schools, day care, offices, and during travelling.

This review is based on multidisciplinary state-of-art reviews of the scientific literature on associations between indoor exposures and asthma and allergies (6, 7, 8, 9), and on results from two ongoing studies in Sweden and Bulgaria, DBH, and ALLHOME. The studies in Sweden and Bulgaria are basically identical, starting with a cross-sectional questionnaire study on small children, allergic manifestations and home environmental factors. The second step has been nested case-control studies including clinical examinations, inspections and environmental measurements.

CAUSES OF THE INCREASE IN ASTHMA / ALLERGIES

Even if genetic change is not the cause of the increase, **genetic predisposition** is an important factor for the risk of getting asthma and allergies. In a questionnaire study of 1,325 children, 7 years of age, Kjellman (10) observed the highest prevalence of atopic disease among children of parents with an identical type of atopic disease (with 72% risk), and the lowest among children of parents without an atopic disease (10% risk). Small boys have a higher prevalence of atopic diseases than small girls, but this changes during puberty.

As allergy means that a person reacts to an **allergen** (e.g. from cat, dog, pollen, mite, mould, cockroaches, specific food etc), the most simple explanation for the increase should be that we are exposed to more allergens today. Even if there are indications of increased allergen levels from mites and moulds (due to tighter, less ventilated, and thus more humid, buildings in northern climate), and, perhaps more pet contacts, there is no scientific data showing that this is an important factor behind the increase in allergies, worldwide.

If the allergen levels can not explain the increase, there must be other environmental changes that are the cause. Either our immune defence is changed (due to e.g. lack of microbial exposure), so that we react to harmless proteins, allergens, more than before (the hygiene hypothesis), or some other exposure (adjuvant factors) makes us more vulnerable (mechanisms not known) for exposure to allergens.

The **hygiene hypothesis** involving factors like family size and number of early infections is, by far, the most popular, discussed and studied explanation for the rising trends in allergy and asthma. It (11) suggests that exposure to infections early in life influences the development of a child's immune system along a "non allergic" pathway, leading to a reduced risk of asthma and other allergic diseases. However, despite numerous studies, (including up to 100 published state-of-the-art reviews), the area is controversial. E.g. the hypothesis does not fit for USA, where the allergy prevalence is very high among children in inner cities (Harlem.), where an increased hygiene is most certainly not a problem The summary of the state-of-the art reviews is that there is very little, if any, consistent evidence for this hypothesis.

If changed hygiene can not explain the increased morbidity, what about new environmental exposures?

Some **outdoor** exposures, such as ozone, nitrogen dioxide, sulphur oxides and particulate matter are known to exacerbate asthma (12, 13, 14, 15, 16). In Bulgaria, Lubomirova et al. (17) reported a higher prevalence of respiratory and allergic diseases among children exposed to air pollution (organic solvents) from refinery and petrochemical plant compared to control children. Outdoor air also constitutes a main source of exposure to air-borne allergens, such as ***pollen*** from plants and ***moulds***. However the role of outdoor air pollution in causing asthma remains controversial (ATS, 2000 (18)). E.g. the prevalence of allergic sensation was three times higher in low polluted Sundsvall (Sweden) than in Konin (Poland), where the levels of common industrial pollutants, SO_2 and smoke particles were much higher (19). In a review of the evidence regarding the link between environmental exposures and the prevalence of asthma, Etzel (3) concluded that outdoor air exposures are not likely to cause the increase in asthma prevalence.

Diet (20), lack of **breastfeeding** (21), **less physical activity** (22) and **obesity** (23) are factors discussed as possible causes behind the increase in asthma/allergies. It is shown that breast-feeding has a protective effect. Otherwise there is no good scientific data behind these ideas.

INDOOR AIR

The air indoor comes from the air outdoor. Outdoor air contains pollutants that are present due to e.g. traffic, soil, vegetation (pollens) and industries. Inside the room, the air receives further contaminations from people, animals, furniture, furnishings and building materials, cooking, vacuum cleaning, combustion processes and smoking as well as from cleaning products, microbial growth, etc.

In two multidisciplinary reviews on moisture related problems in buildings (dampness) and associated health effects it was concluded that **"dampness**" do increase the risk for several health effects such as asthma and allergies, sick building syndrome and airway infections (7, 8). Identified health relevant moisture problems were e.g. visible mould and damp spots, detached or miss-colored flooring materials, condensation on inside of window panes, flooding and bad odor. However, the literature did not show what dampness related exposures that were responsible for the health effects

The results from the DBH and ALLHOME studies are well in line with earlier major studies on dampness and health. In both studies strong and consistent associations were found between moisture related problems indoor and symptoms among children. The risk for symptoms was more than doubled for children living in a home with self reported "dampness" (24, 25).

In Sweden **visible mould or damp spots** were reported from the index child bedroom in 1.4% of homes compared to 35% in Bulgaria. In general no association was found in Sweden between health effects and type of mould, a mouldy odour in the room, glucan, ergosterol, and mVOC. However an association between symptoms and Penicillium in dust, and a strong dose-response relationship between rhinitis and

eczema and inspectors perceptions of a mouldy odour along the skirting board (28). Results from Bulgaria are pending analyses.

The Nordic interdisciplinary review, NORDPET (5), concluded that **pet** exposure in infancy increases the risk for sensitization (OR 1.0-1.5). Pet keeping as a risk factor for asthma and wheezing in children was also reported in the review by Apelberg et al. (27). However, in a study by Lau et al. (28) no relation was found between early indoor pet allergen exposure and prevalence of asthma, wheeze, and bronchial hyper-responsiveness. In a number of studies (28, 29, 30) an inverse relationship between early pet exposure and allergic diseases later in life has been found, suggesting a "protective" effect of pet keeping. Such inverse associations between current or early life pet ownership and symptoms are, however, mainly due to avoidance behaviour in the families (31, 32), i.e. a "healthy pet keeping" effect. In Sweden where a number of information campaigns to the general public, about risk factors for asthma and allergies, there is a strong "protective effect" of pet exposure, while in Bulgaria with no such campaigns, there is no "protection" from pet keeping. Meaning, in countries with a good knowledge about the risk for allergies related with pets, families with allergies tend to get rid of pets, while that is not the case in countries without such public knowledge.

Reduced ventilation rates means increased concentrations of building related pollutants, including moisture. Only few studies on the association between health effects and ventilation rates in homes have been reported (e.g. 36, 37, 38). Oie et al. (33) found that the risk of bronchial obstruction not directly was associated with the ventilation rate in the homes, but that the risk associated with e.g. dampness was greatly increased in homes with a low ventilation rate. Emenius et al. (34) reported that air change rate and type of ventilation system did not affect the risk of recurrent wheezing. However, in a study by Bornehag et al. (35) case children had significantly lower ventilation rates at home than controls and a dose-response relationship was indicated. An important difference between these studies are that the ventilation rate in the study by Emenius et al. are reported to be about the double of that in the study by Bornehag et al. The literature on HDM (36, 37) indicates that inadequate ventilation in homes in cold climate constitutes a major risk factor for infestation of mites and subsequent health effects.

It is well established that ventilation rates in homes in northern Europe have been reduced during the last decades, as a result of energy conservation measures. About 60% of the multi-family houses and about 80% of the single-family houses in Sweden (38) and 36% of all residences in Oslo, Norway (33) had lower than 0.5 air changes per hour.

Indoor smoke from solid fuels (38) and environmental tobacco smoke (39) are significant triggers for asthma symptoms and attacks. The situation with regard to smoking is totally different between Sweden and Bulgaria. In Sweden smoking among pregnant women is rare, and "no one" smokes in a room with a baby, while in Bulgaria 31% of the pregnant women were smoking, and 73% of the children had at least one family member smoking. Smoking is a risk factor for asthma in both countries, but much more pronounced in Bulgaria. Especially a mother smoking during pregnancy, and first year of life of the child were significantly associated with most of the health effects among the children. Adverse effects of both pre- and

postnatal parental smoking on children's respiratory health were recently confirmed by Pattenden et al. (40). Asthma was most strongly associated with maternal smoking during pregnancy, but postnatal exposure showed independent associations with a range of other respiratory symptoms.

CHEMICAL EXPOSURES INDOORS

The home environment has changed considerably during the past 3-5 decades because of the introduction of new building technology, as well as new building materials. Some new surface materials are emitters of chemical compounds with potential allergic properties.

Commonly measured **VOCs** have not been strongly and consistently associated with asthma/allergies. There is, however, some epidemiological evidence for associations between **phthalates** or plasticized products such as PVC and allergic symptoms in the airways (e.g. asthma), nose and skin. Jaakkola et al. (41) found that the total area of PVC surface materials in homes was associated with development of bronchial obstruction in small children in Norway. In another study from Finland lower respiratory tract symptoms, like persistent wheezing, cough and phlegm in children, were associated with the presence of plastic wall materials, while upper respiratory tract symptoms were not (42). Also the relative risk estimated for pneumonia, bronchitis and otitis media were slightly increased in the presence of plastic wall materials. In the first phase of the Swedish DBH-study it was found that PVC as flooring material in combination with moisture problems in the floors was associated with e.g. asthma among children aged 1-6 years (48), the same is valid for Bulgaria. Furthermore, in the second phase of the DBH-study a strong dose-response relationship was found between asthma and di(2-ethyl-hexyl)-phthalate (DEHP) concentration in indoor dust and between eczema and rhinitis and butyl-benzyl-phthalate (BBzP) (43). Oie et al. (1997) (33) elaborated possible mechanisms of respiratory effects by inhalation exposure and concluded that deposition of DEHP in the lungs may increase the risk of inflammation in the airways which is a characteristic feature of asthma. In a population-based incident case-control study among adults (21-63 years), Jaakkola et al (2006) (45) reported that the use of self leveling compounds at home during the past 12 months was a determinant of onset of asthma. They also found that the risk of asthma was significantly related to the presence of plastic wall materials at work. Reviewing existing literature, Nielsen et al (2006) supported the hypothesis that some phthalates may act as adjuvants (46). An adjuvant effect of phthalates for sensitization to common allergens was tested by Glue et al. (2005) (47). None of the phthalates tested was found to induce histamine release per se, however, higher histamine release was observed when the cells first were treated with phthalates and then exposed for allergen. Lee et al. (48) reported that DEHP and DINP (di-isononyl phthalate) enhance allergic responses by enhancement of IL-4 production in CD4+ T cells via stimulation of NF-AT-binding activity which is in line with the discussion in the paper by Chalubinski et al. (2006) (49).

The sources of phthalate esters indoor are ubiquitous plasticized polyvinyl chloride (PVC) materials (floor and wall covering materials), shower curtains, adhesives, synthetic leather, toys, cosmetics and very many other consumer products.

DISCUSSION AND CONCLUSIONS

The increase in asthma/allergies has been dramatic all over the world, in a short time period (decades). The causes must be environmental, as the time period is to short for major **genetic changes**. An easy explanation would be that there has been a major increase in exposure to **allergens**. We are still becoming sensitized to birch, cat, dog, mites etc, and there is no scientific evidence that such sources have increased drastically the last decades. So the cause should be searched for in the way we are reacting more often to pollens, cat dander, mites today.
The most common explanation is the **"hygiene hypothesis".** Our environment is too clean; we are not exposed to "dirt", including microbes, occupying the immune defense. Instead the immune system reacts to harmless proteins, allergens, inducing asthma/allergies. In spite of two decades of research on this hypothesis, no consistent positive confirmation has been found, rather the contrary. Probably many of the findings can be explained by selection bias. In families that have a member that gets sick when exposed it is natural to avoid pets, resulting in a seemingly "protective" effect of pet-keeping in cross-sectional or cohort studies ("the healthy pet keeping effect"). This effect is obvious in a country like Sweden, with a number of national campaigns, informing everyone about risk factors for allergies (including pets), but not existing in Bulgaria (no campaigns, pets is a real risk factor!). The same selection bias could be found among farmers, and other groups that are used as evidence of the "hygiene hypothesis". In the same field of research we have the protective effect of endotoxin. As endotoxin is strongly associated with pet keeping, we have the same strong selection bias involved. The idea that it is cleaner today (more hygienic) in homes, schools than 50 years ago, is also against common experience. We have a society were most are working outside the home. It is reasonable to assume that homes are dirtier today. For schools, day care centers, offices the development has been the same. There is in most buildings less cleaning today than was usual decades ago, as cleaning is a major cost for building owners (much more important than energy use). More frequent early infections should prevent asthma/allergies, according to the "hygiene hypothesis", but in the Swedish study (DBH), it's the opposite! The more early infections (day care attendance), the more asthma/allergies.

If not the hygiene hypothesis, what environmental change could be responsible for the increase in atopic morbidity? :
Outdoor air, Yes for exacerbations; No for incidence!
Food, in spite of two decades of research, No! (part of the hygiene hypothesis)
Indoor air:

- ETS, Yes!
- Ventilation rate, possibly!
- Dampness, Yes!
- Mould, maybe?
- Phthalates, maybe?
- Other new chemical exposures, possibly

There are most certainly other chemical compounds, new in our environments, which may be as important as phthalates. This is where new science should start.

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Bio-aerosols as exposure agents in indoor environments in relation to asthma and allergy

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INTRODUCTION

Asthma, asthmatic symptoms and allergic sensitization are linked with a number of indoor exposures, such as VOCs, phthalates, tobacco smoke and biological agents. This paper focuses solely on the biological exposures. Exposures to allergens, microbial agents and other biological particles are risk factors to these health effects, but the exact causal connections or the mechanisms underlying the symptoms are still not well understood. Among the open questions are e.g. the importance of genetic-environmental interactions in the development of allergy, pathophysiological mechanisms of the development of asthma and the role of various exposing agents as causal or adjuvant factors of these diseases.

In general, any foreign protein may be a potentially sensitizing agent but not all agents are equally important as indoor exposures. Agents that have major importance as indoor allergens are listed below. In addition, many other biological molecules, such as lipopolysaccharide (endotoxin) have immunological potential, although they do not sensitize humans via IgE-mediated allergic pathway. The different types of key exposure agents of the indoor environment that may have a role in development of allergy and asthma are listed in the following.

Microbial agents of indoor environments:

- Endotoxin of Gram negative bacteria
- 1,3-beta-glucan of fungi
- Fungal spores and fragments
- Bacterial cells, spores and fragments
- Microbial metabolites, e.g. fungal and bacterial toxins and MVOC

Allergens of the indoor environments:

- House dust mites
- Storage mites
- Cockroaches, rodents, other pests
- Pets: dog, cat, rabbit, mouse, rat
- Fungal allergens

MICROBES IN THE INDOOR ENVIRONMENT

This paper deals with non-infectious microbes, the major groups of which are fungi and bacteria. Fungi contain allergens, and individuals may become sensitized to them. Among the fungal allergens that have been isolated are water-soluble glycoproteins, some of which are enzymes; some of them may also be high-molecular weight carbohydrates (1). Fungi and bacteria also contain other immunologically potential material that may have a role in the development of asthma or its symptoms. The sources from which fungi and bacteria derive may have importance in their health effects. Therefore, a short overview of their major sources is presented in the following.

The main source of fungi in the indoor environment is outdoor air. Outdoor fungi have remarkable diurnal and seasonal variations (2), the concentrations being highest in summer and fall, and lowest during the cold seasons. In climates with snow cover, the wintertime outdoor concentrations of fungi may be very low (3). Indoor concentrations follow in the seasonal fluctuations of outdoor air (4). As for the species profiles, outdoor air mycobiota is well represented in the indoor air but there are also indoor sources of fungi with somewhat different species profiles.

Among the common fungal genera present in indoor air are *Penicillium, Aspergillus, Cladosporium* and yeasts. The aerodynamic size of fungal spores is usually 2-10µm, although much larger spores also exist. Thus, fungal spores are retained relatively well in the filtering systems of the building ventilation, but in buildings with no filtering system, the spores enter indoors freely through open windows and doors. Those spores that enter the indoor environment, settle relatively easily on surfaces. Fungal spores are removed from the indoor environments by cleaning and by exhaust ventilation.

There also are indoor sources for fungi. Several everyday activities actually act as sources of indoor fungi. For example, handling firewood has been shown to elevate the fungal levels remarkably for several hours. Other such normal sources are handling of vegetables, fruit, houseplants and other organic material (5). Fungi are also carried indoors on people's clothing and on dog and cat fur.

All these sources of fungi are regarded as "normal" everyday phenomena. Fungal spores and particles from such normal sources may accumulate into the indoor environment and lead to elevated exposures. For example, settled house dust may contain large amounts of fungi and once the settled dust is resuspended, also fungal material may become airborne. The control of the fungal material from "normal" sources is limited to the filtering of intake air and proper cleaning practices.

For bacteria, humans are important sources in indoor environments. This has been shown using both cultural methods and by measurements of muramic acid, a chemical marker of bacterial biomass, the concentration of which has been shown to follow the number of pupils in a classroom (6). The majority of indoor bacteria are gram positive cocci originating from the human skin, but also Gram negative species are involved. Apart from the infectious agents that are not the topic of this paper, the majority of the skin bacteria belong to the normal human bacterial flora. Apparently, they do not have importance as allergens or agents involved in the development of allergy or asthma.

On the contrary, the present understanding is that exposure to bacteria, especially endotoxin and fungi in early childhood may be protective from allergy and asthma (7).

MICROBIAL EXPOSURES RELATED WITH DAMPNESS AND MOISTURE

A quite different source of fungi and bacteria is any moist or wet site where fungal or bacterial spores may germinate and start to grow, or where non-sporing bacteria and yeasts are able to proliferate.

In general, building structures and indoor environments should be dry, without any mechanism that would cause constant or regular wetting of surfaces and structures. However, this is often not the case, and various problems with dampness, moisture and water damage are prevalent in all climates and building types. Such problems are strongly linked with respiratory and other adverse health effects (8, 9, 10). However, the causal agents of the health effects are not yet well understood.

As soon as any material gets wet, microbial growth starts. Microbial spores and cells are present everywhere, and therefore the only factor regulating the microbial growth is availability of water. In general, the microbial types that start their growth on building or finishing materials, originate from outdoor air and other natural sources. However, the substrate on which microbial growth takes place, has a crucial role on the microbial profile that will develop on it. Both the moisture content, availability of nutrients, pH and other characteristics of the material have importance. For example, the species profile that grows on moist wood is different from the species that derive on gypsum board (11).

Among the fungal species that grow typically on moist building materials but are not part of the "normal" microbial content of indoor environments, are *Aspergillus versicolor*, *Aspergillus fumigatus, Stachybotrys chartarum, Acremonium, Aureobasidium, Chaetomium, Phialophora* and *Trichoderma*. Among the bacteria that contaminate moist building materials are *Streptomyces* and *Mycobacterium*. In fact, it is a whole ecosystem that develops on moist building materials, including not only many species of fungi and bacteria but also protozoa such as amoebae (12). Amoebae may allow the growth of *Chlamydia* and other bacteria that do not proliferate alone in environmental habitats.

The factor that makes the difference between growing microbes and those transported in and out by "normal " phenomena, is that growing microbes produce additional pollutants into the indoor air. They produce spores and small fragments of microbial material (13), and secondary metabolites that may be either volatile compounds, often with characteristic smell, or non-volatile compounds many of which are characterized as microbial toxins. Fungi and bacteria isolated from houses with moisture problems have shown both cytotoxic and immunotoxic characteristics (14, 15). Thus, having a source of growing microbes in the building structures or indoor environment means quite different exposure situation.

The evidence between dampness and “mold” and risk of allergy and asthma is strong, but the causal links are yet to be documented. A limiting factor in the health studies has been the lack of sensitive enough methods to characterize the microbial exposures. Currently, development of DNA based and other non-cultural methods will provide better tools to profoundly understand the exposures.

Even if the control of microbial exposures may be difficult due to their complex nature, the solution may be the control of their most important determinant, i.e., dampness and moisture. That can be done without numerical guideline values, based on down-to-earth advice and guidance.

INDOOR ALLERGENS

House dust mites are the best known indoor allergens that have a direct link to allergy and asthma. Mites belong to acarids, and the most common species are *Dermatophagoides pteronyssinus*, *D. farinae* and *Euroglyphus maynei*. Also storage mites are important allergens.

Mites live on skin scales and other organic debris and they are strictly dependent on the relative humidity of air. Mites are very common in humid climates and more rare in dry and cold conditions. For example, mite antigens were found in 38% of nearly 800 homes studied in Ohio, USA (16). Mites typically live in mattresses and padded furniture where they can survive long periods of time even if the ambient humidity has decreased. Mites are present in indoor environments wherever the conditions are favourable, i.e., relative humidity >55%.

Dust mite sensitivity is strongly associated with asthma as already reported in the late 1960ies. In some countries the mite sensitization among the asthmatics is so common that other indoor risk factors may not be very important. Control measures have been developed, e.g., mattress covers that would decrease the allergen exposure, but they have not necessarily been successful. In large cities, cockroach debris or rodent urine may be common allergens in house dust (1). The occurrence of these pests is closely related to housing conditions.

Allergen avoidance has long been part of the practice of asthma and allergy medication and health care and associated patient advice and training. Apart from pet allergens that can be avoided by not having pets, it may be beyond the possibilities of indoor air quality regulation to solve the many problems with allergen exposures.

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Moisture as a source of indoor air contamination

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RATIONALE

Dampness / moisture accumulation into building structures or structural components, or on the surfaces of building materials, may lead to physical, biological or chemical deterioration of building materials. Subsequent damage and microbial or chemical contamination of the building may decrease the indoor air quality of the building. Dampness/moisture damage also poses a serious risk to the performance of the building structures (1). In epidemiological population studies, dampness/moisture damage has been associated with a number of health effects including respiratory symptoms and diseases and other symptoms (2, 3).

While exposure to microbial and/or chemical pollution and health outcomes are consequences, the common denominators for them are different forms of undesired moisture behaviour. The health effects associated with dampness/moisture damage seem to be consistent in different climates and geographical regions (4). However, the technical causes of dampness/moisture damage are often closely connected to the climate. The prevention and control of moisture problems should be addressed in early phases of building design and construction, and in the sustained maintenance of buildings.

This report aims to give an overview of moisture as a source of indoor air contamination, which is related to the health topic Asthma and allergy. Included in each chapter, the author(s) list most important (open) questions, which should be addressed in the discussions for this topic.

MOISTURE SOURCES

Moisture can migrate into and inside a building in several ways, depending on its state (vapour, liquid or ice/snow) (5). Moisture transfer is fundamentally based on a migration of water molecules migration in response to forces acting on them, including intermolecular forces, vapour pressure, and gravity (6).

In general, three major moisture source categories can be identified: the outdoors (e.g. air humidity, precipitation, moisture in the ground), the indoors (e.g. humans, water use), and wet construction materials. Common sources of moisture damage are, for example, roof leakage, flooding, leaking services (e.g. burst pipes, defective pipe joints), spillage (e.g. cleaning and washing activities), and construction moisture (e.g. fresh concrete structures, plaster, mortar, timber, etc.).

Moisture conditions in buildings and their surroundings are in a continuously fluctuating stage. Factors affecting moisture transfer include temperature, air/vapour pressure, and moisture content of the surrounding air and materials. The mechanisms of how moisture affects building structures and causes different forms of damage are generally known. As a consequence, risk of dampness/moisture damage can be estimated using meteorological data on moisture related parameters in outdoor air, information on the regional parameters, information on water production and activity indoors, information on the construction and structural design, and information on the performance of the building and its systems. It is far less understood how dampness/moisture damage alters the indoor air quality that is critical from a health point of view.

- How dampness/moisture damage alters the indoor air quality that is critical from a health point of view?
- Are the causal links between dampness/moisture damage related exposures and health sufficiently well known?

HOUSING CHARACTERISTICS ASSOCIATED WITH DAMPNESS/MOISTURE DAMAGE

Several housing characteristics have been associated with dampness/moisture damage. These include age, size and type of building, type of foundation, building frame material, amount of thermal insulation, lack of central heating, use of natural ventilation or poor ventilation, use of humidifiers, indoor RH, and overcrowding (4, 7-12).

Ventilation is a focal housing characteristic that has an important role in building moisture dynamics but is also associated with occupant health in general, especially at low air exchange rate levels (13). Ventilation may also play a crucial role affecting the concentrations of indoor air pollutants (14). If there are no indoor sources of pollutants, mechanical exhaust and supply ventilation systems with adequate air filtration can reduce concentrations of pollutants entering or infiltrating into a residence (15). On the other hand, inadequate outdoor air supply may associate with elevated concentrations.

- How could information about housing characteristics associated with dampness/moisture damage be translated into moisture protective policies?
- What is the role of ventilation in control of dampness/moisture damage?

OBSERVATIONS AND CHARACTERISTICS OF DAMPNESS/MOISTURE DAMAGE

Occurrence and characteristics of dampness/moisture damage can be estimated various ways. Most commonly, the estimates rely on observations made by building occupants or by independent inspectors. Depending mainly on material properties and

the stress to which material is exposed to, there are many typical signs indicating dampness/moisture damage. These signs include condensation on cold surfaces such as window panes, or signs of staining, discoloration, peeling, blistering, shrinkage, or expansion of a building material, or decay from micro-organisms. However, it is also important to notice that dampness/moisture damage is often hidden and can not be observed without dismantling structures.

There are only a few studies comparing occupant reports on moisture damage and on-site building investigations performed by trained inspectors, and even fewer studies assessing the variation between different inspectors' observations. Dharmage et al. (16) concluded that the data collected by questionnaires were accurate as compared to independent inspector's reports. Douwes et al. (17) suggested that occupants' reports were more reliable in estimating "dampness" than inspectors' reports. A conflicting study by Williamson et al. (18) reported occupants having a tendency to underestimate "dampness". Nevalainen et al. (19) concluded the same, suggesting one explanation to be a result of a trained eye of the inspectors to rate their observations together with their knowledge of what represents critical problems. Haverinen-Shaughnessy et al. (20) studied moisture damage observations made by both occupants and independent inspectors and concluded that the inspectors observed more damage sites than the occupants, and the overall agreement between the inspector and the occupants was poor, whereas the agreement between the two inspectors was higher.

Regardless of the observer, dampness/moisture damage observations can be further evaluated based on several characteristics. From the exposure point of view, according to two Finnish studies, characteristics of importance may include location of damage with respect to building occupants, duration of damage, type of damage observation (i.e. signs of visible mould, odours or other biological or chemical processes that may lead to indoor air pollution), and damaged material (21- 23).

- What is the measure of dampness / moisture damage and how it is verified?
- What are the most important characteristics of dampness / moisture damage with respect to occupant health?

PREVALENCE OF DAMPNESS/MOISTURE DAMAGE

Survey based prevalence estimates of dampness/moisture damage vary from approximately 2 to 85% depending on the study, climate and definition used; a large part of the variation may also be related to the method used in the estimation. Therefore, it is difficult to make European level estimates on the proportion of the population that may be adversely affected by dampness/moisture damage. For example, Eurostat (24) defines damp as "rot in the house or damp or leaky roof" and reports percentage of total population exposed to these types of problems in 13 countries varying between 4.2% (Finland) to 35.7% (Portugal) in 2001. However, the definition as such seems to exclude many types of dampness and moisture problems, which may be a reason why, for example, several cross-sectional studies conducted in Finland have reported much higher prevalence values (19, 21).

European community respiratory health survey investigated associations between housing characteristics related to dampness, mould exposure and house dust mite levels and asthma in 38 study centres in 18 countries (4). Centres were located both in Europe (14 countries), and outside Europe (four countries). The information on housing characteristics was obtained in an interview, and included information on water damage, presence of water collecting on the basement floor, and mould or mildew on any surface inside the home. During the year prior to the interview water damage was observed in 12.4% (range 4-32%), water on basement floor in 2.2% (0-16%) and mould or mildew in 22.1% (5-56%) of the dwellings.

LARES survey was undertaken in eight European cities in 2002 and 2003, consisting of data on roughly 400 dwellings from each city (25). According to the dwelling inspections conducted by trained surveyors, visible mould growth was detected in at least one room of almost 25% of all visited dwellings. Country specific data was not reported in the preliminary overview of LARES findings. Findings related to other dampness/moisture related variables (including smells of dampness and signs of condensation) were not included in the report.

Many global phenomena such as climate change and increase in energy consumption have been recognized worldwide. Increase in prevalence and incidence of asthma and other diseases possibly attributed by environmental factors has come under intensive public concern. All of these issues have links to dampness and indoor air quality. Needs for data improvements relate especially to the prevalence of dampness/moisture damage (population exposed), and causal links between exposures and health. Once the prevalence of dampness/moisture damage and the causal links are sufficiently well know, risk assessment can be conducted and correct policy actions can be taken to protect the public and their housing quality attributable to health. Current knowledge is sufficient to give practical advice for building owners and occupants for taking corrective and preventive actions in order to avoid dampness/moisture damage in buildings.

- What is the prevalence of dampness /moisture damage (population exposed) within EU?

REMEDIATION OF MOISTURE DAMAGED BUILDINGS

Dampness/moisture damage of buildings does not have a homogenous appearance but each building needs to be examined individually. Although there are uniform phenomena seen in the microbial contamination of the indoor environment and health effects of the occupants, the original causes of moisture problems and the possibilities to eliminate them vary.

One should emphasize the importance of continuous maintenance as the best practice to ensure good performance of buildings throughout their life span. Dampness/moisture damage should always be addressed in a timely manner, because extended duration may lead to more damaged buildings and create additional stress among the building occupants. Intervention studies have shown positive effects after remediation, or cessation of exposure, on occupants' health (26-28).

Careful remediation process includes solving the cause(s) of the damage, removing contaminated materials, good quality reconstruction, and follow-up measures (29). Attention should also be paid to protecting construction workers and building occupants against contamination released during the work. Assessing the success of remediation of moisture-damaged buildings focuses on these same issues, and it can be done utilizing various methods (30). During the whole remediation process, it is good to keep an open mind toward problems that may have remained unsolved, and return back to monitor and re-evaluate the success of the remediation as necessary.

One of the final decisions to be made is related to re-occupancy of the remediated facilities. On a rare occasion, either total or partial re-occupancy of a building may not be achieved. Such outcome could become realistic, if the remediation costs would exceed the value of the building and/or total reconstruction. It is also possible that a small portion of occupants become sensitized to building contamination, and in such cases sufficient levels of cleanliness may not be achieved at a reasonable cost. Depending on the number of such occupants, appointing new working / living space may be the most feasible choice.

- Can all damp / moisture damaged buildings be remediated cost-effectively (what is the measure of cost-effectiveness)?
- How is the success of remediation assessed?

PREVENTION OF DAMPNESS / MOISTURE DAMAGE

By estimating ambient temperature, pressure and moisture conditions, and moisture sources, loads and mechanisms of action, buildings can be designed and maintained so that under normal conditions, moisture will not cause damage (31).

In the design phase all equipment, components, and structure types chosen should be checked to be moisture safe during both construction and use. The conditions related to building site and the planned use of the building (level of usage, planned activities) should be taken into account. Basis for moisture control is provided by calculations, specifications or other information that also point out important details to be checked during construction. They include estimation of moisture loads the building is subjected to, technical properties required for materials and products, and design of structures and details (including their performance and risk characteristics and how the risks can be minimized). They take into account building tolerances, deformation, and duration of materials and structural components under the estimated moisture loads. Sufficient quality control measures should also be included (e.g. how and when moisture content of materials should be measured).
The design will establish instructions and routines for materials and components handling during transportation and of the building site, establishing a basis for control of construction moisture. It will also establish instructions on how the building should be used and maintained in order to avoid moisture damage. Special consideration is required for new, not tested solutions.

Following design phase, good construction site planning has a crucial effect on the control of moisture. Moisture control plans and actions should be included in the quality control of the construction site. All important checkpoints (e.g. based on estimated risks) should be defined by clear description of test methods, accepted values, tolerances, etc., followed by real time documentation. These checkpoints may include follow-up of drying out time, handling of materials and equipment, protection of structures and materials, and responsibilities of different parties and individuals in carrying out the plans and documenting the results.

A perquisite for successful moisture control is a realistic and sufficiently detailed timetable which allows keeping construction on schedule. Based on careful calculations and measurements, this timetable aims to promote construction moisture drying out as fast as possible by means of ventilating and heating when needed. For example, in a wet/humid climate, the most effective protection against rain is installation of the first layer of roofing, gutters, and downspouts. In cold climate, effective drying out is achieved by heating, which is possible when the frame of the building is closed (inc. windows and doors). These phases need special attention in scheduling and work planning in order to achieve as fast as possible progress. Unnecessary wetting of building materials during the storage and construction phases should be avoided.

- Is moisture control taken sufficiently into building design and construction process?
- Does moisture control planning require special expertise that is not currently fulfilled in the standard building design / construction operations?

MOISTURE CONTROL POLICIES

In order to minimize risk of dampness/moisture damage, long or frequent periods of high humidity (>80%) should not occur in any part of the building structures. Following moisture control policies and preventive measures that are generally incorporated in the building code in Finland will help to reduce risks (32) (however, they may not invariably prevent dampness/moisture damage in buildings):

Buildings shall be designed, constructed and maintained in such a way that moisture accumulation on building surfaces or inside the structures does not cause health risks for the building occupants or the occupants of the neighbouring buildings. Moisture control plans shall include sufficient maintenance procedures for all building structures and components.

