# **EUROPEAN COLLABORATIVE ACTION**

# URBAN AIR, INDOOR ENVIRONMENT AND HUMAN EXPOSURE

**Environment and Quality of Life** 

Report No 25

# Srategies to determine and control the contributions of indoor air pollution to total inhalation exposure (STRATEX)





EUROPEAN COMMISSION
DIRECTORATE JOINT RESEARCH CENTRE - INSTITUTE FOR HEALTH & CONSUMER
PROTECTION PHYSICAL & CHEMICAL EXPOSURE UNIT

2006 EUR 22503 EN

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# Strategies to determine and control the contributions of indoor air pollution to total inhalation exposure (STRATEX)

Dedicated to late Professor Marco Maroni

prepared by

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The mission of the Institute for Health and Consumer Protection is to provide scientific support to the development and implementation of EU policies related to health and consumer protection. The IHCP carries out research to improve the understanding of potential health risks posed by chemicals, biocides, genetically modified organisms, contaminants released from food contact materials and consumer products.

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MANDATE: European Collaborative Action "Urban Air, Indoor Environment and Human Exposure" (formerly "Indoor Air Quality & it's Impact on Man")

For more than 20 years now the European Collaborative Action ECA "Indoor Air Quality & it's Impact on Man" has been implementing a multidisciplinary collaboration of European scientists the ultimate goal of which was the provision of healthy and environmentally sustainable buildings. To accomplish this task ECA is dealing with all aspects of the indoor environment including thermal comfort, pollution sources, the quality and quantity of chemical and biological indoor pollutants, energy use, and the ventilation processes which all may interact with indoor air quality. The work of ECA has been directed by a Steering Committee.

In order to provide a broader view on air pollution exposure in urban areas, both indoors and outdoors, the ECA Steering Committee decided to put more emphasis on the links between indoor and outdoor air quality and to focus its further work under a new title "*Urban Air, Indoor Environment and Human Exposure"*. The focus of the renewed activity is urban & indoor air pollution exposure assessment, seen as part of environmental health risk assessment and also considering the needs of urban and indoor air quality management. The new approach hosts and supports the activities of the Joint Research Centre's Institute for Health and Consumer Protection in Ispra (Italy) dealing with Physical and Chemical Exposures and Health Effects.

This focussed activity proceeds within the broader framework of (i) health and comfort of the citizens, (ii) building technologies and source controls, and (iii) requirements of sustainability, energy efficiency and conservation of natural resources.

Specific examples of the working areas of ECA are:

- the relative importance of outdoor and indoor sources of pollution,
- the building-related interaction between outdoor urban air and indoor air,
- exposure to pollutants from the different urban outdoor and indoor sources and its relation to health and comfort.

By addressing such topics ECA will lay the ground for air quality management to minimise exposures to air pollutants. It will thus continue to contribute to pre-normative research needed by EC services and national authorities responsible for preventing pollution and promoting health, comfort and quality of life.

In this series the following reports have already been published

- Report No. 1: Radon in indoor air. EUR 11917 EN, 1988. \*
- Report No. 2: Formaldehyde emission from wood-based materials: guideline for the determination of steady state concentrations in test chambers. EUR 12196 EN, 1989. \*
- Report No. 3: Indoor pollution by NO2 in European countries. EUR 12219, EN1989.
- Report No. 4: Sick building syndrome a practical guide. EUR 12294 EN, 1989.
- Report No. 6: Strategy for sampling chemical substances in indoor air. EUR 12617 EN, 1989.
- Report No. 7: Indoor air pollution by formaldehyde in European countries. EUR 13216 EN, 1990.
- Report No. 8: Guideline for the characterization of volatile organic compounds emitted from indoor materials and products using small test chambers. EUR 13593 EN, 1991.
- Report No. 9: Project inventory 2nd updated edition. EUR 13838 EN, 1991.
- Report No. 10: Effects of indoor air pollution on human health. EUR 14086 EN, 1991.
- Report No. 11: Guidelines for ventilation requirements in buildings. EUR 14449 1992, EN.
- Report No. 12: Biological particles in indoor environments. EUR 14988 EN, 1993.
- Report No. 13: Determination of VOCs emitted from indoor materials and products. Interlaboratory comparison of small chamber measurements. EUR 15054 EN, 1993.
- Report No. 14: Sampling strategies for volatile organic compounds (VOCs) in indoor air. EUR 16051 EN, 1994.
- Report No. 15: Radon in indoor air., EUR 16123 EN, 1995.
- Report No. 16: Determination of VOCs emitted from indoor materials and products: Second interlaboratoriy comparison of small chamber measurements., EUR 16284 EN, 1995.
- Report No. 17: Indoor air quality and the use of energy in buildings. EUR 16367 EN, 1996.
- Report No. 18: Evaluation of VOC emissions from building products -solid flooring materials., EUR 17334 EN,1997
- Report No. 19: Total Volatile Organic Compounds (TVOC) in indoor air quality investigations. EUR 17675 EN, 1997
- Report No. 20: Sensory evaluation of indoor air quality, EUR 18676 EN, 1999.
- Report No. 21: European Interlaboratory Comparison on VOCs emitted from building materials and products, EUR 18698 EN. 1999.
- Report No. 22: Risk assessment in relation to indoor air quality, EUR 19529 EN, 2000.
- Report No. 23: Ventilation, Good Indoor Air Quality and Rational Use of Energy, EUR 20741 EN, 2003.
- Report No. 24 Harmonisation of indoor material emissions labelling systems in the EU, Inventory of existing schemes, EUR 21891 EN, 2005.

#### **Abstract**

ECA-IAQ (European Collaborative Action, Urban Air, Indoor Environment and Human Exposure), 2006. Strategies to determine and control the contributions of indoor air pollution to total inhalation exposure (STRATEX), Report No 25. EUR 22503 EN. Luxembourg: Office for Official Publications of the European Communities

It is now well established that indoor air pollution contributes significantly to the global burden of disease of the population. Therefore, the knowledge of this contribution is essential in view of risk assessment and management. The ECA STRATEX 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 should be considered as a framework. This framework may have to be adapted to specific situations by policy makers, risk assessors, and risk managers.

<sup>\*</sup> out of print

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#### FOREWORD AND SCOPE

Human exposure to environmental pollutants occurs via various pathways such as air, drinking water, food, and dermal contact. For many pollutants, especially the volatile ones, air exposure is the dominant pathway. Exposure via air occurs both outdoors and indoors, with diverse types of indoor spaces playing a role, e.g., home, workplace, schools, kindergartens, and passenger cabins of means of transportation.

It is now well established that indoor air pollution contributes significantly to the global burden of disease of the population. Therefore, the knowledge of this contribution is essential in view of risk assessment and management. In principle, the characterisation of population exposure to indoor air pollution can be achieved by issue focussed small-scale studies at local level or large-scale population studies. If the small-scale study approach is selected then ways should be established to extrapolate from these to the general population. As large-scale surveys are normally designed to be representative their results are directly reflecting population exposure. However, large-scale surveys are expensive, time and labour intensive so that often modelling exercises are adopted to characterise and quantify population exposure to indoor air pollution. Under some circumstances the measurement strategy cannot be followed, and a modelling strategy will have to be adopted. To do this, a number of other pieces of information need to be known and considered. Among these are, e.g., the behaviour of people (time-activity patterns), the determinants of indoor air quality (sources and their emissions and sinks), and the building characteristics.

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 should be considered as a framework. This framework may have to be adapted to specific situations by policy makers, risk assessors, and risk managers.

#### **EXECUTIVE SUMMARY**

# **Introduction and objectives**

It is now well established that indoor air pollution contributes significantly to the global burden of disease of the population. Therefore, the knowledge of this contribution is essential in view of risk assessment and management. 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. This framework may have to be adapted to specific situations by policy makers, risk assessors, and risk managers.

# Methodology

In the introductory part (chapter 1) of this report firstly the relationships among indoors vs. outdoor exposures, concentrations and sources as well as the health effects due to indoor air pollution are briefly outlined.

Then, it is emphasized the fact that, in order to implement strategies aimed at preventing health effects related to indoor air pollution, a key element consists in carrying out a systematic risk assessment and risk analysis for indoor-related health effects. Exposure assessment is an essential component of any risk assessment, both as an independent requirement, and as an essential part of epidemiological studies. A framework for risk and exposure assessment has been adopted following the paradigms of NAS (NRC, 1983) and the EC Directive 93/67/EEC (EEC 1993).

According to EC Directive 93/67/EEC formal risk assessment is divided into four activities, which are defined as 'hazard identification', 'dose (concentration) - response (effect) assessment', 'exposure assessment' and 'risk characterization'.

For many of the chemicals present in indoor environments due to infiltration of polluted outdoor air and/or emissions from various indoor sources, the risk to human health is difficult to predict, because of a lack of exposure and toxicological data and dose response characteristics in human or animal models. Consequently, it has been difficult in the past to regulate these substances in indoor air. Therefore, there is an urgent need to develop a strategy to determine and control the contributions of indoor air pollution to total inhalation exposure in Europe.

As a practical example of a risk assessment of indoor air pollutants performed in EU, the INDEX ("Critical Appraisal of the Setting and Implementation of Indoor Exposure Limits in the EU") project is mentioned (Kotzias et al., 2005). The aim has been to identify these priorities and to assess the need for a Community strategy and action plan in the area of indoor air pollution.

In spite of the progress in knowledge that has been gained over the past years, there are still uncertainties on the relations between indoor air pollution and health effects. Reasons for these uncertainties are briefly outlined and thoroughly discussed at the end of chapter 3.

Before discussing any strategies and techniques to be used for determining and controlling the contributions of indoor air pollution to the total inhalation exposure, the factors which determine the IAQ and exposure and their interactions have been firstly investigated (chapter 2). These factors are:

- (a) Sources of chemical pollutants to indoor air (infiltration of ambient air pollutants, pollutants through water and soil, building materials and products, processes occurring inside the buildings, the building occupants and their activities). The principal indoor air chemical pollutants due to these sources include combustion gases (mainly NO<sub>2</sub>, SO<sub>2</sub> and CO), environmental tobacco smoke, particulate matter, ozone, mineral fibres, organic compounds (polycyclic aromatic hydrocarbons, volatile organic compounds and formaldehyde), pesticides, inorganic air pollutants and radon. The sources contributing to indoor air pollution are responsible for either continuous emissions (long-term emission with constant sources strength such as material emissions) or discontinuous emissions (short term emissions with variable source strength such as human activities). Spatial and temporal variations can occur, depending on movement of the air, infiltrations between rooms in a building, etc.
- (b) **The physical factors of indoor climate** (temperature, relative humidity, air movement, ventilation and air pressure) can be important modifiers of the concentration pattern for many common indoor air pollutants, biological agents (house dust mites, fungi, bacteria) and radon.
- (c) **Processes affecting the 'history of air pollutants' indoors** (emission from sources, subsequent diffusion, adsorption/desorption, dilution and removal, transport and spreading of pollutants, aggregation, oxidization, photochemical reactions and catalytic reactions on surfaces).
- (d) **Time-microenvironment-activity of occupants** as a result of their own activities and their own lifestyle options and behaviour.

In chapter 3 follows the core part of the report. The strategies and techniques concerning the determination of exposure are thoroughly discussed and a framework for exposure control strategies is proposed.

Concerning the strategies and techniques to determine exposure, some rapid assessment methods are proposed as radically simpler than real data based and validated total exposure and risk assessment methods, yet they may provide adequate support for many practical decisions. These are: (a) optimally designed **ambient air quality monitoring networks** for providing info relevant to long term exposure, exposure distributions and exposure peaks mainly of outdoor sources, (b) **questionnaires** for qualitative data collection on indoor pollution sources and different microenvironment activity diaries, (c) **source inventories** and (d) **intake fractions** to estimate population doses from different sources in risk assessment and help comparing different risk management options in risk management.

More realistic population exposure distribution data can be obtained however, through specific monitoring techniques (more or less developed for different air pollutants, reactive compounds, microbial compounds and PM and for different time scales (hours to weeks exposures) or employing models (deterministic or probabilistic) to estimate past and predict future exposures for large populations that cannot be achieved by monitoring. The pros and cons and the range of applicability and validity of these techniques and models are critically discussed. Source apportionment techniques are suggested as tools to apportion the total exposure to its sources; this helps evaluating the quantitative roles of regional,

local, traffic and neighbourhood, outdoor and indoor sources to population and individual exposures.

Concerning Risk Management, the factors, which should be taken into account in an exposure based UAQ management, are discussed and the necessity to predict the absolute and relative exposure consequences of different exposure scenarios in evaluating the UAQ management options is emphasized. Any alternative exposure control options can only be evaluated via exposure modelling.

At the end of chapter 3 a few alternatives approaches are proposed in the context of a framework for exposure based control strategies aimed primarily at minimising exposures to harmful agents – as opposed to e.g. meeting indoor air quality standards (which does not consider the presence or absence of people), or eliminating specified indoor sources (which does not consider outdoor sources or other source control alternatives). The advantages and disadvantages of one whole population approach and four specific approaches (Indicator based exposure strategy, source targeted exposure strategy, space targeted exposure strategy and individually targeted exposure strategies) are explored.

Finally, a discussion about the uncertainties that are most relevant for exposure based urban air quality assessment and management is included. These uncertainties relate to the agents of health concern, their sources and the vulnerable target populations as well as to the measurements (sampling and analysis) and modelling.

Practical examples concerning risk assessment and risk management of fine particulate matter, formaldehyde and ozone in indoor air are given in Annexes 1 to 3.

#### **Conclusions**

There is a need for prioritisation of a short list of indoor air pollutants for their indoor related health relevance or as indicators of other potential pollution.

There is also a need for more toxicity info on SVOCs and info on the interaction between reactive air pollutants.

There are no generally accepted procedures for the commissioning or curing period of a building.

No comprehensive and general model concerning the development of indoor exposure and risk assessment has yet emerged.

To efficiently reduce health risks from indoor air pollution, air quality management should be based on health concerns and exposure assessments of individual substances, accounting for indoor air quality, but also all other microenvironments and exposure relevant activities with parallel quantification of the related uncertainties.

# Recommendations

It is recommended the common ventilation dilution model to be developed into a dynamic equilibrium model that takes into account the effects of transport, removal, adsorption and reemission and chemical and biological transformation including reactions of pollutants.

A labelling scheme for construction products that includes SVOCs in addition to the VOCs should be implemented across EU.

An overall policy strategy for indoor air quality management should be part of an integrated sustainable development strategy including public health; it should be combined with decision models and cost effectiveness analyses. It should be pro-active and include systematic preparation and dissemination of information and recommendations for the general public.

For an efficient exposure based air quality strategy focusing on the contribution of indoor air quality to the total inhalation exposure, the simultaneous minimisation of six requirements concerning the balanced combination of sectorial strategies is suggested.

Three ways to achieve reduction of uncertainties as an essential element of an appropriate risk management strategy are finally recommended.

#### 1 INTRODUCTION

# The importance of health in relation to indoor exposure

Health protection and health promotion of the population has become a major concern in our modern society for several reasons:

- Firstly, health is a fundamental pursuit of the human beings acknowledged in all the fundamental constitutional documents at national and international level;
- Secondly, since health is a key determinant for socially active and productive life, the health status of the population has a direct impact on economy, representing a basic condition for the social and economic development of the communities;
- Thirdly, the growing costs associated with treatment of diseases and supports to disabled people are increasingly considered as an unsustainable burden to national economies.

Among the causes of disease in the population, three major causal factors can be identified:

- The genetic predisposition of the individuals,
- The life-style (including dietary and behavioural habits, use of tobacco, alcohol and other toxins), and
- The environmental conditions, including housing, quality of the living and working environments, and exposure to natural or man-made pollutants. Air pollutants both in ambient and indoor air have been demonstrated to represent a major contributor to the total burden of disease of the population (WHO World Health Report 2003).

# Contributions of indoor air pollution to the total exposure

Air pollutants in buildings are linked to building or indoor factors (building structures, surfaces, furnishing, ventilation system, etc.), the specific activities of the occupants, or to sources found outdoors. Major sources of indoor air contaminants are:

- Ambient air pollution (due to traffic, urban and industrial activities) comes into the building through the ventilation system or by infiltration (building envelop permeability),
- Building materials and furnishings (wall and floor coverings, paints, insulation materials etc.),
- The processes that occur within buildings (any combustion processes, heating, ventilation and air conditioning systems, paper processing such photocopying, etc.),
- The occupants themselves and their activities (tobacco smoking, use of cleaning products, plant and pet drugs, cooking etc.),
- Water and soil (air pollutants coming through water supply, radon and contaminated soils.

In contrast to typical industrial exposures, characterised by high concentrations of one or a few chemicals released from the processes/activities, indoor pollution in typical office and residence environments consists of low concentrations of a complex mixture of tens to hundreds of chemical, biological or physical agents. Some of these agents are also sufficiently reactive with each other or on indoor surfaces to produce significant levels of new reaction products in indoor air (e.g. the reactions of limonene with ozone).

# Indoor vs. outdoor exposures, concentrations and sources

In average people spend over 80-90% of time in indoor environments, and the percentage is still overall higher for some specific groups as new-born, elderly, disabled or sick people. Their exposure to air contaminants is therefore drastically determined by indoor conditions,

concerning not only the exposure time but also the nature and the concentrations of the pollutants. In the urban environment and for a large part of the population "indoors" means, along a single day, a series of multiple micro-environments much different from one to another (e.g.: home, transportation, workplace, sport hall, theatre, etc.).

In the absence of significant indoor sources such as gas appliances, tobacco smoking, old photocopiers or dust generating activities, the indoor concentrations of nitrogen dioxide, ozone and suspended particulate matter are usually lower than outdoor concentrations. In these conditions concentrations of the rather non-reactive carbon monoxide and benzene concentrations are similar indoors and outdoors, I/O  $\cong$  1.0. For a majority of indoor air contaminants, particularly in the presence of common indoor sources, however, indoor concentrations usually exceed outdoor concentrations, for some pollutants even with an I/O ratio above 10.

The most common pollutants found indoors and their respective sources are:

- Urban ambient air pollutants (e.g. NOx, CO, O3, and particles) that enter the building through ventilation or by infiltration (building envelop permeability).
- The majority of natural allergens that come from soil and vegetation (pollens, spores...) and the outdoor mould.
- Most of the respirable particles (ECA, n° 12) as well as semi-volatile compounds found outdoors, such as pesticides, might also infiltrate inside the building giving high concentration in the indoor environment.
- Other contaminants are more specific to the indoor air such as formaldehyde, volatile organic compounds (VOC) (ECA, n° 13, 14, 16, 18, 19) and semi-volatile organic compounds (SVOC). Their indoor sources are emissions from building materials and furnishings, vaporisation of household chemicals, and human activities.
- Tobacco smoke, re-suspended dust, mineral fibers and biocides are other important indoor pollutants coming from indoor sources. Microbial pollutants are also associated with water damages in the (often hidden) building structures.

These basic considerations all show the complexity of the outdoor and indoor sources for indoor air pollution. This complexity has to be taken into account to the extent possible in assessing human exposure to air pollutants.

# Health effects of indoor air pollution

Health is a state of complete physical, mental and social well-being and not merely the absence of decease or infirmity. The effects of interest indoors therefore include both adverse effects and reversible changes of well-being (WHO 1999).

The main diseases and adverse health effects which can be caused by indoor air pollutants have been discussed in several reports (see ECA 1991, Katsouyanni et al. 1995 and 1997, Mølhave et al. 1996, 2000a, 2000b, 2002, 2004). The available knowledge is summarised below. Indoor air pollutants can be responsible for an increased occurrence of:

- Cancer,
- Chronic and acute pulmonary diseases,
- Upper airways inflammatory diseases,
- Allergic diseases such as asthma and allergies, particularly to house-dust mites,
- Ocular and mucosal reactions,
- Infectious diseases, and respiratory infections,
- Intoxications.