Building structures and HVAC systems shall be built in such a way that vapour, water or snow from either indoor or outdoor sources can not harmfully get into the structures or indoor spaces. If necessary, structures and building materials should be capable of drying without moisture causing harm, or the drying should be ensured by a specifically designed method.

Building envelope should be sufficiently air tight so that it is possible to prevent uncontrolled air flows and control air pressure within the building as compared to

outdoors. All spaces within the building that are meant to be ventilated should be designed and equipped so that the ventilation air flows through the entire space.

All soil, organic material and debris that can rot or grow mould or otherwise harmfully deteriorate should be removed from the building site before construction. Building elevations should be designed so that surface and ground water can be controlled. Rising damp from the ground should be controlled by sufficient drainage, capillary layers, and/or water proofing.
All spaces, equipment, structures and materials susceptible to and/or severely or repeatable exposed to moisture should be built so that they can be inspected and maintained easily and cost-effectively during their designed life-span. Building materials susceptible to moisture should be protected. Structures under water pressure should be protected with sufficient waterproofing. Building materials exposed to water should be able sustain their qualities. Harmful seepage of water into or through the structures should be stopped. Harmful effects of possible moisture and water damage should be minimized by channelling all leaks to become visible and by preventing moisture accumulation in hidden locations.

Building materials and components should be protected against moisture during transportation, storage, and construction. Building materials should be allowed to sufficiently dry before covering or coating them with anything that may slow or prevent drying.

MORE SPECIFICALLY

Rain and surface water should be directed away from the buildings. In such locations where ground water levels or soil properties may cause a risk for rising damp related problems, such drainage systems should be built that can stop capillary flow of water and keep ground water levels sufficiently low with respect to the floor and wall structures.

Crawl spaces should be sufficiently ventilated so that humidity does not harm a structure's function and durability; and designed/constructed so that water does not accumulate underneath the building and that the crawlspace can be inspected thoroughly. Crawlspaces should be clean of debris and organic material that can decay.

External wall structures and adjoining structures should be sufficiently air tight and vapour resistant so that convection or diffusion does not cause moisture content within the wall to rise too high. Both building moisture and moisture from indoor and outdoor sources that may occasionally get in the structures should be able to dry out without causing damage or health risks. Windows, doors, ventilation equipment and other installations, and adjoining roof, balcony and other structures should be designed / constructed so that rain water or snow can not get into the structures and accumulate.

External wall structures should join to the foundation and floor structures so that moisture migration or accumulation into wall structures is prevented and that the wall

can dry out via the lower end, if necessary. Moisture transfer from floor to wall structures should be stopped by a capillary breaking layer in between the structures.

All wall structures that are built against the ground should have sufficient water barriers that prevent water from getting into the structures.

Roof membrane should prevent rain water and snow from getting into roof and upper most floor structures, walls, and interior spaces. The membrane should sustain loads caused by climate changes, snow and ice, and maintenance work. Roof must be designed / constructed so that water is directed away without causing harm to the building.

Upper most floor and ventilation of the roof should be designed / constructed so that moisture does not harmfully accumulate due to diffusion or air flows and so that the structures can dry out, if necessary.

Bathrooms and other rooms with high water usage and/or moisture loads should be designed / constructed so that water in any form can not migrate to the adjoining structures and rooms. Walls and floors should be protected with sufficient water barriers. All free water should be directed to drain / sewage system.

Plumbing and sewage equipment and HVAC equipment and adjoining systems should be designed, constructed and equipped so that possible water leaks can be noticed in an early phase. Piping, ducts and equipment should be located, insulated, and protected so that water in the systems does not freeze and that water does not condense on the surfaces or that the condensed water can be directed away without causing harm.

- Is moisture control sufficiently incorporated into building codes across Europe?

SUMMARY OF THE MOST IMPORTANT QUESTIONS RELATED TO MOISTURE

- How dampness / moisture damage alter the indoor air quality that is critical from a health point of view?
- Are the causal links between dampness / moisture damage related exposures and health sufficiently well known?
- How could information about housing characteristics associated with dampness / moisture damage be translated into moisture protective policies?
- What is the role of ventilation in control of dampness / moisture damage?
- What is the measure of dampness / moisture damage and how it is verified?
- What are the most important characteristics of dampness / moisture damage with respect to occupant health?
- What is the prevalence of dampness /moisture damage (population exposed) within EU?
- Can all damp / moisture damaged buildings be remediated cost-effectively (what is the measure of cost-effectiveness)?

- How is the success of remediation assessed?
- Is moisture control taken sufficiently into building design and construction process?
- Does moisture control planning require special expertise that is not currently fulfilled in the standard building design / construction operations?
- Is moisture control sufficiently incorporated into building codes across Europe?

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On Strategies to Prevent Condensation in Buildings

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INTRODUCTION

It is recognized that the occurrence of condensation in surfaces inside buildings is a major cause of indoor pollution with relevant negative effects on human health. Scientific reviews on health effects from dampness and moisture in buildings made in recent years [1, 2] present the common view that, despite intensive research efforts, the relationships between the probability of the occurrence of dampness and moulds and the building construction and operation parameters have not been fully identified yet. However, some authors [2] clearly state that "setting limits on indoor relative humidity" does not guarantee a mould-free environment.

Nevertheless, it could be stated that dampness and moulds are cases of pollution sources that could, in theory, be totally removed. This would enable the elimination of those sources and effects by the use of a source control strategy. In fact, if the control of a few physical parameters could be guaranteed, the occurrence of dampness and moulds would be prevented and the principle of source control could be successfully applied.

More than exploring the processes through which dampness and moulds are generated or emit pollutants that are responsible for respiratory diseases this paper aims at contributing to discuss the extreme conditions by which it would be possible to avoid condensation.

In terms of physics, condensation occurs in a surface of a room whenever the temperature of the surface is lower than the saturation temperature of moisture in the surrounding indoor air. Phenomena at the micro-scale may make condensation emergence dependant on the nature, microstructure and micro-geometry (roughness) of the surface material. In addition, when condensation occurs, several phenomena may take place at the microscopic scale in the microstructure of the materials behind the surface such as capillarity effects and others. Dampness effects can also be present at the surface caused by the dynamics of the moisture traveling by capillarity inside the fabrics once condensation occurred and mass transport could take place. This case is not considered in this paper.

Other causes for the occurrence of humidity in the fabrics due to leaks in pipes, infiltration of rain water, drainage or others are not considered in this paper since they can be effectively eliminated by different ways, namely, through proper maintenance and operation of the building envelope and water systems.

STRATEGIES

Preventing the occurrence of condensation requires that the temperature of the surface stays permanently higher than the temperature of saturation of the water vapour in the surrounding indoor air. In terms of Physics, this can be achieved by increasing the temperature of the surface or the temperature of the air or by lowering the humidity contents of the air, or by a mix of some of these factors.

In practical terms, this translates into one condition related to the construction of the envelope and two operational conditions associated with thermal comfort and indoor air quality, all of them ultimately interrelated with the energy use in buildings for heating:

i) To insulate the building envelope to ensure that indoor surface temperatures are kept above a certain threshold;
ii) To increase the temperature set-point of the indoor air in the heating mode (which consequently also increases the temperature of the indoor surface);
iii) To increase the ventilation rate in the space to remove the moisture in the indoor air and lower its humidity by mixing it with some new fresh and dryer air and, therefore, to raise the indoor dewpoint temperature. Of course, it may be ineffective when the outdoor humidity is high and the indoor temperature is about the same as outdoors.

In fact, insulation has been justified in some regulations for two main purposes: a) enhancing comfort by avoiding temperature asymmetries and assuring energy saving by reducing heating needs for comfort in winter; and b) preventing condensation inside the fabrics in order to preserve the building structure and the cladding materials. This condensation criterion can probably become, in some cases, the first condition for insulating as it may contribute significantly for the prevention of condensation while heating, by climatie conditions, may not be a critical issue.

The new question could then be to guarantee the fulfilment of the condensation criterion while assuring the thermal comfort conditions. Besides, there is the problem of the thermal bridges that are associated to discontinuities in the insulation. Those thermal bridges are often left without insulation, and become typically the zones where condensations occur in the first place.

Raising the temperature set-points and the ventilation rates have both negative effects in terms of energy demand for heating, which represents a comfort requirement that clearly prevails in Europe over the cooling demand. Nevertheless, there are minimum requirements due to comfort and health reasons. And there are indications that going beyond the minimum requirement might be positive in terms of productivity [3], although there is no consensus on the extent of such effects.

Regarding the occurrence of condensation, it is not clear to what extent, once solved the thermal insulation problem, each of the two operational conditions indicated, taken one by one, may solve the problem or whether there is always a need for a combination of both. And, in the later case, what are the relative weights from each of the two operational conditions. Figure 1 shows a schematic diagram with the relative

risk of occurrence of condensations as a function of the heating temperature set-point, the ventilation rate and the insulation of the envelope. The quantification of the boundaries is a step that could provide a significant contribution towards the assessment of the effectiveness of each measure and of their combination. This will be sought in the next section.

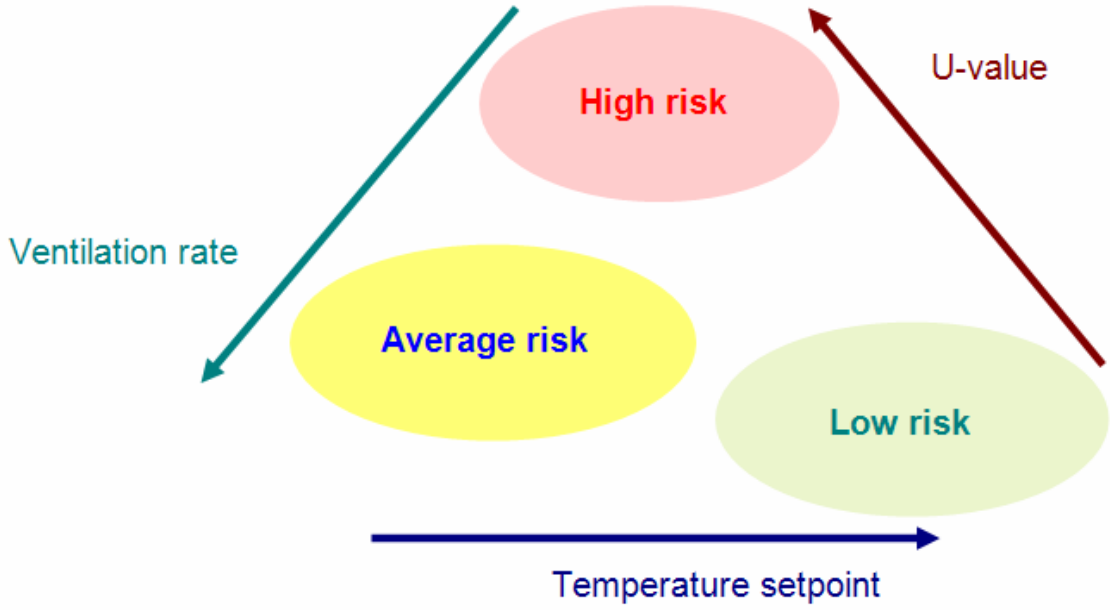


Figure 1: Diagram of risk the occurrence of condensations as function of the insulation (U-value), heating temperature set-points and ventilation rate.

A PARAMETRIC STUDY OF THE CONDENSATION OCCURRENCE

Geometry

A exploratory exercise was made taking as a reference model a room with 15 m^2 of floor area and 3 meters high. The West and the North facades are external; all the other envelope surfaces are considered as connected to similar zones of the building. The room is connected to the exterior through 13 m^2 of opaque wall and 1.65 m^2 of window facing West, plus 8.4 m^2 of wall and 0.6 m^2 of window facing North. A thermal bridge with 0.9 m^2 was considered in the North facade.

Constructions

In terms of construction, the external walls are composed by three layers: an external insulation, an 11 cm thick brick and an internal coating. A thermal bridge made of concrete without any insulation, and with a U-value of 2.7 $W/m^{2o}C$ is considered. The width of the insulation in the main wall is adjusted to vary corresponding to the U-values between 0.8 and 0.2 $W/m^{2o}C$.

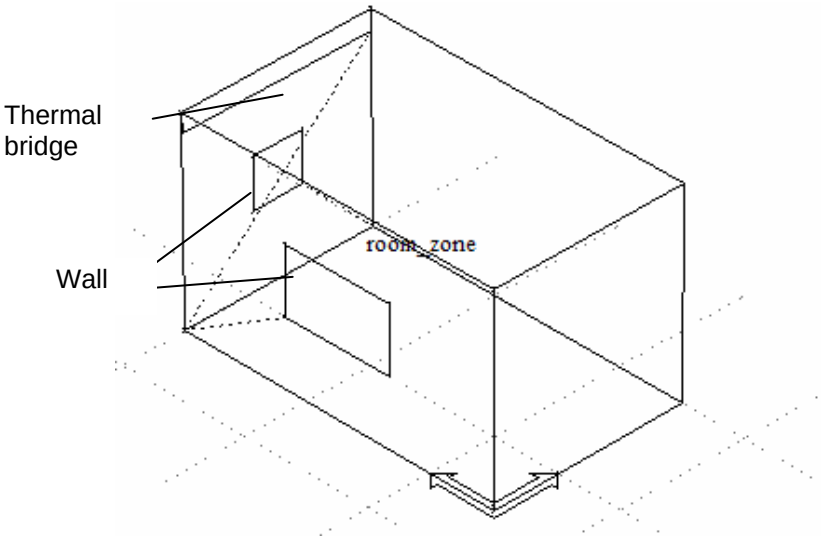


Figure 2: Geometry of the reference room used in the study

Internal gains

The room is considered to be occupied by two persons with a metabolism rate of 115 W/person (70W sensible and 45W latent) from 23 to 24 h and at 70 W/person (50W sensible and 20W latent) from 0 to 8 h. In addition, a general internal gain of 4 W/m^2 due to equipments and lighting is considered.

Software and climate

The thermal behaviour of the room was studied by performing an hourly simulation with the software ESP-r [4]. In order to make the study more representative of an `European average` (and, thus somehow immediately accessible to all), the room was considered to be located in Brussels, with the irrespective geographic coordinates and climate.

Parameters

The parametric study considered variations in the following parameters:

i) U-values of the wall (between 0.2 and 0.8 W/m^2.ºC);
ii) Ventilation rates (between 0.1 and 1.2 air changes per hour);
iii) Temperature set-point (heating mode). Two scenarios were studied for this item:
   a. A control ensuring a minimum of 14ºC at anytime;
   b. A control ensuring a minimum of 18ºC during the occupied period and of 14ºC during the unoccupied period;

By explicitly including a thermal bridge in the model, it was also be possible to assess its effect and therefore compare a “scenario with thermal bridges” and a “scenario without thermal bridges”.

The results of the study, shown in tables 1 and 2, are expressed in number of hours in which condensation might occur for that particular climate and those specific conditions.

Table 1: Number of hours in the year with probable condensation. (temperature set-point at 14 ºC)

U W/m².ºC	air exchange rate									
	0.1		0.2		0.4		0.6		1.0	
	wall	th.bridge	wall	th.bridge	wall	th.bridge	wall	th.bridge	wall	th.bridge
0.8	1403	1967	466	902	93	163	41	65	23	32
0.5	1073	1934	302	766	55	127	29	47	19	25
0.2	656	1489	161	613	33	95	22	37	7	18

Table 2: Number of hours in the year with probable condensation. (temperature set-point at 18ºC)

U W/m².ºC	air exchange rate									
	0.1		0.2		0.4		0.6		1.0	
	wall	th.bridge	wall	th.bridge	wall	th.bridge	wall	th.bridge	wall	th.bridge
0.8	417	1184	55	140	6	11	0	0	0	0
0.5	256	1110	23	100	0	1	0	0	0	0
0.2	84	912	3	58	0	0	0	0	0	0

The results show that, to prevent the physical conditions favourable to the occurrence of condensations at the surface indoors, it is necessary to act simultaneously at the three components: insulation, temperature set-point and ventilation. However, there seems to be a hierarchy in the influence, where the temperature set-point is the factor that influences most, followed by ventilation and finally by the level of insulation.

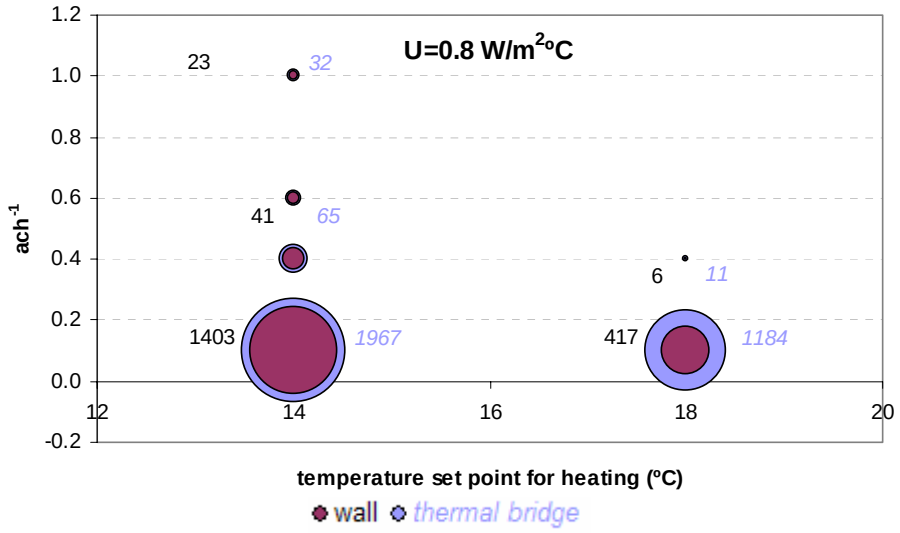
Nevertheless, given the implications in terms of the increase on the energy demand that higher temperatures and ventilation rates will imply, that does not mean that insulation is strategically the least important; on the contrary, it may just be the point where to start from.

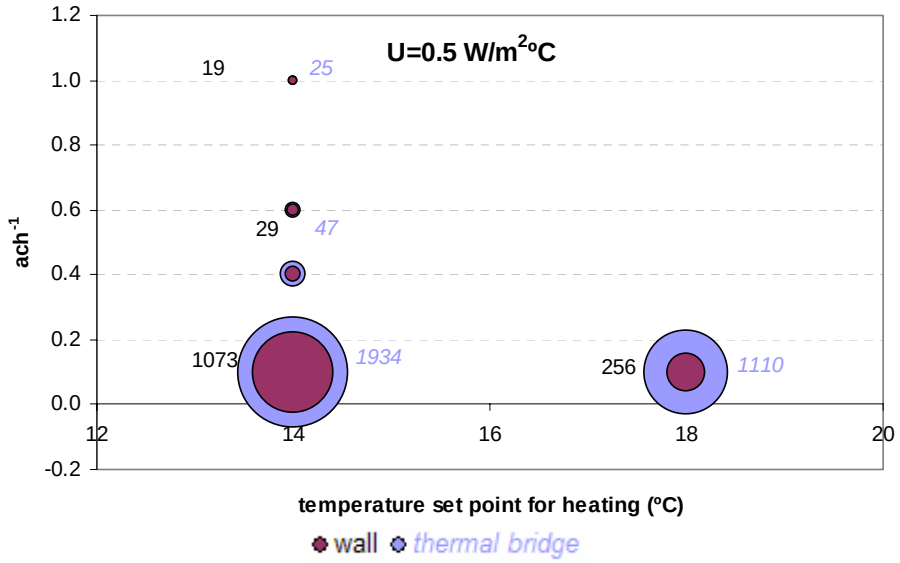
In coherence with this framework, figures 3, 4 and 5 show the number of hours with condensation occurrence for the zones of wall and of thermal bridge, according to the different insulation levels.

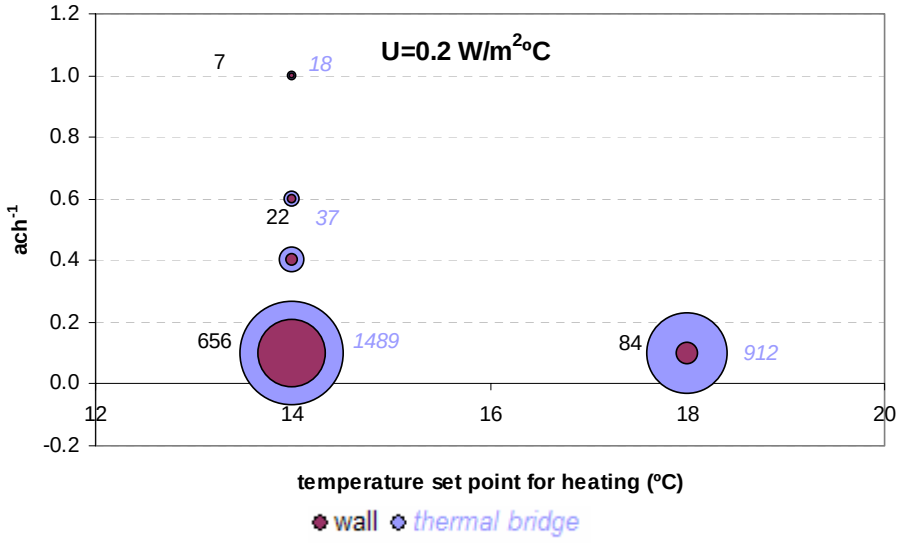
A particularly important observation is that to deal with thermal bridges requires higher ventilation rates. The elimination of the thermal bridges by correct insulation of the building structure thus appears to be the first rationale alternative to adopt.

A second important observation seems to be that, with a 'reasonable' level of insulation and also a 'reasonable' temperature set-point, the ventilation rates recommended by the international standards [5] do solve the problem of condensation at the surfaces. For this room, with 45 m^3 and an occupation supposed to be of 2 persons, the 30m^3/h.person would result in an air change rate of 1.3 ach^{-1}. Therefore the results show that surface condensation could be solved even with much lower air change rates. This issue will however be analysed with further insight in the following section.

An interesting complementary observation is that the strategies to the the problem of condensation also lead to a much better behaviour in terms of the actual relative indoor air humidity. Figures 6 and 7 show the relative humidity of the indoor air for two scenarios, one with high occurrence of condensations (figure 6) and another with a strategy that enables its elimination. Comparing the two scenarios, the second does have about over 3 times more points within the 30%-70% relative humidity range, usually recommended by most international standards.







Figures 3, 4 and 5: Number of hours in the year with probable occurrence of condensations for thermal bridge and wall zone, for three different insulation levels of the external wall.

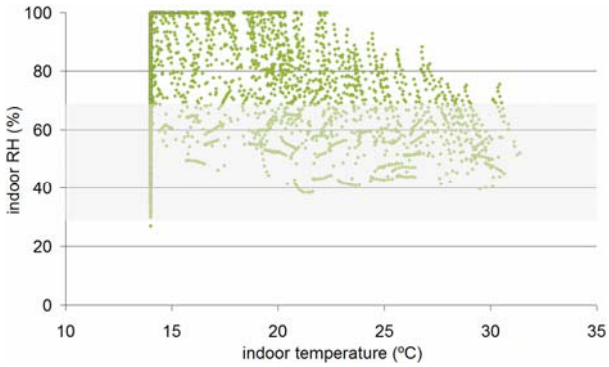


Figure 6 – Indoor temperature *vs* RH at the occupied hours, in a scenario with a U-value of 0.8 W/m^2.ºC, a ventilation rate of 0.1 ach^{-1} and a temperature set-point of 14ºC.

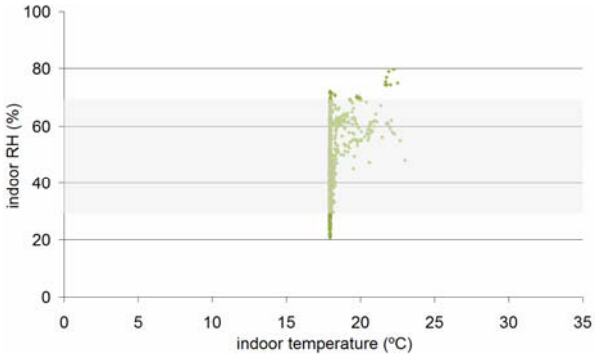


Figure 7 – Indoor temperature *vs* RH at the occupied hours, in a scenario with a U-value of 0.5 W/m^2.ºC, a ventilation rate of 0.6 ach^{-1} and a temperature set-point of 18ºC.

THE INFLUENCE OF OCCUPATION

In order to gain further insight to the influence of occupation, the analysis was extended to study the impact of the density occupation by humans. Only the "central scenario" with a U-value of 0.5 W/m^{2}ºC was considered. The pattern of occupation was shifted from night-time to day-time (8-18 h), since the higher occupation densities are more typical of offices than of domestic rooms. Two set-points, 14 and 18ºC were considered. The metabolic activity was assumed to be equivalent to a moderately office activity with a metabolism rate of 130W/person (75W sensible and 55W latent).

Varying occupation density with constant ventilation

In a first case, the occupation was supposed to vary between 2 and 6 persons (i.e., between 7.5 m^2/person and 2.5 m^2/person), while maintaining the ventilation rate at a constant value of 0.6 ach^{-1} despite the fact that it is against the good practice So this case must be seen essentially as an exploratory exercise to test the physical trends and limits.

The results in table 3 show that, as expected, there is a strong correlation between the occupation density and the likelihood of occurrence of condensations. They also show that even in the most favourable scenarios (7.5 m^2/person and set-point of 18ºC) there is a significant number of hours with probability of occurrence of condensations. This confirms that the 0.6 ach^{-1} is too low for 2 occupants (13.5 m^3/h.person), even when analysing it only from the condensation point of view.

Table 3: Number of hours in the year with probable condensation – wall with a U-value of 0.5 W/m^{2}ºC, ventilation rate 0.6 ach^{-1}

Setpoint	Occupation density (m^2/person)					
	7.5		3.75		2.5	
	wall	thermal bridge	wall	thermal bridge	wall	thermal bridge
14ºC	283	816	1449	1851	1810	2181
18ºC	59	231	1246	1764	1766	2172

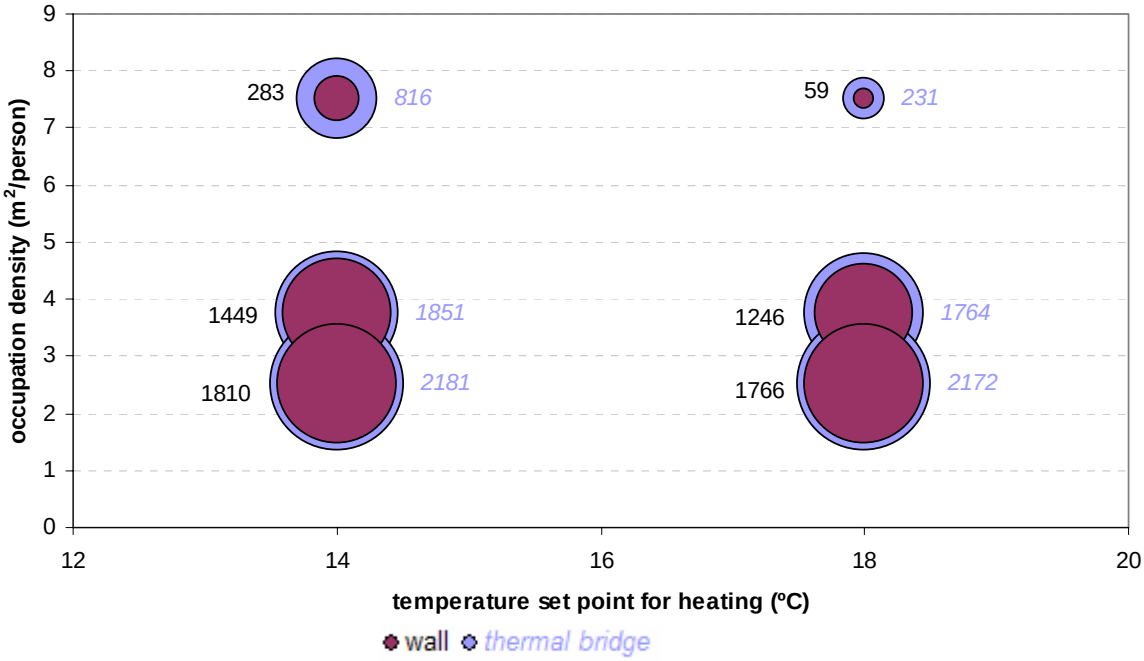


Figure 8: Number of hours in the year with probable occurrence of condensations for thermal bridge and wall zone, for two temperature set-points and three different occupation densities, with a constant ventilation rate of 0.6 ach^{-1}.

Varying occupation density with variable ventilation

A more realistic and in accordance to the good-practice situation consists in adjusting the ventilation rate to the occupation density. This scenario was studied, considering a reference value of 30 m^3/h.person.
The results, in table 4, clearly show that, the adoption of the ventilation rates recommended by the good-practice, guidelines and standards, almost solve the condensation problem. In fact, it seems to be solved if, in addition the walls are well insulated and with the thermal bridges eliminated, and an appropriate level of ventilation is provided.

Table 4: Number of hours in the year with probable condensation – wall with a U-value of 0.5 W/M^2.ºC, ventilation rate of 30 m^3/h.person.

Setpoint	Occupation density (m^2/person)					
	7.5		3.75		2.5	
	wall	thermal bridge	wall	thermal bridge	wall	thermal bridge
14ºC	27	69	27	56	17	40
18ºC	6	9	5	7	2	7

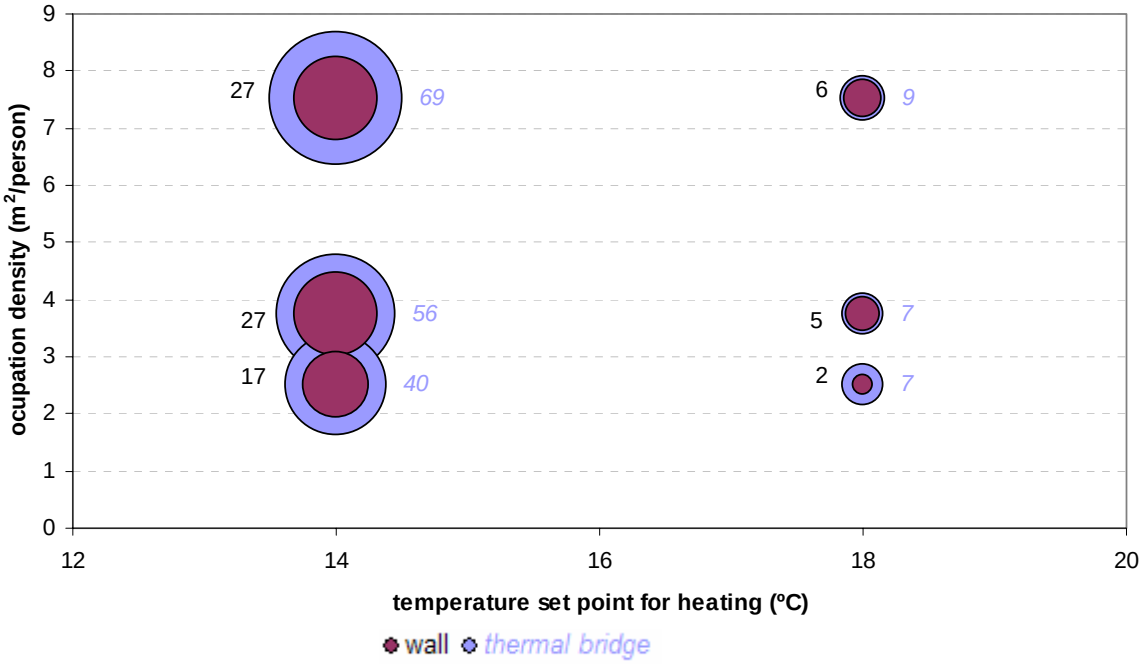


Figure 9: Number of hours in the year with probable occurrence of condensations for thermal bridge and wall zone, for two temperature set-points and three different occupation densities, with a ventilation rate of 30 m^3/h.person

LESSONS FOR POLICY-MAKING

The results of this analysis seem to allow three main conclusions with possible implications on strategic planning and policy-making regarding the elimination of dampness and moulds and consequently eliminate a cause of health effects with an extremely wide and negative impact:

- The prevention of the occurrence of condensations requires combining measures on the insulation, ventilation and temperature heating set-point. Acting on one of the factors alone may not ensure enough good results. For a "central scenario" there is, however, a hierarchy in the effectiveness of measures, increasing from the level of insulation to the ventilation rate and to the temperature set-point. It seems to be wise to take the actual good practices in building construction and in the heating and ventilation systems as a background for the selection of the best combinations for every particular climate condition.

- The existence of thermal bridges in the envelope significantly increases the risk of condensations what implies the need for a particular care in what regards the continuity of the insulation. That may suggest a preference to be given to the external insulation. It is always possible to decrease that risk by increasing the heating set-point and/or the ventilation rate, but this likely means a (unnecessary) significant impact upon the energy demand.

- The ventilation rates around 30 m^3/h.person stated by good-practice guidelines and standards are, combined with reasonable insulation and set-points and

with the absence of thermal bridges, compatible with the removal of the vapour produced by the occupants and the prevention of condensations. In fact it seems that even lower ventilation rates would be compatible, so the prevention of condensations doesn't seem to be the critical parameter to set the ventilation rates.

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Session 4 Infectious diseases

Transmitted infectious diseases in indoor environment

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INTRODUCTION

Except skin, the respiratory tract is the only human organ directly affected by (indoor) air. Therefore it is understandable, that all pollutants from the air can evoke any trouble especially in susceptible people. Microbiological contamination of any environment is common and also indoor air or environment is rich in different microbes and it doesn't make too big difference if pathogenic or not as we must assume, that in common indoor environment will occur many people with immunodeficiency either due their age (ageing people's immune system is mostly weakened), their illnesses (e.g. hereditary malfunction of immune system or acquired immune malfunctions) or their treatment (people after transplantations, with lymphomas or other cancers). These so called immunocompromised people live with us, we are able to safe their lives in hospitals, so we have to guard them in indoor environment, not only hospitals but also in their homes, office buildings, schools etc.

Table 1: Worldwide mortality due to infectious diseases (WHO, 2004)

Cause of death	Deaths 2002 (millions)	% all deaths
All infectious diseases	**14.7**	**25.9%**
Lower respiratory infections	**3.9**	**6.9%**
HIV/AIDS	2.8	4.9%
Diarrhoeal diseases	1.8	3.2%
Tuberculosis (TB)	**1.6**	**2.7%**
Malaria	1.3	2.2%
Measles	**0.6**	**1.1%**
Pertussis	**0.29**	**0.5%**
Tetanus	0.21	0.4%
Meningitis	**0.17**	**0.3%**
Syphilis	0.16	0.3%
Hepatitis B	0.10	0.2%

Tropical diseases (6)	0.13	0.2%
Other causes of death include:		
maternal and perinatal conditions		*5.2%*
nutritional deficiencies		*0.9%*
noncommunicable conditions (cancer, cardiovascular diseases)		*58.8%*
injuries		*9.1%*

In second third of the 20th century people believed that infectious diseases are under control. We had got antibiotics that worked very well; vaccination programmes had fantastic effect on elimination some diseases or at least decreased a number of severe complications and total number of diseased people, especially in developed countries. One of the major killers of the world of the past centuries – smallpox – was even eradicated in the end of 70-ties.

Unfortunately beginning of eighties brought a new challenge – unknown infection agent – HIV. Other new topic followed: SARS. In last several years we are threatened with the potential avian flu mutation into epidemic one.

It seems that the Nature is still one step ahead and when we are sure of our victory, somewhere is hidden at least one new future problem. Some of these problems are results of human's activity, medical efforts or non- responsibility like bacterial strains resistant to antibiotics or Legionnaire's disease.

Regardless of our achievements on the field of infectious diseases, there are still substantial proportions of people dying of various infectious diseases (tab.1). And some of these threats are airborne infections transmitted in indoor environment.

BASICS OF INFECTIOUS DISEASES

Infectious agents are either obligatory pathogens (microbial agents capable of causing disease) or facultative pathogens. In fact we cannot say that any agent it NOT a pathogen as for some people and in some "concentration" (infectious dose) it should be (e.g. for immunocompromised people), so we prefer to call them facultative pathogens.

Transmission of these agents should be direct or indirect from the source. Let take into account mostly the indirect transmission from the unique source: human being. Other possibilities will be mentioned later. Whether in clinical settings, homes, schools, colleges, office buildings, theatres, or airplanes, as long as infected people cough, sneeze, shout, sing, or talk, they can discharge pathogen-filled droplets from their noses or mouths. A single sneeze alone can expel many thousands of infectious respiratory droplets into the air.

Indirect transmission is mediated by contaminated objects (of daily use like towels), by inoculation (e.g. by instruments), by alimentary way and by droplets & air –

airborne infection. Although transmission via droplets is considered to be direct transmission, we can add them, for our purposes, among transmission by air. More over droplets larger than 100µm depending on their resistance to the environment can create contaminated dust. Smaller droplets can stay in air for longer or shorter time. The smaller are the droplets, the further it may be carried from the source. Small respiratory droplets that become aerosolised when people sneeze, cough, laugh or exhale can be carried by air. In addition water droplets aerosolised through air conditioning units may also spread infections. Aerosolised droplets hang in the air and are able to travel considerable distances.

With airborne transmission, direct contact with someone who is infected is not necessary to become ill. The amount of exposure necessary varies from disease to disease. Many airborne pathogens are adapted to spreading in indoor environments, where the temperature, humidity and protection from sunlight protect them in their exposed and vulnerable period when they transmit from one person to the next. For airborne infectious the main entrance of the infectious is the respiratory tract, but for some other it could be e.g. the lesion of skin (skin infectious e.g. furuncle) or mucosa (other than in respiratory tract).

Table 2: Infectious diseases – transmission.

Disease	Infectious agent	Course of illness	Transmission	**Survival in indoor environment**
Upper-respiratory tract	Mainly viruses	Mild	Airborne - droplets	**Short**
Exantematic	viruses	Mild (to severe)	Airborne - droplets	**Short**
Lower respiratory tract	Viruses, bacteria, etc.	Mild to severe	Airborne - droplets	**Short**
Pneumonia	Viruses, bacteria, etc.	Severe	Airborne - droplets	**Short (to long)**
Tuberculosis	*Mycobacterium tuberculosis, aviarum*, etc.	Severe	Airborne - droplets	**Long**
Legionnaire's disease	*Legionella pneumophila*	Severe	Airborne - droplets	**Long (in water)**
Pontiac fever	*Legionella pneumophila*	Mild	Airborne - droplets	**Long (in water)**
Pandemic flu	*Influenza virus*	Mild to severe	Airborne - droplets	**Short**
SARS	*SARS coronavirus*	Severe	Direct contact, airborne, droplets, oral-fecal	**Long? Re-aerosolization**
Anthrax	*Bacillus anthracis*	Severe	Airborne – pulmonary anthrax	**Very long (everywhere)**
Small pox	*Variola major*	Severe	Contact, airborne	**Long**

INDOOR THREATS

In indoor environment some less frequent diseases represent higher risk and higher demands on ventilation systems and environment protection not only in buildings.

WELL-KNOWN SEVERE (INDOOR) INFECTIONS

It is difficult or impossible to require these infections outside indoor environment or the main risk seems to be the transmission via ducts (air-condition systems, ventilation ducts, water ducts).

Tuberculosis

Chronic pulmonary **tuberculosis** caused by *Mycobacterium tuberculosis* is still, despite of the vaccination, severe threat. Over one-third of the world's population now has the TB bacterium in their bodies and new infections are occurring at a rate of one per second. Not everyone who is infected develops the disease and asymptomatic latent TB infection is most common. In developed countries is the prevalence low but in many of them the number of cases is slowly growing up in last years.

Unfortunately the percentage of resistant chains of mycobacterium is increasing and also those of atypical tuberculosis, which are very often multiresistant, too. In most European countries mortality from TB is decreasing but still in some countries (Baltic & Balkan states, but also Portugal, Poland, Finland) TB could be a problem in older age groups (EuroTB). Infectious dose (the amount of microbes necessary for developing an illness) of mycobacteria is in healthy people rather high, so transmission from person to person outdoors is difficult. Survival of agent in indoor environment is long (months).

Legionnaire's disease (Pontiac fever)

Legionellosis is a respiratory disease caused by bacteria *Legionellae*. Most frequently human disease Legionnaire's disease is caused by *L. pneumophila*. The clinical picture is characterized by myalgia, headache, fever, and non-productive cough developing further to pneumonia. Case-fatality rate can be high especially among elderly and immunocompromised individuals. Sporadic cases and outbreaks occur worldwide. In healthy, young people caused *legionella* mostly Pontiac fever – a common cold like disease with none or low risk. *Legionella* is an organism that resides in the environment in pools of stagnant water. Most common route of transmission is airborne. Person to person spread does not occur.

The reservoirs are aquatic systems like cooling towers, evaporative condensers, humidifiers, decorative fountains etc. Legionellosis can be treated effectively with antibiotics. Prophylactic measures include regular cleaning and maintenance of different water systems.

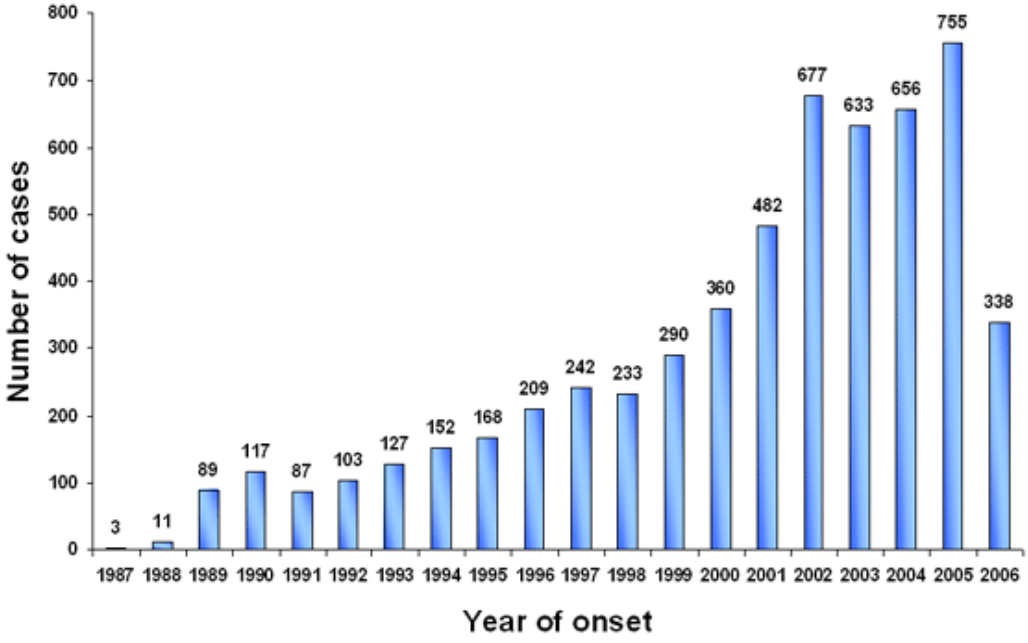
The European Working Group for Legionella Infections (EWGLI) was formed in 1986 with the co-ordinative centre in London. Its members are scientists with an

interest in improving knowledge and information on the epidemiological and microbiological (clinical & environmental) aspects of legionnaires' disease. This is achieved through international surveillance of the disease, as well as developments in diagnosis, management and treatment methods.

The European Surveillance Scheme for Travel Associated Legionnaires' Disease (**EWGLINET**) contents the European Guidelines for Control and Prevention of Travel Associated Legionnaires' Disease. Every European country has a history of *legionella* outbreaks. Sometimes it is difficult to verify the diagnose, especially because nobody believes in it and this is the reason of constitution of this guidelines.

But not only people in hotels in tourist destinations are in risk. Unfortunately other outbreaks were described in hospitals in wards were people with severe diseases were hospitalised. These people are in higher risk than any other.

Figure 1: Graph of Legionnaire's disease cases in Europe by year of onset (EWGLINET)



The disease most often affects the elderly and people with underlying illnesses such as cancer or those with a lowered immune system. Outbreaks of pneumonia have been associated with contamination of water cooling towers in large buildings, with spread of the bacteria mostly through air conditioning systems. Nowadays the new threat comes from **tap water** during shower or aerosolization the tap water, e.g. by spraying etc., so it could be a severe problem of hospital environment.

NEW THREATS

Except these well-known problems, time to time a new one arises somewhere around the world and in a short time it could become a problem of most countries. Frequently is discussing potential epidemic of flu, which is expected for several years, and completely new agent causing SARS.

Flu

Flu pandemic is one of the threats of the end of 20th century and beginning new millenia.
Influenza virus (flu virus) cause diseases with high severity especially for elderly people, with rather high proportion of complications, worsen chronic health problems. Influenza may cause worsening of coronary heart disease or congestive heart failure. Although the incidence of influenza can vary widely between years, approximately 36,000 deaths and more than 200,000 hospitalizations are directly associated with influenza every year in America (1). Every ten to twenty years a pandemic occurs, which infects a large proportion of the world's population, and can kill tens of millions of people (2).

Last several decades a new pandemic strain is expected and there are some "promising" candidates for new reassortment. Influenza reaches peak prevalence in winter. One possible explanation for this seasonal occurrence is that, because people are indoors more often during the winter, they are in close contact more often, and this promotes transmission from person to person. Another is that cold temperatures lead to drier air, which may dehydrate mucus, preventing the body from effectively expelling virus particles.

Anyway the main problem of epidemic flu is not indoor environment as it is highly contagious infectious everywhere. In case of pandemic flu ventilation and especially air-condition systems should play the most important role in transmitting viruses or isolation sick people.

Table 3: Known flu pandemics

Name of pandemic	**Subtype involved**	**Date**	**Deaths**
Asiatic (Russian) Flu	Possibly H2N2	1889–1890	1 million
Spanish Flu	H1N1	1918–1920	40 million (100 millions)
Asian Flu	H2N2	1957–1958	1 to 1.5 million
Hong Kong Flu	H3N2	1968–1969	0.75 to 1 million

Because of high proportion of complications and even death, vaccination is recommended especially for elderly people, people with chronic disease and other immunocompromise people. Virus is extremely variable, so vaccination is necessary every year, due to antigenic drift for every year a new vaccine is necessary.

Severe acute respiratory syndrome (SARS)

In late 2002, a new syndrome was observed in southern China (Guangdong Province). It was named **severe acute respiratory syndrome (SARS)**. The initial outbreak of SARS peaked in April 2003 and by June had tailed off. By that time, there had been about 8,000 cases worldwide and 775 deaths. Respiratory distress leads to death in 3-30% of cases. Via aeroplanes was this disease transmitted to other continents. Transmission of SARS was in most cases observed in indoor environment. In fact the first outbreak was at people living at the same floor of one hotel where doctor from Guangdong province lived. Transmission was possible only via **air-conditioning system**, even airborne spread of SARS does not seem to be a major route of transmission. Also oral-fecal transmission is possible as in other coronaviruses via **sewage systems of the buildings** as coronaviruses were found also in stool of patients.

Bioterorism

Although most of the infectious diseases are rare in 21st century in developed countries, still it is one threat of severe infections of previous centuries. Occurrence of this new threat is dating to the end of twenty century, to the nineties. This is bioterrorism. Still it is at least theoretical chance to get highly danger strains of infectious diseases like smallpox, anthrax or plaque. All these diseases were big killers of the past and are under the control in 21st century.

Yersinia pestis is the agent which causes **plague,** known also as Black Death. The three documented pandemics of plague (Black Death) have been responsible for the death of hundreds of millions of people. The organism in exhaled in cough droplets, infect other humans in close proximity and cause **pneumonic** plague, which more difficult to control and has 100% mortality. Bubonic plague is typical transmissive infection, reservoir are small rodents (well-known are rats) and vector is flea.

Anthrax is a zoonotic disease occurring in wild and domestic animals such as cattle, sheep, goats and other herbivores. It can be acquired by humans either by ingestion, **inhalation**, or skin contact with contaminated animal products. Cutaneous anthrax and gastrointestinal anthrax have lower fatality rates, but still must be treated agressively to assure survival.

Because of the stability of the spore in the environment anthrax is one of the diseases commonly mentioned in relation to germ warfare and terrorist activity. In 2001 several postal workers died of inhalation anthrax after handling B. anthracis-laced.

Pulmonary anthrax results form inhalation of *Bacillus anthracis* spores which are phagocytized by the alveolar macrophages where they germinate and replicate Respiratory distress and cyanosis are manifestations of toxemia. Death results within 24 hours. This form of anthrax is of significance in biological warfare.

Smallpox (also known by the names *Variola* or *Variola vera*) is a highly contagious disease unique to humans. Smallpox is caused by either of two virus variants named *Variola major* and *Variola minor*. The deadlier form, *V. major*, has a mortality rate of 3–35%, while *V. minor* causes a milder form of disease called *alastrim* and kills ~1% of its victims. Long-term side-effects for survivors include the characteristic skin

scars. Occasional side effects include blindness due to corneal ulcerations and infertility in male survivors.

Smallpox was responsible for an estimated 300–500 million deaths in the 20th century. After successful vaccination campaigns throughout the 19th and 20th centuries, the WHO certified the eradication of smallpox in 1977. In most countries the vaccination stopped around 1980, so in fact total population has no protection.

The role of indoor environment is not major but because the important route is re-aerosolization of the dust with scales from scabs, indoor environment could also play important role. Of course also special isolated wards could be highly important.

OTHER DISEASES OF CONCERN (Well-known respiratory diseases)

Some of these diseases are well-known, common, we are familiar with diagnose and treatment of them. They are not harmful, at least for immunocompetent people but could be unpleasant. Many of them are easily transmitted in overcrowded interiors, environment with low air exchange or with pure quality of mechanical ventilation /air conditioning system. In environment with low level of cleaning can persisted infectious agents in dust and can be transferred into breathing zone in consequence of any activity in the environment which can whirl the dust.

These infections are not typically connected with indoor environment and improving the quality of indoor environment probably will not decrease number of sick people. On the other hand - to stop the epidemic we have to **isolate** sick people from healthy ones. Mostly from the beginning all these diseases have symptoms of common cold and rarely are threat for the life. Occurrence of these diseases is common especially from autumn to spring.

Upper respiratory tract illnesses

Cold, common cold
The common cold is caused by a large number of different types of infectious agents, especially viruses. They all result in similar symptoms: sneezing, runny nose, sore throat and cough with or without a low grade fever, muscle aches and malaise. From the medical point of view the health effect of common cold is minor, as complications are rare.
Cause of (common) cold are e.g.: *Adenovirus, Coronavirus, Coxsackie A,B, Rhinovirus, Parainfluenza virus, Respiratory Syncytial Virus (RS).*
Some symptoms could simulate common cold although the cause is different, e.g. *Listeria monocytogenes, Legionella pneumophilla*– Pontiac Fever.

Pharyngitis
Similar to cold is pharyngitis, inflammation of pharynx. The sore throat is highlighted; complications are also rare in immunocompetent people. Problems are usually caused by

different species, e.g.: *Adenovirus, Herpes Simplex Virus 1,2 (HHV1, HHV2), Neisseria gonorrhoeae, Parainfluenza virus, Streptococcus pyogenes.*

Epiglottitis
Haemophilus influenzae is the main cause of life - threatened disease – epiglottitis. Its occurrence is connected with dry air, in young children, mostly younger than 3 years. As *H. influenzae* is so danger, in many countries is used vaccine against this agent.

Laryngitis
The characteristic marker of this disease is hoarseness, loss of voice and pain. Among other causes, one of the most common is *Moraxella catarrhalis.*

Bronchitis, bronchiolitis, bronchopneumonia
These diseases are mostly occurred as a complication of any other, originally upper respiratory tract disease. Course of the disease can vary from mild to severe depending on cause agent and status of the patient. Symptoms are as follow: fever, cough, dyspnoe, shortness of death, cyanosis. Cause (apart others) are: *Moraxella catarrhalis, Parainfluenza virus, Pseudomonas aeruginosa, Respiratory Syncytial Virus (RS), Bordetella pertussis, Nocardia asteroides.*

Otitis media
Otitis media is a problem mostly of young age children and often it is a complication of common cold. In very rare situation it could be danger. Chronic otitis can result in deafness and/or vertigo, in special cases can progress in mastoiditis, meningitis or encephalitis with dramatic development. The most common cause is *Haemophilus influenzae, Moraxella catarrhalis, Streptococcus pyogenes.*

Lower respiratory tract illnesses

Pneumonia has the same symptoms at the beginning but mostly is more dangerous.
Typical symptoms associated with pneumonia include cough, chest pain, fever, and difficulty in breathing. Pneumonia is a common illness which occurs in all age groups, and is a leading cause of death among the elderly and people who are chronically and terminally ill. Causes of pneumonia are several and on the cause depends treatment and also prognosis. Some of the causes are: *Adenovirus, Bacteroides fragilis, Chlamydia pneumoniae, Chlamydia psitacci, Chlamydia trachomatis, Coccidioides immitis, Coronavirus, Coxiella burnetti, Cryptococcus neoformans, Cytomegalovirus* (CMV), *Escherichia coli, Haemophilus influenzae, Histoplasma capsulatum, Influenza virus, Pseudomonas aeruginosa, Klebsiella pneumoniae, Listeria monocytogenes, Moraxella catarrhalis, Mycoplasma pneumoniae, Parainfluenza virus, Proteus mirabilis, Pseudomonas pseudomallei, Respiratory Syncytial Virus* (RS), *Rhodococcus equi, Staphylococcus aureus, Streptococcus agalactiae, Streptococcuspneumoniae, Varicella-Zoster Virus* (HHV3).
Atypical pneumonia is sometimes difficult to diagnose when doctor have no information concerning special lifestyle or hobby (e.g. breeder of parrots). Cause agent could be various like *Adenovirus, Chlamydia pneumoniae, Chlamydia psitacci, Mycoplasma pneumoniae.*

Laryngotracheobronchitis or **croup** is a contagious viral infection causing inflammation and swelling of the larynx and surrounding tissues. It presents with difficulty in breathing especially breathing in and a typical barking cough. It usually affects children between the ages of 6 months and 3 years. Croup can be caused by a number of different viruses. In the fall, it is usually caused by *Parainfluenza virus*. In winter and spring, it is usually caused by *Respiratory Syncytial Virus (RS)* or an *Influenza virus*. Less commonly, croup may be caused by *Measles virus* or other viruses such as *adenovirus, rhinovirus, enterovirus* and *coxsackie virus*. Symptoms are typical: fever, hoarseness, harsh, barking cough, swelling - laryngeal obstruction, dyspnoe.

Herpangina is the name of a painful mouth infection caused mainly by *coxsackieviruses A*. Usually, herpangina is produced by one particular strain of *coxsackievirus A*, but it can also be caused by *coxsackievirus B* or *echoviruses*. It is most common in children. Though herpangina can be asymptomatic, symptoms usually associated are high fever and sore throat.

Other respiratory tract illnesses

Diphtheria is caused by *Corynebacterium diphteriae* and is characterized by an adherent membrane (a *pseudomembrane*) on the tonsil(s), pharynx, and/or nose. Diphtheria is a serious disease, with fatality rates between 5% and 10%. In children under 5 years and adults over 40 years, the fatality rate may be as much as 20%. Nowadays in most developed countries are children vaccinated against diphtheria.

Bordetella pertussis is the only organism of major clinical significance within this genus; it causes **whooping cough** in infants and young children. However, a closely related organism, *B. parapertussis* can also cause a milder form of bronchitis. Despite the vaccination, every 2 -5 years a small epidemic occurred, especially in young adult people, not at children.

Parrot fever is infection transmitted usually via the droppings of infected bird, though it can also be transmitted via feathers and eggs, and are typically either inhaled or ingested. P**sittacosis** - also known as **parrot disease**, **parrot fever**, and **ornithosis** - is a zoonotic infectious disease caused by a bacterium called *Chlamydophila psittaci* and contracted not only from parrots, but also from pigeons, sparrows, ducks, hens, sea gulls, and many other species of bird.

Meningitis

Meningitis is severe complication of various infections and despite antibiotics still kills about 170 000 persons a year (WHO, 2004). It can be caused by several agents: *Coxsackie A,B, Cryptococcus neoformans, Echovirus, Haemophilus influenzae, Herpes Simplex Virus 2* (HHV2), *Leptospita interrogans, Listeria monocytogenes, Moraxella catarrhalis, Neisseria meningitis, Polio virus, Streptococcus agalactiae, Streptococcus pneumonie.*

Children's Exanthema Diseases

Some infections which used to be common, killed hundreds of people, especially children every year, nowadays, due to vaccination, don't present a big risk, at least in developed countries, with one exception – morbilli (measles). Because of travelling around the world, there is a possibility to meat them. Most of these infections are typical diseases of childhood. In younger age there are fewer complications than in adult people, more over vaccination against these "children" infections mostly doesn't assume booster in adult age, so specific immunity is low or lower in middle-age population. Beginning of these infections is similar – common cold and/or typical rush.

Scarlet fever was a threat because of rather frequent complication – rheumatic fever. The disease is caused by *Streptococcus pyogenes* and there is a characteristic rash.

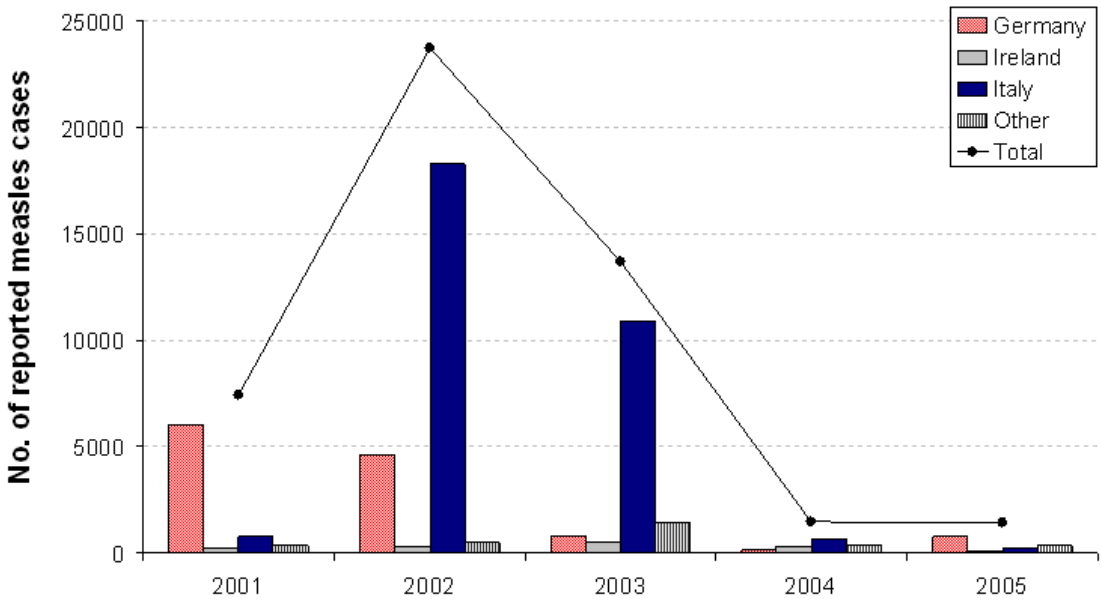
Varicella, chickenpox

Chickenpox is a highly contagious disease that spreads from person to person by direct contact or through the air from an infected person's coughing or sneezing. Chickenpox is rarely fatal but later in life viruses remaining dormant in the nerves can reactivate causing localised eruptions of shingles. This occurs particularly in people with compromised immune system, such as the elderly, and perhaps even those suffering sunburn. Unlike chickenpox which normally fully settles, shingles may result in persisting post-herpetic neuralgia pain. Because of those complications, in several countries vaccine against *Varicella-Zoster Virus* (HHV3), which causes varicella, is used to prevent these later complications.

Rubella (also known as **epidemic roseola**, **German measles**, **liberty measles** or **three-day measles**) is a disease caused by the *Rubella Virus*. It is often mild and an attack can pass unnoticed. Rubella can pose a serious risk as it can also be transmitted from a mother to her developing baby through the bloodstream via the placenta and in this case it caused teratogenic.

The **Measles** are a highly contagious airborne pathogen which spreads primarily via the respiratory system. The *Measles Virus* is transmitted in respiratory secretions, and can be passed from person to person via aerosol droplets containing virus particles, such as those produced by a coughing patient. Complication of measles even in childhood was severe and lethality was rather high. It was estimated that in 1996 about 1 million children died from measles complications.

Figure 2. No. of reported measles cases in 19 EUVAC.NET participating countries since 2001 (EUVAC.NET)



Mumps or **epidemic parotitis** is a viral disease of people causes by *Mumps Virus.* Prior to the development of vaccination and the introduction of a vaccine, it was a common childhood disease worldwide, and is still a significant threat to health in the third world. Despite the vaccination, time to time a small epidemic occurrence of mumps can be observed it is danger especially in young men, because in this age the danger complications like pancreatitis or orchitis and encephalitis.

RISK OF MOULDS, YEASTS (FUNGI)

Very often is indoor environment (or ducts) contaminated by moulds or fungi due to poor maintenance, low air exchange etc. For healthy people this contamination doesn't represent a big harm. If any, so the risk is first of all to evaluate an allergy.

For immunocompromised people moulds could represent a life threat. Generalised or pulmonary aspergillosis can cause severe complications and even death of people with specific treatment of cancer or after transplantation.

CONCLUSIONS

Indoor environment play important role in transmission – ventilation, air-conditioning, water or sewage ducts can transmit several infectious agents to rather long distances.

Also secondary source (water, dust) can play important role even in other type of infections (alimentary – e.g. water-born cholera or some viruses causing alimentary problems).

There are several other facultative or obligatory pathogens without low effect for healthy people who spent their time either in well-maintained indoor environment or mostly outdoors; these agents could be harm for immunocompromised people.
One can assume that if we will be able protect ourselves against these threats, probably we will be successful also in other battles against infectious disease, either those we know or any new still unknown.

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Pathogens

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Pathogens, or in this case better to say, infectious agents are ubiquitous. Some of them are obligatory pathogens, cause of severe diseases and therefore are well-known. Some of them are well-known but they are considered to be either less risky or it is believed that they are under control. Some of them are facultative pathogens, causing fewer infections and therefore mostly are unknown. All of them should cause health problems indoors as the indoor environment is very special:

- People spent indoors most of their life, ill people with higher probability
- Air exchange is mostly inadequate, especially where ill people are concentrated
- Poor maintenance of ventilation/air exchange systems is rather common, so the infectious dosage could be reached easily, if the source of infectious agent is indoors
- Direct & indirect transmission infectious agents is easy in overcrowded interior

A PIECE OF HISTORY

Outbreaks of several infectious diseases occurred during the previous centuries and all of them reduced the population of the civilized countries. People gave them poetic names as “black death” or “red death”. Last such devastating outbreak, with probably higher number of death people than any of the previous, was “Spanish flu” at the beginning of 20th century causing the death of more than 40 million people.

All of these outbreaks, as far as we know, were caused mainly by the airborne infections – transmission from man to man directly or indirectly via droplets. There are some exceptions from this rule: plague is transmitted from man via vector (flea) to other man. Medium for distribution *Vibrio cholerae* is (drinking) water.

Other “plagues” like smallpox or pulmonary plague are distributed by droplets in air.

INFECTION AGENTS

Many people believe that most infectious diseases are under control due to vaccination or/and antibiotics. Unfortunately the reality is less positive. Antibiotics don’t affect viral infections and research and development of new vaccines is not quick enough. Moreover, evolution of the pathogens is probably at least of the same speed as the vaccine development. When people moved from outside in indoor

environment brought also some new problems, arising slowly for centuries. And our "friends" (or pets?) moved with us.

So who share our indoor environment with us? Are these "guests forever" welcomed or even invited? Probably not and we have to change out environment or our way of maintenance, even our way of use of the built environment to protect ourselves against these unwelcome guests.

Infectious agents can be divided according to different points of view. For the purposes of indoor environment and discussion with non-medical professionals, I prefer to combine them according the source localisation & characteristics.

Exposure /causal agents: infectious agents (optional or obligatory) – viruses, germs, fungi/yeasts

Classification of the agents:

According to the source localisation of (potential) infection:

1. in indoor environment, removable
2. in indoor environment, "removable"
3. source is not indoors (this is not matter of this article)

According to the origin:

1. airborne
2. waterborne

According to the transmission:

1. direct (from man to man)
2. indirect (via droplets)
3. transmissive (via vector, mostly insect)

According to the outbreaks (at healthy people):

1. Severe, life threaten infections
2. Mild infections, mostly without complications
3. Dangerous only for immunocompromised people

Table 1: Leading Causes of Death due to Infectious Diseases in 2002 (WHO, 2004).

	No. of death in millions
Lower respiratory infections	**3,9**
HIV/AIDS	2,8
Diarrheal diseases	1,8
Tuberculosis	**1,6**
Malaria	**1,3**
Measles	**0,6**
Pertusis	**0,29**
Meningitis	**0,17**

INDOOR SOURCE OF PATHOGENS

An infectious agent could be a "permanent" part of the environment e.g. in the case of a contaminated ventilation/airconditioning/watersupply systems. An example could be the well-known microorganism *Legionella pneumophila,* which still can cause problems. *Legionella* is waterborne agent, living in cooling towers or humidifiers of AC or in (drinking) water supplies. Via droplets could be transmitted to people, dangerous are mostly for immunocompromised people. Anyway the infection dose has to be high.

The only chance how to avoid this risk is maintenance and disinfection of ventilation/AC and water supply systems.

Poor maintenance of ventilation/AC systems could be the cause of spreading of *Aspergillus sp.,* common mould indoors (also outdoors). For healthy people the risk of developing aspergilosis is low, the risk is getting higher for small children but especially for immunocompromised people. Nevertheless moulds are often the cause of allergic problems and aspergillus sp. and thus are a very pottent allergen.