Moreover, indoor pollutants can increase the occurrence of frequent and severe diseases such as myocardial infarction and other cardiovascular diseases responsible for a great part of mortality and disability of the population.

Less severe, but socially very relevant adverse health effects, include:

- Discomfort,
- Odour perception, sensorial irritation and annoyance and the so-called
- Sick Building Syndrome (SBS), another illness of epidemic nature that may affect occupants of a building.

SBS appears with unspecific symptoms related to nose, eye, respiration, skin, and the nervous system. It is a problem that occurs in buildings worldwide. Contrary to building related illnesses that usually affect one or few subjects in a building, SBS tends to affect a larger number of the building occupants.

SBS symptoms vary in nature, but there are a small number of characteristic symptoms, which may occur singly or in combination:

- Nasal: most commonly nasal obstruction (usually described as nasal stuffiness) or nasal irritation with rhinorrhoea.
- Ocular: dryness or irritation of the eyes.
- Oropharyngeal: dryness of the throat.
- Cutaneous: dryness and irritation of the skin, occasionally associated with a rash on exposed skin surfaces.
- General manifestations: abnormal fatigue or tiredness, general malaise, headache or heavy-headed feeling.

The important point to elicit is the timing and frequency of the SBS symptoms. The symptoms typical for SBS are unspecific symptoms that may also occur for reasons not related to being in a building. To identify SBS, symptoms have to occur at a much higher frequency than normal (e.g. 20% vs. 2-4%) and improve on days away from the problem building and re-occur on return to the building.

There remains a lack of fundamental scientific basis to this condition. Scientific basis for etiology, however, is irrelevant when e.g. sick leaves from a workplace or school are prolonged, repeated and several times more frequent in a "sick building" than in reference buildings. Sheer economic realities demand identification of the cost and remediation of the situation. Impacts of poor indoor air quality on work productivity – and thus occupant economy - have been shown also in numerous buildings which cannot be labelled sick buildings, but nevertheless suffer from the effects of insufficient ventilation, tobacco smoke or traffic fumes, off-gassing materials and furnishings, and poor maintenance.

For an individual the most severe indoor air pollution consequences are of the greatest interest. For the society, however, quite mild effects, upper respiratory symptoms, skin rash, even just discomfort, if they manifest themselves in a large fraction of the population, can be as significant as few cases of the most severe consequences, such as lung cancer.

The occurrence of SBS, also points at the fact that certain subpopulations are more susceptible than others to the harmful effects of indoor air pollution exposures. Women in general respond more frequently than men, asthmatics are a well known risk group, and infants as well as old people with chronic respiratory and cardiovascular diseases are at greater risk to indoor – as well as outdoor - air pollution.

#### Risk assessment and management

A comprehensive conceptual model or scheme for risk assessment and risk management was published by the U.S. National Academy of Sciences in 1983, known generally as the "NAS (1983) Paradigm" (NRC, 1983). It separates risk assessment, based on science, from risk management, which in principle follows from this independent assessment and has more practical goals and constraints, see Figure 1. The risk assessment process was divided into four steps: Hazard Identification, Dose-response Assessment, Exposure Assessment, and Risk Characterization. According to the NAS Paradigm risk management generates risk management options and evaluates them against Risk Characterisation, and then selects and implements a risk management policy. In a dynamic world with imperfect information this policy needs to have a feedback loop to options generation and evaluation so that new accumulating exposure and toxicity information, as well as policy implementation experiences can be incorporated into the evolving policy and its implementation.

Originally the NAS 1983 paradigm set a strict order for these steps, and also separated risk assessment from risk management, so that risk management would follow from a completed risk assessment. The NAS Paradigm succeeded in setting a widely agreed framework for risk assessment / risk management, and it has had broad applications from carcinogenic compounds to non-carcinogens, mixtures, radiations and other situations.

The European Commission directive 93/67/EEC (EEC 1993) "laid down the principles for assessment of risks to man and the environment of chemicals (or substances notified in accordance with Council Directive 67/548/EEC (EEC 1967)". These principles as well as the terminology of this Directive are quite similar to those of the NAS Paradigm. According to EC Directive 93/67/EEC formal risk assessment is divided into four activities, which are defined as 'hazard identification', 'dose (concentration) - response (effect) assessment', 'exposure assessment' and 'risk characterization'.

Exposure assessment is an essential component of any risk assessment, both as an independent requirement, and as an essential part of epidemiological studies. The needs of exposure assessment for risk assessment are still very variable. In some cases the most exposed individual is targeted, in other cases a sensitive subgroup or the whole population. Thus the target population considerations greatly affect the selection of exposure assessment methods.

The most common air pollution exposure modelling approach is the so called time-microenvironment-activity model: Microenvironment exposure is defined as the product of the concentration in a microenvironment (possibly influenced by the activity conducted) and the time spent in that microenvironment, and the total exposure as the sum of the exposures in all microenvironments during the time of interest.

Especially in regulatory risk assessment and management the sequences of and activities in the different microenvironments are often defined in an agreed exposure scenario. Often such a scenario represents a "credible worst case exposure scenario". More recently, however, the interest has grown for probabilistic exposure and risk assessment with the idea of creating whole and realistic distributions of the exposures/risks, from which then e.g. the 95th percentiles can be selected to represent the high-end risk levels. Such exposure scenarios should be based on real life data, representative of the whole range of actual scenarios.

For many of the chemicals present in indoor environments due to infiltration of polluted outdoor air and/or emissions from various indoor sources, the risk to human health is difficult

to predict, because of a lack of exposure and toxicological data and dose response characteristics in human or animal models. Consequently, it has been difficult to regulate these substances in indoor air. Therefore, there is an urgent need to develop a strategy to determine and control the contributions of indoor air pollution to total inhalation exposure in Europe.

In 2002, a European Project called INDEX was started with the aim to identify these priorities and to assess the need for a Community strategy and action plan in the area of indoor air pollution. The INDEX report (Kotzias et al., 2005) provides: 1) a list of priority substances to be regulated in indoor environments on the basis of health impact criteria; 2) suggestions and recommendations on potential exposure limits for these substances; and 3) information on links with existing knowledge, ongoing studies, legislation etc., at world scale.

A proposal on a new EU regulatory framework concerning "Registration, Evaluation and Authorisation of CHemicals (REACH)" was adopted on 29 October 2003 by the EU Commission. REACH aims to improve the protection of human health and the environment while maintaining the competitiveness and enhancing the innovative capability of the EU chemicals industry. REACH requires manufacturers and importers of chemicals, under certain conditions, to make available exposure assessment reports for intended use of their products. This information, when available, will obviously help to implement the strategy presented in this report.

# Uncertainty

In spite of the progress in knowledge that has gained over the past years, there are still uncertainties on the relations between indoor air pollution and health effects. This has several reasons:

- The complexity and variety of indoor air pollutants in different contexts,
- The limited knowledge about the toxicological and sensorial properties of many indoor pollutants at low concentration levels under the given "chronic" exposure conditions,
- The multi-factorial characteristics of several human diseases that can be caused by a multiple combination of factors,
- The possibility of interaction among pollutants that can have synergistic mechanisms of action (cocktail effect),
- The combination of indoor air risk factors with other environmental agents related to food, water, ambient air, noise, and with personal characteristics of the individual subjects.

To implement strategies aimed at preventing health effects related to indoor air pollution, a key element consists in carrying out a systematic risk assessment and risk analysis for indoor-related health effects.

#### **Medical costs**

Although, estimation of medical costs caused by diseases related to indoor air pollution is not a simple task, some analyses have been done. For example, estimated direct annual medical costs in Italy are estimated to be in a range of 152 – 234 millions Euro (see Table 1).

# Examples of exposure determination and control strategies

Examples of exposure determination and control strategies for three common indoor air pollutants particulate matter, formaldehyde and ozone are presented in Annex 1, 2 and 3.

**Table 1.** Estimated direct medical costs (in Euro) yearly attributable to some indoor pollution-related diseases in Italy (Gazzetta Ufficiale della Repubblica Italiana, nr. 276, 27.11. 2001).

| Disease                                       | Pollutant                                     | Impact                         | Direct medical costs |
|---|---|--------------------------------|----------------------|
| Bronchial asthma in children/teenagers        | Allergens (dust mites, moulds, animal dander) | >160,000<br>Prevalent cases/yr | >80 millions         |
| Lung cancer                                   | Radon   | 1,500-6,000 deaths/yr          | 26 -105 millions     |
| Bronchial asthma<br>in children/<br>teenagers |   | >30,000<br>Prevalent cases/yr  | >15 millions         |
| Acute airways infections                      | Environmental<br>Tobacco Smoke                | >50,000 new cases/yr           | >12 millions         |
| Lung cancer                                   |   | >500 deaths/yr                 | >9 millions          |
| Acute heart infarction                        |   | >900 deaths/yr                 | >8 millions          |
| Leukaemia                                     | Benzene                                       | 36-190 cases/yr                | 0.5 – 4 millions     |
| Acute poisoning                               | Carbon monoxide                               | >200 deaths/yr                 | 1 million            |
| TOTAL   |   |                                | >152 - 234 millions  |

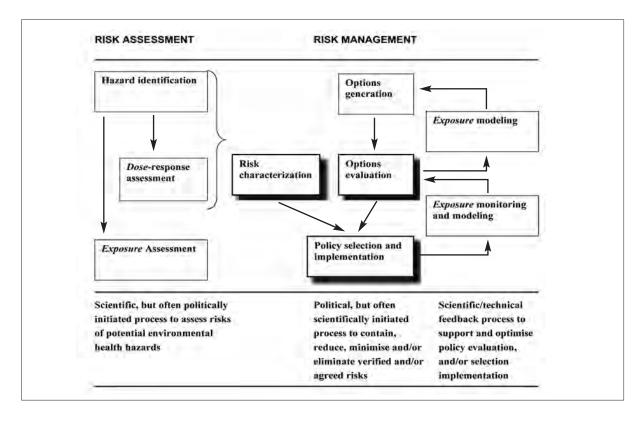


Figure 1. The relationships between exposure assessment, risk assessment, and risk management (NRC, 1983).

# 2 DETERMINANTS OF INDOOR AIR QUALITY (IAQ) AND EXPOSURE

# 2.1 Indoor pollution sources

The indoor environment of a building is a complex system that involves many parameters which impact on health and comfort. The building acts as a shelter comprising a partially or totally enclosed space or cluster of spaces, erected by means of a planned process of forming and combining components and materials. Several spaces may be defined in the building with different environments and, specifically, different air requirements and actual air conditions. The air exchanges to surrounding air compartments or to the ambient air are limited by building components and strategies of operation and use (mechanical ventilation on/off, natural ventilation, etc.).

The building itself through its design, given the appropriate criteria and requirements for each specific application and the materials to put into use, is clearly the primary 'system' to be optimised from the indoor air quality perspective. Indoor air quality depends strongly, on the one hand, on the interaction between the building and its outdoor environment and, on the other hand, on the way the building is used, including the behaviour of the occupants. Airconditioning systems represent a complement to the building itself to guarantee the conditions required in each specific space.

A source is a point of emission of a substance (pollution) or other dysfunction (noise, unacceptable light levels). Different types of sources exist. Source characterization consists of:

- (i) determination of its nature and origin
- (ii) measurement of the intensities of its emissions
- (iii) evaluation or estimation of possible health or comfort effects.

The aim of a good design is to anticipate the fulfilment of the specific minimum requirements of the building or space in the building from the design phase bearing in mind the strength of sources and nature of their emissions and means of attenuating their importance by source control and by ventilation (dilution and removal or filtration). The importance of strategies such as source control depends on the correct identification of the sources and their pollutants. The role of ventilation and systems available to provide fresh air from outside at the appropriate rate and physical state should be better known and integrated in the building design and management.

# Major sources of chemical pollutants in indoor air are:

- Ambient air pollution due to traffic and urban and industrial activities (e.g. Hodgson et al., 1992, Stump et al. 1992, Conner et al., 1995, Quigley et al. 1995, Götschi et al. 2002), which come into the building through the ventilation system or by infiltration (building envelop permeability),
- Water and soil (air pollutants coming through water supply, radon, methane and contaminated soils),
- Building materials (wall and floor coverings, paints, insulation materials etc.) (e.g. Little et al. 1994, Karpe et al. 1995, Hodgson et al. 1993, Kirchner et al. 1993, Kirchner et al. 1997, Wolkoff et al. 1990, Wolkoff et al. 1995a, Volki et al. 1990, Gehrig et al. 1993, Bremer et al. 1993, Plehn 1990, Saarela et al. 1994, Horn et al. 1998) and furnishings (e.g. Salthammer 1997),
- Processes that occur within buildings (combustion, heating, ventilation and air conditioning systems, paper processing such photocopying (e.g. Wolkoff et al. 1993, Wadden et al. 1995 etc.),

• Occupants themselves and their activities (tobacco smoking, use of cleaning products (e.g. Colombo et al., 1990, Person et al. 1990), cooking, pets, plants etc.).

Chemical pollutants of indoor air are mostly represented by complex mixtures of substances. Some of them, such as formaldehyde, originate almost exclusively from indoor sources, while others may enter the indoor space with outdoor air, particularly in case of high outdoor air pollution.

The concentrations of some chemical pollutants indoors are in a range where they can induce toxic effects or significantly interfere with the comfort of the occupants.

The principal indoor air chemical contaminants include combustion gases (mainly NO<sub>2</sub>, SO<sub>2</sub> and CO), environmental tobacco smoke, particulate matter, mineral fibres, organic compounds (polycyclic aromatic hydrocarbons, volatile organic compounds and formaldehyde) and pesticides.

Among nitrogen oxides, nitrogen dioxide is the compound most abundant and important; it is produced by indoor combustion sources such as gas cookers and cigarette smoke. Sulphur dioxide is a gas produced by sulphur-containing fuels, namely fuel oil and some natural gases. Carbon monoxide in indoor air derives from tobacco smoking and unvented combustion sources. Inhalable particles are produced by cigarette smoke, combustion sources and dust produced by the occupants' activities or brought in from outdoor air or through the ventilation systems. The composition of airborne particles generated by combustion processes varies according to the type of fuel and combustion conditions.

Mineral fibres include natural fibrous materials (the most important of which is asbestos) as well as man made mineral fibres, the most used of which in buildings are represented by glasswool, rockwool and other related materials. Both asbestos and its synthetic analogues have found wide use in buildings as insulants and thermal cohibitors and, to a lesser extent, as reinforcing materials for other building coatings or finishing products. Release of asbestos fibres into indoor air of buildings may take place due to a slow deterioration of these products because of a direct damaging action on the coatings or during building modifications and refits.

Many organic compounds, produced by incomplete combustion processes (tobacco smoking and combustion appliances) or deriving from other sources, are commonly found in indoor air of buildings.

Polycyclic aromatic hydrocarbons (PAH) are a wide group of chemicals with two or more aromatic rings. They are present in indoor air both as vapour and attached to particles, and derive from combustion sources (i.e. kerosene boilers) and tobacco smoking. In addition to PAH, several other organic chemicals are present in indoor air; they have been classified into four groups based on their volatility: very volatile organic compounds (VVOC), volatile organic compounds (VVOC), semi-volatile organic compounds (SVOC), and particulate organic matter (POM). This grouping has practical importance as volatility is a major factor influencing the proper technique for sampling these substances in indoor air. The VOC, including substances with boiling point ranging from 50-100 °C to 240-260 °C, are the most important among these chemicals. This group includes several different compounds, namely aliphatic hydrocarbons, aromatic hydrocarbons, chlorinated hydrocarbons, aldehydes, terpenes, alcohols, esters, and ketones. Although formaldehyde and some other aldehydes are

VOC, these substances are considered separately because they require particular techniques for sampling in indoor air, due to their high reactivity.

The occupants and their activities, some materials and products used in buildings, and outdoor air are the main sources of VOC in indoor air. Important sources of VOC in office buildings are represented by tobacco smoke, furnishing, carpets, and textile coating; these materials release VOC particularly soon after their first installation, however a slow emission takes place for months and years along with their progressive wearing. Short high level emissions of VOC can also be originated by printers, photocopiers, cleaning materials and solvents used indoors.

So far more than 900 different VOC have been detected in indoor air of buildings; 250 of them have been measured at concentrations higher than 1 ppm. Generally, a single air sample collected in a building shows 30-50 VOC, the concentrations of which very rarely exceed 50 µg/m³ individually, the total concentration of VOC being usually lower than 1-3 mg/m³.

Formaldehyde is an organic vapour mainly emitted from urea-formaldehyde resins used for insulation purposes, furnishing made of certain types of resins, carpets, and other materials used indoors. This substance is the principal cause of poor air quality in prefabricated houses, caravans, and environments densely fitted with synthetic carpets or furnishing.

Pesticides are a very heterogeneous group of chemicals, generally characterised by low volatility. They are used in buildings to control insects, parasites, and other pests. They can penetrate into the buildings from cracks when they are applied in the basement. A very particular source in buildings may be represented by the biocides used to treat the air-conditioning systems or other components of the ventilation systems. The biocides under these circumstances may spread with the air to the entire building through the ventilation system causing inadvertent exposure of the occupants. High level exposure to pesticides, principally pentachlorophenol, has been documented in residents of log homes or houses internally coated with wood treated with wood-preserving agents.

Environmental tobacco smoke (ETS) is a very complex mixture of gases, particles, organic compounds and products of incomplete combustion of tobacco and paper. Cigarette smoke comprises the "mainstream", inhaled by the active smoker and returned to the environment after lung filtration, and the "side stream", released into the surrounding environment directly from the tip of the cigarette. The latter is more critical for the exposure of passive smokers. About 3800 single chemicals have been identified in ETS; the main toxic substances include carbon monoxide, polycyclic aromatic hydrocarbons, several VOCs, ammonia, volatile amines, hydrocyanic acid and tobacco alkaloids.

The number of pollutants present in the indoor environment and their diversity is immense and probably only a small number of these have been properly characterised. There has been a need to identify a short list of pollutants that could be taken as priority substances to be traced and quantified either as pollutants or indicators of other potential pollution. This kind of prioritisation has been carried out in the recently published INDEX project (Kotzias et al. 2005).

In WHO (2000) the compounds were classified into: 1) organic air pollutants, such as some VVOC (vinyl chloride), VOC (benzene styrene, trichloroethylene) and SVOC (polycyclic

aromatic hydrocarbons (PAH), polychlorinated biphenyls (PCB), polychlorinated dibenzodioxins and dibenzofurans (PCDD/PCDF) and formaldehyde, acrylonitrile, carbon disulfide, and carbon monoxide, 2) inorganic air pollutants (lead, hydrogen sulphide, asbestos), 3) classical air pollutants (nitrogen dioxide, ozone and other photochemical oxidants, particulate matter and sulphur dioxide), and 4) specific indoor air pollutants (environmental tobacco smoke (ETS), man-made mineral fibres and radon).

Volatile Organic Compounds (VOC) have been in the focus of the IAQ, because their levels indoors are higher than outdoors (Wolkoff et al. 1995b). An European audit on 52 buildings (Bluyssen et al., 1996) concluded that the major VOC sources are building materials and HVAC systems. Other sources are cleaning and maintenance products, tobacco smoke and office machines (printers and photocopying machines). Most of the VOC are odorous compounds and can in most cases be detected by the human nose even at the very low levels, which can contribute to personal discomfort. More serious effects are attributable to VOC at higher concentrations: neurotoxic symptoms, respiratory effects, mucous membrane irritation, skin irritation (ECA, 1991). At present benzene and formaldehyde are the only IAQ relevant VOC recognized as carcinogenic and classified as Category 1 (ECA 1997, IARC 2006). Furthermore the different compounds may interact synergistically and cause unexpected toxic effects that could not be expected from the known toxicity of the individual components.