Airborne infections, the main topics when discussing indoor environment risks of infections, are all of the same origin. **Source** of these infections is a person (ill or in incubation period), so the source is "removable". Even when source is indoors, but room is well ventilated and not overcrowded, transmission of infection is not likely to healthy people. Without the host most of these agents do not survive long in the environment (of course with some exception, as usual).

Typically are airborne infections represented by viruses, e.g. those causing (common) cold (Adenovirus, Coronavirus, parainfluenza virus, Respiratory Syncytial Virus, Rhinovirus, Echovirus etc.). Most of them are not causing severe illnesses (in healthy people).

Another group of airborne infections are viruses of so called “children´s exanthema diseases” like Measles Virus, Mumps Virus, Rubella Virus and also Varicella-Zoster Virus (HHV3). In childhood also these diseases are less life threatening, in particular because in most countries there is a vaccination programme against these infections. The main risk occurs in adults people, who meat this infection for the first time.
To avoid this risk is, except vaccination, isolation of sick people not to contaminate the environment. It means mostly at home, only exceptionally it is necessary to admit those people to hospital.

Bacterial airborne infectious of upper respiratory tract are caused also by bacteria like Streptococcus pneumonia, Staphylococcus epidermidis, Staphylococcus aureus, Haemophilus influenzae, Chlamydia pneumoniae, Mycoplasma pneumoniae etc. Many of them caused infections in childhood, can complicate viral infections of upper respiratory tract and also are often cause of illnesses of lower respiratory tract as pneumonia etc. Bordetella pertussis, Corynebacterium diphtheria, are examples of high risky airborne agents. There is a vaccination programme against all of them but still time to time in some part of the world an epidemic occurs. In this case the only chance is isolation, good ventilation (and of course vaccination is the best prevention). Avoidance of risk is again difficult as those bacteria are ubiquitous. So isolation of sick people and good air exchange are probably the most suitable recommendations.
In the recent years in many countries especially among teenagers severe meningitis caused by Neisseria meningitidis has appeared (tabl.1). Occurrence was noticed mainly in barracks or students dormitories with overcrowded rooms and poor ventilation.

Airborne transmission is known to be the route of infection for diseases such as aspergillosis. It has also been implicated in nosocomial outbreaks of MRSA (Methicillin-resistant Staphylococcus aureus); Acinetobacter spp. and Pseudomonas spp., Bacillus cereus or Rotavirus caused problems in different parts of human body including digestive tract.

There are several agents more dangerous than others either due to severity of the disease they can cause, high infectiousness & quick progress or lack of knowledge of treatment.

Probably most common and best known is Mycobacterium (tabl.1), and never mind if *Mycobacterium tuberculosis, Mycobacterium avium intracellularae* or any other. Mycobacterium is resistant, can survive in the environment for several months. Treatment of tuberculosis takes at least a month, sometimes years.

Influenza Virus (Flu Virus) is discussed very often in last several years. Epidemiologists warn against (pandemic) flu as it used to occur every ten years. Focus of infection was traditionally in Asia. Knowing this, there is a chance to avoid spreading infection to other continents.

Notes: The first European influenza epidemic occurred between 1556 and 1560, with an estimated mortality rate of 20%. The Influenza Pandemic of 1918 (or the Spanish Flu) killed 25-50 million people (about 2% of world population of 1.7 billion). Today Influenza kills about 250,000 to 500,000 worldwide each year.

Great lessons we have learned from story of SARS. First outbreaks occurred in hospital personal and family members of patients in 2003 in China but one of the first outbreaks was in hotel in Hong Kong and one of the last was in housing estate also in Hong Kong. Transmission was mediated probably via AC/ventilation system in hotel, but in single building the transmission could be mediated via sewage system. So transmission of this coronavirus is typicaly orofecal as other coronaviruses and also via direct contact and probably dropplets.

Very special role can be played by *Bacillus anthracis* and *Smallpox Virus* (Variola). Variola is nowadays eradicated and anthrax is not common in developed countries but both could be abused for bioterrorist purposes. These pathogens can only be dealth with in isolated high security laboratories.

Special cases are transmissive infections. Source of infection is sick person but these infections cannot be transmitted from person to person. The main condition is a specific vector, which is necessary to transmit the infection agent. These vectors are mostly sucking insects. The best chance to avoid these diseases is source & vector control indoors.

Typical vectors are **fleas** (e.g. Yersinia pestis, cause of plaque), **mosquitos** (e.g. Plasmodium malarie, cause malaria), sucking **louse** (e.g. Rickettsia prowazeki, rickettsia, typhi etc. cause typhus or Rocky Mountains Fever), **fly** (especially tse-tse in some part of the world), **heteropters** and some other vectors, which can transmitt tropical diseases caused by Leishmanias, Trypanosoma etc.

CONCLUSIONS

The main problem of transmission infections agents in indoor environment is poor design and/or maintenance of ventilation/AC/water/sewage systems, also inadequate air exchange and overcrowded spaces.

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Session 5 Cancer, acute cardiovascular effects and COPD

Association between chronic obstructive pulmonary disease (COPD) and indoor air pollution: a review of literature.

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ABSTRACT

COPD is a chronic respiratory disorder responsible for a major burden to the society worldwide. Although the majority of COPD occurs in current or former smokers, a not negligible proportion of the disease also occurs in persons who have never smoked. Available data in the literature indicate that indoor pollution exposure largely affects respiratory health worldwide. Conservative estimates show that between 1.5 million and 2 million deaths per year could be attributed to indoor air pollution, with a significant proportion of deaths due to COPD. In this review of scientific literature, we will describe relevant findings on the association of non-smoking related COPD with the exposure to more common indoor air pollutants, in adults. **Results**: Most of the findings relate to the association of COPD with passive smoke and, in developing countries, biomass combustion exposure. Both these exposures prove to be risk factors for non-smoking related COPD. Mold/dampness exposure results associated to symptoms/signs, who may be related to the presence of COPD or its development. **Conclusion**: In spite of an increased COPD prevalence (predicted to further increase in the next years), and the evidence that other risk factors than smoking may be associated to COPD development, we found relatively few studies that assessed the association between COPD and common indoor air pollution in adult general population. It would be important to improve awareness on adverse health effects possibly associated with biomass combustion-related air pollution in developed countries among others because of the increasing interest for wood and other biomasses as potential alternative energy sources.

INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD)

Currently, approximate estimates indicate COPD as the fifth leading cause of global morbidity (1). In 2010 the disease is expected to rank as number three (2, 3).

According to World Health Organization (WHO) estimates, 80 million people have moderate to severe COPD. More than 3 million people died of COPD in 2005, which corresponds to 5% of all deaths globally. Total deaths from COPD are projected to increase by more than 30% in the next 10 years, unless urgent action is taken to reduce the underlying risk factors. WHO predicts that COPD will become the third leading cause of death worldwide by 2020 (1).

Several different definitions have been used for COPD. Historically, it has been defined symptomatically as chronic bronchitis, anatomically as emphysema, or, most recently, physiologically as airway obstruction (4, 5). ATS and ERS (6) have defined COPD as "a preventable and treatable disease state characterised by airflow limitation that is not fully reversible. The airflow limitation is usually progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases, primarily caused by cigarette smoking. Although COPD affects the lungs, it also produces significant systemic consequences".

Objective demonstration of airflow obstruction by spirometry is mandatory for a diagnosis of COPD. Individuals with chronic cough/sputum production can be at risk for developing airflow obstruction (6). These symptoms, as well as progressive dyspnoea, are common among COPD patients, and they may precede the development of airflow limitation by many years. Thus, all adult individuals (age >40 yrs) with chronic cough/phlegm/progressive dyspnoea, especially if smokers, should be carefully evaluated (6).

Variable definitions and lung function criteria for COPD have made it difficult to quantify the prevalence of the disease around the world (7-9). In addition, a large proportion of patients with COPD in the community remain undiagnosed. In US, about 90% of subjects with undiagnosed airflow obstruction had mild impairment and 10% moderate to severe impairment (10). In Spain, among the subjects with airflow obstruction, previous diagnosis of COPD had been made in only 21.7% of cases (11), and in UK 18.8% of COPD people were undiagnosed (12). The under-recognition and under-diagnosis of COPD lead to significant under-reporting.

Halbert et al (4) have recently published a quantitative summary of the world literature on COPD prevalence, with estimates for COPD in important subgroups defined by age, smoking status, sex, WHO region, study setting (urban or rural), and quality study. It was not possible to locate any spirometric studies reporting COPD prevalence in the African or Eastern Mediterranean regions. The pooled prevalence has been valuated 7.6%, 4.5% in Americas, 11.4% in South-East Asia, 9.0% in Western Pacific, and 7.4% in Europe. The European Lung White Book (13) reports the prevalence of clinically relevant COPD varies in Europe from 4-10% of the adult population.

Active smoking is the most important risk factor for COPD. It has been estimated that about 70% of COPD related mortality is attributable to cigarette smoking (14). Other risk factors than smoking may play an important role in pathogenesis and development of chronic bronchitis and COPD (15). There is enough evidence that poverty, nutritional factors, age, familial and genetic factors, airway hyperresponsiveness, childhood infections, passive smoking, specific occupational

exposure, outdoor and indoor air pollution, are risk factors that increase the probability of developing airway obstruction, independent from smoking status (16).

Although the majority of COPD occurs in current or former smokers, a not negligible proportion of the disease also occurs in persons who have never smoked.

Halbert et al estimated a pooled prevalence of COPD diagnosis of 9.2%, in adults over 40 years and of 4.3% (95% Confidence Interval, CI 3.2-5.7) in never-smoker subjects (4). Recent analyses on the Third National Health and Nutrition Examination Survey (NHANES III) data reveal that never smokers represent a significant proportion of airway obstruction in US adults (23% of obstructed subjects), and only one fifth of the obstruction in this group is explained by presence of asthma (17). Results by a recent Japanese Study indicate airflow obstruction in 5.8% of never-smokers (18). Also in Europe it has been observed a sizeable proportion of never-smoker people with COPD, defined by either airflow obstruction or presence of chronic bronchitis/emphysema. In Spain, the prevalence of COPD in never-smoker people resulted 4.1% (19), and 23% of COPD subjects had never smoked (11). The prevalence of obstruction in lifelong nonsmoking subjects was 8.7% in UK (12), 12% in Poland (20), and even 20.4% in Austria (21). In Italy, COPD in never-smokers of a general population ranged from 10.4% to up 38.8%, when different spirometric criteria for defining COPD were used (22). In non-smoker adult Swedes, the prevalence of COPD varied from 3.4 to 24.5%, according to different spirometric cut-off points for COPD (23).

Chronic cough/phlegm was present in 16% of never-smoker Italian women selected by a general population sample (24). Other Italian Study on young adults of the general population showed that 30% of the subjects with chronic cough/phlegm were never-smokers (25). In Finland, about 50% of the women with chronic bronchitis/emphysema had never regularly smoked (26). In Sweden, chronic bronchitis/emphysema was present in about 10% of the general never-smoking population (27).

Indoor air pollution

Indoor exposure more frequently occurs at home, in social private/public settings, or in workplaces. Indoor environments contribute significantly to human exposure to pollutants, because people spend most of their time indoors. Today, indoor air pollution is globally ranked tenth among the preventable risk factors causing burden of disease (28).

Common indoor pollutants and related sources are summarized in Table 1.
Available data in the literature indicate that indoor pollution exposure largely affects respiratory health worldwide. Conservative estimates show that between 1.5 million and 2 million deaths per year could be attributed to indoor air pollution, with a significant proportion of deaths due to COPD (28).

Table 1. Main indoor pollutants and related sources (28)

Type	Pollutant	Typical sources
Combustion products:	*Carbon monoxide (CO)*	Gas ranges and pilot lights, unvented kerosene and gas heaters, wood and coal combustion, tobacco smoke
	Nitrogen dioxide (NO_2)	Gas ranges and pilot lights, unvented kerosene and gas heaters
	Respirable Particulate Matter (PM)	tobacco smoke, wood and coal combustion, fireplaces
	Environmental Tobacco Smoke (ETS)	Tobacco cigarettes and cigars, pipes
Volatile organic compounds (VOCs)	*- Aldehyde (formaldehyde)* *- Aliphatic halogenated hydrocarbons* *- Aromatic hydrocarbons* *- Terpenes*	Furniture, solvents, paints, adhesives, cleaning products, tobacco smoke, insulation materials
Major indoor allergens	*Acarids*	
	House dust mites	Dust, bedding, carpeting
	Pets:	
	Cats or Dogs	Dandruff
	Birds	Feathers
	Insects:	
	Cockroaches	Floors
	Fungi (moulds)	Dampness
	Pollens	Plants
	Rodents	Mice

The aim of this paper is to describe relevant findings, available in scientific literature, on the association of non-smoking related COPD with the exposure to more common indoor air pollutants, in adults.

METHODS

We performed a review of the literature by focusing on COPD, defined as either airflow obstruction or chronic bronchitis/emphysema. Chronic cough or phlegm and dyspnoea have been also considered as health outcomes. Longitudinal studies have confirmed that cough/phlegm are associated to higher risk for COPD development. In the European Community Respiratory Health Survey (ECRHS), in subjects with chronic cough/phlegm both at baseline and at 8-years follow-up, the incidence of COPD was four-fold higher than in subjects who had never reported these symptoms at baseline (29). Lindberg et al, who prospectively studied the incidence of COPD in people with normal lung function (FEV_1 (forced expiratory volume in one second)/FVC (forced vital capacity) ratio ≥ 70%) at baseline, concluded that bronchitic symptoms and dyspnoea were significant risk factors for developing COPD, and they persisted after adjustment for possible confounders (30).

We mainly considered studies on the health effects of indoor air pollution to which the general population may be commonly exposed. Specific indoor occupational

exposures, that regard only some groups of workers, have been considered only marginally.

RESULTS

In general, we found that few studies investigated the association of non-smoking related COPD with indoor air exposure. Most studies assessed the relationship between COPD and specific occupational exposure, or the health effects of ETS exposure. Biomass combustion was widely investigated as risk factor for COPD, in developing countries. Few studies evaluated the effects by directly measuring levels of pollutants. Information on such exposure has been more likely collected by interview with questions on the presence of known sources of indoor pollution.

Environmental tobacco smoke (ETS)

ETS is produced by tobacco combustion and contains over 4,500 compounds in both vapour and particle phases, many of them being known carcinogens and irritants. ETS is a common major source of indoor PM. Significantly higher concentration of PM has been measured in indoor places where people smoke than in smoking-free indoor environments. The effects of passive smoking have been widely investigated (28). Based on the evidence by literature, the US Environmental Protection Agency (US EPA) (31) concluded that ETS exposure may increase the frequency of respiratory symptoms in adults, and that these effects are estimated to be 30-60% higher in ETS exposed compared to unexposed nonsmokers. Between 10 and 50% of European adults are exposed to ETS (32, 33). Preventable policy legislation has been applied in several countries to reduce ETS exposure at work and in public settings, but no legislative intervention has so far been made in dwellings. In addition, a study performed in some European cities shows that, even in places where smoking is prohibited, the concentration of nicotine indicate that some residuals of tobacco smoke can still be found (34).

Table 2 shows recent studies on the relation of ETS with COPD in never smokers. Chronic bronchitis was the diagnosis more frequently linked to ETS exposure, and the highest risk was reported for never smoking Chinese women exposed to ETS both in childhood and adulthood.

Table 2. Association between ETS and COPD in never smoker adults (OR=odds ratio, CI=Confidence Interval)

Author, *Source* (sample)	**Country**	**Exposure**	**Disorder**	**OR**	**95% CI**
Simoni M, *Respir Med 2007* (women)(24)	Italy	at home and work	Dyspnea CB/emphysema Cough/Phlegm	1.61 2.24 1.52	1.20-2.16 1.40-3.58 1.07-2.15
Jindal SK, Indian J Chest Dis Allied Sci 2006 *(35)*	India	any	CB	1.40	1.21-1.61
David GL, *Thorax 2005* (women)(36)	China	in childhood and adulthood	CB Chronic Phlegm Chronic Cough	2.87 2.38 2.80	1.58-5.22 1.82-3.12 1.61-4.87
Larsson ML, Eur Respir J 2003*(33)*	Estonia	outside home	Dyspnoea CB/Emphysema	1.65 1.54	1.20-2.27 1.13-3.00
Radon K , *Chest 2002* (37)	German	at work	CB	1.90	1.16-3.11
Iribarren C, J Epidemiol Community Health 2001 *(men)(38)*	US	at home or in other places	Chronic Cough Emphysema	1.60 3.02	1.22-2.10 1.22-7.34
Jedrychowski W, *Int J Occup Environ Health 1995* (39) (elderly women)	Poland	any	Dyspnoea	2.23	1.45-3.44
Leuenberger P, *Am J Respir Crit Care Med 1994* (40)	Swiss	any	Dyspnoea CB	1.45 1.65	1.20-1.76 1.28-2.16
Dayal HH Environ Res 1994 *(41)*	US	at home	ORD	1.86	1.21-2.86

CB=Chronic Bronchitis; ORD=obstructive respiratory diseases.

A recent review of the literature estimated the pooled risk for chronic cough in never smokers heavily exposed to ETS: Odds Ratios (ORs) were similar in both men (1.60, 95%CI 1.22-2.10) and women (1.68, 1.17-2.34) (42). Significant relations between ETS exposure and COPD development have been found in the elderly, too, with an OR range of 1.68-5.63 (43). A French study on never smoker adults found a significant inverse association between ETS exposure and both FVC and FEV_1, with a decrement of 3.16% and 2.90%, respectively, in exposed subjects. To be exposed to ETS at home or at work represented an increased risk for abnormal low FVC (OR 2.71, 1.09-6.75) (44). Also in Scotland there was evident decrement of FVC and FEV_1 in non smoker subjects exposed to ETS, when compared to unexposed ones (45). A dose-response effect was reported by Eisner et al, in a study on the general population in US: chronic bronchitis/emphysema/COPD resulted associated with higher ETS

lifetime exposure at home (OR 1.55, 1.09-2.21) or at work (1.46, 1.08-1.96), after controlling for smoking history and sociodemographic characteristics; the association was significant also after discarding from the analyses the subjects who reported chronic bronchitis alone (2.38, 1.42-3.90; and 1.79, 1.21-2.65, respectively (46). A significant dose related increase in the risk for developing dyspnoea has been observed in young adults for an average exposure of 10 cigarettes/day (OR 2.37, 1.25-4.51) (47).

Biomass fuels

Indoor air pollution from biomass (wood/coal) use for either cooking or heating is an important risk factor for COPD, especially in women. About 50% of world's households burn these products for cooking in open fire or with inefficient stoves in poorly ventilated rooms (48). It occurs especially in developing countries, where the production of PM and CO (a proxy for $PM_{2.5}$) by biomass combustion is dramatically high (49). Through an extensive review of epidemiological studies around the world, the estimation of the risk by biomass use for COPD results in ORs of 1.8 (1.0-2.8) in males and 3.2 (2.3-4.8) in females (50). In US, the presence of coal stove increased the risk for chronic inflammatory and obstructive respiratory symptoms, in non smoker adults, with OR ranging from 1.8 to 3.3 (C.I. 1.0-5.9) (51). A selection of studies concerning the association of biomass fuel use with COPD is reported in Table 3.

Table 3. Association between biomass fuel and COPD in adults (OR=odds ratio, CI=Confidence Interval).

Author - *Source* (sample)	**Country**	**Exposure**	**Health outcome**	**OR**	**95% CI**
Shengming L, *Thorax 2007* (never smoker women)(52)	China	biomass fuel	$FEV_1/FVC<0.70$	3.11	1.63-5.94
Orozco-Levy M, *Eur Respir J 2006* (women)(53)	Spain	wood and charcoal smoke	Dyspnoea CB	1.45 1.65	1.20-1.76 1.28-2.16
Ekici A, Environ Res 2005 *(women) (54)*	Turkey	biomass vs GPL	$FEV_1/FVC<0.70$ or CB	2.5	1.5-4.0
Golshan M, *Respir Med 2002* (never smoker women)(55)	Iran	biomass fuel	CB	2.91	2.08-4.40
Dennis RJ, *Chest 1996* (women)(56)	Columbia	wood-smoke	$FEV_1/FVC<0.70$	3.9	1.7-9.1
Xu X, *Rev Respir Dis 1993* (non smokers)(51)	US	Coal stove use: for both cooking and heating	Chronic Cough Chronic Phlegm	1.8 2.0	1.0-3.3 1.2-3.4

CB=Chronic Bronchitis; FEV_1=forced expiratory volume in one second; FVC=forced vital capacity.

Most studies have been performed in developing countries, mainly on women. Biomass fuel use resulted associated with airflow obstruction in women living in Turkey, Columbia, and China. In Turkey, after adjusting for possible confounding factors, the risk for COPD, defined either as $FEV_1/FVC<0.70$ or chronic bronchitis, was higher in women who used biomass fuel than in those who used GPL (liquid petroleum gas), and the attributable fraction of COPD to biomass smoke was 23.1% (54).

In addition (not shown in Table 4), other two studies performed in Turkey found: 1) women exposed to biomass fumes more likely to suffer from chronic bronchitis and COPD than those unexposed, even though the prevalence of current smoking was higher among the latter (57); 2) never smoker housewives exposed for 30+ years to biomass fuel to be at higher risk for developing COPD than those never exposed (OR 6.61, 2.17-20.18) (58). In Mexico, the exposure to biomass smoke has been associated with chronic bronchitis and chronic airflow obstruction, in adults. Among never smoker women, those exposed to wood smoke had a five-fold risk as compared to the unexposed (59), and women exposed domestically to biomass developed COPD with clinical characteristics, quality of life, and increased mortality similar in degree to that of tobacco smokers (60). In China, coal smoke derived from home heating was associated with high reporting of persistent cough and phlegm (61).

Recently, Orozco-Levy et al have evidenced that biomass fuel may be a risk factor for COPD also in Europe. In their Spanish case-control study in women, exposure to wood or charcoal smoke was associated with COPD after adjusting for age and smoking. Wood or charcoal alone independently increased the risk of COPD (OR 1.8 and 1.5, respectively), but only the combination of both was statistically significant. The association between length of exposure and COPD suggested a dose-response pattern (53).

Mould/dampness

Building dampness may lead to emission of odorous or irritation compounds from microorganisms or chemical degradation of building materials, such as formaldehyde (VOC).

Reported prevalence rates of home mould/dampness range widely around the world: from 10 to up 50% (62).

There is evidence that long-term exposure to mould/dampness is linked to higher risk for cough, phlegm, or dyspnoea, in adults. Dales et al, in Canadian adults, found that dampness/moulds were associated with respiratory symptoms, including cough, phlegm, or dyspnoea, with an OR of 1.62 (1.48-1.68)(63).

The Institute of Medicine (IOM) of the National Academy of Sciences has published, in 2004, a critical review of the scientific literature pertaining to the association of indoor dampness and mould contamination with adverse health effect (64). Recently, through a quantitative meta-analysis of the studies reviewed by IOM, Fisk et al estimated the pooled OR for cough in adults to be 2.10 (1.27-3.47) (65).

Table 4 reports details of the studies considered in the meta-analysis. All the studies have been performed in European Nordic countries. Another study performed on Swedish adults found, by meta-analysis, that an exposure of at least 3 years to damp or mouldy odour at home was associated with persistent cough with OR ranging from 1.32 to 5.86 (95%CI from 1.22 to 6.19)(70).

Table 4. Association between mould/dampness at home and cough/phlegm in adults (OR=odds ratio, CI=Confidence Interval).

Author - *Source* (sample)	**Country**	**Exposure**	**Disorder**	**OR**	**95% CI**
Brunekreef B, *Allergy 1992* (66)	Netherlands	damp	Chronic Cough: men	2.56	1.94-3.38
			women	1.75	1.30-2.36
			Chronic Phlegm: men	2.56	1.94-3.38
			women	1.66	1.16-2.38
Gunnbjornsdottir MI *Respir Med 2003* (youg adults)(67)	Sweden	visible mould/ water damage	Chronic Cough	2.23	1.24-4.00
Koskinen OM, Eur Respir J 1999 *(68)*	Finland	mould	Cough	1.60	1.01-4.01
Pirhonen I, *Eur Respir J 1996* (69)	Finland	mould/damp	CB	1.51	0.96-1.35
			Cough	1.37	0.99-1.88
			Phlegm	1.36	1.01-1.85

CB=Chronic Bronchitis.

Gas/kerosene fuels

Some studies have evidenced associations of COPD with gas/kerosene fuel use for both heating or cooking. Gas/kerosene combustion mainly produce nitrogen dioxide and carbon monoxide. In UK, decrements in FEV_1 (-70mL) and in FVC (-35mL) have been observed in young adults using gas fuel when compared to those using electricity for cooking (71). In Poland, never smoker elderly women exposed to high gas cooking showed an elevated risk for dyspnoea (OR 7.16, 5.02-10.2) (39). In US, kerosene heaters use in never smoking women living in nonsmoking households was associated with increased cough (OR 1.05, 1.01-1.09)(72). In Italy, in a rural general population sample, the use of bottled gas for cooking, resulted related to higher risk of chronic cough in males (OR 1.66, 1.12-2.46) and dyspnoea in males (OR 1.81, 1.15-2.85) and females (OR 1.45, 1.00-2.10)(73). The association between chronic cough and use of bottled gas, instead of natural gas, was also confirmed in Italian male nonsmokers of a urban general population (OR 2.82, 1.12-7.10); the presence of stoves for heating (mainly non-natural gas stoves) inside the home resulted a risk factor for attacks of shortness of breath in nonsmoker women, when compared to those who lived in dwellings with central heating (stoves outside the home)(OR 1.72, 1.11-2.65)(74).

Objectively measured indoor pollutants and COPD

As above reported, few studies on the relationships between COPD and indoor pollution were based on direct measurements of pollutants concentrations, except for specific occupational exposure. In Mexico, non smoking rural women, long-term exposed, when cooking, to peaks of PM_{10} > 2.6mg/m^3 , showed a borderline significantly higher risk for having FEV_1/FVC <70% and FEV_1<80% predicted (OR 3.5, 0.94-16.3) than those exposed to lower concentration (75). In China, in those exposed to elevated indoor PM_{10} level, higher prevalence of chronic cough and phlegm (76) and adverse effects on the lung function have been observed (77). Effects on lung function have been found in Italy, too. In a general population of adults, the exposure to high concentration of $PM_{2.5}$ resulted in both increased maximum amplitude (OR 1.38, 1.24-1.54) and diurnal variation (1.37, 1.23-1.53) of peak expiratory flow (78).

Occupational exposure

A brief comment has to be devoted to specific occupational exposure. Even if it involves only specific groups of persons and can not be defined as common indoor air exposure for the general population, it often occurs in indoor environments. Occupational exposure is an important risk factor for COPD independently of tobacco smoke, and several studies report a causal association between specific work-related exposures and COPD. Dust or chemical agents to whom some categories of workers are exposed result in inflammation, a key factor in the pathogenesis of COPD. Chronic inflammation throughout the airways, parenchyma, and pulmonary vasculature are hallmarks of the disease process and lead to the pathologic changes characteristic of COPD (79). Blanc and Toren (80), in their recent review, estimated a population attributable risk (PAR) of 15% due to occupational factors, when the outcome analyzed was either chronic bronchitis or airflow obstruction. Thus, they have confirmed the figures previously published by the ad hoc committee of the ATS (81). In Spain, among the workers in the textile industry, lung function impairment resulted related to exposure duration, being independent of the effect of smoking. (82). Data from the ECRHS Study showed that occupational exposures to dust/fumes, vapours, or gas were risk factors for chronic cough/phlegm (relative risk ratio (RRR) 1.47, 1.31-1.65) and for COPD (by GOLD criteria, 1.62, 1.24-2.12), also after adjustment for sex, ETS exposure, smoking status, socio-economic status, and respiratory infection (83). The European Farmer Study (Denmark, Germany, Switzerland, and Spain), found a COPD prevalence of 17% in never-smoker farmers working inside animal confinements buildings, and higher risk for having COPD in subjects highly exposed to indoor dust (OR 6.6, 1.1-39.5) (84). Finally, in Poland, among the workers in a pesticide producing factory, chronic bronchitis/emphysema or COPD (by GOLD criteria) was more prevalent in exposed to pesticides than in unexposed ones (19.3 vs 3%)(85).

COPD Exacerbations

Acute exacerbations that increase both morbidity and mortality are common among the subjects with diagnosis of COPD. Exacerbations are accompanied by increased specific symptoms (i.e. dyspnoea or phlegm), and frequently require medical intervention or hospitalization. Studies report significant relationships between acute

exacerbations of COPD and exposure to increased levels of outdoor particulate pollution. Considering that indoor levels of PM may be several fold higher than outdoors, high indoor levels of PM might be associated with COPD exacerbations, too. Indeed, among Spanish COPD patients, ETS - one of the major sources of indoor PM - resulted associated with increased hospital readmission for COPD exacerbations (OR 1.63, 1.04-2.57)(86). A similar result was found in a study on US never smoker adults, who were more likely to report exacerbation of chronic respiratory disease (including chronic bronchitis and emphysema) when they were exposed to ETS (OR 1.44, 1.07-1.95) (87).

CONCLUSION

In spite of increased COPD prevalence (and its predicted increasing in the next years), and the evidence that other risk factors than smoking may be associated to COPD development, we found relatively few studies that assessed the association between COPD and common indoor air pollution in adult general population, except for studies on ETS and, in developing countries, biomass combustion exposure. Both these exposures prove to be risk factors for non-smoking related COPD. It would be important to improve awareness on adverse health effects possibly associated with biomass combustion-related air pollution in developed countries among others because of the increasing interest for wood and other biomasses as potential alternative energy sources.

Mold/dampness exposure results associated to symptoms/signs, which may be related to the presence of COPD or its development.

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Cardiovascular effects of indoor air pollutants

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In the last decades, research on the causes of cardiovascular disease has made great progress. Multiple pharmaceutical and surgical approaches have been devised to prevent, treat, or otherwise manage heart disease, yet it remains the leading cause of death both in Europe and United States. There are important gaps in the understanding the leading causes of cardiovascular disease and the underling pathological mechanisms. Prevention is likely to provide the most effective gains against highly unpredictable events, such as acute myocardial infarction, stroke or arrhythmia, so it is essential to identify the preventable and the modifiable causes of heart disease. Lifestyle choices such as smoking, diet, and exercise are viewed as the most important factors, but in the past years many studies has focused on the contribution of air pollutants in the onset and/or exacerbation of cardiovascular disease.

DEFINITION OF CARDIOVASCULAR DISEASE

Cardiovascular disease (CVD) is a broad term used to describe a range of diseases that affect heart and the circulatory system. Heart disease develops as a result of complex interactions between genes and environment. The most frequent forms of CVD are coronary heart disease and stroke, and other forms include hypertensive heart disease, arrhythmia and heart failure.

EPIDEMIOLOGY, TIME TREND AND SOCIAL COST

Cardiovascular disease is the leading cause of death in the industrialized world: CVD accounts for over 4.35 million deaths (49% of all death) each year in Europe and over 1.9 million deaths (42%) in the European Union (EU). The most common forms of cardiovascular disease are coronary heart disease (CHD) and stroke that are by themselves the two most common causes of death in the EU: accounting respectively for over 744,000 (17%) and 490,000 (11%) deaths in the EU each year. CVD mortality, incidence and case fatality are falling in most Northern, Southern and Western European Countries but either not falling as fast or rising in Central and Eastern European countries.
Overall CVD is estimated to cost the EU economy €169 billion a year. Of the total cost of CVD in the EU, around 62% is due to health care costs, 21% due to productivity losses and 17% due to the informal care of people with CVD (European

cardiovascular disease statistics 2005 edition
http://www.heartstats.org/uploads/documents%5CPDF.pdf).

TRADITIONAL RISK FACTORS

The seminal Framingham Heart Study framed determinants of heart disease as "risk factors" that can quantitatively predict cardiovascular disease [1] [2]. Major risk factors for CVD could be classified in fixed and modifiable. Fixed risk factors are age (older than 65), gender (male) and heredity (including race). Factors that could be modified are hypertension, high blood cholesterol levels (in particular low-density lipoprotein), diabetes mellitus (especially adult-onset or Type 2 diabetes), obesity and overweight, cigarette smoke, physical inactivity.

Besides major risk factors, other exposures, like stress and high alcohol intake (called contributing risk factors) have been associated with increased risk of cardiovascular disease, but their significance and prevalence have not yet been precisely determined.

OTHER FACTORS that could be involved in CVD determination and/or exacerbation: the role of environment

The so called major risk factors identified in the frame of Framingham Heart Study account for a major portion of but not for the total CVD risk [3, 4]. Many patients suffering from heart disease have no established risk [5], suggesting that quantitatively important determinants of CVD are currently unknown [6]. Moreover, the identification of modifiable risk factors, such as smoking and diet, fosters the perception that the environment significantly influences cardiovascular health. This view is further reinforced by studies showing that CVD rates differ 5- to 100-fold among population groups of similar genetic background. These rates change quickly within the same ethnic group, and they increase when populations migrate from low to high-risk environments [7, 8].