Semi-volatile organic compounds (SVOC) are now receiving much more attention than before. Their major sources are in the outdoor air that may also infiltrate the building giving high level of concentration in the indoor environment. The trend nowadays, with the new building materials becoming cleaner, is that the VOC levels tend to decrease and the SVOC levels tend to increase, reflecting a substitution of VOC with SVOC to decrease exposure concentrations. However there is a need for more toxicity information for many such SVOCs.

In most situations the major source of airborne particulate matter is the outdoor air (urban traffic, industry) and some activities carried out indoors (smoking, cooking). Gases and vapours may be adsorbed by respirable particles and then transported into the human respiratory system (mainly the lungs) where they can produce negative health effects (ICRP 1994). It should be noted that aliphatic acids and aldehydes are normally found in house dust, in addition to other VOC and SVOC that were identified in office dust (Clausen et al, 1994; Wilkins et al. 1993).

The major source of  $NO_2$  is the outside air (vehicle emission) (Bostrom 1993). Local peak concentrations in residences originate from specific sources, e.g gas stoves, wood stoves, fireplaces, kerosene heaters and candles. In offices, electrostatic equipment (photocopying machines and laser prints) emits  $NO_2$  (Olander 1992). Tobacco smoke is another source.  $NO_2$  can also react with alkenes in the presence of ultraviolet light, giving rise to products that cause mucous membrane irritation (Kane and Alarie, 1978).

The major part of ozone in indoor air comes from the outdoor air. However, in offices, electrostatic equipment such as copy machines and laser printers may emit ozone. Ozone may also react with VOCs (e.g. terpenes) to give rise to irritant compounds (Wolkoff et al. 2000). For example, the exposure of a carpet to ambient air containing ozone may as a consequence cause an increase of the emission of aldehydes (Wolkoff 1995b). In many cases the products of the reactions are stronger irritants than their precursors. Interactions between other reactive air pollutants are expected to be frequently occurring but little general information is available.

The major source on indoor radon is the ground of the site with building materials generally making a smaller contribution. Outdoor air usually acts as a diluting factor due to its low radon concentration but in some cases, as in high-rise apartments built with materials having low radon content, it can become the principal contributor to indoor concentration (ECA, 2000).

The sources contributing to indoor air pollution are responsible for either continuous emissions (long-term emission with constant sources strength such as material emissions) or discontinuous emissions (short term emissions with variable source strength such as human activities). Spatial and temporal variations can occur, depending on movement of the air, infiltrations between rooms in a building, etc. In conclusion, it may be stated that all ventilation aspects are of extreme importance in the concentrations levels obtained in a building.

# 2.2 The physical indoor climate and its interaction with IAQ

The principal physical factors of the indoor climate are temperature (air and enclosure surfaces), relative humidity, air movement, ventilation and air pressure. While not "climatic"

in the traditional meaning of the word, light and noise levels may also be usefully added to this list. Within a building these indoor climate components will display spatial and temporal variations both due to effects on the building arising from the outdoor ambient climate and as a result of occupant behaviour and requirements. To fulfil comfort requirements there are several options according to the needs: limitation of the lowest temperature (heating); limitation of the highest temperature (cooling); control of humidity; provision of clean and circulated air (ventilation).

| Factor  | Target Values          |
|---|------------------------|
| Room temperature, winter                              | 21-22 °C               |
| Room temperature, summer                              | 22-25 °C               |
| Floor temperature                                     | 19-29 °C               |
| Vertical temperature difference                       | <2 °C                  |
| Air change rate (residence)                           | > 0.8 hr <sup>-1</sup> |
| Air velocity, winter 21 °C                            | < 0.10 m/s             |
| Air velocity, summer 24 °C                            | < 0.15 m/s             |
| 27 °C   | < 0.20 m/s             |
| Relative humidity of air, winter                      | 25-45 %                |
| Relative humidity of air, summer                      | 30-60 %                |
| Noise level of heating and air conditioning equipment |                        |
| Offices   | < 30  dB(A)            |
| Living and bedroom                                    | < 25 dB(A)             |

Several factors may simultaneously influence the indoor climate in particular heating, ventilation and air conditioning. The materials and methods used in the construction of the building as well as its operation, use and maintenance will also have considerable impact on the indoor climate. In the present context of IAQ most of the components of the indoor climate, either individually or in combination, may be important determining factors for the concentration of many indoor air pollutants. Before considering these aspects of the indoor climate it is instructive to describe the characteristics of an acceptable physical indoor climate for common work and living spaces. A good example are those classified as Category I by the Finnish Society for Indoor Air Quality and Climate which when used as target values should give a high satisfaction to and result in a low number of complaints from the occupants. (Seppänen et al. 1995).

The physical factors of indoor climate can have a major influence on the concentrations of many common indoor air pollutants. For instance, the rates of emission of many VOCs from building furnishings and fittings is temperature dependent and, in general, if the temperature of an occupied space increases one may expect a consequent increase of emission of such

substances into the air spaces (Van Der Wal et al. 1997, Haghighat and De Bellis 1998). This may be of particular importance at the commissioning phase of a building when adhesives, paints and solvents etc., present in many fittings, may not be adequately cured or stabilised and thus are likely to produce larger emissions of organic pollutants initially than at a later time. Acceptable models for the curing or commissioning period are missing.

A most immediately obvious and common IAQ problem in buildings is that the ventilation rate may be insufficient to reduce the air concentrations of pollutants from indoor sources to an acceptable level. Poorly maintained and unclean ventilation systems may also contribute to indoor air pollution (Bluyssen et al., 1996). Ventilation has also a major influence on indoor air pollutants in other ways. If, for example, the make-up air drawn into a building from outside is already contaminated with a pollutant then an increase in the concentrations of the pollutant in the indoor air may be expected unless it is removed from the make-up air by filtration or some other cleaning technology.

Physical factors of the indoor climate may also produce conditions conductive to an increase in the indoor populations of certain biological agents that can affect IAQ. This is true in particular of air temperature and relative humidity where in combination they may cause an increase in such biological agents as house dust mites, fungi and bacteria all of which can make their respective contributions to indoor air pollutant levels. It is known that some examples of building materials, when attacked by fungi, emit odorous VOCs, the so-called MVOC (microbiologic volatile organic compounds) that result from the metabolism of bacteria and fungus (Norback et al. 1995, Smedje et al. 1996, Thogersen et al. 1993, Rocha et al. 1996, Clausen and Oliveira Fernandes, 1997). The increase of the humidity can also be a direct factor on the emission of such compounds which were not emitted from dry building materials. It was verified that vinyl materials exposed to high levels of humidity produce 2-ethyl-1-hexanol, phenol and cresol (Gustafsson 1992). Such interactions are expected to be frequent but little general information is available.

While the air pressure in a building is not usually considered as an important component of the climate from an IAQ perspective it can have an influence on the concentration of some indoor air pollutants. This is true in particular of indoor radon gas levels where the principal source is usually the soil gas beneath the building with normal building materials making a smaller contribution. As the air pressure in a building is generally slightly lower than that outdoors a negative pressure differential between indoor air and soil gas will exist thus causing a micro-flow of soil gas into the building. This is considered to be the main mechanism for transporting radon gas from the soil into buildings and is generally more important than diffusion in this regard. In a similar manner for buildings on landfill sites this differential pressure driven flow of soil gas may cause gases such as methane to reach an unacceptable or even a dangerous level in near ground level air spaces in some buildings.

# 2.3 Factors determining exposure to indoor air pollutants (sources, sinks, dynamics of IAQ and ventilation)

By nature of its construction and the activities going on in the building, air pollution will appear indoors. Air pollution is the presence of unwanted material (air pollutants) in the air. The term "unwanted material" here refers to material in sufficient concentrations, even if not detectable by any instrument, present for a sufficient time, and under circumstances to interfere significantly with comfort, health or welfare of persons or with the full use, enjoyment and maintenance of property.

In this context, exposure is any measurable environmental factor, which can result in a dose received by a target organism from the atmospheric environment. Quite commonly exposure is defined as the product of the pollutant air concentration and time. The exposure is assessed through determination of the emissions, pathways and transformations to estimate the concentrations/doses to which humans are or may be exposed.

This chapter deals with the factors affecting the 'history of air pollutants' indoors. This includes processes such as emission from sources, subsequent diffusion, adsorption / desorption, dilution and removal, transport and spreading of pollutants from the sources to the occupants thus creating an exposure to these. These processes are the key elements to be considered in the prevention of IAQ problems and in the regulation of indoor air pollutants. Models that take these factors into account will be essential for the development of indoor exposure and risk assessment. Some attempts have been made to develop models but no general model has yet emerged.

Emission is a measure of the discharge of a pollutant from a source, commonly expressed either as a rate (amount per unit time) or as the amount of pollutant per unit volume of gas emitted. A source emission rate, or emission factor, is the amount of air pollutants emitted per unit time (sometimes also per unit source) from a source. Emitted pollutants will disperse in the building through infiltration, diffusion and natural air movements. All these processes are dependent on gradients of air temperature, on air pressure variations in the building spaces or on direct dynamic effects from ventilation. As a result of these influences pollutant concentrations will suffer both temporal and spatial variations within a building.

Infiltration is caused by a leaky envelope due to a locus on a wall, ceiling or floor where a fitting or component passes completely through the wall etc., e.g. window, light fitting, pipe work etc.

Diffusion of gases causes molecules of all gases to move freely and tend to distribute themselves equally within the limits of the vessel enclosing the gas; thus all gases diffuse within the limits of any enclosing walls, and are all perfectly miscible with one another. Thermal gradients within the enclosure may cause some stratification. However due to surfaces with different temperatures, in the enclosure, natural convection may induce air movements that might enhance the diffusion effect on the transport of pollutants indoors.

Important factors, besides the transport by air movements, are the removal of pollutants through dilution, filtration, adsorption or deposition, absorption and re-emission from sinks, chemical or biological changes of pollutants in the sinks or in the air. These factors all combine to a dynamic equilibrium, which in principle can be described mathematically but is very difficult to model realistically.

Ventilation is the process of supplying and removing air by natural or mechanical means to and from any space. Natural ventilation is due to incoming air through openings or leaks on the envelope of the space or the building. It is caused by the dynamic pressure effects of wind direction and velocity on the façade and thermal effect on the gravity field (Archimedes effect). The provision of air to an enclosed space must be sufficient for the needs of the occupants or of any process taking place in the enclosed space. The ventilation efficiency describes the ability of a mechanical (or natural) ventilation system to remove/dilute pollution originating in a room, either at steady state or transient rates.

Ventilation is measured through the ventilation rate, which is the number of room air

changes per unit time of replacement or make-up air. It is usually expressed in air changes per hour, or as the volume flow rate of the replacement or make-up air coming into a room or building per unit volume (usually m³/s.m³ or 1/s.m³).

Infiltration rate is the rate of which outside air infiltrates through leaks into a room or building. This may be viewed as equivalent to a fresh air change rate, and is usually expressed in ach (air change rate) or 1/s.

General ventilation acts mainly by dilution which is the removal of contaminated air and its replacement with clean air from other parts of the general building area as opposed to local ventilation, which is specific air changing in the immediate air of a contamination source. An example of local ventilation is a laboratory fume hood. This air movement will carry pollutants away from the sources. Dilution is used to reduce the concentration of an air pollutant by the administration of a neutral volume of air.

Ventilation using only natural motive force such as wind pressure or differences in air density is called natural ventilation. Mechanical ventilation is ventilation by means of one or more fans. A HVAC system is the heating, ventilation and air conditioning mechanical system used in buildings. To operate properly it must handle sufficient air masses to control, both, air cleanliness, temperature and moisture. As temperature control often requires much more air than pollutant dilution, some air is re-circulated for temperature control. The re-circulated air causes old room air - removed by natural airflow or the mechanical ventilation system, to be reintroduced into the rooms via HVAC system cupboards, floor spaces, etc.

In summary the emitted pollutants are distributed within the building and the concentrations vary time wise and according to location.

During the transport of pollutants through the building the air and pollutants will make contact with surfaces to which they may adhere and thus be removed from the air. Some of these processes are built in features of the building such as filters and air cleaners, other are more natural physical processes.

Some filters can specifically address one pollutant or may such as activated charcoal address most pollutants. Filters have limited capacity and efficiency and require constant maintenance. Electrostatic precipitation is a widely used method of controlling the particulate pollution of air. The air, containing solid or liquid particles suspended in it, is subjected to a mono-directional electrostatic field so that the particles are attracted to, and deposited upon, the positive electrode.

In general, absorption is a physical process in which one material (the absorbent) takes up and retains another (the absorbate) with the formation of a homogeneous mixture having the attributes of a solution. Chemical reaction may accompany or follow absorption and the absorbed pollutants tend to remain on the absorber. Adsorption is a physical process in which molecules of gas, of dissolved substances or of liquids adhere in an extremely thin layer to the surfaces of solid bodies with which they are in contact. In the present context this process is the most useful property of activated charcoal. Chemical reaction may accompany or follow adsorption, giving an irreversible character to the process. These types of processes are part of what are called indoor sink effects.

Mathematical models exist which allow estimations of the absorption/adsorption processes from multi component air pollution as it is found indoors.

As a result of these processes a fraction of the pollutants are removed from the air and therefore cannot contribute to human exposures indoors. The effects of these processes mostly are localized and thus further complicate the description of indoor exposures.

In indoor air chemistry, a sink is an area or part of the building and its atmosphere in which, or a process of which, one or more pollutants are removed from the air. The adsorption of air pollutions to and subsequent re-emission from materials is called a sink effect. The compound may be re-emitted (by temperature increase, for example) and thus again contribute to the air pollution. These processes strongly affect the time course of exposures indoors. Some mathematical modeling is possible (e.g. Dunn and Tichenor 1988, Evans 1996; De Bortoli et al, 1996, Little and Hodgson 1996; Kephalopoulos 1999; Guo 2002). Nevertheless there is a lack of knowledge of the actual adsorption coefficients as they depend on the coupling substance (adsorbate)/ material (adsorbant). Only a few coefficients are actually known (Meninghaus et al., 2000; Kirchner et al., 1999).

During the transport through the air physical and chemical reactions and other processes may change the pollutants thereby removing the contributions of some of them from the original spectrum of exposures. At the same time these reactions may create new exposures to new compounds. Important processes in this regard are aggregation, oxidisation, photochemical reactions and catalytic reactions on surfaces.

Aggregation causes separate particles to fuse into clumps or masses. Literally, oxidation is a reaction in which a substance combines with oxygen provided by an oxidizer or oxidizing agent. An oxidation reaction may occur even when free oxygen is not present. An oxidation reaction is always accompanied by an offsetting (balancing) reduction reaction in which (1) oxygen is removed from a compound; or (2) atoms, molecules, or ions gain electrons. Ozone is a well-known oxidizer. Photochemical reaction is a chemical reaction that is initiated as a result of absorption of light.

The likelihood of a pollutant to undergo reactions depends not only on its own concentration but also on the presence of oxidizers or light and on the concentration of catalysts. The likelihood of reactions are described by means of the reactivity of the compounds which describes tendency of a substance to undergo chemical reaction either by itself or with other materials accompanied by a release of energy. These processes are complex and poorly understood for indoor air pollutants.

The emission equilibrium concentration is the concentration of a pollutant in a room after ventilation and thermal equilibrium is achieved and the emission rate has achieved equilibrium following exposure to the new environment in the room.

The processes illustrated above may combine into a dynamic equilibrium in which opposing processes are going on at the same rate in the indoor system, thus keeping the system unchanged. In such a situation the system is said to be in dynamic equilibrium.

Some attempts have been made to develop the ventilation equation to take these factors into account. Such a model will be essential for the development of indoor exposure and risk assessment but no general model has yet emerged.

# 2.4 Time-microenvironment- activity, behaviour

Characterization of the air existing in a space is a very complex issue, because of all the

factors involved. Infinitely different combinations including occupant behaviour and options can be obtained in order to achieve different compositions in the indoor air, including particles and bio-contaminants.

Another variable that is very important is time. The age of the materials has consequences on the emitted compounds: the major part of materials experience some decrease of VOC emissions with time. Some of these decrease curves are faster like in the case of paints; others are slower like for carpets. Many models were developed with the aim of describing the life of a material (Dunn and Tichenor, 1988, Clausen et al., 1993, Chang et al. (1992 and 1997). Some of these models include sink effects, but the validation of these models with experimental data in most cases has not yet proved to be possible, since data for that purpose is very scarce. In addition thousands of interactions between compounds present in the indoor air are occurring all the time both among themselves and between them and the indoor surfaces. For reactions among indoor pollutants to occur there must be sufficient time for the pollutants to interact. Ventilation largely determines the time available for such interactions, thus ventilation affects indoor chemistry. The smaller the ventilation rate, the longer the residence time of a specific condition of the indoor air. A longer residence time means a longer interval during each indoor pollutant can decompose or react with one another, generating new products. Ventilation also influences indoor chemistry, in a secondary manner, by affecting the concentration of reacting species. As the ventilation rate decreases the concentration of pollutants from indoor sources increase indoors while the indoor concentrations of pollutants from outdoor sources increase. (Weschler and Shields 2000).

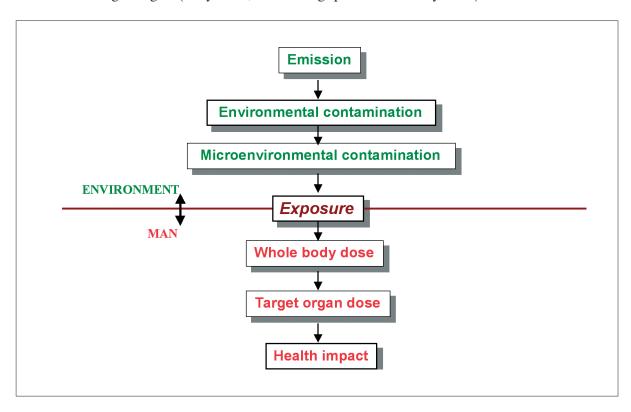
Finally, there is the impact of the human occupants not only arising from their own emissions but also as a result of their own lifestyle options and behaviour. Where heating, humidification / de-humidification or cooling functions are on, in addition to the ventilation effect, the situation can be controlled automatically or be subject to human intervention. These options may affect the indoor air quality in a decisive way. There are opinions that support the control of the personal environmental conditions instead of the room conditions (Fanger, 2000). Nevertheless the whole spectrum of control options between the use of natural ventilation through the desk-controlled air conditioning implies a wide variety of indoor air quality conditions.

A new approach regarding the quality of spaces is needed: each space in a building that can integrate many different functions and services has to have its specific requirements clearly defined, according to the language of EU Directive 89/106 on Construction products. Then, at the design stage, there is a need to identify the potential sources of indoor air pollution and select the most appropriate strategies to make sure that the requirements stated at the beginning will be fulfilled. Among the strategies the most important is the source control strategy that leads to select clean materials and to place potential sources in confined areas with ways of removal of pollutants as close to the emission as possible. Ventilation is a necessary complement. It allows for two other strategies: the removal of pollutants in the air by filtration and the dilution of the pollution indoors. Nevertheless the fact that ventilation requires energy for its operation implies that the strategies associated with ventilation must be used with care. Otherwise, the link between indoor air quality and outdoor air will become 'interactive' in the sense that as energy depends mostly on fossil fuels more energy being needed for ventilation implies a deterioration of outdoor air quality and may conflict with sustainability principles.

# 3 DETERMINATION OF EXPOSURE: STRATEGIES AND TECHNIQUES

# 3.1 General principles for exposure assessment

Probably the most widely accepted definition for exposure in environmental health is the one presented by Wayne Ott: "the existence of a person and an agent (contaminant) in the same micro-environment at the same time (in potential contact with each other)". Another definition locates exposure at the man-environment interface in the chain of events or flow of molecules from source via environmental concentration, exposure and absorbed dose to dose in the target organ (Lioy 1990, and Georgopoulos and Lioy 1994).



This figure shows the man-environment interface position of exposure, and defines the roles of exposure assessment (from source to exposure; environmental chemistry and physics, human activity) and dose response assessment (from exposure to health impact; toxicokinetics and pharmakodynamics) in environmental health risk assessment (the whole chain of events from source to health impact).

#### General

For many air pollutants no threshold values can be defined under which the risk would be zero. This means that exposure reductions are likely to be beneficial to health at all levels of exposures. Therefore, not just the exceedance of a particular exposure level, but the whole exposure distribution is relevant for population level risk assessment. We need such information for the general populations as well as for population subgroups of particular interest.

Management of outdoor air is better developed than for indoor air. Urban air is an umbrella concept combining ambient and indoor air. It may be broken into indoor, outdoor and

commuting microenvironments, residential, occupational and public microenvironments, each requiring different approach and risk management strategies.

The legal and administrative framework for regulating air pollution - except in ambient air and certain workplaces - is very diverse and still mostly undeveloped.

Public health risk management requires that different exposure patterns and dose-response relationships for different air contaminants and population subgroups are assessed. This is because different microenvironments contribute differently to the exposures of different population groups - due to both microenvironmental concentration ranges and the time fractions spent in each. To optimise the source control investments (with the aim of reducing public health risks), the relative contributions of the sources to microenvironments and microenvironments to exposures and risks should be known.

Development of exposure based urban air quality management should integrate the health relevant air pollution impacts from all common urban microenvironments and individual's activities. Such an intersectoral policy could focus on the most efficient mitigation alternatives for maximum public health benefits, minimum cost or minimum intervention.

The necessary tools for urban air quality management have to cover the following elements:

- Ambient air quality monitoring and modelling
- Micro-environment monitoring and modelling
- Time-microenvironment-activity monitoring and databases
- Exposure scenarios
- Exposure monitoring and modelling.

#### 3.2 Exposure to what

A monitored air pollution compound may be a source marker, an indicator or a causal agent for health consequences. In risk management one needs to differentiate between these roles. It is important to carefully assess the appropriateness of using indicators or proxies in each application. E.g. an indoor  $NO_2$  exposure indicator can be the use (h & min per day) or just presence of a source (gas stove). A yet simpler indicator does not assign either concentrations or compounds, like when using damp building or presence of a smoker as a proxy (or label) of exposure. When indicators are used in regulation, it is essential to differentiate between causal (such as smoking) and associative (such as  $NO_2$  – probably) indicators. Regulating non-causal associative indicators (proxies) may accomplish significant indicator reductions, and yet be quite inefficient for risk management.

Consider also that outdoor and indoor pollution, and personal exposures have demonstrated impacts on individual, corporate and public economies, not only disease and/or prevention costs but also quality of life and productivity losses. Resistance to new policies and policy changes can sometimes be overcome by explicitly addressing these facts.

Several studies have indicated the presence of many chemicals in indoor environments due to infiltration of polluted outdoor air and of emissions of various indoor sources (e.g. building materials, activities of the occupants, consumer products, smoking, etc). Some studies have been performed for assessment at local or national level, but very few at European level to allow for comparisons across Europe. For many of these chemicals, the risk to human health and comfort is difficult to assess, because of a scarcity of toxicological data, especially at low

concentration levels and human dose response characteristics. At the same time, there have been only quite limited data on prevailing population exposures in Europe. Recently, however, such data has become available and therefore, risks of many of these chemicals in indoor air can now be assessed and regulation of the risks is becoming feasible. Therefore, there is an urgent need to develop a strategy for the identification of priorities in testing, risk assessment and regulation. In 2005, a European Project INDEX identified these priorities and provided suggestions for the forthcoming European Commission strategy and action planning in the area of indoor air pollution. This was achieved by: 1) setting up of a list of priority substances to be regulated in indoor environments on the basis of health impact criteria; 2) providing suggestions and recommendations on potential exposure limits for these substances; and 3) providing information on links with existing knowledge, ongoing studies, legislation etc., at world scale (Kotzias et al. 2005).

# 3.3 Rapid assessment methods (Risk Management)

There are several more or less widely applied methods for rapid assessment of the presence or absence of an air pollution issue, as well as for the selection of proper mitigation actions. These methods are radically simpler than real data based and validated total exposure and risk assessment methods, yet they may provide adequate support for many practical decisions.

# 3.3.1 Ambient air quality

Optimally designed monitoring networks are capable of providing - for certain air pollutants and source contributions - information which is directly relevant to long term exposure, exposure distributions and even exposure peaks. For non-reactive air pollutants with no significant indoor or personal sources ambient air monitoring & modelling is often sufficient for both population exposure assessment and for risk management options evaluation. Even for reactive pollutants with non-dominant indoor sources ambient concentrations may provide useful surrogates for long term population exposures of ambient origin particularly in relation to cumulative, chronic risks and stochastic effects like cancer. Besides, ambient monitoring is essential for assessing ambient concentration trends and for outdoor (exposure component) source apportionment. Ambient monitoring is, of course, of no help when assessing the risks of indoor pollution sources.

#### 3.3.2 Questionnaires

Questionnaires are the fast and easy way to obtain information about the presence/absence of significant indoor pollution sources, such as tobacco smoking, gas stoves, use of certain household products etc.

Questionnaires are well suited for qualitative, but less reliable for quantitative information collection. While the presence or absence of an attached garage in the house can be acquired quite reliably, a reliable quantitative estimate of its use or emission is already more difficult to obtain, and e.g. information of workplace exposure to tobacco smoke has been found to be highly subjective. For one subject, it means someone smoking frequently in the same room, for another the detection of the smell of tobacco while walking to the lavatory. Such biases are not random, but depend on the past/present smoking or residential ETS exposure status, gender, age and health of the respondent.

It is therefore essential to use validated questionnaires, experienced survey staff and on the spot checks in questionnaire based exposure assessments.

Quantitative questionnaires - different time-microenvironment-activity diaries - are by far the most used techniques for time-microenvironment-activity data collection.

#### 3.3.3 Source inventories

Source inventories can replace questionnaires focused on the presence or absence of sources in microenvironments. They can be based on existing databases (of e.g. gas using households and their gas bills), or be performed by trained survey staff visiting the microenvironments. The advantage of the latter is better comparability across the study population (e.g. differentiation between vented and unvented gas appliances), the disadvantages are cost and cross sectional view lacking longer perspective (e.g. how often and how long time was gas stove used). The two methods can, of course, be combined.

# 3.3.4 Intake fractions

Only small fractions of the emitted air pollutant molecules usually end up in the body of any human individual. This so called intake fraction (iF) varies from ca. 0.5 for active smoking,  $10^2$ -  $10^3$  for residential indoor sources,  $10^4$ -  $10^5$  for neighbourhood and local outdoor sources, to ca.  $10^6$  -  $10^8$  for regional and distant sources of non-reactive / reactive emissions. When air quality management actions are aimed at reducing the exposures to and health effects of air pollution, the broad range of iF values indicate huge differences to the benefit from reducing the same emission quantity from different sources.

Emissions from residential and occupational indoor sources have about two orders of magnitude higher intake fractions than near field sources outdoors. Consequently controlling even rather small indoor sources can be more effective for public health than controlling much larger outdoor sources, especially if they are non-urban and elevated. Individual intake fractions – especially for indoor sources – vary over several orders of magnitude. At total population level, however, the iF values tend to be rather robust.

In risk assessment iF estimates can be used to estimate population doses from different sources, in risk management they can help compare different risk management options.

# 3.4 Exposure and microenvironment monitoring and modelling

Realistic population exposure distribution data are essential, because they provide information of the high-end exposure levels, frequencies and causes, and because (an estimate of) the population disease burden from air pollution is the product of population exposure distribution and the respective dose/response function.

At least some exposure monitoring techniques are available for most of the regulated air pollutants. For reactive compounds, a number of organic compounds or microbial compounds, and the coarse fraction of PM<sub>10</sub>, exposure monitoring is least developed. Regarding the time scales, most information is available for 12-h to 1-week exposures, while long term or short time resolution data is lacking. Further limitation is the lack of integration across the microenvironments, as well as the great gaps in information on time-microenvironment-activity patterns, for many geographic areas (e.g. rural), population and age groups (e.g. infants between 1 year and school age), and microenvironments/activities of interest (recreational microenvironments and out of home hobbies). Large differences in the availability of tools and information exist between countries.

Direct exposure measurement is invasive, expensive and consequently a rare luxury in indoor air exposure assessment. The personal exposure monitoring devices that people carry with them must be lightweight, silent, highly autonomous and still one week is about the maximum time that any population representative sample of individuals will comply with personal exposure measurements. Most air pollution exposure studies have measured just one air pollutant (CO, benzene, NO<sub>2</sub>), although some have measured many gaseous and particulate pollutants such as PM<sub>2.5</sub>, CO, NO<sub>2</sub>, BS, PM<sub>2.5</sub> elements, VOCs simultaneously. Such large-scale multi-pollutant air pollution exposure study in Europe has been for example the EXPOLIS study (Jantunen et al. 1998, Koistinen et al. 1999, Edwards et al. 2001). In the German Environmental Surveys (GerESs) run at regular intervals since 1985/86 (Seifert et al. 2000) in addition to air and house dust, and tap water samples collected indoors, human biomonitoring samples and - to some extent - dietary samples are also analysed. In the United States the USEPA NHEXAS (Sexton et al. 1995, Pellizzari et al. 1995) assessed besides multiple air pollution exposures also some dietary and dermal exposures. An interesting finding from the exposure studies is that some pollutants are statistically close to each other (e.g. xylenes and ethylbenzene), while others occur independently of others (e.g. naphthalene, and phenol), obviously due to the same or different sources.

Yet even EXPOLIS, after measuring ca. 60 gaseous, volatile and particulate phase compounds, did not capture the whole air pollution exposure, nor does NHEXAS capture the total multi-pathway exposure. The question remains, did they capture the exposure which is relevant for a particular given issue or problem.

In addition to measurements, exposures can be modelled. Models can estimate past and predict future exposures, as well as exposures for large populations, that cannot be achieved by monitoring. In predicting the future, the alternative to modelling is guessing. Exposure assessors need models also for efficient assessment of the total exposure (exposure from all sources and in all microenvironments) to different air pollutants.

Because most individuals spend much of their time in the same usually well-mixed indoor space (home, office), microenvironment measurement closely approximates personal exposure while being in that space.

When the source of concern and ventilation (and other removal) rates are known, deterministic models can be used to compute indoor air level for a room or, with more information, for several rooms. The microenvironment exposure models group the main outdoor (e.g. street, garden, parking lot), indoor (home, workplace, restaurant) and commuting microenvironments and exposure relevant activities (e.g. smoking, cooking with a gas stove) into typically 5 - 21 categories. For each such microenvironment-activity category a concentration (or for population a concentration distribution) is assumed, modelled or measured. Similarly a real or modelled time (or for population a time distribution) spent in such microenvironment-activity is determined. The daily exposure is now computed as the sum of the daily concentration-time contributions from each microenvironment-activity category. In probabilistic population exposure modelling, the whole input data distributions for the respective population are used to generate respective output data distributions (Hänninen et al. 2003 and 2005a).

Microenvironment concentrations and exposures cannot be transferred from one city or region and applied to another city or region. Validated exposure models, source impacts in microenvironments or the impact of personal activities, e.g. smoking, developed for one region can, however, be transferred and applied elsewhere, provided that their validity has been re-evaluated with local data.

# 3.5 Source apportionment

Apportioning the total exposure (or indoor pollution concentrations) to its sources helps evaluate the quantitative roles of regional, local, traffic and neighbourhood, outdoor and indoor sources to population and individuals exposures. A multitude of source apportionment techniques is in use - in fact, have been used for decades. They can be divided into three main categories, logical, deterministic and statistical, and are not described here in more technical detail. A comprehensive review of source apportionment techniques and marker substances available for identification of personal exposure, indoor and outdoor sources of chemicals can be found in Bruinen de Bruin et al. 2006. Only by linking the exposures on one hand to sources and on the other hand to consequent health effects, the burden of disease arising from the different pollution sources can be determined, cost efficient policies developed, and verifiable gains for public health achieved.

Obviously, controlling sources, which have minimal contributions to exposures, can bring minimal risk reductions at best. Conversely, the highest health benefits can be expected from controlling those sources, which dominate the exposures. Often, but not always, the exposures are dominated by the nearest indoor, neighbourhood and traffic sources.

# 3.6 Risk management

Although buildings and their ventilation systems can be used to significantly reduce exposures to many ambient air pollutants, most air pollution control actions focus at the sources.

Exposure based UAQ management has the highest potential to act upon the most relevant sources and activities. Yet, no risk management body has currently responsibility for urban air in this broad sense. To improve we will have to take into account the following factors:

- Regulatory bodies are responsible for some microenvironments and for some they are lacking
- For monitoring techniques it does not matter if the microenvironment is public or private, but it does for policies and management
- In public spaces, large populations are affected, but exposure times are mostly short. In private residencies and workplaces, fewer people are affected in each, but the exposure times can be quite long.

To develop effective UAQ management options, time budgets of whole populations as well as time-microenvironment-activity patterns of the individuals must be known, and the exposures need to be attributed accordingly to microenvironments, activities and sources. In evaluating the UAQ management options, we need to predict the absolute and relative exposure consequences of different exposure scenarios, i.e. to model exposures from each source and activity to differentiate between the options in terms of the direct benefits, costs and other, often indirect consequences of the interventions. Alternative exposure control options can only be evaluated via exposure modelling.

While total exposure to an agent is the best indicator for its health effects and impact assessment, it is usually difficult to put into a legislative framework and implement, because total exposure is acquired in multiple indoor, outdoor, private, occupational and public, stationary and transport environments, which are controlled under different jurisdictions.

All in all there is a common agreement that exposure models should be further improved and their capacities exploited especially in connection to extensive surveys of health effects.

#### 3.7 Framework for exposure control strategies

Exposure based urban air quality management strategies that focus specifically on the contribution of indoor environments need to consider:

- contributions of indoor sources vs. outdoor sources to total exposure
- contribution of indoor exposure (originating from outdoor and indoor sources) vs. outdoor exposure
- objectives of the overall air quality management strategy, and the role of indoor air quality strategy in the whole.

These objectives are normally based on some commonly agreed principles, such as precautionary principle, polluter pays principle, as low [risk] as reasonably achievable—principle, and justification principle. Besides there are technical, economic and legal feasibility requirements, and additional societal and legal objectives such as sustainability, economic efficiency, social justice, and individual's rights, which must be considered in every risk management strategy. Exposure based strategies aim primarily at minimising exposures to harmful agents—as opposed to e.g. meeting indoor air quality standards (which does not consider the presence or absence of people), or eliminating specified indoor sources (which does not consider outdoor sources or other source control alternatives)—but such minimising can be guided according to any prioritisation of the above mentioned principles and objectives. In the following we will take a look at a few alternative approaches.

# 3.7.1 Whole population approach

This approach aims at minimising the total population exposure. It is the most relevant for pollutants for which long term accumulated exposure determines the risk and we can assume that risk is directly proportional to exposure with no threshold. It is commonly applied for genotoxic carcinogens and radiation risks. The total population risk is assumed to be directly proportional to the integrated population exposure, and the risk (e.g. lung cancer) may or may not manifest itself in any individual regardless of that individual's personal exposure. Exposure increases the probability, but not severity of the outcome. Consequently all exposure reductions would be beneficial for the population regardless of whether high or low exposures are reduced.

Examples include general ventilation and building codes that aim at reducing radon entry into and enhancing radon dilution from all buildings. In principle such a strategy would not reduce the relative differences in radon concentrations between buildings - all levels would be lowered - but because most people change their indoor spaces that they occupy many times during their life virtually all will benefit.

The disadvantage of this approach is that, in spite of population risk reduction, some individuals may remain at much higher risk than others. Besides, the approach is almost certainly based on an oversimplification of reality.

#### 3.7.2 Specific approaches

These approaches focus at exposures that are specific to certain agents, sources, spaces or target groups. These are based on the assumptions that the overall public health risks can best be reduced by targeting on a major portion of the total risk or most efficient risk reduction potential. The following examples should clarify the idea.

**Indicator based exposure strategy:** Many air pollutants from incomplete combustion of fuels are harmful to health. Benzene and some PAHs are carcinogenic, CO is acutely toxic, soot particles increase cardiopulmonary mortality, etc. Of these compounds CO is most abundant, inert and easiest to measure. Regulation to minimise indoor exposure to this target compound, CO, would indirectly also reduce other exposures to incomplete combustion products by eliminating or modifying their sources, indoors and even outdoors, with public health benefit potential far beyond the reduced acute toxicity of COHb in blood.

**Source targeted exposure strategy:** Attached garages, indoor tobacco smoking and unvented gas appliances expose the individuals within the premises to significantly elevated levels of VOCs, fine PM, CO and NO<sub>2</sub> concentrations. It can be argued whether elimination of these sources from residential, school or occupational buildings are exposure strategies, but they do efficiently reduce exposures of the most exposed populations. Such strategies are also simple to implement, in the sense that compliance is easy to verify. Even smoking restrictions, which require behavioural change of a significant part of the population, have turned out to be surprisingly easy and after some adaptation periods well over 90% efficient where applied.

**Space targeted exposure strategy:** Even the most active people spend 50-65%, and the most vulnerable people (infants, the elderly and chronically or temporarily ill) can spend over 90% of their time at home. Consequently air pollution exposures are dominated by exposures in the home, even when the sources and higher concentrations may be found outdoors. Exposure strategies focussing in the homes may be difficult to implement, because of the multitude and privacy of the residences, but they offer the highest potential for exposure control. Exposures in new and renovated residential buildings can be indirectly regulated when building codes are set and implemented. In rather extreme cases exposures can be also interfered with when health and sanitation inspectors are called to investigate rented apartments and homes. For most part, however, residence targeting exposure strategy must rely on risk communication to the occupants, who both cause and control their own exposures via their own decisions and actions.

Depending on age people may spend significant portions of their time, and thus may acquire significant portions of their daily exposures in day-care centres, schools or workplaces. The numbers of these buildings are smaller than those of residences, and public authorities have better legal instruments to influence the safety of these indoor environments.

Finally, there are indoor environments with exceptional sources and exposures, where only a fraction of people spend only a fraction of time, and yet they may present significant risks. One such example is a poorly ventilated ice skating rink, where one uncontrolled internal combustion engine powered ice resurfacing machine can generate acutely toxic indoor air CO and NO<sub>2</sub> exposures to hundreds of ice skaters and spectators.

**Individually targeted exposure strategies** are by necessity similar to the previous space targeted examples, except that they can be more focussed to the indoor exposures of vulnerable population groups such as asthmatics, chronically ill, or individuals with immune deficiencies. Therefore such strategies can be, albeit expensive per unit, cost efficient for the society. Examples range from blocks of apartments with materials (flooring, paints, wall constructions), ventilation systems (two way forced air with high efficiency filtration), occupant restrictions (no smoking or grilling), and maintenance requirements designed for asthmatics, to isolated hospital departments.

#### 3.8 Uncertainties

Focusing on the most significant causal agents is the first key to efficient – knowledge based - risk management (regulating a wrong agent is non-productive, yet as expensive as regulating the right one), identifying the sources which contribute most to the respective exposures is the second (regulating a source with marginal contribution to exposure has only marginal exposure reduction potential), and identifying the vulnerable target populations the third (when feasible, focusing on the exposure of the most vulnerable population brings the greatest benefit, and usually reduces everyone else's exposure, as well). Therefore, the uncertainties, which are most relevant for exposure based urban air quality assessment and management, relate to the agents of health concern, their sources and the vulnerable target populations.