Despite these studies, our understanding of environmental influences has been limited to lifestyle choices such as diet, smoking, and exercise, and it is only in the last few years that disparate lines of evidences have congealed into a coherent idea that environmental exposure to pollutants and chemicals contribute to CVD risk [9-11].

Indoor air pollutants that have been associated, or could be related, to an increase risk of CVD are secondhand smoke, carbon monoxide, particulate matter, ozone, nitrogen oxides, carbon monoxide and sulphur dioxide.

Sources of indoor air pollution are both indoor and outdoor. While many studies have been conducted on carbon monoxide and secondhand smoke effects on heart, less scientific work has been done on CVD risk related to exposure to indoor particulate matter and gaseous pollutants other than CO. Several studies have shown some link between outdoor PM and gases exposure and cardiovascular disease mortality and morbidity [10].

Secondhand smoke

Secondhand smoke (SHS), also known as environmental tobacco smoke (e.g. spousal smoking, cohabitant smoking, work exposure), is a complex mixture of gases and particles that includes smoke from the burning cigarette, cigar, or pipe tip (sidestream smoke) and exhaled mainstream smoke [12].

Many reviews have been published summarizing the epidemiological studies about the association between SHS and increase risk for CVD, here we summarized the most important and recent ones. Law and collegues [13] conducted a meta-analysis of all 19 studies of risk of ischaemic heart disease in lifelong non-smokers who live with a smoker and in those who live with a non-smoker and concluded that people who have never smoked have an estimated 30% greater risk of ischaemic heart disease if they live with a smoker, The Australian 1997 NHMRC Working Party Report [14] reviewed the data from 22 analysis from 16 studies of SHS and CHD, finding a statistically significant increase in the risk of coronary events in nonsmokers exposed to SHS. The Californian 1997 CalEPA Report [15] considered 10 cohort studies and 8 case-control studies of SHS and CHD and concluded that epidemiological data in western and eastern countries are supportive of a causal association between SHS exposure from spouses and CHD mortality in nonsmokers, in both genders. The U.S. 2001 Surgeon General's Report *Women and Smoking* [16] reviewed 10 cohort and 10 case-control studies concluded that data from these studies support a causal association between SHS and CHD mortality, morbidity and symptoms. The U.S. 2006 Surgeon General's Report *The Health Consequences of Involuntary Exposure to Tobacco Smoke* [17] reviewed 9 cohort and 7 case-control studies (between June 1998 and April 2002) concluded that the evidence is sufficient to infer a causal relationship between exposure to SHS and increase risk for CHD morbidity and mortality.

All these reviews concluded that the estimate risk for CHD related to SHS is about 25-30 percent and is within range of risk estimates observed for active smoking and CHD[14, 15, 17] [16]

In the 2006 USDHHS report were also reviewed 6 studies (4 case-control, 1 cross-sectional and 1 cohort) about the association between SHS and risk of stroke, and 12 studies about the link between SHS and subclinical vascular disease, particularly carotid arterial wall thickening. The conclusion was that the analysed studies were "suggestive but not sufficient to infer a causal relationship between exposure to second hand smoke" and an increased risk of stroke and atherosclerosis [17].

Carbon monoxide

See the paper *Health effects of carbon monoxide intoxication* by P. Carrer et al. on page 187 of this proceedings.

Particulate matter

Particulate matter (PM) is a complex mixture of airborne solid particles and liquid droplets (aerosols) that vary in size and composition, depending upon the location and time of its source. PM is generally divided, according to the aerodynamic diameter

(D_a), into PM_{10} (D_a < 10 μm), $PM_{2.5}$ (D_a < 2.5 μm), ultrafine particles (UFPs; D_a < 100 nm). Despite its modest contribution to overall volume, the ultrafine fraction represents the largest number of particles and, therefore, presents the largest surface area.

Indoor sources of PM include fuel/tobacco combustion, cleaning operations and cooking [18]. Moreover, fine and ultrafine particles may be formed by reactions between ozone and some VOCs (the so called *indoor chemistry*), in particular terpenes. The highest terpene concentrations also produced high particle levels [19-21]. Particles from outdoor air may contribute to particle load in indoor air, and exposure studies carried out in the United States and Europe showed that particles in outdoor air contributed substantially to personal exposures and to temporal variation in personal exposures, *also in the indoor environment* [22].

The concern about indoor particulate matter cardiovascular effects arises from the epidemiological evidences of health effect of exposure to PM. During the past 15 years, the magnitude of evidence and number of studies linking outdoor air pollution to cardiovascular diseases has grown substantially [23, 24] and there is concern that the association of airborne particles (PM_{10} and $PM_{2.5}$) with adverse cardiovascular outcomes is causal, as summarized in a review by a committee of the American Heart Association [10].
Long-term exposure to $PM_{2.5}$ have been demonstrated to be independently related to cardiovascular mortality in general [25], and in particular to mortality for ischemic heart disease, arrhythmia, heart failure and cardiac arrest [26].

Short-term effects of PM_{10} exposure include an increase in the overall cardiovascular mortality [27, 28]. Observations in Europe [29, 30] and North America [31, 32] have demonstrated higher rates of hospitalizations for all cardiovascular causes. Direct associations have also been identified with respect to incidence of ischemic heart disease, arrhythmias, and heart failure. Elevations in air pollution have also been associated with increased blood pressure during a prolonged air stagnation episode in Europe[33]. Finally, recent studies from Seoul, South Korea [34], Taiwan [35] and Kuopio, Finland [36] have reported higher incidences of ischemic strokes in direct relation to changes in ambient particle concentrations. In summary, these findings imply that short-term elevations in ambient particle levels are capable of evoking cardiac arrhythmias, worsening heart failure, and triggering acute atherosclerotic/ischemic cardiovascular complications.
To date, there have been only a limited number of studies on the association of measures of ultrafine particles with risk of cardiovascular effects [37-42]. The available literature suggests that ultrafine particles may induce cardiovascular health effects immediately, with a 2–4-day lag, and in association with cumulative exposures [18].

Gaseous pollutants

Gaseous pollutants, other than carbon monoxide, that could affect cardiovascular system are ozone, nitric dioxide and sulphur dioxide.

Ozone (O_3) and other photochemical oxidants are pollutants that are not directly emitted by primary sources. Rather, they encompass a group of chemical species formed through a series of complex reactions in the atmosphere driven by the energy transferred to nitrogen dioxide (NO_2) molecules when they absorb light from solar radiation. In most buildings indoor ozone has been transported from outdoors [43]. Indoor ozone concentrations track outdoor concentrations with a slight time lag that depends on the air exchange rate.

There is solid evidence that ozone acutely increases morbidity [18]. To date, data about cardiovascular effects of ozone exposure are poor also because in studies of acute responses to pollutants in humans it is generally not possible to separate effects due to peaks in PM concentrations from those that may be due to ozone. In a review presented in the Air Quality Guidelines of WHO – Europe 2005, 10 of the 15 reviewed studies, focusing on cardiovascular diseases, showed no significant effects of ozone. In addition, there is no clear positive effect of ozone on any of the particular end-points evaluated (myocardial infarction, sudden death, stroke, congestive heart failure and peripheral arterial diseases). Thus, on the basis of the available information, it is clear that the effects of ozone on cardiovascular morbidity need further evaluation [18]. However a recent analysis of the link between ambient air pollution and the risk of hospital cardiac readmissions of MI survivors suggests that the strength of associations with same-day CO, O_3, or NO_2 was similar to that for PM_{10} (von Klot S 2005) suggesting a significant contribution of ozone among gaseous co-pollutants.

Nitrogen dioxide (NO_2) is a reddish brown gas with a characteristic pungent odour. Nitric oxide spontaneously produces the dioxide when exposed to air. Nitrogen dioxide gas is a strong oxidant, and reacts with water to produce nitric acid and nitric oxide. Significant human exposure to NO_2 can occur in non-occupational indoor settings [44, 45]. Gas-burning appliances, such as unvented furnaces and stoves, are the principal sources of indoor NO_X, although kerosene space heaters and tobacco smoke may also play a role. [46]. In urban areas, infiltration of ambient NO_2 from vehicular emissions may also influence indoor exposures.

Epidemiological evidences of cardiovascular effects of NO_2 exposure proceed form studies on outdoor air pollution. Moreover, it is very difficult to differentiate the effects of nitrogen dioxide from those of other pollutants in epidemiological studies.

Short-term effects of NO_2 exposure have been investigated in time-series studies on mortality and morbidity in Europe and North America [28, 29, 31, 47-60]. These studies suggest that daily concentrations of nitrogen dioxide are significantly associated with increased cardiovascular mortality. Moreover, the results of time series include an increase in mortality for cardiovascular disease, and in hospital admissions for heart failure, arrhythmia and ischemic heart disease. Controlling for other pollutants at times lowers the effect estimates and at others makes them not statistically significant, and this makes the conclusions less clear. To date, no cardiovascular long-term effect of NO_2 has been demonstrated.

Sulfure dioxide (SO_2) is a colourless gas that is readily soluble in water. Sulfur dioxide is derived from the combustion of sulfur-containing fossil fuels. In nonoccupational

settings, SO_2 is generally found at substantially lower concentrations indoors than outside; however, the use of kerosene space heaters can generate significant indoor concentrations.
Literature about cardiovascular effects of SO_2 is poor, and it prevalently include studies on outdoor air pollution health effects. A review of literature on Health effects of outdoor air pollution in developing countries in Asia [61] is suggestive for a positive association between SO_2 levels and hospital admission for cardiovascular disease, in studies from Hong Kong. The European APHEA 2 project single pollutant models resulted in positive and significant sulfur dioxide risk estimates for all of the cardiac outcomes except stroke. However, these estimates were reduced when carbon monoxide, nitrogen dioxide, black smoke or PM_{10} were included in the model. The authors noted that sulfur dioxide could be a surrogate of urban pollution mixtures that in some cases is more strongly associated with cardiovascular hospital admissions than particles [62-65]. In an analysis of morbidity after the step-change in ambient sulfur dioxide concentration in Hong Kong, Wong et al. [66] concluded that for sulfur dioxide concentrations in the 5–40-μg/m3 range in Hong Kong, there were non-threshold and nearly linear relationships between sulfur dioxide on the one hand and cardiac admissions on the other, but no trends for ischemic heart disease.
Moreover, the influence of SO_2 levels on PM_{10} risk estimates has been investigated in the U.S. NNMAP [52] (re-analysis by Schwartz et al in 2003 [67]). The authors concluded that there was little evidence of PM_{10} effects confounded by sulfur dioxide.

IDENTIFICATION OF SUSCEPTIBLE POPULATION SUBGROUPS

People who already have heart disease are at especially high risk of acute events if exposed to SHS [17].

It is now reasonably well established that both short-term and chronic air pollution (including PM and gaseous pollutants) exposures are related to cardiovascular diseases. Whether there are specific individuals or subsets of patients at increased relative risk is less well documented. Some observations have suggested that people suffering from cardiovascular diseases are more vulnerable to particles and NO_2 and persons suffering from asthma and other respiratory diseases are more susceptible to particles [18, 61, 68, 69]. Morover, the elderly [25, 28, 70-72] and those with less than a high school education (low socioeconomic status) may be particularly susceptible populations [71, 73]. According to a few recent studies women gender seems to be more prone to cardiovascular effects of PM than men [74, 75].

CONCLUSIONS

Environmental cardiology is a emerging field of research. The identification of modifiable risk factors for cardiovascular disease such as smoking and diet, supports the perception that the environment significantly influences cardiovascular health. The indoor environment represents an important microenvironment in which people

spend a large part of their time each day, so that exposure to cardio-toxic indoor air pollutants could have a role in the cardiovascular etiopathology.

There are consistent evidences that SHS exposure is associated with increase risk of cardiovascular disease, in particular CHD (similar risk estimates observed for active smoking).

Cardiovascular effects of particles, in particular PM_{10} and $PM_{2.5}$, have been suggested in a consistent number of studies on outdoor pollution, and short-term elevation of particles seem to evoke cardiac arrhythmias, to worse heart failure, and to trigger acute atherosclerotic/ischemic cardiovascular complications. As highlighted by Hänninen and Jantunen in a recent letter to the Journal of Epidemiology and Community Health, the observed seasonal variation in mortality due to particles could be due to the variation in the infiltration of outdoor air particles indoors, that mostly depends on ventilation via open windows[76].
More research is needed to identify the role of the ultrafine fraction.
Cardiovascular toxicity of gaseous pollutants, i.e. ozone, nitrogen dioxide and sulfur dioxide, has been investigated in the outdoor environment. To date, literature about cardiovascular effects of the exposure in indoor environments to these pollutants is too poor, and no conclusions could be made.

All together, these studies suggest a potential role of indoor particles, both generated indoor or infiltrated from outdoor, in the causation and/or exacerbation of cardiovascular disease. Additional studies on gaseous pollutants effects on cardiovascular system are much needed and more refined epidemiological studies on indoor pollutants effects on cardiovascular system are required.

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Quantitative estimation of lung cancer deaths attributable to passive smoking from spousal exposure in Europe

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INTRODUCTION

Lung cancer is the most common cancer in the world and accounts for 12.3% of all new cancer in Europe. About 375,000 new cases of lung cancer were estimated for Europe in 2000; 303,000 in men and 72,000 in women. The number of deaths was about 347,000 (280,000 in men and 67,000 in women). However, there are substantial differences in incidence of lung cancer in the different regions and populations within Europe (Tyczynxki, 2003). Estimates for the year 2000 indicate that the highest age-standardized incidence rates in men (per 100,000 inhabitants) are in Hungary (95.5), Croatia (82.5), Bosnia Herzegovina (82.2) and Yugoslavia (80.9). The lowest rates are in Sweden (21.4), Iceland (31.5), Portugal (33.9) and Norway (35.1). In women the highest rates are observed in Denmark (27.7), Iceland (23.8), Hungary (22.6) and the UK (21.8). In women, the lowest incidence rates are observed in Spain (4.0), Belarus (5.0), and Portugal (5.5).

There are also differences in temporal trends. In men, lung cancer mortality is declining in Northern and Western Europe (UK and Finland), although it is already low and fairy stable in Sweden and Norway. In Central and Eastern Europe, however, lung-cancer mortality is increasing. In women, there was high and increasing mortality in the UK until the end of the 1980s. Since then, however, a plateau has been reached and rates have started to decline. In Sweden and Norway, mortality has been increasing during the past 25 years, although it is still much lower than in the UK. In Southern Europe, mortality from lung cancer is either quite low and stable in countries like Greece, or increasing at a moderate rate in Italy and Portugal.

Epidemiological studies indicated cigarette smoking as the predominant cause of the disease, but there are sound scientific data that air pollution, both indoor and outdoor, may cause lung cancer. The lung cancer risks associated with indoor and outdoor air pollutants need to be considered in the context of cigarette smoking, that is the leading cause, and other causes of lung cancer. Workplace exposures contribute substantially, either independently or by modifying the risk of smoking. Indoor air is contaminated by multiple pollutants generated by combustion sources, biological sources, gaseous pollutants released from household products, furnishings and building materials, and by entry of pollutants in outdoor air. These pollutants consist of a number of carcinogens, including several that have been linked to lung cancer, such as tobacco smoke (ETS), radon, asbestos and other fibers.

Environmental tobacco smoke (ETS) indicates the mixture of sidestream smoke and exhaled mainstream smoke that contaminates indoor air when smoking is taking place. The inhalation of ETS by nonsmokers is generally referred to as involuntary or passive smoking. The exposures of involuntary and active smoking differ quantitatively and, to some extent, qualitatively. Nevertheless, tobacco smoking in indoor environments increases levels of respirable particles, nicotine, polycyclic aromatic hydrocarbons, carbon monoxide, acroleine, and many other substances. Measurements of components of tobacco smoke in public and commercial buildings, various workplaces, and residences have shown widespread contamination by ETS. Studies using biomarkers of exposure including nicotine and its metabolite, cotinine, have further shown that ETS components are inhaled and absorbed by nonsmokers.

The adverse effects of exposure to environmental tobacco smoking (ETS) are well established (Office of Environmental Health Hazard Assessment, 2005). Several well-conducted studies have shown higher risk of coronary artery diseases, lung cancer, respiratory diseases and stroke associated with exposure to passive smoke. ETS exposure could occur in private households, work and public places. Several countries have enacted legislation that prohibits smoking in work and public places, but the interest towards policies to address exposure in households is more limited. Studies conducted in the '90 have elucidated the relationship between exposure to ETS from spouse and lung cancer risk and relative risks (RR) have been provided, resulting in 1.36 for men and 1.22 for women (Boffetta et al, 1998).

The aim of the present work is to examine the overall impact of ETS exposures on lung cancer mortality in 25 European countries. The resulting figures are helpful to quantify the overall burden of indoor environment on cancer mortality.

METHODS

We considered the population of the 25 EU countries aged 35+ years in the year 2000. Statistics of lung cancer mortality by gender were available from an extensive publication reporting estimates of the health burden due to active smoking (Peto et al, 2006).

We calculated the number of lung cancer cases attributable to ETS from spouse, i.e. the Proportional Attributable Risk (PAR), by applying the following formula:

$$PAR = Pe\,(RR-1) / Pe(RR-1) + 1$$

where :

Pe = *Prevalence of people exposed to ETS from spouse*.

RR= *Relative Risk of having lung cancer being exposed to ETS from spouse*

Country specific data on prevalence of exposure to ETS from spouse were estimated. We applied the proportion of exposure to ETS from spouse derived from the large European case-control study (Boffetta et al, 1998) (63% for women and 13% for men) to the prevalence of smoking of each country (WHO, 1999-2001). A correction factor

to consider the proportion of married subjects and that smokers tend to marry smokers was applied. The relative risks applied (and 95% Confidence Intervals, CI) were those resulting from the same study, namely 1.36 (1.02-1.82) for males and 1.22 (1.12-1.32) for females. These values are consistent with a comprehensive meta-analysis: 1.24 (95% CI: 1.13-1.36) (Hackshaw, 1997)

The number of attributable cases was derived by multiplying the PAR by the number of lung cancer cases "*not attributable to active smoking".* These figures were derived from the publication referenced above (Peto et al, 2006) as the difference between total lung cancer deaths and smoking attributable lung cancer deaths. All the calculations were made separately for men and women.

RESULTS

Table 1 and 2 illustrate the relevant figures for males and females, respectively. The total EU25 population aged 35+ was more than 118 millions males and about 132 millions females. A total of 170,343 lung cancer deaths were observed in the year 2000 in men and 52,847 in women. Lung cancer deaths not attributable to smoking were about 15,000 in men and 18,500 in women. Active smoking was more prevalent in Central-Eastern Europe for males and Western and Northern Europe for females.

A total of 916 (54-1928) lung cancer cases due to exposure from spouse were estimated for males and 2,449 (1,424-3,357) for females. The largest burden is for Western and Southern Europe for males (especially Germany and UK) and females (especially Germany, Italy, and France). These figures correspond to an attributable proportion of 0.5% in males and 4.6% in females.

DISCUSSION

This indicates that exposure from passive smoking exposure at home is relevant factor contributing to lung cancer etiology in Europe. In this study, we considered only exposure from spouse and no other individuals in the family. In addition, ETS exposure at work and in other places was not considered. Our figures are then an underestimate of the true attributable cases.

As a limitation of the present study, it should be considered that several figures were estimated and they were based on information collected during the nineties. Changes in prevalence of active smoking, changes of smoking behaviors in public places and indoor, changes in family composition may have altered the exposure distribution of the population and thus the number of attributable lung cancer cases. New data should be collected for a more recent period.

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Cancer and cardiovascular effects from exposure to combustion products

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BACKGROUND

The key indoor air exposures leading to cancer and severe cardiovascular consequences are radon, certain volatile organic compounds (VOCs) and incomplete combustion generated particulate matter (PM and PAHs) and carbon monoxide. Of these radon [1] and carbon monoxide [2] are dealt with in focused presentations, PM and VOCs are covered in the current presentation.

Complete oxidation of a hydrocarbon fuel would result in only water and carbon dioxide as combustion products. Combustion of even the simplest fuel, methane (over 90 % of natural gas), however, is a complicated chemical process, let alone combustion of the infinitely more complex solid fuels, coal and wood. Complete oxidation is never achieved even in the most advanced industrial scale boiler plants, which are built upon generations of engineering sciences, are equipped with advanced flue gas cleaning technologies, and employ professional operating teams using a network of sensors and automated feedback control systems.

In comparison, the domestic combustion devices to heat, cook and entertain (fireplaces, incense, candles) represent much simpler technologies, use more heterogeneous fuels, are operated by untrained lay persons, and are hardly ever equipped with any feedback control or flue gas cleaning. Consequently the harmful emissions per generated heat unit of small scale combustion devices are orders of magnitude higher than those of industrial and power generating stations (NO_X is an exception). A fact that highlights the consequences of this difference is that per unit operating time the emissions of particle phase polycyclic aromatic hydrocarbons (PAH [1]) are equally large for a residential woodstove and a coal fired generating station with 10.000 times larger heat output.

The total emissions of combustion particles to outdoor air are orders of magnitude higher than into indoor air, but the intake fraction (*iF*) of the particles emitted into indoor air that is inhaled by people, and could thus harm their health is in the order of $10^{-2}..10^{-3}$, i.e. 100 – 10.000 times the intake fraction of particles emitted into outdoor air [3, 4]. Therefore by far the highest exposures to combustion particles (also vapours and gases) occur in indoor air, and originate from indoor sources.

[1]) PAH:s are characteristic for visible smoke particles from incomplete combustion and contain most of the combustion generated carcinogens which make e.g. tobacco smoke carcinogenic. Polyaromatic organic matter (POM) is an often used broader definition that includes PAH.

COMBUSTION PARTICLES

Combustion processes generate both fine particles (aerodynamic diameter ≤ 2.5 μm, $PM_{2.5}$, measured as mass of the particulate matter per 1 m^3 of air) and ultrafine particles (UFP#, usually measured as the number count of particles < 0.1 μm per 1 cm^3 of air. The count is insensitive to the upper but very sensitive to the lower size limit of the particles counted, typically 0.02 – 0.03 μm).

The compounds that characterise the combustion generated PM are elemental carbon (EC, also black smoke, BS, black carbon, BC), organic carbon (OC, including the previously mentioned PAH and POM). In an indoor and outdoor, urban and rural $PM_{2.5}$ in Hong Kong, the indoor and outdoor concentrations of $PM_{2.5}$ mass and EC were highly correlated but the correlation was much lower for OC. All concentrations were higher in indoor than outdoor air, but the difference was highest (I/O = 1.8) for OC, indicating significant contributions from indoor sources, most likely combustion [5] and cooking.

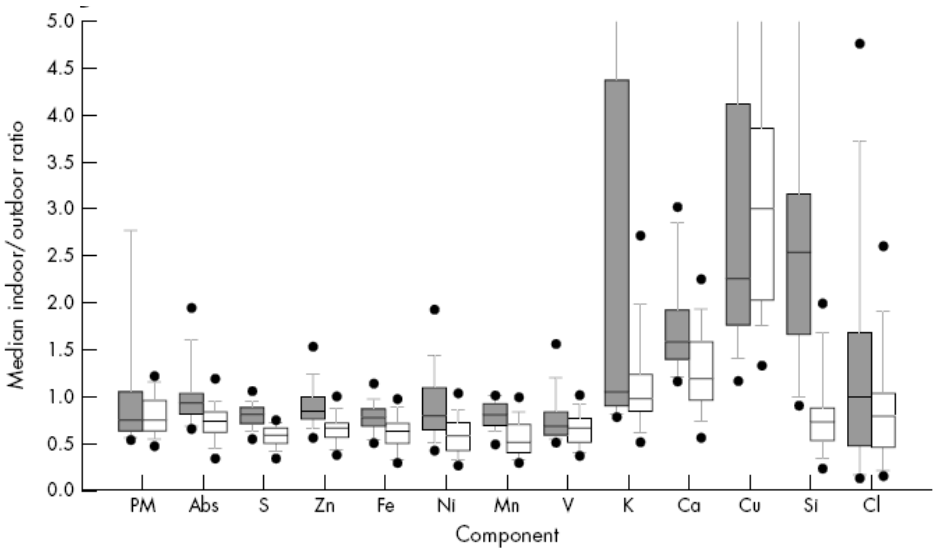

Figure 1. I/O ratios of several elemental constituents of $PM_{2.5}$ in Helsinki and Amsterdam. ULTRA-study [6]

In the ULTRA study in Helsinki and Amsterdam I/O ratios were analysed for a number of $PM_{2.5}$ constituents inside and outside of non-ETS residents. Only Cu was consistently dominated by indoor sources. I would speculate that its apparently omni-present, yet highly variable indoor source is electric machinery: vacuum cleaners, kitchen blenders, etc. In addition, K, indicating wood combustion, was dominated by indoor sources in many residences in Amsterdam and some in Helsinki. Indoor source contributions were also often significant for the indoor concentrations of Ca and Si, which indicate soil dust [6]. In the Western European context and in the absence of smoking, the average contribution of indoor sources to combustion particles is minimal. Instead the main indoor source contributions are mineral dusts and – surprisingly – phosphate particles, probably originating from detergent residues in clothing [7,8].

In a regression modelling study, using *EXPOLIS* data from six European cities, the presence of indoor combustion sources explained 16 % (smoking) and 14 % (gas stove) of the variation of the 48 h indoor concentrations of $PM_{2.5}$ [9]. In a French regression modelling study indoor combustion processes explained 36 % (smoking) of the variation of the $PM_{2.5}$ exposures [10].

In a study focused on the short-term indoor PM concentrations, peak concentrations of 30 - 60 μg/m^3 for $PM_{0.02-0.5}$ and 10 - 300 μg/m^3 for $PM_{0.7-10}$. were observed in kitchen during oven cooking, while cleaning activities (8 μg/m^3 for $PM_{0.02-0.5}$ and 30 μg/m^3 for $PM_{0.7-10}$) and mobility of the occupants (4 μg/m^3 for $PM_{0.02-0.5}$ and 20 μg/m^3 for $PM_{0.7-10}$) contributed much less [11]. In the US $PM_{2.5}$ exposure study, RIOPA, meat cooking in particular was speculated to be a source of high indoor concentrations of particulate amides, but also other particulate aliphatics and amines were found in high indoor vs. outdoor concentrations [12].

Combustion particles may form a large fraction of indoor air particulate matter – e.g. 1/3 in the *EXPOLIS* sample in Helsinki, but in the absence of significant indoor combustion sources this PM originates from ambient air [8], and exposure to it can be best prevented by modern building structures and ventilation systems [13]. The highest exposures to combustion particles by far, however, occur indoors in the most primitive conditions [14]. Indoor combustion processes may increase the levels of indoor air ultrafine particles by 2, 3 orders of magnitude in comparison to a situation without the source.

VOCs AND CARBONYLS

After CO, VOCs are found in *highest indoor air concentrations*, and are consequently also responsible for the highest long-term population exposures. VOCs are no doubt the most studied indoor air contaminants. In a review of 68 American and European indoor air studies, Brown [15] lists ethanol, 1,1,1-trichloroethane, toluene, limonene, acetone and xylenes as the indoor air VOCs with the highest average concentrations. In Helsinki, Finland, indoor concentrations of acetone, formaldehyde, toluene, xylenes and limonene were the highest [16]. In a study of 3 German cities, the highest average indoor air concentrations were observed for limonene, toluene and pinene [17]. The top rank lists in these different studies are similar, acknowledging that ethanol was only considered by Brown and formaldehyde - not a VOC - was only considered in the Helsinki list.

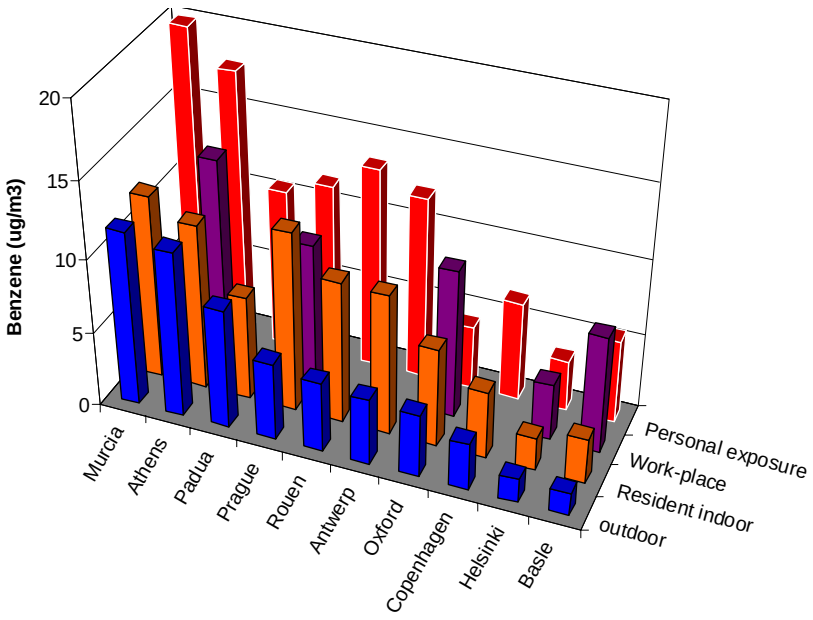

Figure 2. Average personal, indoor (work and home) and outdoor air benzene concentrations in 10 European cities measured in the EXPOLIS [16] and MACBETH [20] studies.

In the German Environmental Survey (GerES II) [18] 74 VOCs were analysed in the personal air (7 days) in a sub-sample of 108 participants representing 36 study locations. The mean exposure to total VOCs was 901 μg/m^3, with 95th percentile as high as 2810 μg/m^3. Mean personal exposure concentrations for the different VOC classes were highest for oxygenated VOCs (308 μg/m^3), followed by aromatics (286 μg/m^3), alkanes (187 μg/m^3), terpenes (98 μg/m^3) and aliphatics (21 μg/m^3) [19].
Some of these VOCs are readily produced in and emitted from incomplete combustion processes (e.g. aromatics, notably benzene, and oxygenated VOCs), others have combustion as one of the sources (e.g. aliphatics, alkanes, aldehydes), and the sources of yet others are clearly non-combustion (terpenes, halogenated). With the exception of the last category and outside of the most obvious cases, it is in practice difficult to define the proportion of the different VOCs that originate from combustion sources. E.g. aromatic VOCs are the main components in many solvents used in household products, although benzene – because it is an IARC classified human carcinogen – is currently found in the solvents only as a low level impurity. Benzene can, therefore be regarded as a useful indicator for the VOCs from combustion sources, and the indoor / outdoor ratio of benzene as an indicator of the contribution of indoor combustion sources.
Comparison of the average indoor and outdoor benzene concentrations in 10 European cities [16, 20], see Figure 2, reveals that the average indoor source contribution is quite small, in the order of 10% or less in Murcia, Athens, Padua, Copenhagen and Helsinki. On the other hand the average residential indoor contribution is in the order of 50% or more in Antwerp, Prague and Antwerp, with particularly high workplace contributions in Basle and Oxford. While the lowest indoor source contributions indicate also low combustion source contribution, it is

unclear how much of the high indoor source contributions originate from combustion processes.

Contributions of indoor sources to indoor exposures

The highest personal exposures to VOCs are often due to high residential indoor concentrations. In the *EXPOLIS* study, e.g., in Helsinki, workplace and home indoor concentrations were the leading factors for 1/3 of the higher than average benzene exposures. [21]
According to Brown [15] the indoor sources contribute of all VOCs the least (½ or less) to benzene, butanal and carbon tetrachloride. In Helsinki the contributions of residential and occupational indoor sources to total personal exposures were highest for aldehydes, terpenes and xylenes, lowest for benzene, toluene and nonane [16].
In the GerES II -study multivariate analysis was carried out to determine and quantify the major sources of personal exposure to various VOCs, with main focus on aromatics. The only significant indoor source for benzene was tobacco smoke, i.e. biomass combustion. [19]

Differences between regions

Between cities: According to the *EXPOLIS* study differences between the average residential indoor air VOC concentrations in 7 European cities were remarkable, and in the non-smoking indoor spaces with hardly any combustion heating or cooking devices in Helsinki, the indoor source contribution to benzene exposure was marginal, and to the other aromatics also small (22, 23)

Table 1. Average VOC concentration differences between 6 European cities, note the high proportion and variation of aromatics, which have the highest contribution from combustion processes (EXPOLIS) (μg/m^3) [16]

VOC Group	Lowest avg.	Avg	Highest avg.
Alcohols	2 (Basel)	25	60 (Milan)
Aldehydes	10 (Basel)	25	63 (Milan)
Alkanes	18 (Helsinki)	48	127 (Milan)
Aromatics	**43 (Helsinki)**	**163**	**463 (Milan)**
Esters	3 (Bas.&Hel.)	16	70 (Milan)
Halogens	2 (Bas.&Hel.)	28	130 (Milan)
Miscell.	21 (Basel)	59	98 (Mil.& Ath.)

The high average indoor concentrations in Milan in particular were due to a small number of very high levels. The indoor levels reflected the respective outdoor air VOC levels, but were in average 50 % (alkanes, aromatics, exters) ... 150 % (alcohols, halogenated VOCs) higher.