For some indoor air pollutants we know specifically the agent of concern and its toxic effect mechanism (e.g. CO-toxicity, benzene-carcinogenicity). For others we do not know for certain whether they are the harmful agents responsible for epidemiologically observed health impacts, or just exposure indicators (NO<sub>2</sub>, PM). The assessment of uncertainties in exposure assessment should therefore be started by what is often the most fundamental uncertainty, the exposure agent uncertainty (or even metric uncertainty, e.g. for particulate matter; PM<sub>10</sub>, PM<sub>2.5</sub>, PM<sub>1.0</sub>, Ultra Fine Particles, Black Smoke). Targeted research is the tool for reducing the uncertainty concerning both agents and appropriate metrics, and expert elicitation the means of quantifying uncertainty estimates.

In some cases the sources are obvious enough, such as smoking in an office. In most cases, however, there are significant uncertainties in the contributions of different sources (outdoor, indoor, transport, personal) to the exposures. With sufficient databases of microenvironment and personal measurements or source emissions and the use of validated exposure models, exposure concentrations can be attributed to sources, usually by mass balance or factor analysis techniques. The uncertainties in source attribution are determined by the availability and representativeness of the samples, appropriateness of the analysed compounds, measurement errors (sampling and analysis), and statistical limitations of the attribution method.

Correct identification and characterisation of vulnerable population groups (specific for agents of concern and respective risks) can add considerable benefits for providing (i) relevant protective measures for their residences and workplaces, (ii) targeted exposure avoidance guidance for the individuals, and (iii) warning labels for locations and products, which could pose risky exposures. Uncertainties in these identifications and appropriate protective measures thereof can lead to reduced protection due to sub-optimal regulation, or unnecessary limitations to the freedoms of the protected individuals, and non-productive restrictions for spaces, services and products due to excessive regulation, with no added benefits attached to the added costs. Targeted meta-analyses of studies on the vulnerable groups are needed to reduce these uncertainties.

Compared to these agents, source and target group uncertainties, the measurement (sampling and analysis) or modelling uncertainties are usually much smaller.

## 4 CONCLUSIONS

- There are an enormous number and diversity of indoor air pollutants. There is a need to select a short list of pollutants that could be taken as priority substances to be traced and quantified either for their own indoor related public health relevance or as indicators of other potential pollution.
- For new building products VOC emissions tend to decrease. One reason is that VOC in many products have been substituted with SVOC. Consequently, there is an increasing trend of SVOC concentrations in indoor air. However, there is a need for more toxicity information for many such SVOCs.
- In many cases the products of the chemical reactions occurring in indoor air are stronger irritants than their precursors. Interactions between other reactive air pollutants are expected to be frequently occurring but little general information is available.
- At the commissioning phase of a building, adhesives, paints and solvents etc., present in many fittings, may not be adequately cured or stabilised and thus are likely to produce larger emissions of organic pollutants initially than at a later time. There are no generally accepted procedures for the commissioning or curing period.
- Models have been developed that take into account factors such as emission from sources, subsequent diffusion, adsorption/desorption, dilution and removal by ventilation, transport and spreading of pollutants from the sources to the exposure of the occupants. Such models are essential for the development of indoor exposure and risk assessment. No comprehensive and general model, however, has yet emerged.
- To efficiently reduce health risks from indoor air pollution, air quality management should be based on health concerns and exposure assessments for individual substances, accounting for indoor air quality, but also all other microenvironments and exposure relevant activities.
- Exposure based and the current ambient-based air quality management strategies are built upon similar principles. For the exposure-based assessment, however, additional information needs to be incorporated, particularly concerning the indoor microenvironments and individual activities.
- The uncertainties, which are most relevant for exposure based indoor air quality assessment and management, relate to the pollutants of health concern, and the vulnerable target populations. The measurement (sampling and analysis) or modelling uncertainties are usually much smaller in comparison.

## 5 RECOMMENDATIONS

- The common ventilation dilution model should be developed into a dynamic equilibrium model, which takes into account the effects of:
  - Transport of pollutants by air movements and removal of pollutants through dilution,
  - Removal of pollutants through filtration, absorption or deposition,
  - Adsorption into and re-emission of pollutants from sinks,
  - Chemical or biological transformations of pollutants in the air or in the sinks, including reactions.
- The labelling schemes of construction products that are aimed at promoting the use of cleaner materials, should consider including SVOCs in addition to the VOCs, which may interfere with human health or well-being.
- A labelling scheme for construction products should be implemented in all countries of EU. Such scheme should encompass all types of building materials.
- An overall policy strategy for indoor air quality management should be part of an
  integrated sustainable development strategy including public health; it should be
  combined with decision models and cost effectiveness analyses. It should be pro-active
  and include systematic preparation and dissemination of information and recommendations for the general public.
- An efficient exposure based air quality strategy focusing on the contribution of indoor air quality to the total inhalation exposure must be a balanced combination of sectorial strategies, so that it simultaneously requires the minimising of:
  - harmful exposures (from both outdoor and indoor sources),
  - health risks (exposures of the most vulnerable),
  - overall costs (of investment and operation, to individuals, organisations and the society),
  - loads on the environment (energy, materials, sustainability),
  - unhelpful restrictions to building materials, furnishings and technologies, and
  - non-productive restrictions to the activities of individuals and organisations.

Clearly, these six requirements are not commensurate, and not simple to fulfil.

- An essential element of an appropriate risk management strategy is reduction of the uncertainties. It is recommended to be achieved such reduction by:
  - targeted research concerning exposures to and health effects of harmful substances in indoor air.
  - improved study designs with emphasis on (i) representativenes of the acquired samples, (ii) appropriateness of the analysed compounds, (iii) minimizing of the measurement errors (sampling and analysis), and (iv) statistical power analyses to ensure attribution of the health effects to substances at exposure as well as the exposures to their sources, and
  - correct identification and characterization of the vulnerable population groups through targeted meta-analyses of studies concerning these groups.

#### REFERENCES

Bluyssen, P.M., Oliveira Fernandes, E., Groes, L., Clausen, G., Fanger, P.O., Valbjorn, O., Bernhard, C.A., e Roulet, C.A., 1996. European indoor air quality audit project in 56 office buildings. Indoor Air, 6: 221-238.

Bostrom C.-E. (1993). Health risk evaluation of nitrogen oxides. Nitrogen oxides in ambient air – properties, sources and concentrations, Scandinavian Journal of Work, Environment & Health, 19, 9-13.

Bremer, J., Witte, E. e Schneider, D., 1993. Measurement and characterisation of emissions from PVC materials for indoor use. In Indoor Air'93. O.Seppanen, J. Railio e J. Sateri (Eds.). Espoo, 2: 419-424

Bruinen de Bruin, Y., Koistinen, K., Yli-Tuomi, T., Kephalopoulos, S., Jantunen, M., 2006. A review of source apportionment techniques and marker substances available for identification of personal exposure, indoor and outdoor sources of chemicals. EUR 22349 EN. Luxembourg: Office for Official Publications of the European Communities.

Chang J.C.S. and Guo Z. (1992). Characterization of organic emissions from a wood finishing product – wood stain. Indoor Air, 2: 146-153.

Chang J.C.S., Tichenor, B. A., Guo, Z., Krebs, K.A. (1997). Substrate effects on VOC emissions from latex paint. Indoor Air, 7(4): 241-247.

Clausen P.A, Laursen B., Wolkoff P., Rasmusen E., Nielsen P.A, (1993). Emission of volatile organic compounds from a vinyl floor covering. In Modeling of Indoor Air quality and Exposure. ASTM STP 1205. Niren L. Nagda (Ed.). American Society for Testing and Materials, Philadelphia, 3-13.

Clausen P.A., Wilkins, C.K. and Jeppesen, P. (1994). Determination of fatty acids and fatty acids salts in office dust quantified as the methylesters by on-column capillary gas chromatography. In: Sandra, P. and Devos, G (eds) Sixteen International Symposium on Capillary Gas Chromatography, Heildeberg, Hugit GmbH, 1, 364-370.

Clausen, G. and Oliveira Fernandes, E., (1997). Final Report, European Data Base on Indoor Air Pollution Sources in Buildings. European Commission (DGXII), Brussels.

Colombo, A., De Bortoli, M., Knoppel, H., Schauenburg, H. e Vissers, H., (1990). Determination of volatile organic compounds emitted from household products in small test chambers and comparison with headspace analisys. In Indoor Air'90. D.S. Walkinshaw (Ed.). Toronto, 3: 599-604.

Conner, T.L., Lonneman, W.A e Seila, R.L., (1995). Transportation-related volatile hydrocarbon source profiles measured in Atlanta. J. Air Waste Management Association, 45: 383-394.

De Bortoli, M., Knoppel, H., Colombo, A. and Kefalopoulos, S. (1996). Attempting to characterize the sink effect in a small stainless steel test chamber. In Characterizing Sources of Indoor Air Pollution and Related Sink Effects. ASTM STP 1287. Bruce A. Tichenor (Ed.). American Society for Testing and Materials, pp. 307-320.

Dockery DW, Pope C, Xu X, Spengler J, Ware J, Fay M, Ferris B and Speizer F (1993). An association between air pollution and mortality in six U.S. cities. *NEJM*, 329:753-1759

Dunn, J.E. e Tichenor B.A. (1988). Compensating for sinf effects in emissions test chambers by mathematical modeling. Atmospheric Environment, 22: 885-894.

ECA (European Collaborative Action "Indoor Air Quality and its Impact on Man") 1991. Effects of indoor air pollution on human health. Report nr. 10, EUR 14086 EN. Luxembourg: Office for Official Publications of the European Communities.

ECA (European Collaborative Action "Indoor Air Quality and its Impact on Man") 1997. Evaluation of VOC emissions from building products – Solid flooring materials. Report Nr.18, EUR17334 EN. Luxembourg: Office for Official Publications of the European Communities.

ECA (European Collaborative Action "Indoor Air Quality and its Impact on Man") (2000). Risk Assessment in Relation to Indoor Air Quality. EUR19529 /EN, Report nr. 22.

Edwards R, Jurvelin J, Saarela K, Jantunen M. (2001). VOC concentrations measured in personal samples and residential indoor, outdoor and workplace microenvironments in EXPOLIS-Helsinki, Finland. Atmos Environ 35:4531-4543.

EEC (1967). Council Directive 67/548/EEC of 27 June 1967 on the approximation of laws, regulations and administrative provisions relating to the classification, packaging and labelling of dangerous substances. Official journal 1967 p. 169.

EEC (1993). Commission Directive 93/67/EEC of 20 July 1993 laying down the principles for assessment of risks to man and the environment of substances notified in accordance with Council Directive 67/548/EEC Official journal N° L 227, 08/09/1993 P. 0009 – 0018.

Evans W. C. (1996). Linear systems, compartmental modelling, and estimatibility issues in IAQ studies. In Characterizing Sources of Indoor Air Pollution and Related Sink Effects. ASTM STP 1287. Bruce A. Tichenor (Ed.). American Society for Testing and Materials, pp. 239-262.

Fanger P. O. (2000). Indoor air quality in the 21st century: search for excellence. Indoor Air, 10(2): 68-73.

Gazzetta Ufficiale della Repubblica Italiana (2001). Suppl. Ord. al n. 276 del 27.11.2001, n. 252, pag.47 (In Italian).

Gehrig R., Hill M., Zellweger C., Hofer P. (1993). VOC-Emissions from wall paints – a test chamber study. In Indoor Air'93. O.Seppanen, J. Railio e J. Sateri (Eds.). Espoo, 2: 431-436.

Georgopoulos PG and Lioy PJ (1994). Conseptual and theoretical aspects of human exposure and dose assessment. J Expo Anal Environ Epidemiol, 4:253-285.

Götschi T, Oglesby L, Mathys P, Monn C, Koistinen K, Hänninen O, Georgoulis L, Polanska L, Jantunen MJ, and Künzli N (2002). Comparison of Black Smoke and PM2.5 levels in indoor and outdoor environments of four European cities. Environmental Science & Technology, 36:6:1191-1197.

Guo, Z. (2002). Review of indoor emission source models. Part 1. Overview, and Review of indoor emission source models. Part2. Parameter estimation. Environmental Pollution, 120 (2002) 533-549, 551-564.

Gustafsson, H. (1992). Building materials identified as major sources for indoor air pollutants – A critical review of cases studies. D10. Swedich Council for Building Research, Estocolmo.

Haghighat, F. and De Bellis, L. (1998). Material emission rates: literature review and the impact of indoor air temperature and relative humidity, Building Environment, 33(5), 261-277.

Hänninen O, Kruize H, Lebret E and Jantunen M. (2003). EXPOLIS Simulation Model: PM2.5 Application and Comparison to Measurements. J Exposure Anal Environ Epidemiol, 13, 75-85.

Hänninen, O., Alm, S., Katsouyanni, K., Künzli, N., Maroni, M., Nieuwenhuijsen, M.J., Saarela, K., Srám, R.J., Zmirou, D., Jantunen, M.J. (2004). The EXPOLIS Study: Implications for exposure research and environmental policy in Europe. Journal of Exposure Analysis and Environmental Epidemiology, 14: 440-456.

Hänninen, O., Lebret, E., Tuomisto, J.T., Jantunen, M.J. (2005a). Characterization of model error in the simulation of PM2.5 exposure distributions of the working age population in Helsinki, Finland. JAWMA 55:446–457.

Hänninen, O., Palonen, J., Tuomisto, J., Yli-Tuomi, T., Seppanen, O., Jantunen, M.J., (2005b). Reduction potential of urban PM2.5 mortality risk using modern ventilation systems in buildings. Indoor Air 15 (4): 246-256.

Hodgson A.T., Garbesi K., Sextro R.G., Daisey J.M. (1992). Soil-gas contamination and entry of volatile organic compounds into a house near a landfill. J. Air Waste Management Association, 42(3): 277-283.

Hodgson A.T., Wooley J.D., Daisey J.M. (1993). Emissions of volatile organic compounds from new carpets measured in a large-scale environmental chamber. J. Air Waste Management Association, 43(3): 316-324.

Horn W., Ullrich D., Seifert B. (1998). VOC Emissions from cork products for indoor use. Indoor Air, 8: 39-46.

IARC (2006). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Vol. 88 Formaldehyde, 2-Butoxyethanol and 1-tert-Butoxy-2-propanol (in preparation). Updated 7 September 2004. Available in the internet at

http://monographs.iarc.fr/ENG/Monographs/vol88/volume88.pdf. Accessed on June 21, 2006.

International Commission on Radiological Protection (ICRP) (1994). Human Respiratory *Tract Model for Radiological Protection*, Ann. ICRP, 24, Publ. 66.

Jantunen M, Hänninen O, Katsouyanni K, Knöppel H, Künzli N, Lebret E, Maroni M, Saarela K, Srám R, Zmirou D. (1998). Air pollution exposure in European cities: the EXPOLIS study. J Expo Anal Environ Epidemiol. 8:495-518.

Kane L.E. and Alarie Y. (1978). Sensory irritation of select experimental photochemical oxidants. Archives of Environmental Health, 33, 244-250.

Karpe P.H., Kirchner S., Hubert S. (1995). Chemical and sensory evaluation of building materials using TD/GC/MS/FID/Sniffer multicoupling analytical method. In Proceedings of the Second Int.Conf.Indoor Air Quality, Ventilation and Energy Conservation in Buildings, F. Haghighat (Ed.). Toronto, 1: 145-154.

Katsouyanni K, Zmirou D, Spix C, Sunyer J, Schouten JP, Pönkä A (1995). Short term effects of air pollution on health: A European approach using epidemiological time series data. *Eur Respir J*, 8:1030-1038.

Katsouyanni K, Touloumi G, Spix C, Schwartz J, Balducci F, Medina S, Rossi G, Wojtyniak B, Sunyer J, Bacharova L, Schouten JP, Ponka A, Anderson HR (1997). Short term effects of ambient sulphur dioxide and particulate matter on mortality in 12 European cities: Results from time series data from the APHEA project. BMJ, 314:1658-1663

Kephalopoulos S (1999). Mathematical modelling of test chamber kinetics. In: Organic Indoor Air Pollutants: Occurrence, Measurements, Evaluation, Chapter 2.3 (1999) 153-168, T. Salthammer (Ed.) Wiley-VCH.

Kirchner S., Karpe P., Cochet C. (1993). Characterization of volatile organic compounds emission from floor coverings. In Indoor Air'93. O.Seppanen, J. Railio and J. Sateri (Eds.). Espoo, 2: 455-460.

Kirchner S., Karpe P., Rouxel P., Kephalopoulos S., Knoeppel. H., De Bortoli M., Bluyssen P., Van der Wal J.F., Cornelissen H.J.M., Hoogeven A.W., Kjaer U., and Tirkkonen T. Characterization of adsorption-desorption of organic pollutants on wall and floor coverings surfaces. Proceedings of the 4th International Conference on Characterization and Control of Emissions of Odors and VOCs, Montreal, 20-22 October 1997, pp. 270-282, Ecole Polytechnique de Montreal, Montreal (Canada).

Kirchner S., Badey J.R., Knudsen H., Meninghaus R., Quenard D., Saarela K., Salle H., and Saarinen A. (1999). Sorption capacities and diffusion coefficients of indoor surface materials exposed to VOCs: proposal of new test procedures, Indoor Air'99, 1, 430-435.

Koistinen KJ, Kousa A, Tenhola V, Hänninen O, Oglesby L, Künzli N, Georgoulis L and Jantunen MJ (1999). Fine particle (PM2.5) measurement methodology, quality assurance procedures and pilot results of the EXPOLIS study. J Air Waste Manage Assoc, 49:1212-1220.

Koistinen KJ, Hänninen O, Rotko T, Edwards R, Moschandreas D, Jantunen MJ (2001). Behavioral and Environmental Determinants of Personal Exposures to PM2.5 in EXPOLIS-Helsinki, Finland. Atmos. Environ. 2001, 35(14), 2473 - 2481.

Koistinen KJ, Edwards RD, Mathys P, Ruuskanen J, Kuenzli N, Jantunen MJ (2004). Sources of PM2.5 in Personal Exposures and Residential Indoor, Outdoor and Workplace Microenvironments in EXPOLIS-Helsinki, Finland. Scandinavian Journal for Work, Environment and Health, 30:suppl. 2, 36-46.

Kotzias D, Koistinen K, Kephalopoulos S, Schlitt C, Carrer P, Maroni M, Jantunen M, Cochet C, Kirchner S, Lindvall T, McLaughlin J, Mølhave L, Fernandes EO and Seifert B. (2005). Final Report of the INDEX Project, Critical Appraisal of the Setting and Implementation of Indoor Exposure Limits in the EU. Office for Official Publication of the European Communities, Luxembourg. EUR 21590 EN, ISBN 92-894-9353-4. pp. 331.

Kousa A, Oglesby L, Koistinen K, Künzli N, Jantunen M (2002). Exposure chain of urban air PM2.5 - associations between ambient fixed site, residential outdoor, indoor, workplace and personal exposures in four European cities in the EXPOLIS-study. Atmos Environ, 36:3031-39.

Lioy PJ (1990). Assessing total human exposure to contaminants. Environ Sci Technol, 24:938-945.

Little J.C., Hodgson A.T. and Gadgil A.J. (1994). Modeling emissions of volatile organic compounds from new carpets. Atmospheric Environment, 28(2): 227-234.

Little J. C. and Hodgson, A. T. (1996). A strategy for characterizing homogeneous, diffusion-controlled, indoor sources and sinks. In Characterizing Sources of Indoor Air Pollution and Related Sink Effects. ASTM STP 1287. Bruce A. Tichenor (Ed.). American Society for Testing and Materials, pp. 294-304.

Meninghaus R. Gunnarsen L. and Knudsen H. (2000). Diffusion and sorption of volatile organic compounds on indoor surface materials – Impact on indoor air quality, Environ. Sci. Technol., 34, 3101-3108.

Mølhave L, Kjærgaard SK, Attermann J, Pedersen OF (1996). Sensory and neurotoxic effects of airborne exposures to house dust. In: Frang T, editor. Allergistämma 96, tema innomhusmiljö. Stockholm: Folkhälsainstituttet, p. 5-6.

Mølhave L, Kjærgaard SK, Attermann J (2000a). Sensory and other neurogenic effects of exposures to air borne office dust. Atmos. Environ. 34:4755-66.

Mølhave L, Schneider T, Kjærgaard SK, Larsen L, Norn S, Jørgensen O (2000b). House dust in seven Danish offices. Atmos. Environ. 34:4767-79.

Mølhave L, Kjærgaard SK, Attermann J (2002). Effects in the eyes caused by exposures to office dust. Indoor Air 12:165-74.

Mølhave L, Kjærgaard SK, Attermann J. (2004). Respiratory effects of experimental exposures to office dust. Indoor Air 14:376-84.

National Research Council (NRC) (1983). Risk Assessment in the Federal Government: Managing the Process. National Academy Press. Washington, DC.

Norback D., Wieslander G., Strom G., Edling C. (1995). Exposure to Volatile Organic Compounds of Microbial Origin (MVOC) during indoor application of water-based paints. Indoor Air, 5: 166-170.

Olander L (1992). Laser printers and air contaminants – a review, Arbete och Hälsa, 10, 1-38. Özkaynak H, Xue J, Spengler J, Wallace L, Pellizzari E, Jenkins P (1996). Personal Exposure to Airborne Particles and Metals: Results from the Particle TEAM Study in Riverside, California. *J Exposure Analys and Environ Epidemiol*, 6: 57-78.

Pellizzari E, Lioy P, Quackenboss J, Whitmore R, Clayton A, Freeman N, Waldman J, Thomas K, Rodes C, Wilcosky T. (1995). Population-based exposure measurements in EPA region 5: a phase I field study in support of the National Human Exposure Assessment Survey. J Expo Anal Environ Epidemiol. 5(3):327-58.

Person A., Laurent A.M., Louis-Gavet M.C., Aigueperse J., Anguenot E.F. (1990). Characterization of volatile organic compounds emitted by liquid and pasty household products via small test chambers. In Indoor Air'90. D.S. Walkinshaw (Ed.), Toronto, 3: 605-610.

Plehn W. (1990). Solvent emission from paints. In Indoor Air'90. D.S. Walkinshaw (Ed.), Toronto, 3: 563-568.

Pope CA III, Thun MJ, Namboodiri MM, Dockery DW, Evans JS, Speizer FE and Heath CW Jr. (1995). Particulate Air Pollution as a Predictor of Mortality in a Prospective Study of U.S. Adults. *Am J Respirat Crit Care Med*, 151:669-674.

Quigley C.J., Corsi R.L. (1995). Emissions of VOCs from a Municipal Sewer. J. Air Waste Management Association, 45, 395-403.

Rocha S., Delgadillo I., Correia J.F. (1996). GC-MS study of volatiles of normal and microbiologically attacked cork from Quercus suber L. J. Agric.Food Chem., 44(3), 865-871.

Saarela K., Tirkkonen T., Tahtinen M. (1994). Preliminary data base for materials emmissions. 04 E. Nordic Committee on Building Regulations, Helsinki.

Salthammer (1997). Emission of volatile organic compounds from furniture coatings. Indoor Air, 7, 189-197.

Seifert B, Becker K, Hoffmann K, Krause C, Schulz C (2000). The German Environmental Survey 1990/1992 (GerESII): a representative population study. J Expo Anal Environ Epidemiol. 10:103-114.

Seppänen, O et al. (1995). "The Classification of Indoor Climate, Construction and Finishing Materials", Vol 3, 1667-1673, Proceedings of Healthy Buildings '95 Conference. ISBN 88-900086-0-1.

Sexton K, Kleffman DE, Callahan MA (1995). An introduction to the National Human Exposure Assessment Survey (NHEXAS) and related phase I field studies. J Expo Anal Environ Epidemiol. 5(3):229-32.

Smedje G., Norback D., Wessen B., Edling C. (1996). Asthma among school employees in relation to the school environment. In Indoor Air'96. S. Yoshizawa, K. Kimura, K. Ikeda, S. Tonabe e T. Iwata (Eds.). Nagoya, 1: 611-616.

Stump F.D., Knapp K.T., Ray W.D., Snow R., Burton C. (1992). The composition of motor vehicle organic emissions under elevated temperature Summer driving conditions (75 to 105°F). J. Air Waste Management Association, 42(2): 152-158.

Thogersen K., Gunnarsen L., Nielsen P.A. (1993). The effect on indoor air quality by water-damaged chipboards. In Indoor Air '93. O.Seppanen, J. Railio e J. Sateri (Eds.). Espoo, 2: 537-542.

Van Der Wal, J.F., Hoogeven, A.W. e Wouda, P. (1997). The Influence of temperature on the emission of Volatile Organic Compounds from PVC flooring, carpet and paint. Indoor Air, 7: 215-221.

Volki, S., Gewbefugi, I.L., Korte, F. (1990). Emission of Volatile Organic Compounds from coatings into indoor air. In Indoor Air'90. D.S. Walkinshaw (Ed.), Toronto, 3: 701-705.

Wadden R.A., Scheff P.A., Franke J.E., Conroy L.M. (1995). Determination of VOC emission rates and compositions for offset printing. J. Air Waste Management Association, 45: 547-555.

Wallace L, Özkaynak H, Spengler J, Pellizzari E and Jenkins P. (1993). Indoor, outdoor and personal air exposures to particles, elements and nicotine for 178 southern California residents. Proceedings of Indoor Air '93, 3:445-450.

Wallace L (1996). Indoor Particles: A Review. J AWMA, 46:98-126.

Weschler CJ and Shields HC (2000). The influence of ventilation on reactions among indoor pollutants: modelling and experimental observations, Indoor Air, 10, 92-100.

WHO (1999). Health21 - The health for all policy framework for the WHO European Region. Copenhagen, Denmark, WHO Regional Office for Europe (European Health for All Series; No. 6, ISBN 92 890 1349 4).

WHO (2000) Air quality guidelines for Europe, 2nd Edition. WHO Regional Office for Europe, Copenhagen. http://www.who.dk/WHO-Euro/tech/.

Wilkins C.K., Wolkoff P. Gyntelberg F., Skov P. and Valbjorn O. (1993). Characterisation of office dust by VOC and TVOC release identication of potential VOC by partial least square analysis, Indoor Air, 3, 283-290.

Wolkoff P., Clausen P.A., Nielsen P.A., Molhave L. (1990). The danish twin apartment study; Part I: Formaldehyde and long-term VOC measurements. In Indoor Air'90. D.S. Walkinshaw (Ed.), Toronto, 2: 657-662.

Wolkoff P., Wilkins C.K., Clausen P.A., Larsen K. (1993). Comparison of Volatile Organic Compounds from processed paper and toners from office copiers and printers: methods, emissions rates, and modelled concentrations. Indoor Air, 3 (2): 113-123.

Wolkoff, P., Clausen P.A., Nielsen P.A. (1995a). Application of Field and Laboratory Emission Cell "FLEC" – Performance study, intercomparison and case study of damaged linoleum in a office. Indoor Air, 5(3): 196-203.

Wolkoff P (1995b). Volatile Organic Compounds – sources, measurements, emissions, and the impact on indoor air quality, Indoor Air, Suppl.3.

Wolkoff P, Clausen PA., Wilkins CK, Nielsen GD (2000). Formation of strong Airway Irritants in Terpene/Ozone Mixtures, Indoor Air, 10(2), 82-91.

#### **ANNEX 1** Fine Particulate Matter in Indoor Air

#### 1 INTRODUCTION

The scope of this example is to present a general risk assessment for particulate matter (PM) in indoor air in a Western urban context.

The most important source of indoor air particles - in the absence of smoking - is usually outdoor air. Compared to e.g. VOCs, indoor sources play a smaller relative role in the exposure to particulate matter. Penetration of particles from outdoor air into indoor spaces is therefore a matter of great interest for indoor air exposure, and it will be dealt here before assessing the indoor sources.

## 2 IDENTIFICATION OF HAZARD AND SOURCES

The hazard identification of PM is based on outdoor air and environmental tobacco smoke (ETS) exposure studies (see PM Exposure-Response Assessment). Because ETS is the most important indoor air PM source and because more than half of the non-ETS PM indoors usually originates from ambient outdoor air, this hazard identification is considered relevant for indoor air PM also. Particulate air pollution is - in sampling, regulation and research usually divided according to particle size into coarse (2.5 - 10  $\mu$ m aerodynamic diameter), fine (PM<sub>2.5</sub> < 2.5  $\mu$ m) and ultrafine (< 0.1  $\mu$ m) fractions. Indoor air PM has two different origins.

Coarse particles (>  $2.5 \mu m$  diameter) are produced by resuspension of floor dust, handling of textiles and cleaning activities. They contain mostly soil minerals, non-volatile organics and textile fibres. Much of the coarse PM settles rapidly out of the air, but is also easily resuspended. Outdoor air coarse particles are generated by mechanical erosion; wind, traffic, and materials handling and they penetrate poorly into indoor environments.

Fine particles ( $< 2.5 \, \mu m$ ) are produced by tobacco smoking, cooking, unvented kerosene heaters and wood burning, but there is also a significant mineral dust source of PM<sub>2.5</sub> indoors. They contain mostly semivolatile organics (SVOC), polyaromatic hydrocarbons (PAH) and elemental carbon (soot, EC) and inorganic minerals. Fine particles do not settle out of indoor air. They move freely with air currents, stick to any surface they touch and are only poorly resuspended. PM<sub>2.5</sub> from outdoor air they penetrate effectively indoors through most ventilation systems.

Ultrafine particles (<0.1  $\mu$ m) are usually measured by count (UFP#, #/cm³, often illogically called 'ultrafine particle number'), but interest on the mass concentration (UFPM or PM<sub>0.1</sub>,  $\mu$ g/m³) and total surface area (m²/m³) of ultrafine particles is increasing. Indoor sources of UFP are combustion processes, smoking, cooking, mostly same as for PM<sub>2.5</sub>. The indoor fate of ultrafine particles is dominated by their high Brownian velocities: at high concentrations they agglomerate rapidly into larger particles; at lower concentrations they are scavenged through impaction on larger particles and on any indoor surfaces. Because UFP# (but not necessarily PM<sub>0.1</sub> mass) has a much shorter half life than PM<sub>2.5</sub> (mass), however, the relative short term impact of a source is greater on UFP# than on PM<sub>2.5</sub>.

## 3 PM EXPOSURE-RESPONSE ASSESSMENT

Particles larger than 10 µm do not penetrate into the alveoli even in mouth breathing, but particles smaller than 2.5 µm may penetrate deep into the lung. About half of the small

particles are retained in the respiratory tract and, if insoluble, are only slowly removed from the alveolar tissue. Particles between 2.5 and 10 µm show intermediate behaviour that depends on the breathing intensity (Bates *et al.* 1966).

The dose/response for exposure to PM is based on numerous epidemiological studies, which have linked significant mortality differences to different levels of outdoor PM between different days in one city (dozens of time series studies, e.g. Katsouyanni et al. 1995 and 2001) between different cities (cohort studies, Dockery et al. 1993, Pope et al. 1995), as well as demonstrated the reduced mortality for ambient PM reduction (Intervention studies, Pope et al. 1989, Clancy et al. 2002, Laden et al. 2006).

Based on extensive review of the literature, WHO Air Quality Guidelines (WHO 2005) concludes that a daily outdoor air  $PM_{2.5}$  increase of  $10 \mu g/m^3$  or  $PM_{10}$  increase of  $20 \mu g/m^3$  increases daily total mortality by 6 (2-11) %. According to long term cohort studies, typical urban outdoor air levels of  $PM_{10}$  and  $PM_{2.5}$  appear to not only affect the date of death, but also significantly reduce life expectancy. The result is consistent between different studies, and seems to affect at least babies and the elderly.

ETS, the most significant indoor PM source, is also a fully established health hazard (IARC 1986, NRC 1986).

# **Exposure-Response Assessment for Indoor Air Particles?**

The presence of indoor and personal PM sources have typically much stronger immediate impact on personal PM exposures than the ambient air PM levels, because 1 g of indoor PM emission causes 100 - 1000 time higher PM exposure than the same emission to outdoor air. A particular example is ETS; the average fine PM exposure in smoking environments is 2-3 times higher than in non-smoking environments. The ambient air PM, however, forms the large scale exposure baseline on which the impacts of the more variable near field, indoor and personal PM sources are superimposed.

The key question is, how can risk estimates based on the statistical association of population mortality and morbidity with outdoor air PM levels be used to estimate the health risks of indoor PM, where people spend ca. 90% of their time. In the absence of ETS 50 - 65 % of indoor air fine PM comes of outdoor origin, the rest comes from indoor sources, such as dust resuspension, cooking and combustion sources. Therefore, most of the PM exposure is to outdoor air particles. Consequently the overall uncertainty as to the health effects of indoor air PM relative to the health effects of outdoor air PM is not larger than a factor of 2.

## 4 PM (INDOOR) EXPOSURE ASSESSMENT

On one hand the recent epidemiological findings about the public health impacts of atmospheric PM, and on the other hand the uncertainties and costs involved in significant reduction of the present PM exposures of the people living in the industrialized world lead to increasing demand for better information about;

- which chemical and physical characteristics and hereby sources of the PM are most significant for the health consequences observed,
- which environmental, microenvironmental and individual characteristics are most significant for personal PM exposures, and
- how much can the PM related health hazards be reduced by different control actions.

Personal exposure modelling, validated by data from representative exposure studies, is essential for answering these questions, and as a consequence of the time use of people, such models and studies assess mostly indoor exposures to PM of both outdoor and indoor origin.

According to the USEPA P-TEAM study (Riverside, California) variations of the outdoor central site  $PM_{10}$  levels explain only 37 % of the variations in daytime and 54 % in night time personal  $PM_{10}$  exposures, and 1/2 to 2/3 of the personal  $PM_{10}$  exposures are explained by indoor, microenvironmental (e.g. in traffic) and/or personal sources (Wallace et al. 1993, Özkaynak et al. 1996).

In Europe the EU funded EXPOLIS study measured personal, residential, workplace and outdoor concentrations of dozens of air pollutants, including  $PM_{2.5}$  in seven European cities. The results show great variation both between and within the cities. Indoor and personal  $PM_{2.5}$  levels follow outdoor  $PM_{2.5}$  levels closer than  $PM_{10}$  concentrations. Annual city average  $PM_{2.5}$  exposures were quite close to respective ambient air concentrations, but leisure time personal exposures showed only moderate correlations to night time (17:00 – 07:00) ambient air concentrations ( $R^2 = 0.2 - 0.35$ ), and workday (incl. commuting) exposures did not correlate with daytime ambient air concentrations (Kousa et al. 2002, Hänninen et al. 2004). Therefore the contributions of both outdoor and indoor sources to indoor PM exposure need to be considered.

## Infiltration of ambient particles into indoor spaces

Most epidemiological studies point out to  $PM_{2.5}$  the most harmful ambient urban air particle size fraction. In non-smoking indoor spaces most of the  $PM_{2.5}$  originates from outdoor air. Most of the population exposure to and mortality and morbidity from outdoor air  $PM_{2.5}$  is, consequently, caused by indoor exposure to  $PM_{2.5}$  of outdoor origin. An American epidemiological study (Janssen et al. 2002) pointed out that, indeed, increased fraction of buildings with central air conditioning within the community significantly reduces the association between ambient air PM and serious health effects (CVD, COPD, P Pneumonia). These facts open one interesting avenue for reducing urban  $PM_{2.5}$  mortality risk.

Also in the EXPOLIS study in Helsinki (Finland) the highest contributions to personal exposures of non-ETS exposed individuals, came from outdoor air (Koistinen et al. 2001). The most significant sources were combustion generated primary particles, secondary particles, and soil dust (half of which originated from indoor sources) (Koistinen et al. 2004). Of the commonly monitored particle size fractions,  $PM_{2.5}$  infiltrates best from outdoor to indoor air (Indoor-outdoor concentration ratio, I/O = 0.3 - 0.9). Indoor infiltrations of ambient coarse ( $PM_{2.5-10}$ ) and ultrafine particles are lower.

In non-smoking homes in Boston, MA, minimum infiltrations were observed for ultrafine and coarse, maximum for 0.1 - 0.5 µm particles. Infiltration efficiency depends also on the season (higher in the summer than winter, if high air exchange rate (AER) in the absence of air conditioning (AC) and) and home characteristics (lower in summer than winter, if low AER in houses equipped with AC) (Long et al. 2001).

In studies made in two very different climate zones and building types, namely residences in Brisbane, Australia (Morawska et al. 2001), and offices in Helsinki, Finland (Kulmala et al. 1999), indoor concentrations in both followed the outdoor concentration changes in a smoothed and delayed pattern. In the former, however, the I/O ratios were 0.8 - 1.0 for both UFP# and PM<sub>2.5</sub> mass concentrations, in the latter the I/O ratios fell within a much broader range, 0.05 - 0.75. In another Finnish office building with a mechanical ventilation system equipped with EU7 class

filters, infiltration of outdoor air particles was highest (I/O = 0.25 - 0.3) for 0.2 - 0.5  $\mu$ m particles, lower (0.15 - 0.2) for 0.1  $\mu$ m, and lowest (0.02 - 0.06) for 0.01  $\mu$ m particles. (Koponen et al. 2001) In an American study of two retirement homes (in Baltimore, DE, and in Fresno, CA), residential HVAC filter efficiencies were 30 - 65 % for coarse particles, dropped to 10 - 20 % for 0.06 - 1.0  $\mu$ m particles but increased again up to and over 25 % for particles smaller than 0.01  $\mu$ m. I/O of PM<sub>2.5</sub> in air conditioned apartments was, in average, only 0.45, but reached 0.8 when AC was turned off and homes were ventilated through open windows (April - May in Fresno, CA). Personal exposures of the residents followed closely the indoor air concentrations with a mean 3  $\mu$ g/m³ increase from personal cloud (Rodes et al. 2001).

I/O ratios of RSP (respirable suspended particulates, approximately PM<sub>4.5</sub>) depend also on outdoor meteorological conditions, which influence both ventilation methods and air exchange rates. In Hong Kong the I/O was strongly increased with increased ambient temperature and humidity, weakly increased with solar irradiation and no effect was seen for wind speed and atmospheric pressure (Chan 2002).

In modern residential and office buildings with sealed buildings envelopes, balanced ventilation and intake air filtration, the infiltration of outdoor air  $PM_{2.5}$  is significantly lower (by 20 - 50%) than in older buildings with natural ventilation via open windows and vents (Hänninen et al. 2005). A probabilistic population exposure simulation exercise demonstrated that reducing the distribution of  $PM_{2.5}$  infiltration into all buildings in the city of Helsinki to the level of the office buildings built after 1990, would reduce the population exposure to  $PM_{2.5}$  from outdoor origin as well as its adverse health effects by 27%, in fact almost as much as total elimination of all traffic sources from within the metropolitan area.

## **Indoor sources of PM exposure**

Outdoor and indoor air concentrations of and personal exposures to  $PM_{2.5}$  in Helsinki, Finland, were attributed to sources in the *EXPOLIS* study using both statistical (PCA) and deterministic source reconstruction techniques (Koistinen et al. 2004). In the absence of ETS, indoor  $PM_{2.5}$  concentrations were in average 10% lower than the respective outdoor concentrations. The dominating indoor source was human activity, which significantly elevated the average levels of indoor air mineral dust levels (2.5  $\mu$ g/m³) compared to outdoor air (1.6  $\mu$ g/m³). Another distinct indoor source contribution turned out to be detergent particles from poorly rinsed clothes, marked by phosphorus, which was not detected in outdoor air, but reached quite high levels - up to 2  $\mu$ g/m³ - in some residential environments. In most other cases phosphorus levels were quite low.