Urban - rural: In a German study outdoor and indoor concentration differences for BTEX-concentrations between urban (High traffic area in the city of Hannover) and rural (in Wedemark, no major traffic nearby) areas were assessed [24, 25]. Although the average outdoor air concentration were about 10 times higher in the urban vs. rural

area, the respective indoor air concentrations of benzene were only about 50% higher in the city. For the other aromatics the average indoor concentrations in the urban and rural areas were even closer. I.e. for BTEX compounds indoor sources all but eliminated the urban-rural indoor exposure differences. The high indoor vs outdoor benzene concentrations in the rural area indicate that combustion is probably the most important source.

Unlike the particulate matter in general, and combustion generated $PM_{2.5}$ in particular, the indoor concentrations of VOCs are typically far higher in indoor than outdoor air. For combustion generated VOCs, benzene as a marker, this difference is, however, the smallest. The presence of indoor sources and inappropriate ventilation may still result in indoor concentrations that are one or two orders of magnitude higher than those in outdoor air.

CONCLUSIONS

- Compared to outdoor sources, benzene and fine PM from indoor sources cause 100 … 10 000 times more exposure per unit mass released.
- Most exposure to benzene and fine PM from combustion sources occurs in indoor environments but – in the absence of smoking - has outdoor origins.
- The contribution of indoor combustion sources to exposure varies greatly, not only between buildings and residences, but also between the European cities.
- The highest and sometimes acutely dangerous exposures to combustion products, however, have indoor origins and occur indoors.

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Combustion sources

Derrick Crump

BRE, May 2007

INTRODUCTION

Combustion processes are an important source of a range of air pollutants as follows;

- Carbon monoxide (CO),
- Nitrogen dioxide (NO_2),
- Sulphur dioxide (SO_2),
- Particulates and associated inorganic and organic chemicals,
- Organic vapours e.g. formaldehyde, acetaldehyde, and benzene.

Sources of these are present in both ambient and indoor environments. In European countries emissions from major anthropogenic sources are controlled by legislation. This includes regulation of industrial emissions (e.g. Directive on Integrated Pollution Prevention and Control, Large Combustion Plant Directive) and emissions from motor vehicles including directives arising from the Auto/Oil programme (e.g. emission limits defined by 'Euro 4' for CO, hydrocarbons and nitrogen oxides from petrol fuelled vehicles, and additionally particles from diesel fuelled vehicles under EC Directive 98/69/EC), and limits on the sulphur content of some liquid fuels and the benzene content of petrol. Under the Air Quality Limit Values Regulations member states are required to take necessary measures to ensure that air quality limit values are not exceeded by defined dates in zones that have a population of more than 250,000. Table 1 summarises objectives for England set out in the Air Quality Regulations. Also, under the EU strategy to combat acidification national emission ceilings have been set for SO_2, nitrogen oxides, volatile organic compounds (VOCs) and ammonia.

The concentrations present in the ambient air provide a baseline for the level of pollutant found indoors as this air enters indoors by processes of infiltration and ventilation. However the concentration indoors will be modified by processes of sorption to surfaces and chemical reaction depending on the chemical and physical properties of the pollutant and internal surfaces. Also sources of these pollutants indoors will result in direct emissions to the indoor environment and resulting concentrations will depend upon rates of emission, dilution by internal space and removal by air exchange with the outside.

COMBUSTION SOURCES

The main combustion sources are summarised for each pollutant as follows:

Carbon monoxide (CO) is a colourless, practically odourless and tasteless gas that is the product of incomplete combustion of carbon containing fuels (Kotzias et al. 2005, WHO 1987). Anthropogenic emissions are responsible for about two thirds of the CO in the atmosphere and natural emissions account for the remaining one third. The most important source in ambient air is exhaust of gasoline powered motor vehicles. Other common ambient sources include heat and power generation, especially when using coal, industrial processes such as carbonation of fuel and the incineration of refuse. Urban CO concentrations vary with traffic density and most cities have peak concentrations that coincide with rush hour traffic.

The most common cause of high exposure of people to CO is the smoking of tobacco and inhalation by the smoker. Gas stoves, unflued gas room heaters and exhaust from vehicles in attached garages are important indoor sources. Faulty domestic cooking and heating appliances inadequately vented to outside air can cause high concentrations. For example in the 16 month period from January 2006 to the end of April 2007 there were 102 reported CO poisoning incidents in the UK. These resulted in 50 deaths and a further 218 injuries (CORGI 2007) although actual numbers of incidents are considered to be higher because of undiagnosed cases and others where blood levels of occupants were elevated, but the source of CO in the building could not be identified. 67% of deaths occurred in the peak heating months of November to February. Many of the incidents were avoidable, the most common causes being faulty gas appliances, installation faults, blocked chimneys and owner error.

Use of gas for heating and cooking is widespread in developed countries; for example the Indoor Air Quality Survey of England involving nearly 900 homes found that 80% of homes used gas as the main heating fuel and 57% used gas for cooking (Coward et al. 2001). 6.5% of homes reported using a gas cooker to provide heating either sometimes or regularly during the two week sampling period. Regular smoking was reported to occur in 33% of the homes and in 8% of homes more than 20 cigarettes per day were smoked indoors. Figures collated for the EU report that 25% of the UK population smoke with the average for the EU being 29% (ASH 2006). Incense burning in public buildings and homes has also been reported to be a significant source of CO. For example Jetter et al. (2002) report the rate of emission of CO from 23 different types of incense rope, cones, sticks, rocks and powder as between 144 and 531 mg h^{-1}. In developing countries in particular solid fuel burning stoves are an important indoor source.

Nitrogen dioxide (NO_2) is a reddish brown gas in colour and a strong oxidant with a characteristic pungent odour (Kotzias et al. 2005). The main ambient sources of nitrogen oxides (NOx) include intrusion of stratospheric NOx, bacterial and volcanic action and lightning. Fossil fuel power stations, motor vehicles and domestic combustion appliances emit nitric oxide (NO) which is a reactive compound that is oxidized to NO_2. There are also some specific non-combustion industrial sources such as welding. Urban outdoor levels of NO_2 vary according to the time of day, the season of the year and meteorological factors. Typically low background levels occur onto

which are superimposed peaks corresponding to rush hour traffic emissions of nitric oxide.

The most important indoor sources of NO_2 include gas appliances and unflued kerosene heaters. Owing to the widespread use of unvented combustion appliances concentrations in homes may considerably exceed those found outdoors. Figure 1 summarises data for two week average concentrations measured in the Indoor Air Quality Survey of England. Concentrations were found to be higher in homes where gas ovens were used for cooking compared with those with only a gas hob (and both groups had higher concentrations than homes using electricity for cooking). NO_2 is also a component of tobacco smoke.

Sulphur dioxide (SO_2) is a colourless gas with a characteristic pungent smell; it is produced by burning sulphur-containing fuels such as coal and oil. In the absence of indoor sources, concentrations of these combustion products would normally be slightly lower indoors than outdoors because of absorption by surfaces inside buildings. However, where indoor sources are present, concentrations can be much higher indoors.

Particulates and associated inorganic and organic chemicals in the air may arise from a wide variety of sources, both natural and related to human activity (DOE 1995). Natural sources consist for example of forest fires, volcanic eruptions, sea spray and the erosion of soil and rocks by wind. Man-made particles result mainly from combustion and industrial processes, the working of soil and rock and from the attrition of road surfaces by motor vehicles.

Particles larger than 1 μm generated indoors are mainly from mechanical processes such as cleaning and physical activity by occupants. Particles in the submicron ranges are generated during combustion as well as from secondary processes such as gas to particle conversion and nucleation or photochemical processes. The main indoor sources of these submicron particles include smoking, cooking, and the operation of gas burners, ovens and electric toasters (Morawska L (2004). A number of studies have investigated rates of emission of particles from combustion sources and for example a typical value for emission from a cigarette would be 10 -20 mg per cigarette, but consideration must be given to the size fraction reported when comparing data (Morawska and Salthammer 2003).

Particles are commonly categorised by size;
PM_{10}: the mass of particles that pass through a size-selective orifice with a 50% collection efficiency cut-off at 10 μm aerodynamic diameter. This fraction represents the "thoracic fraction", i.e. those particles most likely to penetrate the human larynx and into the lungs.
$PM_{2.5}$: the mass of particles that pass through a size-selective orifice with a 50% collection efficiency cut-off at 2.5 μm aerodynamic diameter.
Ultrafine or nanoparticles: particles below 100 nm in aerodynamic diameter and these are usually produced by combustion or by chemical reactions of gases.

Concentrations of particles in the air and particle size distribution can be considered either in terms of particle number or mass. In terms of number the vast majority are in the nanoparticle range. The particle size distribution can change over time as for

example for environmental tobacco smoke (ETS) which is a highly dynamic and reactive mixture (Morawska 2004). Ross et al. (1999) reported that large numbers of ultrafine particles were generated both by the combustion of gas and from cooking processes. In urban outdoor air where motor vehicle emissions are a dominant pollution source, over 80% of particulate matter in terms of number is in the ultrafine range. Outdoor particles contribute significantly to indoor concentrations and the particle number concentration is dominated by the smallest particles. However most of the mass of airborne particles is associated with large particles (Morawska 2003).

The chemical composition of particles is complex (WHO 2000). The outdoor coarse fraction with an aerodynamic diameter of larger than about 2.5 μm is largely composed of soil and mineral ash that are mechanically dispersed into the ambient air. They can be further characterised as coal and oil fly ash, metal oxides of crustal elements, calcium carbonate, sodium chloride, sea salt, pollen, mould spores, plant/animal fragments and tyre wear debris. The composition of the fine fraction includes sulphate, nitrate and hydrogen ions, elemental carbon, organic compounds (e.g. PAHs) particle-bound water and metals such as lead, cadmium, copper, zinc and iron.

Owing to their irregular shape the majority of particles present in an indoor environment have large surface areas which provide an opportunity for particles to serve as sinks for a variety of organic species. Semi-volatile substances occur in the particulate and vapour phases and compounds with very low vapour pressure are adsorbed almost exclusively.

Organic vapours are formed during incomplete combustion of carbon containing substances. Complete combustion would produce carbon dioxide and water but during burning of any organic matter, material or fuel a wide range of volatile organic compounds are formed. In the ambient air of developed countries a number of toxic compounds such as benzene and 1,3-butadiene are regulated to control the amount in air - often through requirements for fuel composition, the mode of combustion and the cleaning of exhaust gases.

In the indoor air there is a multitude of sources such as emissions from construction products, furnishings, office equipment and consumer products (Crump 1995, Salthammer 1999). Burning of fuels for heating and cooking as well as candles and incense are sources of compounds such as formaldehyde, acetaldehyde and benzene (Crump and Gardiner 1989, Madany and Crump 1994). Emission rates from one type of incense (‘Bukhoor’) reported by Madany and Crump (1994) were formaldehyde (4.4 mg g^{-1}), benzene (0.94 mg g^{-1}), toluene (1.0 mg g^{-1}), linalool (1.8 mg g^{-1}), phenethyl alcohol (6.6 mg g^{-1}) and diethylphthalate (3.1 mg g^{-1}). ETS contains several thousand organic compounds including some carcinogenic compounds such as benzene and benzopyrene. It is a mixture of gases and particles generated by combustion of tobacco products including cigarettes, cigars and pipes. Most ETS originates from the smouldering tobacco between puffs, although exhaled mainstream smoke contributes (Nazaroff and Klepeis 2003). Once released to the environment the particles and gases are subject to physical and chemical processes such as deposition, dispersion and reaction that alter the component concentrations, physical form and chemical composition.

A number of studies have shown ETS to be a significant source of benzene in the indoor air, including the Indoor Quality Survey of England. Figure 2 shows data for Scotland where the benzene concentration in the air of 54 homes was measured over a 12 month period during 2005 – 2006 (Crump et al. 2007). 24 of the properties were investigated because they were situated on land contaminated with pollutants, including benzene, resulting from previous use of the site as a gas works. The other 30 (control) homes were located nearby, but not on the historic gas works site. Information about the household characteristics and occupant activities were recorded during the study. There was no significant difference between the benzene concentrations in the homes situated on the contaminated land and the control properties. However the study did show that benzene concentrations were higher in the homes of smokers than those where tobacco smoking did not occur in the home.

IMPORTANCE OF INDOOR SOURCES

An indicator of the significance of indoor rather than outdoor sources for determining the concentration indoors is provided by the indoor to outdoor concentration ratio. In the absence of an internal source the ratio for NO_2 and SO_2 is generally less than unity because these compounds are readily sorbed by internal surfaces whereas for CO it is close to unity because it is not absorbed. The ratio for particles is reported to depend upon particle size as this influences the tendency of particles to deposit on surfaces and, in the absence of indoor sources, is less than unity (Morawska and Salthammer 2003). In the presence of an internal source the indoor outdoor ratio is considerably higher than unity and will be greatest when peak levels are generated by the indoor source such as during cooking or release of environmental tobacco smoke.

The European INDEX project undertook a critical appraisal of the setting and implementation of indoor exposure limits in the EU and identified a high priority list composed of five substances to be regulated in indoor environments (Kotzias et al. 2005). All of these compounds have indoor combustion sources and these are particularly important for CO, NO_2 and benzene and also contribute to concentrations indoors of formaldehyde and naphthalene.

Emissions from indoor combustion sources are controlled by legislation such as that implementing building regulations and product standards as well as by advice given to the public about the safe use of products and appliances. Examples of such controls are;

- Requirements for safe installation and servicing of fuel burning appliances,
- Use of flued appliances to vent fumes to outdoors,
- Extractor fans and hoods for venting cooking fumes,
- Requirements for fuel quality,
- Provision of means for adequate ventilation such as opening windows and trickle ventilators,
- Smoking bans in workplaces and enclosed public places,
- Advice to the public about threat to health caused by tobacco smoking.

The significance of a particular source for determining the concentration of pollutant in air will vary between countries. Ambient air quality is strongly related to the degree of industrialisation and urbanization. Also as the nature of indoor environments will differ in countries with contrasting climates and those with differing cultures and wealth then the significance of indoor sources will vary. For example there are strong differences in the types of fuel used in developing and developed countries, the latter primarily gas (usually with flues to vent combustion products from main sources such as heating appliances) or electricity.

By contrast the majority of households in developing countries, representing about half of the world's population, burn biomass fuels in poorly functioning earth or metal stoves or use open pits, often in an open fire configuration (Balakrishnan et al. 2004). Incomplete combustion in poorly ventilated kitchens produces high concentrations of pollutants. The amount and characteristics of pollutants produced during fuel burning depends on the composition of fuel, combustion conditions (temperature and air flow), mode of burning and shape of the fireplace. Toxic pollutants reported to be released are particulates, CO, NO_2, SO_2, and organics (formaldehyde, acetaldehyde, phenols, pyrene, benzo[α]pyrene, benzopyrenes, dibenzopyrenes, dibenzocarbazoles and cresols). In addition, associated cooking fumes such as from the heating of cooking oils contain potentially harmful chemicals such as aldehydes, ketones, hydrocarbons, fatty acids, alcohols, aromatic compounds and heterocyclic compounds (Hao 2004).

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Table 1. Objectives for the purpose of air quality management in England and Wales

Pollutant	objective
benzene	16.25 µg m^{-3} running annual mean by 31/12/03. 5 µg m^{-3} running annual mean by 31/12/10.
1,3 butadiene	2.25 µg m^{-3} running annual mean by 31/12/03.
CO	10 mg m-3 daily running 8 hour mean by 31/12/03.
lead	0.5 µg m^{-3} annual mean by 31/12/04. 0.25 µg m^{-3} annual mean by 31/12/08.
NO_2	200 µg m^{-3} 1 hour mean by 31/12/05, not to be exceeded more than 18 times per year. 40 µg m^{-3} annual mean by 31/12/05.
Particulates (PM_{10})	50 µg m^{-3} 24 hour mean by 31/12/04, not to be exceeded more than 35 times per year. 40 µg m^{-3} annual mean by 31/12/04.
SO_2	350 µg m^{-3} 1 hour mean by 31/12/04, not to be exceeded more than 24 times per year. 125 µg m^{-3} 24 hour mean by 31/12/04, not to be exceeded more than 3 times per year. 266 µg m^{-3} 15 minute mean by 31/12/05, not to be exceeded more than 35 times per year.

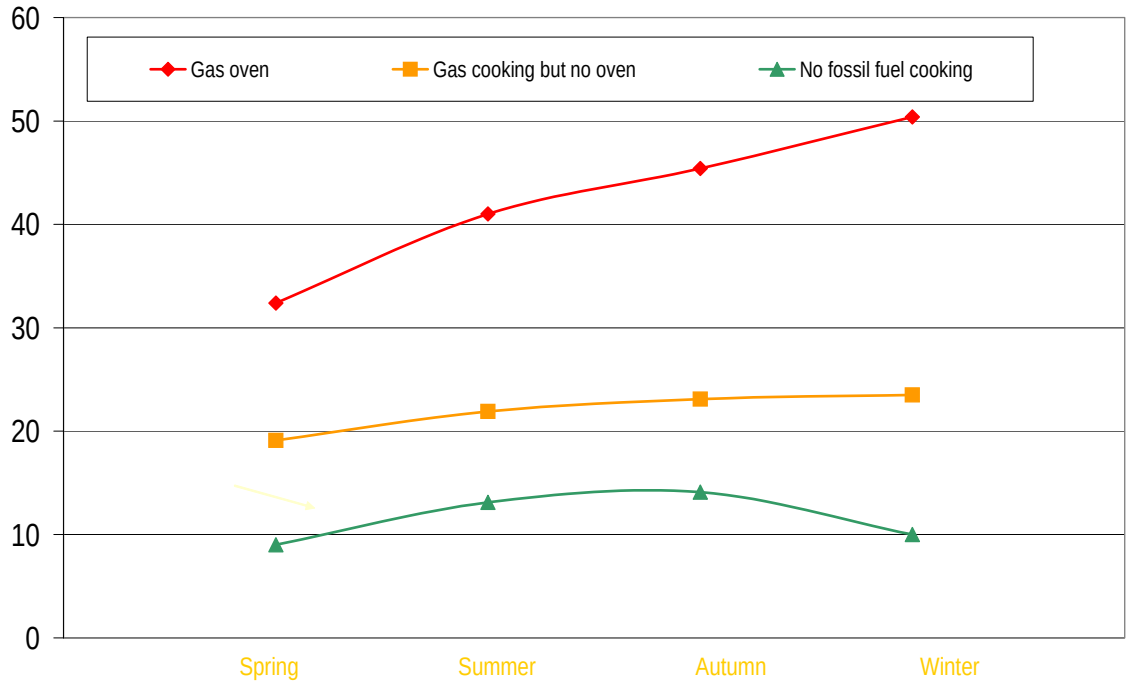

Figure 1. Nitrogen dioxide (µg m^{-3}) in kitchens by cooking type and season in the BRE Indoor Air Quality Survey of England

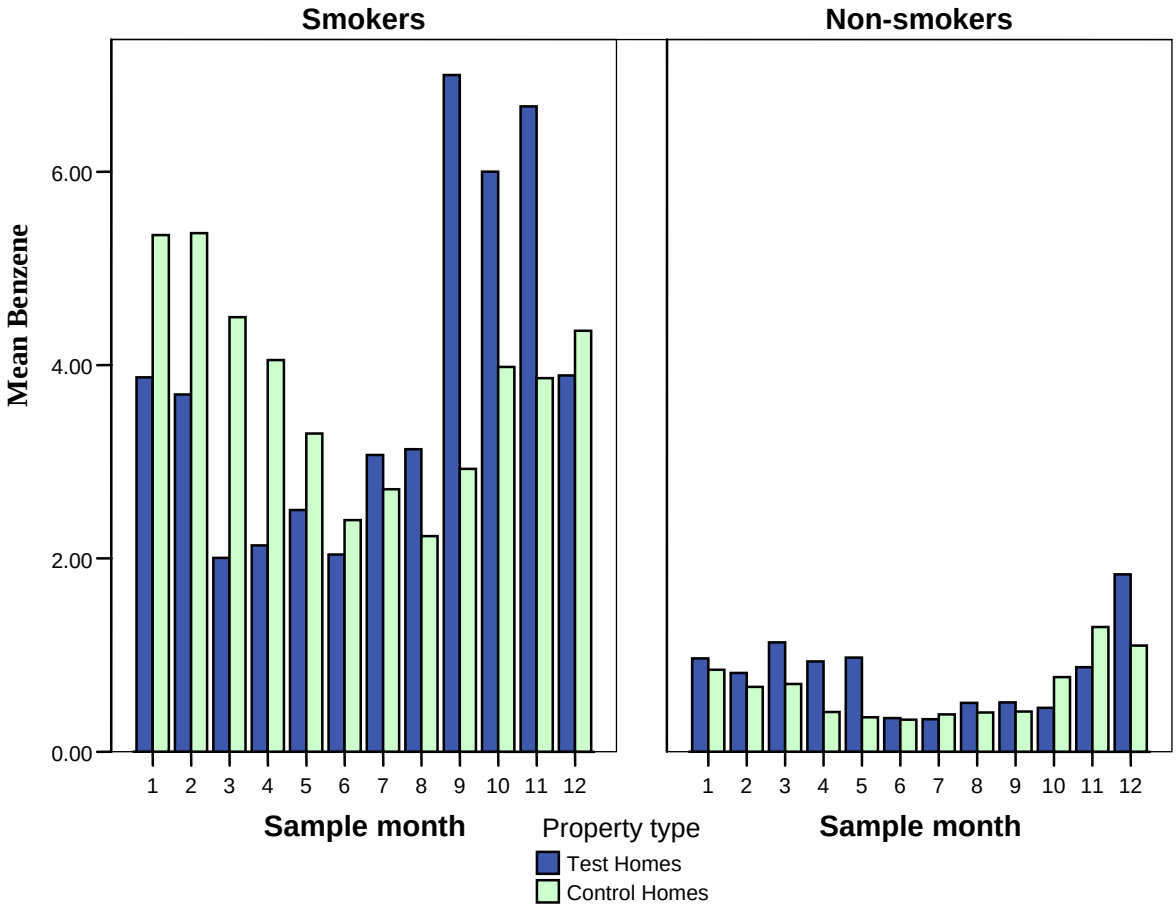

Figure 2. Benzene concentrations (µg m^{-3}) in the indoor air of homes in Scotland situated on contaminated land (test homes) and nearby (controls) categorized by whether occupants are smokers or non-smokers.

In Focus: Health effects of carbon monoxide intoxication

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INTRODUCTION

Carbon monoxide is a colourless, practically odourless and tasteless gas that is poorly soluble in water, but it is soluble in alcohol and benzene. It is a product of incomplete combustion of carbon-containing fuels. Carbon monoxide burns with a violet flame and it is classified as an inorganic compound. It has a slightly lower density than air.

HEALTH EFFECTS

TOXYCOKINETICS

After reaching the lungs, inhaled carbon monoxide diffuses rapidly across the alveolar and capillary membranes. It also readily crosses the placental membranes. Approximately 80–90% of the absorbed carbon monoxide binds with haemoglobin, which causes a reduction in the oxygen-carrying capacity of the blood. The affinity of haemoglobin for carbon monoxide is 200–250 times that for oxygen, while the relative affinities of other haem proteins (e.g. myoglobin), cytochrome oxidase and cytochrome P-450 for carbon monoxide are much lower.
When in equilibrium with ambient air, the carboxyhaemoglobin (COHb) content of the blood will depend mainly on the concentrations of inspired carbon monoxide and oxygen. If equilibrium has not been achieved, the COHb concentration will also depend on the duration of exposure, pulmonary ventilation and the COHb originally present before inhalation of the contaminated air.
Carbon monoxide is eliminated unchanged via the lungs. The decline in COHb concentration depends on the rate of carbon monoxide release from haem proteins, alveolar ventilation, oxygen concentration in inhaled air, duration of carbon monoxide exposure, and the level of COHb saturation. The formation of COHb is a reversible process, but because of the tight binding of carbon monoxide to haemoglobin, the elimination half-life while breathing room air is 2–6.5 hours depending on the initial COHb level. The elimination half-life of COHb is much longer in the fetus than in the pregnant mother (1).

EFFECTS OF SHORT-TERM EXPOSURE

CO affects health by interfering with the systemic transport of oxygen to tissues (especially the heart and other muscles and brain tissue). The resulting impairment of O2 delivery cause tissue hypoxia and interferes with cellular respiration. Direct intracellular uptake of CO could permit interactions with haemoproteins such as myoglobin, cytochrome oxidase and cytochrome P-450, and therefore interfere with electron transport processes and energy production at the cellular level. Thus, in addition to observed physiological effects and cardiovascular effects, CO can modify electron transport in nerve cells resulting in behavioural, neurological and developmental toxicological consequences, and may itself play a role in neurotransmission.

The health effects associated with inhaled CO vary with its concentration and duration of exposure. Effects range from subtle cardiovascular and neurobehavioral effects at low concentrations to unconsciousness and death after prolonged exposures or after acute exposures to high concentrations of CO.

Carbon monoxide exposure causes unintentional and suicidal poisonings, and a large number of deaths annually both in Europe and in the United States. It is estimated that more than half of the 6000 annual deaths from fires in the Unites States is caused by CO poisoning (2). It is obvious that such homes exist where CO concentrations are high enough to increase chronic health effects, especially among sensitive populations such as pregnant women, the fetus, children, the elderly, and individuals suffering from anemia or other diseases that restrict oxygen transport between blood and cells (3).
Annual number of deaths due to inddor Co poisoning has decreased in Europe in the last decades, still they represent a major public health issue. Data from Italy indicate that deaths varied from 135-150 cases per year in the first part of the 80s to 40-105 cases in the very last years (4). Data from France are similar, indicating that deaths attributable to indoor CO poisoning passed from 260/280 cases in the first part of the 80s to 88/107 cases in the first years of this century (5).

First signs and symptoms on healthy individuals, such as decreases in work capacity and decrements of neurobehavioral functions start at [COHb] of 5%, whereas first signs of CO poisoning appear at [COHb] concentrations of 10%.
However, the variability within the human population must be considered high. A [COHb] of about 15 % only leads to slight symptoms, such as headache, in healthy adults. In contrast, the same [COHb] can cause long-lasting defects in the cognitive development and behavioural alterations in children or even contribute to death from myocardial infarction in individuals with coronary artery disease (6).

Cardiovascular effects

In apparently healthy subjects, the maximal exercise time and the maximal oxygen consumption have decreased at COHb levels as low as 5%. The regression between the percentage decrease in maximal oxygen consumption and the percentage increase in COHb concentration appears to be linear, with approximately a one percentage

point fall in oxygen consumption per one percentage point rise in COHb level above 4% (1).

Patients with cardiovascular disease, especially ischaemic heart disease, are expected to be particularly sensitive to carbon monoxide. Atherosclerotic narrowing of the coronary arteries and impaired dilatation mechanisms restrict blood flow to the myocardium and prevent physiological compensation for lowered oxygen delivery caused by elevated levels of COHb. In exercise, these subjects experience myocardial ischaemia, which can impair myocardial contractility, affect cardiac rate and rhythm, and cause angina pectoris (1).

Early studies have suggested that low level carbon monoxide exposures resulting in COHb levels of 2.5–3.0% shorten the time to onset of exercise-induced chest pain in patients with angina pectoris. Subsequent studies by other investigators have actually given similar results (1).

The design and results of the five most important clinical studies conducted in patients with ischaemic heart disease show that despite the obvious differences between the studies, they all refer to a significant shortening in the time to onset of angina at mean post-exposure COHb levels of 2.9–5.9% which represent mean incremental increases of 1.5–4.4% COHb from the pre-exposure baseline levels (1).

The potential arrhythmogenic effects associated with low-level carbon monoxide exposures have not been fully resolved at COHb levels of ≤5% (1). Hinderliter et al. (7) reported no effects at 3.5% and 4.9% COHb levels (post-exercise concentrations) on resting and exercise-induced arrhythmias in ten patients with coronary artery disease and no baseline ectopia. In contrast, Sheps et al. (8) showed in 41 nonsmoking patients with documented coronary artery disease and various levels of baseline ectopia that the frequencies of both single and multiple ventricular depolarizations increased significantly at a mean post-exercise COHb level of 5.0% but not at 3.5%. Dahms et al. (9) found no additional effect of either 3% or 5% COHb over the exercise-induced increases in single or multiple ectopic beats experienced by patients with myocardial ischaemia and baseline ectopia.

According to some epidemiological and clinical data, carbon monoxide from recent smoking and environmental or occupational exposures may contribute to cardiovascular mortality and the early course of myocardial infarction (1). It is not known whether this contribution is due to arrhythmogenic effects or to some longer-term effects, as suggested by some authors. In patients with severe ischaemic heart disease, carbon monoxide poisonings have been lethal at COHb levels of 10–30%, while usual COHb levels in lethal poisonings are around 50–60% (10).

A number of recent epidemiological studies reported associations between levels of ambient air pollutants (CO, PM, O_3, NOx, SO_2) and hospital admissions for cardiovascular diseases (11). In all the cited studies a positive association was found between CO ambient concentrations and the daily number of cardiovascular disease hospitalizations at the local level.

Often, individuals suffering from CO poisoning are unaware of their exposure because symptoms are similar to those associated with viral illness or clinical depression (2). This may result in a significant number of misdiagnoses by medical professionals. Although the precise number of individuals who suffer from CO poisoning is not known, it is certainly much larger than that indicated by mortality figures. It has been estimated that more than 10 000 people per year in the United States required medical attention or missed at least 1 day of work in the early 1970s because of sublethal exposures to CO. Recent esteems indicate that over 40 000

emergency department visits annually for recognized acute CO poisoning in the United States.

Developmental effects

The pregnant mother, the fetus *in utero* and the newborn infant are at high risk of adverse health effects from atmospheric carbon monoxide exposures. During pregnancy, the endogenous production of carbon monoxide can be elevated as much as 3-fold, the concentration of maternal haemoglobin is often reduced, and the mothers have physiological hyperventilation. As a result of these changes, maternal COHb levels are usually about 20% higher than the non-pregnant values. Carbon monoxide diffuses readily across the placental membranes, and the carbon-monoxidebinding affinity of fetal haemoglobin is higher than that of adult haemoglobin. Moreover, carbon monoxide is cleared much more slowly from fetal blood than from maternal blood. At steady state, fetal COHb levels are up to 10–15% higher than maternal COHb levels (1).
There are theoretical reasons and supporting laboratory animal data to suggest that the fetus and the developing organs are especially vulnerable to carbon monoxide. The developing brain seems to have the highest sensitivity of all organs. There is a well established and probably causal relationship between maternal smoking and low birth weight at fetal COHb levels of 2–10%. In addition, maternal smoking seems to be associated with a dose-dependent increase in perinatal deaths and with behavioural effects in infants and young children. Carbon monoxide is probably one of the most important etiological factors for these effects, although there are numerous other toxic pollutants in tobacco smoke.
A case-control study of the association between low birthweight infants and maternal CO exposures in approximately 1000 cases in Denver failed to detect a relationship between CO exposure (estimated form fixed-site outdoor monitoring data) during the last 3 months of pregnancy and lower birth weights. Mean CO levels ranged from 0.6 to 4.1 mg/m^3 (0.5 to 3.6 ppm) at 8 monitoring locations in metropolitan Denver. The 5th and 95^{th} percentile concentrations at the site with the highest (4.1 mg/m3) mean were 1.8 and 5.5 mg/m3 (1.6 and 4.8 ppm), respectively. The odds ratio at the highest concentration site was 1.1 and the 95% confidence interval was 0.8-1.6. This study did not directly account for unmeasured sources of CO exposure, such as smoking, emissions from gas appliances and exposures to vehicular exhaust, which are limitations of the study design.
A more extensive study of a cohort of 125573 children born to women living in the Los Angeles area (1989-1993) found that exposure to ambient concentrations > 6.3 mg/m3 (3 mo average) during the last trimester of pregnancy was associated with a significantly increased risk of low birthweight (odds ratio = 1.22; confidence interval =1.03-1.44) after adjustment for potential confounders (12). Fetotoxicity has been demonstrated in laboratory animal studies. Altered brain neurochemical development and growth retardation have been demonstrated in rats exposed to CO in utero (13).

Neurological and neurobehavioural effects

Central nervous system (CNS) effects in individuals suffering acute CO poisoning cover a wide range, depending on severity of exposure: headache, dizziness, weakness, nausea, vomiting, disorientation, confusion, collapse, and coma.

At low concentrations, CNS effects include reduction in visual perception, manual dexterity, learning, driving performance, and attention level. Earlier work is frequently cited to justify the statement that CO exposure sufficient to produce COHb levels of ca. 5% would be sufficient to produce visual sensitivity reduction and various neurobehavioral performance deficits. In a recent literature re-evaluation, however, the best estimate was that [COHb] would have to rise to 15–20% before a 10% reduction in any behavioral or visual measurement could be observed (2). This conclusion was based on: critical review of the literature on behavioral and sensory effects, review and interpretation of the physiological effects of COHb on the CNS, extrapolation from the effects of hypoxic hypoxia to the effects of CO hypoxia, and extrapolation from rat behavioral effects of CO to humans.