In a multiple regression modelling study, using *EXPOLIS* data from six European cities, the indoor determinants identified for the 48 h indoor concentrations were smoking (16% of the variance) and gas stove (1.4%) for PM<sub>2.5</sub>, and smoking, building type and gas stove (all < 4%) for BS, (Lai et al. 2006). In a French regression modelling study of the determinants of PM<sub>2.5</sub> exposures of asthmatic children, the statistically significant variables were indoor smoking (36% of variation), ambient air PM<sub>10</sub> (24%), pets (particularly rodents, 21%) and traffic exposure (12%) (Gauvin et al. 2002).

In a study, which focused on the short-term impacts of indoor PM sources, various means of cooking contributed drastically to indoor PM concentrations (with peak concentrations of 30 -  $60~\mu g/m^3$  for  $PM_{0.02\text{-}0.5}$  and 10 -  $300~\mu g/m^3$  for  $PM_{0.7\text{-}10}$ . Oven cooking was the strongest source for indoor  $PM_{0.5}$ , sautéing for  $PM_{10}$ . Cleaning activities (8  $\mu g/m^3$  for  $PM_{0.02\text{-}0.5}$  and  $30~\mu g/m^3$  for  $PM_{0.7\text{-}10}$ ) and mobility of the occupants (4  $\mu g/m^3$  for  $PM_{0.02\text{-}0.5}$  and  $20~\mu g/m^3$  for  $PM_{0.7\text{-}10}$ ) contributed much less (Abt et al. 2000).

## **Summary of PM Exposure and Microenvironmental Studies**

A comprehensive review of the indoor air PM studies was prepared by Wallace (1996). A summary of the personal fine particulate matter exposure levels and corresponding levels measured in microenvironments such as homes, workplaces, adjacent outdoor environments and central ambient air monitoring sites from the studies mentioned above are presented in Table 1.

**Table 1.** The observed median \*) levels for  $PM_{2.5}$  in European and  $PM_{10}$  in American personal and microen-vironmental PM exposure studies.

|                         | $PM_{2.5} (\mu g/m^3)$ | $PM_{10} \left(\mu g/m^3\right)$ |
|-------------------------|------------------------|----------------------------------|
| Personal exposures      | 9 - 44                 | 33 - 129                         |
| Home indoor levels      | 8 - 32                 | 22 - 78                          |
| Home outdoor levels     | 8 - 30                 | 18 - 83                          |
| Central monitoring site | 7 - 36                 | 38 - 76                          |

As a summary of the impacts of certain indoor activities on personal PM exposures and indoor concentrations (Table 2), the most significant is, of course, smoking. An average  $PM_{2.5-10}$  level increase in ETS exposed environments is 30 - 40  $\mu$ g/m³ or doubling of the non-ETS level. Cooking may increase the daily mean PM exposures by 7 - 26  $\mu$ g/m³, unvented kerosene heaters 5 - 30  $\mu$ g/m³ and wood stoves 0 - 10  $\mu$ g/m³.

Table 2. Contributions of different sources to indoor and outdoor air PM combined from a body of literature. Note that, due to the data availability, the contributions to indoor air levels are reexpressed in (%) of the total PM mass in indoor air, and to the outdoor air levels in (μg/m³). Except for smoking, other data are for conditions without smoking.

| Source category   | Indoor air PM <sub>2.5-10</sub> | Outdoor air PM <sub>2.5-3.5</sub>           |
|-------------------|---------------------------------|---|
| smoking           | 24 - 71 % (*                    | negligible                                  |
| cooking           | - 25 - % (*                     | - 3 (μg/m³) (not fire, but frying fur       |
| wood burning      | 3 - 21 %                        | $1 - 4  (\mu g/m^3)$                        |
| soil dust         | 4 - 50 %                        | $1 - 23 \ (\mu g/m^3)$                      |
| traffic emissions | 5 - 30 %                        | 5 - 17 (µg/m <sup>3</sup> ) (mostly diesel) |

## **Risk Management - Recommended Ideal Practice**

The first exposure and risk management option for indoor air pollution originating from indoor sources should always be indoor source elimination or isolation. The second option is dilution by ventilation.

When a safe threshold level for fine PM exposure cannot be established, and mechanistic information or information about the chemical characteristics that define the observed PM

toxicity remains limited, a risk manager would normally ask for more research, facts and time before making any decisions. When at the same time, however, independent epidemiological studies of different designs provide consistent evidence that fine PM is probably responsible for 1-2 % of the overall mortality, and seems to decrease the average lifetime by an order of 1 year, there are strong reasons to search for effective, "no regrets", exposure reducing policy alternatives.

Because all fine PM exposures seem to be harmful the RM policy should aim at reducing all exposures, and not just searching and cutting the highest or peak levels that exceed a certain level.

In indoor environments the requirements for effectiveness, no regrets and ALARA (As Low As Reasonably Achievable -policy) point at first towards the most significant indoor sources, and secondly towards reducing the entry of outdoor air fine PM into indoor environments. The most obvious first target is ETS. Eliminating smoking in indoor environments effectively halves the PM exposures of all affected non-smoking individuals. It is difficult to imagine any other exposure reduction measure with comparable cost/effectiveness. Bans and restrictions on smoking at workplaces, public spaces, transport environments and even restaurants are increasing across Europe, and extremely significant for exposure control.

Decreasing the infiltration of ambient air particles to indoor spaces is not an option for managing the risks of PM from indoor sources, but it is a powerful option for reducing the overall risks from ambient air PM. Its main attractions are the facts that - unlike local/urban emission reduction actions - (i) it will reduce exposure to all PM of outdoor origin, be it from neighbourhood, local, or regional and transboundary sources, and (ii) it can be selectively focused to protect vulnerable population groups. This option should not be seen as an alternative to local or larger scale emission reducing and ambient air quality improving actions, but rather as a means of providing additional and more focused protection.

These measures can only slowly be implemented across the urban building stock. Elimination of smoking indoors, updating of the building codes, good maintenance of the ventilation system and education and training of the building operators can reduce PM exposures (and presumably also the related health risks) of the affected populations more than any locally achievable urban outdoor air quality management measures in many years.

#### REFERENCES

Abt E, Suh HH, Allen G, Koutrakis P (2000). Characterisation of indoor particle sources: A study conducted in the Metropolitan Boston Area. *Environ. Health Perspect.* 108, 35-44.

Bates D V, Fish B R, Hatch T F, Mercer T T, Morrow P E (1966): Deposition and retention models for internal dosimetry of the human respiratory tract (The NCRP Lung model). *Health Physics*, 12:173-207.

Chan AT (2002). Indoor-outdoor relationships of particulate matter and nitrogen oxides under different outdoor meteorological conditions. *Atmos. Environ.* 36, 1534-1551.

Clancy L, Goodman P, Sinclair H, Dockery DW (2002). Effect of air-pollution control on death rates in Dublin, Ireland: an intervention study. *The Lancet* 360, 1210-1214.

Dockery DW, Pope CA III, Xu X, et al. An association between air pollution and mortality in six U.S. cities. N Engl J Med 1993;329: 1753-9.

Gauvin S, Reungoat P, Cassadou S, Déchenaux J, Momas I, Just J and Zmirou D (2002). Contyribution of indoor and outdoor environments to PM2.5 personal exposure of children - VESTA study. Sci. *Tot. Environ*. 279, 175-181.

Hänninen O, Palonen J, Tuomisto JT, Yli-Tuomi T, Seppänen O, Jantunen MJ (2005). Reduction Potential of Urban PM2.5 Mortality Risk Using Modern Ventilation Systems in Buildings. *Indoor Air* 15 (4), 246-256.

Hänninen OO, Jantunen MJ (2004). The *EXPOLIS* Study: Experiences and implications for PM exposure research and environmental policy in Europe (Die *EXPOLIS* Untersuchung: Ehrfahrungen und Konsequenzen an Staub Exposition Forschung und Umweltspolitik in Europa). Staub und Staubinhaltstoffe: Emission, Immission, Innenraum, Quellen, Gesetze. KRdL-Experten-Forum 10./11. November 2004, Düsseldorf. KRdL-Schriftenreihe Band 33. Kommission Reihaltung der Luft im VDI und DIN – Normenausschuss KRdL, Düsseldorf, pp 31-54 (/308).

IARC (1986). Tobacco smoking, Vol 38, pp. 309-314.

IARC (2006). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Vol. 88 Formaldehyde, 2-Butoxyethanol and 1-tert-Butoxy-2-propanol (in preparation). Updated 7 September 2004. Available in the internet at

http://monographs.iarc.fr/ENG/Monographs/vol88/volume88.pdf. Accessed on June 21, 2006.

Janssen NAH, Schwartz J, Zanobetti A and Suh HH (2002). Air Conditioning and Source Specific Particles as Modifiers of the Effect of PM10 on Hospital Admissions for Heart and Lung Disease. *Environ. Health Perspect.* 110, 43-49.

Katsouyanni, K. et al, "Confounding and effect modification in the short-term effects of ambient particles on total mortality: results from 29 European cities within the APHEA2 project", *Epidemiology*, vol. 12, no. 5, pp. 521-531 (2001).

Katsouyanni K, Zmirou D, Spix C, Sunyer J, Schouten JP, Pönkä A (1995). Short term effects of air pollution on health: A European approach using epidemiological time series data. *Eur Respir J*, 8:1030-1038.

Koistinen KJ, Edwards RD, Mathys P, Ruuskanen J, Künzli N and Jantunen MJ (2004). Sources of fine particulate matter in personal exposures and residential indoor, residential outdoor and workplace microenvironments in the Helsinki phase of the EXPOLIS study. Scand. J. Work *Environ. Health* 30, suppl. 2, 36-46.

Koistinen KJ, Hänninen O, Rotko T, Edwards R, Moschandreas D, Jantunen MJ (2001). Behavioral and Environmental Determinants of Personal Exposures to PM2.5 in EXPOLIS-Helsinki, Finland. Atmos. Environ. 2001, 35(14), 2473 2481.

Koponen IK, Asmi A, Keronen P, Puhto K and Kulmala M (2001). Indoor air measurement campaign in Helsinki, Finland 1999 - the effect of outdoor air pollution on indoor air. *Atmos. Environ.* 35, 1465-1477.

Kousa A, Oglesby L, Koistinen K, Künzli N, Jantunen M (2002). Exposure chain of urban air PM2.5 - associations between ambient fixed site, residential outdoor, indoor, workplace and personal exposures in four European cities in the EXPOLIS-study. Atmos Environ, 36:3031-39.

Kulmala M, Asmi A and Pirjola L (1999). Indoor air aerosol model: the effect of outdoor air, filtration and ventilation on indoor concentrations. *Atmos. Environ.* 33, 2133-2144.

Laden F, Schwartz J, Speizer FE, Dockery DW. Reduction in Fine Particulate Air Pollution and Mortality - Extended Follow-up of the Harvard Six Cities Study. Am J Respirat Critical Care Med. 173 (2006) 667-672.

Lai HK, ApSimon H, Bayer-Oglesby L, Götschi T, Jantunen MJ, Künzli N, Kulinskaya E, Nieuwenhuijsen MJ, Schweizer C, Colvile R (2006). Determinants of indoor air concentrations of PM2.5, black smoke and NO2 in six European cities (EXPOLIS study). *Atmos. Environ.* 40, 1299-1313.

Long CM, Suh HH, Catalano PJ and Koutrakis P (2001). Using time- and size resolved perticulate data to quantify indoor penetration and deposition behaviour. *Environ. Sci. Technol.* 35, 2089-2099.

Morawska L, He C, Hitchins J, Gilbert D, Parappukkaran S (2001). The relationship between indoor and outdoor airborne particles in the residential environment. *Atmos. Environ.* 35, 3463-3473.

NRC, 1986. Environmental Tobacco Smoke: Measuring Exposures and Assessing Health Effects. National Academy Press, Washington DC.

Özkaynak H, Xue J, Spengler J, Wallace L, Pellizzari E, Jenkins P. (1996). Personal Exposure to Airborne Particles and Metals: Results from the Particle TEAM Study in Riverside, California. *JEA&EE*, 6:57-78.

Pope CA III. Respiratory disease associated with community air pollution and a steel mill, Utah Valley. Am J Public Health 1989;79: 623-8.

Pope CA III, Thun MJ, Namboodiri MM, et al. Particulate air pollution as a predictor of mortality in a prospective study of US adults. Am J Respir Crit Care Med 1995;151:669-74.

Pope CA III, Burnett RT, Thun MJ, et al. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA 2002;287:1132-41.

Rodes CE, Lawless PA, Evans GF, Sheldon LS, Williams RW, Vette AF, Creason JP and Walsh D (2001). The relationship between personal PM exposure for elderly populations and indoor and outdoor concentrations for three retirement center scenarios. *J. Exposure Analys. Environ. Epidemiol.* 11, 103-115.

Wallace L, Özkaynak H, Spengler J, Pellizzari E and Jenkins P (1993). Indoor, outdoor and personal air exposures to particles, elements and nicotine for 178 southern California residents. *Proceedings of Indoor Air* '93, 3:445-450.

Wallace L (1996). Indoor Particles: A Review. J AWMA, 46:98-126.

WHO: WHO air quality guidelines: Global update (2005). Report on a Working Group meeting, Bonn, Germany, 18-20 October 2005, 30 pp.

# **ANNEX 2** Formaldehyde in Indoor Air

## 1 INTRODUCTION

The scope of the formaldehyde example is to introduce a general risk assessment for formaldehyde in indoor air.

## 2 IDENTIFICATION OF HAZARD AND SOURCES

Hazard identification of formaldehyde is based on a number of epidemiological and cohort studies. There are data from clinical studies and cross-sectional surveys of human populations, as well as experimental studies carried out with laboratory animals, to show the irritant effects of formaldehyde on the eyes, nose, throat and respiratory tract (WHO 2000, Kotzias et al. 2005).

In 2004, IARC assessed that there was sufficient evidence that formaldehyde causes nasopharyngeal cancer in humans and re-classified it as a Group 1, known human carcinogen (Kotzias et al. 2005).

There are several indoor sources that can increase human exposure to formaldehyde including cigarette smoke, insulating materials, particle board or plywood furniture containing formaldehyde-based resins, water based paints, fabrics, household cleaning agents, disinfectants, pesticide formulations, paper products and adhesives containing formaldehyde used for plastic surfaces, parquet, carpets and other building materials containing ureaformaldehyde resins. Also gas cookers and open fireplaces emit formaldehyde to indoor air (WHO 1989, COMEAP 1997, Jurvelin et al. 2001, EPA/Cal 2003).

Humidity and temperature may change the emission profiles of formaldehyde for some materials and products. Emission rates of 55 diverse materials and consumer products were determined in a chamber study in California (Kelly et al. 1999). The tests showed that among dry products highest formaldehyde emissions were found from bare pressed-wood materials made with urea-formaldehyde (UF) resins, and from new permanent press fabrics. An acid-cured floor finish showed the highest emissions from wet products, clearly exceeding those of any dry product.

Increased formaldehyde concentrations may be present also in hospitals and scientific facilities where formaldehyde is used as a sterilising and preserving agent (WHO 1989).

## 3 EXPOSURE-RESPONSE ASSESSMENT

Non-carcinogenic and carcinogenic effects of formaldehyde were reviewed in the INDEX project (Kotzias et al. 2005). Table 1 summarises the health effect levels of acute and chronic exposure for non-carcinogenic effects.

**Table 1.** Non-carcinogenic health effect levels of acute and chronic exposure to formaldehyde (Kotzias et al. 2005).

| NOAEL<br>mg/m³          | LOAEL<br>mg/m³  | Target system; critical effects  | Remarks   | Study  | Source; Ref.<br>Value mg/m <sup>3</sup> |
|-------------------------|---|--|---|--|---|
| Short-term              | exposure  |  |   |  | l                                       |
| 0.6<br>EXPOSURE         | 1.2 <sub>EXP</sub> 0.53 <sub>BC05</sub> 0.94 <sub>Th-ADJ-BC05</sub> | Mild and moderate eye irritation   | Volunteers, 3h  | Kulle et al. (1987)  | OEHHA 2006;<br>REL: 0.094               |
|                         | 0.5 ехр   | Nasal and eye irritation   | Volunteers, 2h<br>(potentially<br>sensitive<br>population)                        | Pazdrak et al.<br>(1993)   | ATSDR 1999;<br>MRL: 0.05                |
|                         | 0.24 STAT   | Respiratory tract irritation   |   | cited in Sloof et<br>al. (1992)  | RIVM 1992                               |
|                         | 0,1   | Nose and throat irritation   | Human, short-<br>term   | cited in<br>IPCS/WHO, 1989   | WHO 2000                                |
| 11                      | (0.037)   | Respiratory symptoms (depending on study)  | Children;<br>additional<br>investigation<br>requested                             | Krzyzanowski et al., (1990);<br>Wantke et al. (1996); Garrett et al. (1999);<br>Franklin et al. (2000) | UCLA 2001                               |
| Long-term               | exposure  |  |   |  | J.                                      |
| 0.09 Study<br>0.032 ADJ | 0.26 Study<br>0.093 ADJ   | Nasal and eye irritation, nasal obstruction, and lower airway discomfort; histopathological nasal lesions including rhinitis, squamous metaplasia, and dysplasia | Occupational, 10y average;  | Wilhelmsson and<br>Holmstrom, 1992;<br>supported by<br>Edling et al., 1988                             | OEHHA 2006;<br>REL; 0.003               |
|                         | 0.3 Study   | Mild irritation of the eyes and<br>upper respiratory tract and mild<br>damage to the nasal epithelium  | Occupational,<br>10.4y  | Holmstrom et al.,<br>1989  | ATSDR 2006;<br>MRL: 0.01                |
|                         | 0.31 <sub>STAT</sub>  | Sensory irritation   | for low but<br>significant<br>percentage of<br>exposed workers                    | Weighting the total body of data   | NIWL 2003                               |
|                         | 0.12  | Symptoms of irritation   | LOAEL may be<br>lower only for a<br>very small<br>proportion of the<br>population | well conducted<br>studies  | Health Canada<br>1999; (noTC)           |

Carcinogenicity: IARC: 1 (15.06.04); U.S.EPA: B1; Unit risk (EPA-IRIS): 1.3E-5 (µg/m³)-¹; An association exists between cytotoxic, genotoxic, and carcinogenic effects. Most probably HCHO cannot act as a complete carcinogen at non-cytotoxic concentrations. If exposure is accompanied by recurrent tissue damage at the site of contact, it may be assumed to have carcinogenic potential in humans. Evidence of carcinogenicity in rats: induction of squamous cell carcinoma at > 18 mg/m³.

In June 2004, IARC announced there was sufficient evidence that formaldehyde causes nasopharyngeal cancer in humans and re-classified it as a Group 1, known human carcinogen (previously classified as 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.

**Genotoxicity:** Mutagenic in vitro, in vivo only in tissue of contact at doses adapt to give cytotoxic effects (no primary mutagen).

**Odour threshold:** range: 0.03 - 0.6 mg/m3 (WHO); 0.035 mg/m3 (Devos); > 6 mg/m3 untolerable to most people

Recommended WHO Air Quality Guideline value is  $100 \mu g/m3$  as a 30-min average (WHO 2000).

**Susceptible population:** Wide variability in response to HCHO irritancy; Asthmatics: no significant changes in pulmonary function and non-specific airway reactivity at 3.7 mg/m3; population with allergic contact dermatitis (3-6%) seem not to react differently at 0.5 mg/m3 (Pazdrak, 1993: 9 volunteers with formalin skin sensitization).

## 4 EXPOSURE ASSESSMENT

Exposure to formaldehyde has been typically assessed by measuring indoor air concentrations in workplaces and at homes. There is limited information available about personal exposures to formaldehyde in general population and especially in quantitative source apportionment in indoor settings. Although, common sources of formaldehyde indoors are known, it is still not known what are the most important source and behavioural factors in large populations.

## 5 RESULTS OF EXPOSURE AND MICROENVIRONMENTAL STUDIES

Residential indoor air concentrations of formaldehyde in European large scale population based surveys were reviewed in the INDEX study (Kotzias et al. 2005). Residential concentrations varied between 1 and 171  $\mu$ g/m³. Figure 1 shows the cumulative distributions of residential formaldehyde concentrations in some European studies (from 5<sup>th</sup> to 95<sup>th</sup> percentiles).