In controlled human studies involving patients with documented coronary artery disease, mean postexposure COHb levels of 2.9–5.9% (corresponding to postexercise COHb levels of 2.0–5.2%) have been associated with a significant shortening in the time to onset of angina, with increased electrocardiographic changes and with impaired left ventricular function during exercise. In addition, ventricular arrhythmias may be increased significantly at the higher range of mean postexercise COHb levels (8). Epidemiological and clinical data indicate that carbon monoxide from recent smoking and environmental or occupational exposures may contribute to cardiovascular mortality and the early course of myocardial infarction (1). According to one study there has been a 35% excess risk of death from arteriosclerotic heart disease among smoking and nonsmoking tunnel officers, in whom the long-term mean COHb levels were generally less than 5% (14). Current data from epidemiological studies and experimental animal studies indicate that common environmental exposures to carbon monoxide do not have atherogenic effects on humans (1).

During pregnancy, endogenous production of carbon monoxide is increased so that maternal COHb levels are usually about 20% higher than the non-pregnant values. At steady state, fetal COHb levels are up to 10–15% higher than maternal COHb levels (1). There is a well established and probably causal relationship between maternal smoking and low birth weight at fetal COHb levels of 2–10%. In addition, maternal smoking seems to be associated with a dose-dependent increase in perinatal deaths and with behavioural effects in infants and young children.

Subpopulations at increased risk of adverse effects

At CO levels typically encountered in indoor and outdoor environments, health effects are most likely to occur in individuals who are physiologically stressed, either by exercise or by medical conditions that can make them more susceptible to low levels of CO. Subpopulations at increased risk of adverse effects are:

1. Individuals with cardiovascular diseases: COHb levels of 2-6% may impair the delivery of oxygen to the myocardium causing hypoxia and increasing coronary blood flow demand by nearly 30%. When myocardial oxygen demands are increased, as in exercise, the hypoxic effects of CO may exceed the limited coronary reserve producing adverse health effects including earlier onset of myocardial ischaemia, reduced exercise tolerance in persons with stable angina pectoris, increased number and complexity of arrhythmias, and increased hospital admissions for congestive heart failure.

2. Fetuses are more susceptible to CO exposure for several reasons: CO crosses the placenta; fetal Hb has greater affinity for CO than maternal Hb; the half-life of COHb

in fetal blood is three times longer than that of maternal blood, and the fetus has high rate of oxygen consumption and lower oxygen tension in the blood than adults. Also, maternal smoking during pregnancy exposes the fetus to greater than normal concentrations of CO leading to a decrease in birth weight.
3. Children develop acute neurotoxic effects (e.g. headaches, nausea), long-lasting neurotoxic effects (e.g. memory deficits) and impaired ability to escape (i.e. syncopes) at lower [COHb] than adults. Children have greater activity levels and smaller body masses than adults and should therefore experience higher levels of CO uptake than will adults for the same average exposure concentration.
4. Pregnant women have increased alveolar ventilation, increasing the rate of CO uptake from inspired air. Also, a pregnant woman produces nearly twice as much endogenous CO.
5. Individuals with chronic obstructive pulmonary disease such as chronic bronchitis, emphysema and chronic obstructive pulmonary disease are more susceptible to CO effects, since their lungs are less efficient at oxygenating the blood.
6. Individuals with reduced blood haemoglobin concentrations, or with abnormal haemoglobin, will have reduced O2 carrying capacity in blood. In addition, disease processes that result in increased destruction of red blood cells (haemolysis) and accelerated breakdown of haemoproteins accelerate endogenous production of CO, resulting in higher COHb concentrations than in normal individuals. For example, patients with haemolytic anemia have COHb concentrations 2 to 3 times those seen in normal individuals.
7. Certain occupational groups are at risk from ambient CO exposure including those who work on city streets (street repairmen, street cleaners, street vendors, deliverymen, and garage attendants, taxi and bus drivers). Individuals who work in industrial processes including those exposed to other chemical substances (e.g. methylene chloride) that increase endogenous CO formation.
8. Individuals who have not adapted to high altitude and are exposed to a combination of high altitude and CO.

A synthesis of adverse health effects of CO exposure is presented in Table 1.

Table 1 - Carboxyhaemoglobin levels resulting from steady-state exposure to increasing concentrations of CO in ambient air and associated symptoms in healthy adult humans and susceptible (adapted from U.S.EPA, 2000; Ellenhorn and Barceloux, 1988)

[CO] in atmosphere		**[COHb]**	**Signs and symptoms**	
ppm	**mg/m**	**%**	**Healthy adults**	**Susceptible subpopulations**
0	0	0.4 – 0.7	Physiologic background concentration	
10	11.5	2	Asymptomatic	
17	19.5	2,9		during physical exertion reduced time to onset of angina and electrocardiogram signs of myocardial ischaemia in subjects with

				coronary artery disease
		5-6	Decreases in work capacity and decrements of neurobehavioral function	Increase in cardiac arrythmias in subjects with coronary artery disease
42	48	7		Headache, nausea in children
		3-8	Background concentration in smokers	
70	80	10	No appreciable effect, except shortness of breath on vigorous exertion; possible tightness across the forehead; dilation of cutaneous blood vessels.	
		13		Cognitive development deficits in children
		15		Myocardial infarction in subjects with coronary artery disease
120	137	20	Shortness of breath on moderate exertion; occasional headache with throbbing in temples	
		25		syncopes in children - stillbirths
220	252	30	Decided headache; irritable; easily fatigued; judgment disturbed; possible dizziness; dimness of vision	
350-520	401-595	40-50	Headache, confusion; collapse; fainting on exertion	
800-1220	916-1400	60-70	Unconsciousness; intermittent convulsion; respiratory failure, death if exposure is long continued	
1950	2230	80	Rapidly fatal	

EFFECTS OF LONG-TERM EXPOSURE

There is not enough reliable information on effects of chronic exposures to low concentrations from either controlled human studies, ambient population-exposure studies, or from occupational studies (1). Chronic exposures to low CO concentrations may not pose as much a problem as high, acute exposure due to physiological compensation, tolerance, or adaptation.

EMISSION SOURCES AND EXPOSURE LEVELS

The most common cause of high carboxyhaemoglobin concentrations in man is the smoking of tobacco and the inhalation of the products by the smoker. Faulty domestic cooking and heating appliances, inadequately vented to outside air, may cause high indoor concentrations of CO. Also gas stoves, water heaters, and exhaust from vehicles in attached garages might be important indoor sources.

The most important source of carbon monoxide in ambient air is the exhaust of gasoline-powered motor vehicles. The emission rate depends on the type of vehicle, its speed, and its mode of operation.

Other common ambient sources include heat and power generators, especially when using coal, industrial processes such as the carbonisation of fuel, and the incineration of refuse (6).

The EXPOLIS project (15) found indoor concentrations typically lower in Northern Europe than in Central Europe, where they were again lower than in Southern Europe. Average residential indoor concentration in Helsinki was 1.2 mg/m^3 for non-ETS population. Average 48-hour exposure to CO, being 1.4 mg/m^3, was slightly higher than the respective indoor concentration. In Basle and Prague average exposures to CO were higher than in Helsinki, but lower than in Milan and Athens. The highest geometric mean exposure concentrations were found in a subpopulation of smokers in Athens, being 4.0 mg/m^3 (15). Average residential indoor CO concentrations in Milan vary from 2.1 to 3.9 mg/m^3.

Based on the EXPOLIS results average residential indoor CO concentrations in Milan were the lowest when no special indoor sources were present and the highest if gas cooking and environmental tobacco smoke (ETS) were present (16;17). The highest short-time peak concentration was found during gas cooking, 7.4 mg/m^3. Average 15-min ambient CO concentration, 2.6 mg/m^3, was higher than the respective indoor concentration when no indoor sources were present, but lower if gas cooking or ETS was present. Average 1-hour exposures to CO were higher, 8.4 mg/m^3, than the respective ambient concentrations, 5.7 mg/m^3. Instead, average 8-hour and 48-hour ambient concentrations were at the same level than the respective exposures, being 3.8 and 2.4 mg/m^3, respectively.

Short time carbon monoxide concentrations related to some typical indoor sources such as tobacco smoke, gas cooking and commuting in five European cities showed that much higher levels were found in a Finnish study determining personal exposures of preschool children in Helsinki. The highest exposures to carbon monoxide were as high as 80 mg/m3, 69 mg/m3 and 28 mg/m^3 for 15-min, 1-hour and 8-hour averages, respectively. Elevated exposures were related to gas stoves, mothers' smoking and living in high rise buildings.

The highest maximum values ranging 121 – 182 mg/m3 were measured in homes when using a gas grill attached to the gas stove (3), in the underground parking facilities and in a home with a faulty boiler. Elevated concentrations were also found in other microenvironments such as motor vehicles, indoor ice arenas, bars and restaurants.

Lately attention is being paid to incense burning in homes and other public buildings including stores and shopping malls. Jetter et al (18) reported emission rates of 23 different types of incense such as incense rope, cones, sticks, rocks, powder etc. that are typically used indoors. The measured emission rates of carbon monoxide ranged 144 - 531 mg/h. The authors estimated a peak concentration of 9.6 mg/m^3 caused by incense burning and, therefore concluded that carbon monoxide concentrations could exceed the US EPA's National Ambient Air Quality Standard (NAAQS) 10 mg/m^3 for an 8-hour average depending on the room volume, ventilation rate and the amount of incense burned. Especially, incense burning might be a significant contributor to population exposure in such cultures, where incense is burned frequently, for example in religious rituals.

INDEX STANDARDS

The INDEX project "Critical appraisal of the setting and implementation of indoor exposure limits in the EU (2002–2004)" was funded by the European Commission´ DG SANCO and JRC was given the assignment to identify priorities and to assess the needs for a Community strategy and action plan in the area of indoor air pollution (19).
CO was included in a list of five priority compounds with potential of high indoor concentrations, uncontested health impacts, and effective risk management were selected to be regulated with priority.
Available exposure data from the INDEX project confirmed that Carbon Monoxide (CO) sources in EU-residences are contributing to short-term rather than to long-term exposures. Personal exposure outcomes averaged over 1-hour were considered of moderate concern even for the most susceptible subpopulations. Increased exposures could be expected for residences in the vicinity of busy city streets. In addition, there was no evidence that long-term CO exposures in EU residences contribute to carboxyhaemoglobin levels in blood higher than the baseline levels resulting from endogenous production in normal, non-smoking individuals.
On the other hand carbon monoxide causes a considerable number of deaths and acute poisonings in the general population (with complications and late sequelae). Also, individuals suffering from CO poisoning are often unaware of their exposure because symptoms are similar to those associated with viral illness or clinical depression. In indoor environments, these health risks are nearly completely associated with the incorrect use of combustion devices or faulty unvented gas appliances.
As to carbon monoxide, proposed guideline values are 10 mg/m^3 (8-hour) and 30 mg/m^3 (1-hour). Management options suggested were to connect each combustion equipment/appliance to chimney or vented hood, to ensure sufficient local extract

ventilation in kitchens with gas stove, mandatory inspection and maintenance of indoor combustion devices, and CO alarms.
Following general recommendations have been suggested: • Restrict tobacco smoking in all indoor spaces; • Restrict the construction of attached garages, or isolate them from living and working spaces; • Ensure that ventilation dilutes predictable indoor emissions below the guideline levels; • Raise public awareness about indoor air risks.

CONCLUSIONS

CO sources in EU-residences are contributing to short-term rather than to long-term exposures. CO health effects vary from very light effects to death. Some groups of subjects (pregnant women, children, elderly and individuals with anemia, peripheral vascular disease or chronic obstructive lung disease) are more susceptible to adverse effects of CO. Recommendations and management options are necessary to avoid the development of adverse effects due to CO exposure.

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In Focus: Radon and lung cancer

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ABSTRACT

In the European Union lung cancer death is the most common cause (circa 20%) of total cancer deaths. For 2006 it is estimated that 236,000 lung cancer deaths occurred in the EU 25 with the majority of these being due to active cigarette smoking. From the pooling of 13 residential radon epidemiological studies in 9 EU countries it has been estimated that about 9% of lung cancer deaths may be due to radon exposure in the home. In this paper an account is given of the lung cancer risk estimates derived from these and other residential radon epidemiological studies. A summary account is also given of the mechanisms by which radon can cause lung cancer. Based on the epidemiological studies it is estimated that in 2006 in the EU 25 about 21,000 lung cancer deaths were due to radon exposure. The important role of smoking in radon related lung cancer is discussed. Also discussed are sources of indoor radon as well as practical strategies that may be adopted to reduce residential radon exposures and the associated lung cancer risks.

INTRODUCTION

In the EU as in most developed regions of the world lung cancer is the most common cause of death from cancer. It is estimated that 19.7% of all cancer deaths in the EU in 2006 were due to lung cancer (1). The vast majority of these lung cancer deaths are attributable to cigarette smoking but residential radon studies estimate that radon exposure may be responsible for a not insignificant percentage of these deaths. The U.S. Surgeon General has cited radon to be the second cause of lung cancer after active smoking and radon has been classified as a Group 1 carcinogen by IARC (2, 3). It has been tentatively suggested and is being investigated that radon exposure may be associated with other health endpoints such as leukaemia in children or adults but at present the only health effect established for radon is that it does cause lung cancer (4).

In indoor air radon produces a series of short-lived decay products which may attach to aerosol particles present in the air or deposit on room surfaces. It is the inhalation and deposition of the airborne short-lived radon decay products which gives rise to irradiation by alpha particles of sensitive cells in lung tissue such as the basal cells of the bronchial epithelium (5). From considerations of their respective radioactive half

lives as well as their physical and chemical properties lung dosimetry models show that the radiation dose delivered to the lung is dominated by the alpha particles emitted by the short-lived radon decay products Po-218 (E_α = 6.00 MeV) and Po-214 (E_α = 7.68 MeV). Because these alpha particles have respective ranges of only 48 μm and 71 μm in tissue they deliver a high density of ionization damage to cells in these short distances. It is this lung dose that is considered to be the cause of radon induced lung cancer either on its own or jointly with tobacco smoke carcinogens. This is supported by animal studies. Due their respective size dependent spatial deposition patterns in the human respiratory tract radon decay products unattached to aerosols (the unattached fraction) deliver a greater alpha radiation dose to sensitive lung tissue in the bronchial region compared to those attached to aerosols (the attached fraction).

MINER AND RESIDENTIAL STUDIES OF RADON HEALTH EFFECTS

There have been numerous studies over past decades into the effects of elevated radon exposure on underground miners both those in uranium mines and in other types of mines (5). Due to differences in study design and in particular to large errors in measuring radon and its decay products in these mines the lung cancer risk factor estimates from these studies cover a range of values. All of them, however, showed a clear dose-related increased risk due to radon exposure. Information on smoking status was available only for a fraction of miners of some of these studies. For smoker miners, the relative risk per unit radon exposure were found to be about 2–3 times higher than the relative risk for all the miners (6,7). This means that the combined risk of smoking and radon was found in these studies to be sub-multiplicative but to be more than additive, thus suggesting synergism between radon and tobacco smoke. In absolute terms the estimated risks per unit radon exposure to smokers was found to be greater than for non-smokers in the mining cohorts. Attempts have been made to transfer or apply the miner studies' risk factors to members of the public exposed to radon in their homes or to the general workforce in above ground workplaces, but this has proved to be somewhat problematic. This is primarily because the miner studies only give estimated risks for adult male miners whose breathing rates, lung morphometry, etc, differ from that of the general population. Moreover, miners were exposed to some more risk factors for lung cancer than are the general population in their homes. In addition aerosol characteristics, degree of equilibrium between radon and its decay products and other aspects of underground mines which influence radon progeny behaviour and consequent deposition pattern in the respiratory tract differ considerably from those present in homes. Nevertheless, Lubin et al. and the U.S. National Research Council BEIR VI Committee took data on residential radon exposure in the U.S. together with data on lung cancer mortality from 11 cohorts of underground miners and on this basis evaluated that the best estimate of the contribution from residential radon exposure to lung cancer deaths in the U.S. is about 10% or 15%, depending on the model used to fit miner data, with a 95% confidence interval of 3%-21% (7, 8). As stated above in this approach, there are many sources of uncertainty in extrapolating from the miner occupational studies to the public. An alternative approach to such use of miner studies or of the more theoretical approach of lung dosimetry modeling for estimating the radon lung cancer risk to the public has

been to directly determine the lung cancer risk from residential radon exposure studies.

Since the 1980s a number of case-control residential radon epidemiological studies have taken place in North America, in Europe and in China. A review of these can be found in (9). Some of the individual studies yielded results which were equivocal. A meta-analysis, however, of the summary odds ratios for these studies showed a slightly significant association between the lung cancer risk and residential radon exposure which was consistent with the results from the occupationally exposed miner studies (10). However, heterogeneity among these studies occurred, probably due to different control of confounding factors which cannot be controlled uniformly in a meta-analysis, whereas it can be done with a pooled analysis (11, 12).

More recently the results of a pooling of North American residential radon studies in a combined analysis of 7 North American case-control studies has been published (11). In this pooling study the radon measurements were based on long-term alpha track radon detectors placed in current and former homes of study subjects. Data was gathered on modifying factors, including age, sex, and smoking habits of the subjects. The study involved 3,662 cases of lung cancer and 4,966 controls. Collaborative analysis of individual data was carried out and data on each separate individual in the seven studies were collated centrally and analyzed with uniform methods.
The odds ratios for lung cancer was found to be increased with increasing radon exposure categories, with an odds ratio of 1.37 (95% CI = 0.98–1.92) for concentrations exceeding 200 Bq/m^3 relative to concentrations under 25 Bq/m^3. Using a continuous linear model to fit data, the overall estimate of the excess odds ratio for lung cancer per 100 Bq/m^3 was 11%, which was slightly significant (95% CI = 0%-28%). No substantial differences was observed in the excess odds ratio by categories of cigarette smoking, number smoked per day, duration of smoking, or time since quitting. The data obtained in this pooling provides direct evidence of an association between residential radon exposure and lung cancer in keeping with extrapolation from the miner studies.

In Europe a similar pooling of residential radon studies has also taken place in recent years and, like their North American counterpart, has clearly demonstrated and estimated the lung cancer risks associated with radon exposure in homes. Moreover, due to the larger total study size and the higher radon exposure levels of the European studies, a higher statistical power and therefore smaller confidence intervals were obtained and further analyses were possible to be carried out. This collaborative analysis involved 13 European epidemiological studies from nine EU Member States (Austria, Czech Republic, Finland, France, Germany, Italy, Spain, Sweden and the United Kingdom) and included individual data on 7,148 lung cancer cases and 14,208 controls without lung cancer (12,13). Each of these European case-control studies of residential radon and lung cancer had over 150 people with lung cancer and 150 controls without lung cancer. These studies incorporated detailed smoking histories of all subjects and sought radon measurements in homes inhabited by these individuals during the past 15 years or more. As in the North American pooling study data on each separate individual in the thirteen European studies was analyzed with uniform methods and was collated centrally. Radon measurements were obtained from residences occupied during the 5-34 year period prior to lung cancer diagnosis or acceptance as a control.

In this collaborative study a proportionate increase in risk was found not to be strongly influenced by any one study. The dose-response relationship appeared linear with no evidence of a threshold, and a significant relation remained even among those whose average measured radon concentrations were below 200 Bq/m^3. A non-regulatory Reference Level of 200 Bq/m^3 for residential radon has been in common use in some European countries for many years, originally recommended by the European Communities for future dwellings (14). The absolute risk to smokers and recent ex-smokers was not unexpectedly found to be much greater than that to lifelong non-smokers. This study has provided strong direct evidence of a statistically significant association of residential radon exposure and lung cancer, as predicted by extrapolation from the miner studies. The risk of lung cancer after stratification for study, age, sex, region of residence, and smoking increased by 8.4% (95% CI = 3.0%–15.8%) per 100 Bq/m^3 increase in measured radon concentration. No evidence was found that the excess relative risk varied with age, sex or smoking history. When corrections were applied to remove the bias arising from random uncertainties in radon exposure assessment, the dose-response relation was found to remain linear but increased twice in magnitude to 16% (95% CI = 5%–31%) per 100 Bq/m^3 increase of the estimated mean corrected radon concentration. While the estimated excess relative risks were independent of smoking status, in absolute terms the risks to smokers at any level of radon exposure were much greater than those to lifelong never smokers. For example, taking the risk to lifelong non-smokers exposed to a radon concentration of 0 Bq/m^3 to be 1.0 the relative risk for a habitual smoker of 15-24 cigarettes per day relative to this was estimated to be 25.8, 29.9 and 42.3 at radon concentrations of 0, 100 and 400 Bq/m^3 respectively. For lifelong non-smokers the corresponding risks are estimated to be 1.0, 1.2 and 1.6 respectively. While the very high risks for smokers exposed to radon may seem to indicate that the risk from radon exposure is only important for smokers this is not the case. Taking the absolute lifetime risk to 75 years of lung cancer for lifelong non-smokers not exposed to radon to be about 0.41% (or 1 in 250) then on the basis of the Darby et al study for continuous exposure to radon concentrations of 400 Bq/m^3 and 800 Bq/m^3 this risk will be increased by factors of about 1.6 and 2.3, respectively. In the latter case at 800 Bq/m^3 the estimated absolute risk to a lifelong non-smoker will have increased to 0.93% (or close to 1 in 100). Even allowing for the many uncertainties in such an estimate an involuntary risk of this magnitude of contracting a fatal cancer cannot reasonably be considered to be trivial.

In the context of radon and smoking it should be noted that an interaction between passive smoking and exposure to radon has also been estimated, although the combined risk would be much lower than for active smoking and with a larger confidence interval. Therefore, in this paper we will consider synergism between radon and active smoking, only. It should be noted that a pooling analysis of all the Chinese, North American and European studies which is presently underway is expected to be more informative than the previous regional ones.

In 2006 lung cancer was the most common cause of cancer death in Europe with an estimated 334,800 (19.7% of total) deaths (1). Its major cause is smoking but on the basis of the Darby et al study it is estimated that in Europe, exposure to radon in the home may account for about 9% (95% CI = 3%–17%) of deaths from lung cancer and 2% of all deaths from cancer (12,13). This major collaborative study of 13 residential radon epidemiological studies in 9 EU Member States therefore forms a very solid

basis for policy makers both at EU and Member State levels to formulate and develop effective radon risk management strategies.

ESTIMATING RADON RELATED LUNG CANCER DEATHS IN THE EU

The collaborative pooled analyses of epidemiological studies in North America and in Europe have provided strong evidence that residential radon is an important cause of lung cancer. The European collaborative analysis in particular has quantified the radon related risk of lung cancer to smokers and former smokers relative to that of lifelong never smokers. This study gives a firm basis in principle for estimating the burden of radon related lung cancer deaths in the EU. The process of making a realistic estimate of this burden, however, requires the existence and availability of reliable data bases on indoor radon concentrations and also of smoking prevalence in all Member States.

It should be noted in Table 2 that mean indoor radon concentrations throughout the EU are quite variable. Large variability in indoor radon concentrations may also be present within individual countries. There are many contributory factors to such variability. As indoor radon in most houses originates in the soil or rock subjacent to the house the geological and soil characteristics in a region are a strong determinant of indoor radon levels. Building design, air-tightness of houses and also ventilation preferences of the occupants can also be major influences on the indoor radon level. These factors combined with the geographical distribution of the population in a country can also contribute to the variability. A good example is the UK where high indoor radon values are present in the Devon and Cornwall peninsula but the mean population weighted national indoor radon level at 21.7 Bq/m^3 is one of the lowest in the EU. This is primarily due to the fact that a large fraction of the UK population lives in the London region which is mainly built on clay with low radon emanating and permeability characteristics.

In the case of smoking habits the data bases available also show there is considerable variability in smoking prevalence throughout the EU. As shown in Table 1 the percentage of adults who smoke in the EU ranges from 17.5% in Sweden to 45% in Greece (15). The EU average is 29% but despite wide variations in smoking prevalence among member states, the overall average for the 25 member states is broadly the same as it was before enlargement in 2004. While the average percentage of non-smoking adults in the EU can be taken from Table 2 to be 71% it should be noted that the non-smoking cohort is composed both of lifelong never smokers and former smokers. As the risk of radon related lung cancer is strongly influenced by smoking status and as the lung cancer risk decreases with time since quitting smoking in order to make a realistic estimate of radon related lung cancer incidence in the EU good information on former or ex-smokers is needed in addition to data on present active smokers (16). Where national data on former smokers is available it usually simply given as their percentage in the population with little or no additional information such as the time since they stopped active smoking or indeed the duration and extent of their previous active smoking habits. In spite of these and other limitations in the available radon and smoking data it is possible using the findings of the Darby et al collaborative study to

make an estimation of the lung cancer impact due to radon in the EU. As already stated above in this study it was estimated that in Europe, exposure to radon in the home accounts for about 9% of deaths from lung cancer and perhaps up to 2% of all deaths from cancer. More accurate estimates on the radon lung cancer burden in Europe are presently being made but are not yet completed. As lung cancer deaths in Europe are estimated to have been 334,800 in 2006 this implies that perhaps up to 30,000 of these deaths may have been caused by exposure to radon in the home (1). The corresponding estimated figures in 2006 for the EU 25 are 236,000 and about 21,000 respectively. In considering these putative radon related EU lung cancer deaths the following three important qualifying observations must be made:

(1) The majority of these estimated radon related lung related cancer deaths occur in active smokers exposed to radon.

(2) It should also be noted that, due to the near log-normal distribution of indoor radon levels found in all national surveys the majority of these deaths will occur to persons (both smokers and non-smokers) exposed to indoor radon levels well below the indoor radon Reference Level of 200 Bq/m^3 used in most European and EU countries.

(3) Residential radon studies have shown that the risk of lung cancer due to the combined effects of smoking and radon exposure are much greater than the additive effect of both individual risks. Therefore in estimating the global lung cancer burden in a country or region good data is needed on not only the indoor radon distribution but also on smoking prevalence. As Table 2 shows smoking prevalence is quite variable throughout the EU. While the EU mean is 29% the percentage of active smokers ranges from 17.5% in Sweden to 45% in Greece.

These three observations have important implications for policy makers in the EU formulating policies and strategies aimed at managing the lung cancer risk from indoor radon.

Table 1. SMOKING PREVALENCE IN THE EUROPEAN UNION (EU 25)*

EU Member State	**Total % of Smokers**	**% of Men**	**% of Women**	**EU Member State**	**Total % of Smokers**	**% of Men**	**% of Women**
Austria	29	32	26	Belgium	27.5	33	22
Cyprus	23.5	39	8	Czech Rep.	30.5	38	23
Denmark	27	30	24	Estonia	31.5	45	18
Finland	22.5	26	19	France	30.5	36	25
Germany	32.5	37	28	Greece	45	51	39
Hungary	35.5	42	29	Ireland	27	28	26
Italy	24	31	17	Latvia	31	49	13
Lithuania	28	44	12	Luxembourg	37.5	39	26
Malta	25.5	30	21	Netherlands	30	33	27
Poland	31	39	23	Portugal	20–23	31	9 ?
Slovakia	40	48	32	Slovenia	24	28	20
Spain	32	39	25	Sweden	17.5	16	19
UK	25	26	24	**EU Average**	**29**	**35**	**22**

*(15)

EXPOSURE TO RADON

There are a wide range of both passive and active radon measurement techniques available. As radon is a gas its concentration in a building can be quite variable both diurnally and seasonally due to changes in meteorological parameters, ventilation practices etc. Due to this variability it is generally the case that an assessment of radon exposure in a building is best achieved by making a long-term passive measurement of radon. Typically this is done using alpha track–etch detectors (17). In many EU Member States such long–term indoor radon measurements are usually made over a period of at least three months and preferably in the heating season when radon levels are usually at their highest. In these cases, the annual average can be obtained by applying seasonal correction factors. In some other EU Member States one-year measurements are preferred to obtain the annual average radon concentration. A common approach is to place one detector in the main living room of a house and a second one in the principal bedroom.

In most of the older EU Member States extensive and representative surveys of indoor radon have taken place while in many of the recent accession countries representative nationwide indoor radon surveys have yet to take place. Table 1 gives a summary of the indoor radon data in the EU 25 expressed in units of Bq/m^3. Because of differences in the characteristics of these surveys it is not possible to calculate a population weighted EU average indoor radon concentration but it is probably close to 50 Bq/m^3. The distribution of indoor radon in most countries approximates well to a log-normal distribution. While they are very rare a small number of homes with indoor radon levels of some tens of thousands of Bq/m^3 have been found in a number of countries.

Table 2. INDOOR RADON IN THE EUROPEAN UNION (EU 25) *

EU Member State	Arithmetic Mean Bq/m^3	Geometric Mean Bq/m^3	EU Member State	Arithmetic Mean Bq/m^3	Geometric Mean Bq/m^3
Austria	102	n/a	Belgium	48	38
Cyprus	7	7	Czech Rep.	118	n/a
Denmark	53	29	Estonia	120	92
Finland	120	84	France	62	41
Germany	50	40	Greece	55	52
Hungary	107	82	Ireland	91	37
Italy	70	52	Latvia	n/a	n/a
Lithuania	32	22	Luxembourg	110	70
Malta	n/a	n/a	Netherlands	23	18
Poland	41	32	Portugal	62	45
Slovakia	87	n/a	Slovenia	87	60
Spain	45	42	Sweden	108	56
UK	22.7	9.7	**EU Average**	**n/a**	**n/a**

* (18,19)

SOURCES OF INDOOR RADON

Radon-222, commonly referred to as “radon”, is a chemically inert radioactive gas which is a member of the uranium-238 naturally occurring radioactive decay series. Its immediate parent in the decay series is radium-226. It is produced in most rocks and soils from which it may enter the indoor air of houses**.** There are a number of possible sources of indoor radon. The most important source for most buildings is soil gas infiltration. It is well established that this is driven by the positive pressure gradient that usually exists between the subjacent soil gas and the indoor air spaces of a building (20). In assessing the risk potential of soil for high indoor radon concentrations in future buildings the main determinants are the subjacent soil permeability, its radium-226 activity concentration and the associated concentration of radon in the soil gas. In some EU member states such as the Czech Republic and Sweden soil radon risk classification based on such soil characteristic is in use (21). In most EU member states, while soil and geological characteristics are taken into account, strategies to achieve low radon levels in future buildings in an area are largely based on surveys on indoor radon levels in existing buildings and on the use of radon proof construction technologies.

In general the contribution to indoor radon levels due to radon emanation from building materials is minor compared to the contribution from soil gas. There are exceptions to this, for example, in parts of Italy where high radium content volcanic tuff is used as a building material or in Sweden where alum shale containing elevated levels of radium has been used in the past as aggregate in aerated concrete products (17).

RADON CONTROL OPTIONS

While exposure to indoor radon gives rise to a lung cancer risk this risk in principle can be controlled or reduced. At the level of an individual house it is technically feasible, in most cases, to ensure that the indoor radon level is kept at or brought down below a reference or action level set by the national radiation regulatory agencies. In principle the use of ventilation as a means to reduce indoor radon levels appears to be an obvious radon control strategy. It should be noted, however, in the majority of buildings with a radon problem the source of the radon is soil gas which enters the building by pressure driven flow. Therefore if ventilation is used care must be taken to ensure that the ventilation regime does not increase the pressure driven flow thus increasing indoor radon levels. A ventilation solution to an indoor radon problem also may carry an energy penalty. The preferred approaches to controlling indoor radon levels are active soil depressurization by means of sub-floor radon sumps coupled to extraction fans and/or the installation of radon impermeable barriers or membranes in the building foundations (22).

As already mentioned above the most common residential radon reference level being used in EU countries is 200 Bq/m^3. This reference level is a recommended value and is not a mandatory regulatory level unlike an Action Level such as 400 or 500 Bq/m^3 for radon in workplaces set by some Member States in their implementation of the EU Basic Safety Standards Directive (23). In the case of an existing house found to be above such a reference level remedial action might involve the installation of a sub-floor sump coupled to an extractor fan or some other appropriate remedial technology, such as a radon membrane barrier, to reduce soil gas radon entry to the house living spaces (22). The cost of such remedial action will vary considerably from one house type to another but experience in some EU countries would indicate that remediation costs should be between € 500 and € 2000. In the case of future houses the incorporation of radon control building technologies into the construction is less costly than their retrofitting in existing houses and would represent a very small fraction of the cost of new house construction. The incorporation of such building technologies in all new houses is already part of the existing building codes in some EU member states such as Ireland (24). WHO Air Quality Guidelines for Europe also suggest that building codes should include sections to ensure that radon daughter levels do not exceed 100 Bq/m^3 EER (Equilibrium Equivalent Radon concentration) which is similar to a radon concentration of about 200–250 Bq/m^3 (25).

Apart from these building technology aspects there are a number of different strategies that can be adopted at a national level to control indoor radon with the objective reducing the lung cancer risk associated with long term radon exposure. These strategies may be divided into the following three principal categories:

(A) Identification of houses with high radon levels and the remediation of these houses. This is rather like the concept often used in radiation protection where a critical group of the most exposed persons is considered a protection priority and the main objective is to reduce *individual* high risks.
In most countries a house with an indoor radon level above 1000 Bq/m^3 would be classified as “high” as the estimated lifetime lung cancer risk, even for a lifelong never smoker, would be considered unacceptable by most standards of health protection. On the basis of European national radon surveys which show that the

distribution approximates closely to a log-normal distribution the percentage of dwellings in most EU states likely to have a radon level above 1000 Bq/m^3 will be very low. For example in Ireland, where the mean indoor level is 91 Bq/m^3 it is estimated that in < 0.1% of houses is the radon level above 1000 Bq/m^3. Obviously where high houses are found at random in an area householders should be strongly advised to take action and the competent regulatory agencies should carry out more detailed local surveys to find other high houses that may be present in the area. The problems and costs of finding all high houses on a national basis would not appear in most countries to be justified both from a practical perspective and also from a cost-benefit analysis perspective. On the other hand having a strategy to find high radon houses may be justified in a defined region known to have a high radon potential due to its geological and soil characteristics.

(B) As a consequence of the characteristics of log-normal distributions and the fact that national average indoor radon levels in the EU are mostly below 100 Bq/m^3 the best strategy in principle to reduce the *collective* risks, i.e. the radon related number of lung cancers in the population, should be to reduce the average indoor radon level in a country. For the existing housing stock this is not a practical or cost effective option. The reduction of radon levels in new build future houses by the introduction of appropriate radon preventative building regulations is perhaps therefore the only effective strategy that can over time effectively reduce the national risk from radon related lung cancer. In regions known to have a high radon potential particularly stringent radon prevention building regulations might be considered.

(C) Due to the demonstrated synergism between radon and smoking in terms of causing lung cancer a strategy that should be considered is to couple radon reduction strategies with national strategies aimed at reducing the consumption of cigarettes.

In most EU Member States where there are well developed radon control policies a mixture of the above strategy options (A) and (B) are usually in operation together with radon risk communication programmes. However, having a combined strategy of reducing smoking and radon exposure is presently not part of the public health programme in any EU Member State.

CONCLUSION

It has been demonstrated by residential radon studies that exposure to radon increases the risk of lung cancer. Even though the estimated excess relative risk factor of 16% per 100 Bq/m^3 was found not to vary with age, sex or smoking history the absolute lung cancer risk associated with unit radon exposure is much greater for active smokers than for lifelong never smokers. In the EU it is estimated that radon related lung cancer deaths account for about 9% (95% CI = 3%–17%) of the total and similar estimates can be obtained from North American studies. Radon levels in homes are controllable by various building technology options such as the installation of active radon sumps and radon proof membranes in the foundations of houses. Coupled to the introduction of indoor radon control regulations there is a need at EU level to establish strict protocols and training programmes to ensure the effective use of these techniques. While radon levels in high radon homes should be reduced it is more cost-effective at a national level to adopt building regulation strategies aimed at reducing

the average radon levels in new houses below the current national average level. In the case of radon risk communication programmes, however, information on the exacerbation of the lung cancer risk in smokers by radon exposure should be emphasised.

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Session 6 Indoor air policy perspectives

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INTRODUCTION

Although there is a long history of regulating and controlling outdoor air quality, the indoor environment has generally been neglected, even though it is well understood that there are significant sources of pollution in the indoor environment and it is indoors where people spend the vast majority of their time.

Pollutants encountered indoors are known to have the potential to cause adverse health impacts (1), so it is reasonable to consider what policies might be helpful for preventing or mitigating these effects. A number of possible approaches to improving indoor air quality are discussed in this paper in relation to a wider strategic approach, including: assessing the impact of particular indoor air pollutants through an appraisal of hazard, exposure and risk; source control and ventilation, development of exposure guidelines and product and appliance emission standards; legislation; information dissemination (e.g. guidance for the public), education and training; and influencing the designers, builders and managers/ operators of buildings.

This paper focuses on policies for non-industrial buildings such as homes, schools and offices, but the principles apply also to enclosed spaces such as vehicles. Industrial and other regulated environments are excluded from consideration. The principal concern is indoor climate and chemical and biological contaminants of indoor air; other aspects such as noise, lighting and electromagnetic radiation are not included.

THE NEED FOR INDOOR AIR POLICY

There is a large body of evidence on the hazardous nature of indoor air pollutants, on their sources, the conditions leading to human exposure, and the significance of the associated health effects. Consequently, indoor air quality is now acknowledged to be an important determinant of health and comfort (2), as evidenced for example by the inclusion of indoor air quality in the European Commission research strategy for the Framework 7 Programme. However, the complexity of pollution sources and the multitude of parties responsible for generating and potentially acting to control indoor air pollution make the coherent development of risk reduction strategies difficult. To be effective, any policies directed at improving indoor air quality need to be part of a comprehensive management strategy involving governments, institutions, professional bodies and individuals. Plans need to be directed at both new and existing buildings and involve action at both local and national levels. Important

considerations include outdoor climate and air quality, building materials and styles, knowledge and behaviour patterns of the occupants, energy and sustainability policies, and building system technologies. In recent years the importance of sustainable energy usage in the context of climate change has come increasingly to the fore.

RESPONSIBILITIES FOR IMPROVING INDOOR AIR QUALITY

The quality of indoor air in any particular building or location is dependent on a number of factors that are themselves governed by a range of different influences. These include the quality of the outdoor air, the condition of the building, ventilation exchange rates, the furnishings present, and the occupiers' lifestyle, habits and behaviours - including their management of the building and use of products. Although acknowledgement of the importance of indoor air quality has often led to calls for legislation, specific legislation for this purpose is often not available (see below), or is perceived as too 'intrusive' on individuals' lifestyle. Thus very often no single profession or authority has overall responsibility for indoor air quality - and the same invariably applies at the governmental level. In the UK, for example, responsibilities for indoor air quality fall variously to the Department of Health, the Department of Communities and Local Government, the Health and Safety Executive, the Department of Trade and Industry, and the Department for Environment, Food and Rural Affairs.

ASSESSING HAZARD AND RISK

Much work has already been done to assess the exposure of individuals to particular indoor air pollutants and to evaluate the risks to health of such exposures. Such understanding is a prime prerequisite for policy formulation. Research of this nature involves monitoring indoor environments and assessing personal exposure, toxicological assessment of chemical hazards, monitoring health effects related to the indoor environment (either in situ or in experimental studies), and health impact assessment. The risk assessment process is crucial and involves the identification of factors that impact on the health and well-being of occupants, quantification of human exposure to these factors, assessment of human responses to these factors, and characterisation of risk. Risk assessment may then lead to specific recommendations for control, mitigation and/or remediation, or more general policy recommendations for improving indoor air quality through identified management options (see below).

There are many publications reviewing the impact of indoor pollution on health, and the work of the European Collaborative Action "Urban Air, Indoor Environment and Human Exposure" has been particularly significant in advancing understanding in this area.

PRINCIPLES OF ACTION

Effective action is best achieved if it is based on good scientific evidence, but there are other important considerations in the establishment of indoor air policy. The following principles are relevant (2):

The precautionary principle: A responsible indoor air policy should not be restricted to combating the danger or repairing the damage, but should be pro-active in addressing emerging problems.

The principle of individual responsibility: Individuals should be informed about hazards inherent in the indoor environment and should then assume responsibility by behaving and acting in such a way as to minimise harm to themselves and to others.

The cooperation principle: Central and local governments and other interested parties – including professional bodies and commercial organisations - should aim to cooperate in formulating sustainable indoor air policies and in implementing appropriate actions.

The 'polluter pays' principle: This principle requires the costs of removing the source of indoor air pollution, or compensation for the damage caused, to be borne by the polluter.

The 'right to know' principle: People using indoor spaces are entitled to know the possible harm to health arising from the materials, appliances and products in use around them.

Limitations of action: Due consideration must be given to the fact that application of the above principles may conflict with other external principles (e.g. right to personal freedom), which may on occasion limit the action that can be taken.

MANAGEMENT OPTIONS

Risk management for indoor air quality can involve regulatory or non-regulatory strategies. Examples of possible regulatory strategies include bans of chemicals or products, emissions limits, labelling requirements, exposure limits, building design standards, building operation and maintenance requirements and ventilation standards. Non-regulatory approaches include guidelines, market and fiscal incentives, population information campaigns, training and education of involved parties, support of sustainable non-polluting technologies.

Legal tools

Nationally there is little specific legislation aimed at the regulation of indoor air (one primary exception being the ban on smoking in public places now implemented in several countries). Those regulations that do exist are largely associated with building codes (including ventilation provision), control of dangerous appliances, and product safety – for example there are rafts of national and international legislation to regulate

the quality, marketing and use of construction products, consumer products and chemicals. The applicability of current regulation and its effectiveness in improving indoor air quality is generally rather limited, and only relatively recently has concerted attention been given to approaches for specifically measuring, assessing and reducing emissions from construction products into indoor air. Certainly it is problematic to deal with all the facets of indoor air quality in one regulatory system because of the wide range of pollutants, sources and causes.

Guidelines, guidance and labelling schemes

Health-based standards exist for outdoor air and there are good arguments for developing equivalent indoor air quality guidelines (3). In the UK, the requirement for guidelines for indoor air was recognized as early as 1991 by the House of Commons Select Committee which, in its report on indoor pollution, recommended that the Government "develop guidelines and codes of practice for indoor air quality in buildings which specifically identify exposure limits for an extended list of pollutants…".

Setting guidelines or standards for indoor air quality involves some difficult issues, for example: Where are the guidelines to apply? What is the basis for the establishment of the guideline (i.e. health or comfort)? Who are the guidelines meant to protect? Who is responsible for monitoring and regulating identified indoor pollutants? What are the legal implications? Can indoor air guidelines be different from outdoor air guidelines for the same pollutant? However, there are very clear advantages that the establishment of indoor air guidelines can bring. For example, they could inform the public and other interested parties on typical indoor pollutant levels – i.e. what is 'normal' – as well as on levels likely to be hazardous to health. Establishment of numerical guideline levels would in turn allow informed development of product emission standards (and also product labelling schemes – see below) and could be used in housing assessment and rating systems. Some nations, for example Germany, Norway and Poland, have already established target concentrations for various indoor pollutants, and the UK recently issued guidance on indoor air pollutants that includes numerical standards for nitrogen dioxide, carbon monoxide, formaldehyde, benzene and benzo(a)pyrene. Some countries (e.g. Australia) have adopted the approach of identifying indicators of good air quality rather than defining quantitative limits (3).

Importantly, having led the way by defining outdoor air quality guidelines for Europe (which are often, by default, used by individuals assessing indoor air quality), WHO is now in the process of establishing guideline values specifically for application to the indoor environment.

Whatever the potential pitfalls of numerical standards or guidelines for indoor air pollutants, there is much scope for issuing guidance to assist homeowners, building managers, etc., achieve good air quality. Consideration should be given to extending such guidance (e.g. through professional bodies) to others with a role to play, including architects and engineers. For example, the Finnish Society of Indoor Air Quality and Climate, with others, has published the wide ranging report 'Classification of Indoor Climate 2000: Target Values, Design Guidance and Product Requirements' (4). However it as achieved, education and training should be an

integral part of any strategic policy on indoor air pollution. The principal objectives of such a programme should be to: raise public awareness and help citizens make appropriate choices; ensure appropriate education and training for professionals; and promote the exchange of information between science and policy (2).

A number of labelling schemes are presently in operation; these are to be encouraged as they have a significant role to play in encouraging consumers (and builders/tradesmen) to make informed choices when selecting materials and products for use within buildings - and thereby influence manufacturers. The ECA report 'Evaluation of VOC emissions from building products' (5) presents ways of distinguishing between acceptable and non-acceptable materials and how to label these accordingly, and in Denmark and Finland, for example, a special labelling scheme for construction products (based mainly on material emission rates) has been introduced that takes account of impacts on indoor air quality (2).

Other approaches

There are a number of other approaches that can be considered when formulating indoor air strategies, including cost-benefit analysis, economic instruments, incentives for the building sector, planning controls, encouraging sustainability, and research to fill knowledge gaps.

EXISTING RESOURCES

A number of reports have been published that provide useful information and pointers on indoor air policy setting. These include 'The Right to Healthy Indoor Air' (6), 'Strategic approaches to indoor air policy-making' (2), and the various publications of the European Collaborative Action on Urban Air, Indoor Air and Human Exposure (e.g. 5) and the series of reports from the pilot study on indoor air quality by the NATO Committee on the Challenges of Modern Society (e.g. 7).

FORMULATING A NATIONAL STRATEGY

Requirements for the establishment of a national strategy include prior justification, goal setting, options appraisal, and political willingness.

Justification

This needs to address the questions: Why is a policy needed? What are the priorities? Can health impacts be prevented? Who is responsible? Until these questions are satisfactorily answered, the policy is unlikely to carry conviction.

Goal setting

The policy must have clear goals on what the policy aims to achieve, what the short, medium and long term objectives are, and which tools and indicators will be used to monitor success.

Management options

These are detailed above and include legal tools, guidelines and guidance, labelling schemes, economic instruments and incentives.

Political willingness: current drivers

There are a number of current developments that are strongly pushing the drive to improve indoor air quality. For example, a major outcome of the Fourth Ministerial Conference on Environment and Health was the publication of the Children's Environment and Health Action Plan for Europe (CEHAPE). In this document, under Regional Priority Goal III, there is explicit reference to indoor air and the specific pronouncement "We aim to achieve a substantial reduction in the morbidity and mortality from acute and chronic respiratory disorders in children and adolescents by developing indoor air quality strategies that take into account the specific needs of children [and] applying and enforcing regulations to improve indoor air quality, especially in housing, child care centres and schools..." There is now a requirement on Member States to produce their own Action Plans. The full text of this document is available at http://www.euro.who.int/document/e83338.pdf. Also the European Commission has announced that efforts to monitor indoor pollution and its impact on human health are to be intensified, and the Commission's Joint Research Centre (JRC) is taking the lead to develop new approaches and research methods. The aim is to gather data that may in future "provide the basis for policy initiatives at EU and national level".
The recently released 'Preliminary report on risk assessment on indoor air quality' from the Scientific Committee on Health and Environmental Risks (SCHER) – available at http://ec.europa.eu/health/ph_risk/committees/04_scher_scher_cons_01_en.htm – is likely to further stimulate debate on the impact on indoor air quality on human health and how such impacts can best be addressed.

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IAQ Case examples

Wood preservatives xylamit as a source of indoor air pollution

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INTRODUCTION

Oily wood preservatives Xylamit were used in the residential and public building industry in Poland in the 1960s and 1970s for impregnation and fungicidal treatment. They were a source of air pollution due to the emission of toxic compounds causing the deterioration of hygienic conditions indoors. The most serious negative consequences, felt to this day, were caused by the use of these preparations to impregnate porous fibreboards laid in the ceilings of buildings as insulation in the industrial systems of residential building industry (the so-called "large panel buildings"). At the time, it was used in a great number of flats (their number is estimated at around 400,000).

The consequences of using Xylamit in the building industry are felt to this day. Despite the fact that in the 1980s conditions to remove such defects from buildings were created, there are still an unknown number of flats in Poland with fibreboards impregnated with Xylamit. The Environmental Protection Department in the Building Research Institute performs several expert opinions each year on the presence of Xylamit in buildings in different cities in Poland.

XYLAMIT TOXICITY

Xylamits were preparations used for impregnating and eliminating fungi in construction timber and wood-derived materials, made in 1958-1986. Their main components were coal tar and petroleum distillates (coal and petroleum residuum oils), used as solvents for the active components, dissolving well in oils. The main biologically-active components used in Xylamits were chlorophenols and polyphenols, chlorobenzenes, chlorinated coal residuum oils, 1-chloronaphthalene. These were usually waste materials - technical pentachlorophenols, post-distillation chlorophenol and regenerated chlorophenols - used in amounts from 4 to 20% (1,2). The quantitative composition of these raw materials varied and is not fully known. Tests performed in later years showed that technical chlorophenols could contain the halogen-derivatives dibenzo-p-dioxin and benzofurans, formed as a result of side reactions. These compounds are present in the form of numerous isomers and are

characterised by their ability to bio accumulate in the fat tissue of humans and animals. Some are considered to be the strongest acting poisons.

In the building industry during peak usage mainly the so-called dark Xylamits based on coal residuum oils were used. The toxicity of Xylamits was not determined at the time. Due to the great complexity of the chemical composition of these preparations and the varying quality of raw materials used, its evaluation posed a problem. Toxicity assessments of Xylamits carried out in 1971 and 1978 for the needs of occupational hygiene made it possible to include preparations from this group in the 4th class of harmfulness, i.e. substances with a weak toxic effect. The grounds for this classification were the small strength of the general toxic effect (acute oral toxicity 3900 mg/kg of body weight, acute inhalation toxicity 5000 mg/kg of body weight). Tests of short-term chronic toxicity carried out at the time showed that the preparation absorbed over a longer period of time, in small doses may damage solid organs, as well as cause disorders in the central and peripheral nervous system. Chlorophenols, polyphenols and chloronaphthalene, the main biologically active components in the discussed group of wood preservatives, are characterised by good solubility in fats and lipids, which is why in the body they show large affinity to lipid-rich organs and nervous tissue, causing disorders of their functions and pathological changes. (3, 4)

Xylamits were characterised by a very strong, specific chemical odour. This odour was described by the producer as “non-lasting”, which among others decided on their use in the residential building industry. Today, from a perspective of over thirty years that have passed since its usage, we can say that the smell is practically ever-lasting.

INDOOR AIR POLLUTION RESULTING FROM XYLAMIT USE

In the late 1960s - early 1970s, Xylamits were introduced in the industrialised residential building industry, developing dynamically at this time. Those wood preservatives were used to protect soft porous fibreboards used as insulation material in insulating ceilings between floors. Insulation building paper, concrete blind floors and external flooring material were placed right next to the impregnates.

There were also cases where Xylamit was used to impregnate wooden floors in public buildings (schools, offices) or wooden structures of walls or roofs in the traditional building industry. (5)

Complaints started to appear quite quickly from the users of such premises, who said that specific chemical odour were present in the flats and public buildings, which caused a series of symptoms of ill-being. The reason for these complaints, as it turned out, was the use of Xylamit impregnated fibreboards used as insulation in ceilings between floors. The characteristic odour of preservatives was clearly felt in the premises and was a great nuisance for the dwellers. As a result of indoor air pollution tests carried out at the time, it was found that the air contained phenolic compounds. (6,7,8) The evaluation of the degree of air pollution was hindered by the lack of sufficiently precise analytical methods, which would make it possible to identify and determine the concentrations of all volatile components of those wood preservatives in the air. Colorimetric methods were in general use and these only made it possible to

determine the sum of phenolic compounds in the air, without any possibility of identification. Chromatographic methods enabling such analysis appeared in laboratories studying air pollution only in the 1980s. Tests conducted by the Building Research Institute ITB since the early 1980s showed that the buildings, in which Xylamit was used, were contaminated with naphthalene, methylnaphthalenes, 1-chloronaphthalene, phenol and cresol vapours. Chlorophenols as non-volatile compounds were not detected in the air in buildings in many cases. On the other hand, cholorobenzenes, as the most volatile compounds, evaporated from the impregnated products after a year or so.

The growing number of complaints from dwellers, the substantial nuisance of the odour found beyond any doubt, and the exceeding of permitted concentrations of phenol compounds in the air showed that Xylamits were a serious cause for the deterioration of hygienic and sanitary conditions in the premises. The withdrawal of products from the Xylamits group from use in the building industry lasted several years - from the introduction of restrictions in using them for impregnating window and door joinery in 1977, to the complete ending of production of these preservatives in 1986. (9) The process of removing the consequences of using these wood preservatives in the building industry started in 1984. The removal of these defects was made possible on the basis of the Resolution of the Council of Ministers, by financing with redeemed bank credits some defects of buildings, among others caused by the emission of toxic compounds in residential and public buildings. Many investors took advantage of this opportunity and removed products impregnated with Xylamit from the buildings. Most of these works were carried out in 1984-1992. For various reasons, these renovations did not encompass all of the buildings. One of the reasons for this was the inability to determine, on the basis of an analysis of the building works documentation, what type of wood preservative was used. The dwellers were often unaware that materials impregnated with Xylamit were present in the building.

Many years have passed since the introduction of the ban on using Xylamits inside buildings, but the negative consequences of its use have not been eliminated to this day. Dwellings with Xylamite impregnated felt-boards have existed till now and occupants have been exposed for many years. The problem usually appears when the dwellers change or the building is modernised.

Each year, the Environmental Protection Department in ITB performs several expert opinions on the presence of Xylamit in premises. In the framework of these expert opinions, samples of all flooring materials are collected, in order to determine whether the ceiling contains fibreboards impregnated with Xylamit. Next, these samples are examined in laboratory conditions, in test chambers, in accordance with ITB Laboratory procedures. Chemical compounds found in the air are adsorbed on the surface of a solid adsorbent, desorbed by solvent and identified using the gas chromatography and liquid chromatography method. Standard compounds included naphthalene, phenol, cresol and their chlorine derivatives.

In Table 1 the results of expert opinions performed in 1996-2006 are presented. The air of the laboratory chambers was usually found to contain naphthalene, methylnaphthalenes, phenol, chloronaphthalene, less often cresols. In some cases, the concentration of the examined compounds was very high and exceeded many times

the permitted norms, in others, it was low, even on the limit of detection. However, in all of the tested flats there was the odour characteristic of preparations from the Xylamit group, only its intensity differed. This odour continued to be perceptible, despite the fact that very often over 30 years had passed from the time people moved in. Often in the case of samples collected in different flats in the same building, or even in different rooms in the same flat, depending on the amount of the wood preservative used and the quality of the impregnation, very large differences were found in the concentrations of chemical compounds.

Air pollution tests in flats are not performed as often. Many years of experience of both the Environmental Protection Department ITB and other institutions concerned with air pollution tests indicate that despite the fact that the odour is clearly felt in the premises, test results usually do not show that the permitted air concentrations of the components of Xylamit, i.e. cholorophenols, naphthalene, chloronaphthalene or cresols, are exceeded. Table 2 and diagram 1 present the results of tests of air pollution in premises, in which materials impregnated with Xylamit were used, carried out by the Environmental Protection Department in the last few years.

In 2002 and 2003, the Environmental Protection Department tested the air in 2 buildings during major renovation works. In these buildings, the ceilings contained built-in fibreboards impregnated with Xylamit. The tests were carried out during the renovation, after all the flooring materials were removed and the plaster was chiselled off. These works were carried out a couple of weeks before the air samples were collected. The temperature during the tests was 9-14°C, as the buildings were not heated.

Despite the total elimination of the source of pollution in the form of fibreboards impregnated with Xylamit, volatile components of the wood preservative were found in the air. The source of emission was secondarily contaminated floor slabs.

Table 1. Results of laboratory tests of volatile compound emissions from samples of flooring materials carried out in 1997-2003

Year	No. of tested samples	Range of concentrations of volatile compound vapours in chamber air [µg/m^3]			
		naphthalene	methyl-naphthalene	phenol	1-chloro-naphthalene
1997	17	2 - 829	1 -276	-	-
1998	13	5 -133	5 - 40	-	-
1999	5	2 -300	1 -255	1 -124	51
2000	8	1 - 2058	2 -322	1 - 121	1 -42
2001	4	3 -1669	5 -297	1 -17	-
2002	2	18-244	28-99	15-22	2-30
2003	1	25	4	3	74
2005	1	31	31	-	13
2006	3	3-102	2-111	1-69	3-53
Admissible values in category A[2] premises		**100**	**Not established**	**20**	**15**

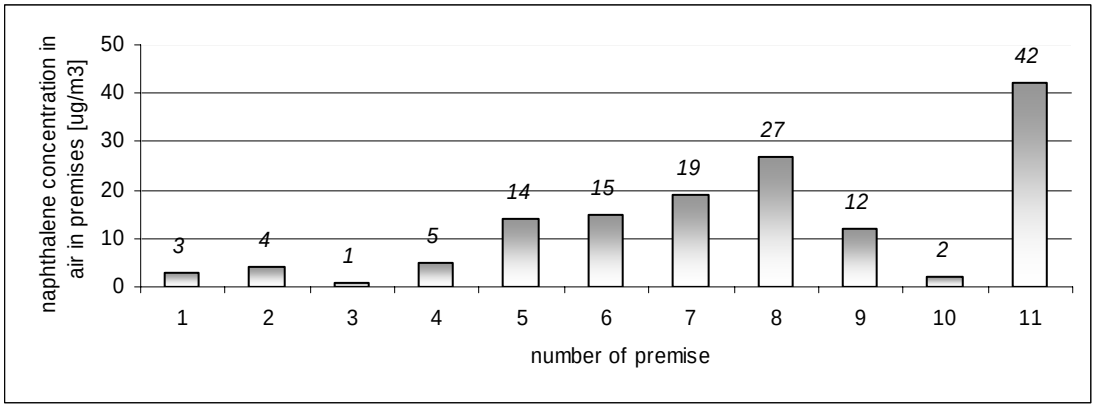

Diagram 1. Concentration of naphthalene vapours in 10 office premises in a building containing materials impregnated with Xylamit

[2] Admissible values according to the Regulation of the Minister of Health and Social Care of 12 March 1996 on permitted concentrations and intensities of factors harmful to health emitted by building materials, fittings and fixtures in premises intended for humans (Polish Monitor no. 19 item 231) – category A premises – residential premises, premises for permanent residence of the ill in health care institutions and for permanent residence of children and youth in educational buildings, as well as premises intended for storing food products, category B premises - all other premises, in this premises intended for people in public buildings, other than those included in A category premises and auxiliary premises

Already during the first renovation works, carried out in the 1980s, it turned out that the removal itself of al of the flooring materials and laying a new floor does not completely eliminate the nuisance. In many cases, a specific odour remained in the premises, though at a much lower intensity, upon the completion of the renovation works.

Table 2 Results of air pollution tests in premises containing materials impregnated with Xylamit

Year	Test object	Range of concentrations of volatile compound vapours in air in premises [μg/m^3]			
		naphtha-lene	methyl-naphtha-lenes	phenol	1-chloro-naphtha-lene
2001	Flat in municipal building. In the 1960s-it was subject to fungicidal treatment (3 measurements)	13-17	7-8	3-5	5
2002	Multi-family building during renovation. All flooring materials, plasters removed. Temperature during tests – 10/14^0 C (5 premises	2-8	1-11	-	2-19
2003	Multi-family building during renovation. All flooring materials, plasters removed. Temperature during tests – 9-15^0 C (5 premises)	11-68	16-69	3-13	0 – 6
2004	Office rooms in office building (10 premises)	1-44	1-15	<1-5	<1-6
2004	Rooms in students' house (12 premises)	1-7	1-5	-	<1-4
Admissible values in class A category premises[1]		**100**	**Not established**	**20**	**15**

The reason for this was the secondary contamination of floor slabs with the wood preservative Xylamit, caused by long-lasting contact with the fibreboards saturated with the preparation. The degree of contamination depended on the amount of the preparation used to impregnate the fibreboards. As a result of further observations, it was found that in order to carry out the renovation effectively, it was necessary to maximally remove the surface of floor slabs secondarily contaminated with the wood preservative Xylamit and use neutralising preparations on the floor slab and a part of the walls that could be in contact with the impregnated slab. The preparation that turned out to be an effective neutraliser was only developed in the early 1990s, and was called NEUTRAL. In the last few years, this preparation is being used increasingly often during renovations of buildings connected with removing impregnated materials.

The use of the preparation neutralising the residues of Xylamit in floor slabs prevents the emission of volatile components from surfaces secondarily contaminated with the wood preservative. This makes it possible to achieve the appropriate hygienic conditions in the renovated premises and their further use. However, it should be pointed out that the undertaken measures will only be effective if the renovations are

carried out correctly. This encompasses: a careful removal of all impregnated materials, thorough cleaning of the floor slab, secondarily contaminated (chiselling off or grinding off of the surface is recommended), thorough cleaning of the premises, precise covering of the slab with the neutralising substance, maintaining the required seasoning period of the premises before continuing with the renovation works (4-6 weeks). This is illustrated in the results of tests given in Table 3. It presents the results of air pollution tests in a multi-family 5-floor residential building, in which major renovations of the entire building were carried out, including the removal of materials impregnated with Xylamit and neutralisation. Several months after moving in, the dwellers of some of the premises began complaining about arduous smells in their flats. Air pollution tests showed increased amounts of vapours of volatile components of the wood preservative Xylamit in these flats (diagram 2). In these flats, the renovation works and neutralisation were repeated. During these renovations, it was found that the impregnated materials were not completely removed from these flats and the neutralisation was not performed precisely. Following the repeated renovation, these flats were freed of the arduous odour and the air was no longer contaminated with vapours of volatile components.

Table 3 Results of air pollution tests in the premises of a building after renovation

No. of test	Test object	Range of concentrations of volatile compound vapours in air in premises [μg/m^3]			
		naphtha-lene	methyl-naphtha-lenes	phenol	1-chloro-naphtha-lene
I	Flats. in which dwellers informed about the presence of a distinct odour (10 flats)	2- 14	2- 8	1-4	3-13
II	Flats, in which dwellers did not inform about the presence of a distinct odour (5 flats)	<1-3	<1-2	<1-1	<1-2
III	Flats after repeated renovation works (6 flats)	1-4	<1-1	<1	<1-2
Admissible values in category A premises[1]		**100**	**Not established**	**20**	**15**

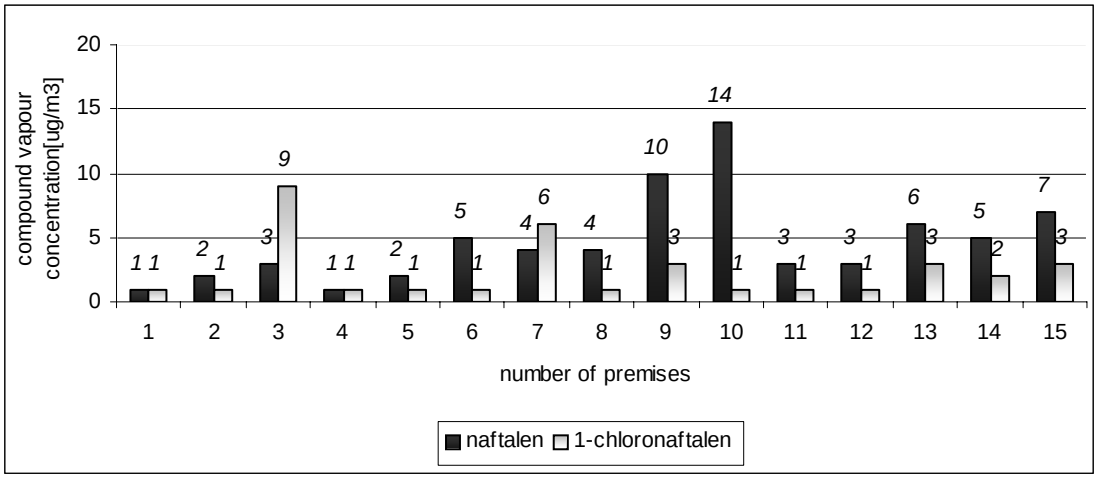

Diagram 2. Concentration of naphthalene and 1-chloronaphthalene vapours in the air in 15 flats after renovation connected with the removal of impregnated materials

Neutralisation makes it possible to achieve the appropriate hygienic conditions in flats containing materials impregnated with Xylamit enabling their further exploitation.

SUMMARY

In the period of their most extensive use in the building industry, the toxicity of wood preservatives Xylamit was not determined. The decision, based on erroneous presumptions, on introducing them in the industrialised systems of the building industry caused serious economic consequences connected with the costs of renovations lasting to this day and financed initially by the state budget, and later by the owners of the buildings. However, the health effects for the dwellers of these buildings in which fibreboards impregnated with Xylamit were used, seem to be much more serious. For a dozen or so or even tens of years they were in contact with toxic compounds - volatile components of Xylamit, such as phenol, chlorophenols, naphthalene, methylnaphthalene and chloronaphthalene, present in indoor air. These compounds are characterised by their toxic effect on living organisms and their constant presence in the air in flats most probably also affected the health of the dwellers.

Despite the passing of almost thirty years from the introduction of the ban on using Xylamit in the residential building industry, there continues to be an unknown number of buildings containing materials impregnated with Xylamit. It is impossible to state on the basis of an analysis of the building works documentation, where Xylamit was used. Other impregnating substances, such as those based on salt, were also in use at the time. The results of air pollution tests in flats, in which the presence of impregnated materials was found, carried out in the last few years have not shown any volatile components of wood preservatives to be present in the air in amounts exceeding the permitted norms. However, it should be pointed out that their presence in the air is always found. This shows that the dwellers of these flats for many years were in contact with polluted air.

The example of Xylamit shows how important it is to evaluate the properties of building materials with respect to their chemical composition and how long-lasting and costly can be the effects of introducing an untested product on the market. Products basing on chemical raw materials should undergo judicious hygienic evaluation based on emission tests or an examination of harmful substance content.

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