There is a lack of personal exposure data measured in general populations in Europe. Figure 2 shows cumulative distributions from Finnish and Swedish studies.

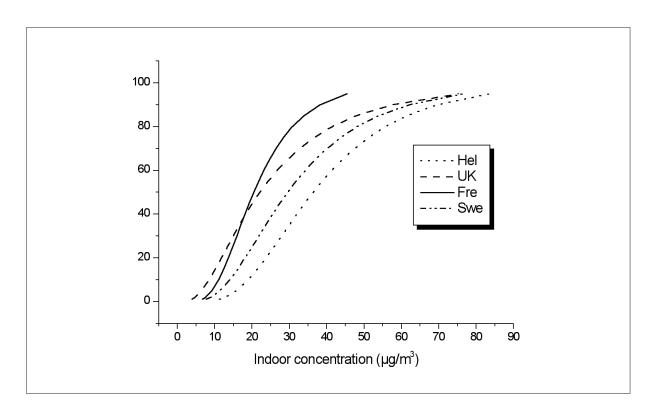
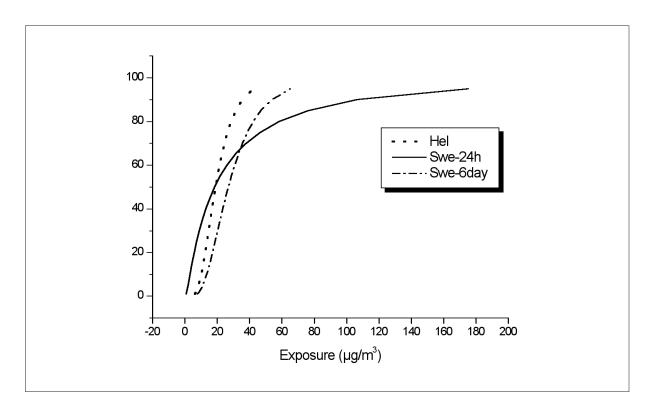


Figure 1. Cumulative frequency distributions of indoor air concentrations of formaldehyde in Helsinki (Hel, n=15, Jurvelin et al. 2001), in England (UK, n=833, Brown VM et al. 2002), in the French Survey (Fre, n=201, Kirchner 2004), and in Sweden (Swe, n=40, Gustafson et al. 2004).



**Figure 2.** Cumulative frequency distribution of personal exposure concentrations of formaldehyde in Helsinki (Hel, 48-hour average, n= 15, Jurvelin et al. 2001) and Sweden (Swe-24h = 24-hour average, n=24, and Swe-6day = 6-day average, n=40, Gustafson et al. 2004).

# 6 RISK MANAGEMENT OF FORMALDEHYDE – RECOMMENDED IDEAL PRACTISE

The INDEX (Critical Appraisal of the Setting and Implementation of Indoor exposure Limits in the EU) Working Group (WG) has estimated the no-effect level (acute and chronic) at  $30 \mu g/m^3$  as a 30-minute average. This WG has also recommended keeping formaldehyde concentrations as low as reasonably achievable pending the outcome of the IARC revision of the carcinogenicity of formaldehyde (Kotzias et al. 2005).

Specific recommended risk management options are:

- Minimise the emissions of formaldehyde from consumer products, building materials, furnishings and household/office chemicals.
- Require product labelling to inform about formaldehyde content and potential formaldehyde release from household and building products
- Discourage the use of any building, furnishing, and consumer products which contain and release formaldehyde, or which, when exposed to certain temperature or humidity conditions, or to co-pollutants, will form and release formaldehyde into indoor air.
- Raise public awareness and provide information to the public about the sources, nature and levels of risks of formaldehyde in indoor air.

In addition to the specific recommendations and management options, the following general recommendations shall be applied to formaldehyde:

- Ban tobacco smoking in all indoor spaces under public jurisdiction, and working places. Raise public awareness on the hazards of tobacco smoke, and discourage smoking in the homes, particularly in the presence of children.
- Raise public awareness about the various acute and long term risks of exposure to formaldehyde from indoor sources by campaigns focused on specific and concrete issues and on relevant target populations.

#### REFERENCES

ATSDR - Agency for Toxic Substances and Disease Registry (2006). Available in the Internet at http://www.atsdr.cdc.gov/phshome.html.

Brown VM, Coward SKD, Crump DR, Llewellyn JW, Mann HS, Raw GJ 2002. Indoor air quality in English homes – formaldehyde. In: Proceedings of the 9<sup>th</sup> International Conference on Indoor Air Quality and Climate, vol 4, 473-476.

COMEAP Committee on the medical effects of air pollutants (1997). Handbook on air pollution and health. Department of health, committee on the medical effects of air pollutants. Her Majesty's Stationary Office, London, UK.

Edling C, Hellquist H, Odkvist L. 1988. Occupational exposure to formaldehyde and histopathological changes in the nasal mucosa. Br. J. Ind. Med. 45 11:761-765.

EPA California, USA (2003). OEHHA Office of Environmental Health Hazard Assessment, <a href="http://www.oehha.ca.gov/air/chronic\_rels/">http://www.oehha.ca.gov/air/chronic\_rels/</a>. Accessed in November, 2003.

Devos M, Patte F, Rouault J, Laffort P, Van Genert LJ, (1990). Standardized Human Olfactory Thresholds. IRL Press at Oxforf University Press.

Franklin P, Dingle P, Stick S. 2000. Raised exhaled nitric oxide in healthy children is associated with domestic formaldehyde levels. Am J Respir Crit Care Med. 2000 May;161 5:1757-9.

Garrett MH, Hooper MA, Hooper BM, Rayment PR, Abramson MJ. 1999. Increased risk of allergy in children due to formaldehyde exposure in homes. Allergy 1999 Dec;54 12:1327.

Gustafson P, Barregard L, Lindahl R, Sallsten G 2004. Formaldehyde levels in Sweden: personal exposure, indoor, and outdoor concentrations. J Expo Anal Environ Epidemiol, online publication, 1-9.

Health Canada, Environment Canada. 2001. Priority substances list assessment report: Formaldehyde. Ottawa. Ministry of Public Works and Government Services. Available at <a href="http://www.hc-sc.gc.ca/hecs-sesc/exsd/psl2.htm">http://www.hc-sc.gc.ca/hecs-sesc/exsd/psl2.htm</a>.

Holmstrom M, Wilhelmsson B, Hellquist H, et al. 1989. Histological changes in the nasal mucosa in persons occupationally exposed to formaldehyde alone and in combination with wood dust. Acta Otolaryngol Stockh 107:120-129.

IPCS/WHO (1989). Formaldehyde. In: Environmental Health Criteria 89. Geneva: International Programme on Chemical Safety, World Health Organisation, 1989.

Jurvelin J, Vartiainen M, Jantunen M, Pasanen P. (2001). Personal Exposure Levels and Microenvironmental Concentrations of Formaldehyde and Acetaldehyde in the Helsinki metropolitan Area, Finland. J.Air & Waste Manage. Assoc. 51:17-24.

Kelly TJ, Smith DL, Satola J (1999). Emission rates of formaldehyde form materials and consumer products found in California homes. Environmental Science and Technology 33, 81–88.

Kirchner S 2004. Preliminary results of the French National Survey on Indoor Air Quality. Personal communication.

Kotzias D, Koistinen K, Kephalopoulos S, Schlitt C, Carrer P, Maroni M, Jantunen M, Cochet C, Kirchner S, Lindvall T, McLaughlin J, Mølhave L, Fernandes EO and Seifert B. (2005). Final Report of the INDEX Project, Critical Appraisal of the Setting and Implementation of Indoor Exposure Limits in the EU. Office for Official Publication of the European Communities, Luxenbourg. EUR 21590 EN, ISBN 92-894-9353-4. pp. 331.

Krzyzanowski, M, J.J. Quackenboss and M.D. Lebowitz. (1990). Chronic respiratory effects of indoor formaldehyde exposure. Environ. Res. 52: 117-125.

Kulle TJ, Sauder LR, Hebel JR, et al. 1987. Formaldehyde dose-response in healthy nonsmokers. J Air Pollut Control Assoc 37:919-924.

NIWL - National Institute for Working Life - The Nordic Expert Group for Criteria Documentation of Health Risks from Chemicals and The Dutch Expert Committee on Occupational Standards (2006). Available in the Internet at www.arbetslivsinstitutet.se/.

OEHHA - Office of Environmental Health Hazard Assessment of the Californian Environmental Protection Agency (2006). Available in the Internet at <a href="http://www.oehha.ca.gov/air/acute\_rels/allAcRELs.html">http://www.oehha.ca.gov/air/acute\_rels/allAcRELs.html</a>.

Pazdrak K, Gorski P, Krakowiak A, Ruta U. (1993). Changes in nasal lavage fluid due to formaldehyde inhalation. Int Arch Occup Environ Health;64 7:515-519

Slooff et al. (1992). Exploratory report formaldehyde. National Institute of Public Heatlh and Environmental, editor.RIVM report 710401018. Bilthoven. Ref Type: Report.

Wantke F, Demmer CM, Tappler P, Gotz M, Jarisch R. 1996. Exposure to gaseous formaldehyde induces IgE-mediated sensitization to formaldehyde in school-children. Clin Exp Allergy. 1996 Mar;26 3:276-80.

WHO (1989). Environmental health criteria for acetaldehyde, 89. WHO, Geneva, 1989. Available in the Internet <a href="http://www.inchem.org/documents/ehc/ehc/ehc89.htm">http://www.inchem.org/documents/ehc/ehc/ehc89.htm</a>.

WHO 2000. Air quality guidelines for Europe 2000. WHO Regional Office for Europe, WHO Regional Publications, European Series, No. 91, Copenhagen, 2001.

Wilhelmsson B, and Holmstrom M. 1992. Possible mechanisms of formaldehyde-induced discomfort in the upper airway. Scand. J. Work. Environ. Health 18 6:403-407.

## **ANNEX 3** Ozone in Indoor Air

## 1 INTRODUCTION

The scope of the ozone example is to introduce a general risk assessment for ozone in indoor air

## 2 IDENTIFICATION OF HAZARD AND SOURCES

Hazard identification of ozone has been done typically using results from epidemiological studies based on ambient air concentrations.

There are also some indoor sources of ozone such as ozonizers, which are marketed as "air cleaners", electrostatic air cleaners, photocopying machines and printers.

The common source of ozone present in indoor air is outdoor air. In ambient air ozone is formed in the lower atmosphere by reactions of volatile organic compounds (VOC) and nitrogen oxides (NOx) in the presence of sunlight.

Emissions of tropospheric ozone precursors come mainly from mobile sources i.e. from transport sector. In Western Europe and Central European countries transportation is a source of 52% and 37% of the total precursor emissions in these countries (EEA 2003).

EU (Directive 2002/3/EC) and WHO (WHO 2000) have set a guideline value of 120 μg/m³ (0.06 ppm) for 8-hour average (to be exceeded on no more than 25 days per year) for tropospheric ozone. Considerable part of the Western European population is exposed under special meteorological conditions to peak concentrations exceeding the 8-hour limit value. Although there seems to be decreasing trend in peak ozone concentrations, long-term concentrations are increasing in Western Europe (EEA 2003).

## 3 EXPOSURE-RESPONSE ASSESSMENT

Relatively low amounts of ozone can cause health effects such as respiratory symptoms, pulmonary function changes, increased airway responsiveness, airway inflammation. Ozone may also worsen chronic respiratory diseases such as asthma and weaken the ability to fight against respiratory infections. People vary widely in their susceptibility to ozone. Healthy people, as well as those with respiratory diseases, can experience breathing problems when exposed to ozone. Exercise during exposure to ozone causes a greater volume of ozone to be inhaled, and thus, increases the risk of adverse respiratory effects. Recovery from the harmful effects can occur following short-term exposure to low levels of ozone, but health effects may become more damaging and recovery less certain at higher levels or from longer exposures (EPA 2006, WHO 2000).

Factors expected to increase risk and severity of health effects caused by exposure to ozone are:

- Increase in inhaled exposure concentration to ozone
- Longer duration of exposure for some health effects
- Activities that raise the breathing rate (e.g., heavy physical work, exercise)
- Certain pre-existing lung diseases (e.g., asthma)

WHO (2000) has reviewed toxicological results of dose-response factors for ozone. The results of the review are summarised in Table 1 for two adverse health effects based on controlled exposure experiments. Table 2 presents the relationship between changes in the selected health effects and the peak daily ozone concentration as a result of epidemiological studies.

*Table 1.* Health outcomes associated with controlled ozone exposures (adopted from WHO 2000).

| Health effect  | Ozone concentra<br>which the hea<br>expec | lth effect is |
|--|---|---------------|
|  | Averagin                                  | g time        |
| Change in FEV1 (active, healthy, outdoors,<br>most sensitive 10% of young adults<br>and children): | 1 hour                                    | 8 hours       |
| 5%   | 250                                       | 120           |
| 10%  | 350                                       | 160           |
| 20%  | 500                                       | 240           |
| ncrease in inflammatory changes (neutrophil influx)  |   |               |
| (healthy young adults at >40 litres/minute outdoors)   | 400                                       | 180           |
| 2-fold   |   |               |
| 4-fold   | 600                                       | 250           |
| 8-fold   | 800                                       | 320           |

**Table 2.** Health effects associated with changes in ambient ozone concentration in epidemiological studies (adopted from WHO 2000).

| Health effect  | Change in ozone cor | ncentration (µg/m²) |
|--|---------------------|---------------------|
|  | Averagir            | ng time             |
| Increase in symptom exacerbations among adults or asthmatics (normal activity) | 1 hour              | 8 hours             |
| 25%  | 200                 | 100                 |
| 50%  | 400                 | 200                 |
| 100%   | 800                 | 300                 |
| Increase in hospital admissions for respiratory                                |                     |                     |
| conditions:  |                     |                     |
| 5%   | 30                  | 25                  |
| 10%  | 60                  | 50                  |
| 20%  | 120                 | 100                 |

## 4 EXPOSURE ASSESSMENT

Exposure to ozone is assessed by measuring ambient air concentrations at fixed monitoring sites. In several studies, exposure to ozone has been assessed by measuring ambient concentrations and by collecting additional data such as time spent outdoors, traffic density in the neighbourhood, physical activity, ventilation and air conditioning systems, etc. using questionnaires and time-activity diaries (Monn 2001).

Indoor air concentrations are still rarely studied. There is clearly a lack of information about personal exposures to ozone in general population. Although, there are relatively few indoor sources of ozone, it is important to find out what are the personal exposure concentrations and, especially their determining factors in large populations.

#### 5 RESULTS OF EXPOSURE AND MICROENVIRONMENTAL STUDIES

Kalabokas and Kotzias, (2004) have assessed exposure to ozone in three European cities Athens, Paris and Rome (Table 3). They assessed exposure to ozone by measuring ambient air concentrations in fixed site monitoring stations in comparable location characteristics (urban kerb-site, urban background, urban peripheral, rural). For the estimation of the total ozone exposure the broad activity patterns of the population were considered assuming the indoor concentration being 50% of the outdoor concentration. Since most people spend on average 90% of their time indoors, this activity pattern was used together with the assumption that those working outdoors would spend on average 50% of time indoors. A 20 m³ daily average breathing volume was assumed when staying outdoors. Indoors the breathing volume of 15 m³ was applied assuming less heavy exercise being done indoors compared to outdoors.

The maximum ozone exposure was observed at the periphery of the urban areas and the minimum at the urban centres. The EU health protection standard (120  $\mu g/m^3$  – 8-hour average) can be frequently violated in peripheral and rural sites in all three cities, especially in the Mediterranean capitals of Athens and Rome. For the same activity pattern the ozone personal exposure at the rural stations is higher than at the urban kerb-site stations but it is on the average lower than the exposure at the urban-peripheral sites and comparable with the exposure at the urban background stations. The ozone exposures do not differ substantially in the three cities (urban Athens and Rome being more comparable). Unlike most other atmospheric pollutants, there is a widespread population exposure to ozone, regardless of the place of residence or the type of activity. In order to obtain more accurate and reliable human air exposure estimates, systematic personal ozone exposure monitoring is needed.

Salmon et al. (2000) studied indoor and outdoor concentrations of ozone in five museums in Krakow, Poland. For the continuous measurements using sampling periods from 21 to 46 hours, the indoor levels ranged from 1.5 ppb (3.0  $\mu$ g/m³) to 8.5 ppb (17.0  $\mu$ g/m³), while outdoor levels ranged from 11.0 ppb (22.0  $\mu$ g/m³) to 32.4 ppb (64.8  $\mu$ g/m³). It was concluded that in the museums, rapidly ventilated through many open doors and windows, the I/O ratio was about 0.42-0.44.

Breysse et al. (2005) has studied ozone concentrations in the homes of 100 Baltimore city asthmatic children. The 72-hour time weighted average ranged from 0.8 ppb (1.6  $\mu$ g/m³) to 55.2 ppb (110.4  $\mu$ g/m³) having median at 1.7 ppb (3.4  $\mu$ g/m³).

Chao (2001) studied residential indoor concentrations of ozone in 10 non-smoking apartments in Hong Kong. The indoor ozone concentrations ranged from 0 to 9.78  $\mu$ g/m³ with an average of 5.3  $\mu$ g/m³. Simultaneous outdoor concentrations ranged from 3.91  $\mu$ g/m³ to 31.3  $\mu$ g/m³ with an average of 13.3  $\mu$ g/m³. The indoor/outdoor (I/O) ratios ranged from 0 to 0.83 with an average of 0.40. The average sink strength of the buildings for ozone was estimated to be 1.39 mg/h.

**Table 3.** Average ambient ozone concentrations (in μg/m³) and daily ozone exposures (in μg O³/day) for those who spend 50% and for those who spend 90% of their time indoors in Athens, Paris and Rome (adapted from Kalabokas and Kotzias 2004).

|                     | Average O <sub>3</sub> | Total exposure for 50% indoors | Total exposure for<br>90% indoors |
|---------------------|------------------------|--------------------------------|-----------------------------------|
| Athens              |                        |                                |                                   |
| Peireas             | 68                     | 940                            | 600                               |
| Geoponiki           | 75                     | 1000                           | 660                               |
| Maroussi            | 93                     | 1300                           | 820                               |
| Aliartos            | 70                     | 970                            | 610                               |
| Paris               |                        |                                |                                   |
| Champs de Mars      | 44                     | 600                            | 390                               |
| Eastman             | 51                     | 700                            | 450                               |
| Jardin de Luxemburg | 5.1                    | 700                            | 450                               |
| Rambuillet          | 73                     | 1000                           | 640                               |
| Rome                |                        |                                |                                   |
| Ada                 | 56                     | 770                            | 500                               |
| Cavaliere           | 56<br>69               | 950                            | 600                               |
| Guido               | 89                     | 1200                           | 800                               |

#### 6 RISK MANAGEMENT OF OZONE – RECOMMENDED IDEAL PRACTICE

For managing indoor ozone concentrations, there are two practical management options: 1) to manage the entry of ozone from outdoor air and 2) to minimise indoor sources of ozone.

## **Ventilation**

Typically outdoor concentrations of ozone are higher than indoor concentrations. Therefore, in the absence of indoor sources, outdoor air is the driving force for ozone concentration in indoor air. In practice, this means that keeping windows open or otherwise increasing the air exchange rate, especially in hot summer days, when outdoor ozone is typically at its highest level, will increase indoor ozone levels.

It is also recommended to use mechanical ventilation, which removes ozone by surface reactions in air channels. Especially, if refrigerating cooling of incoming air is not available, the use of active carbon filters is recommended to decrease indoor concentrations of ozone.

#### Source control

Ozonisers should not be used in indoor environments. Indoor ozonisers are marketed as indoor air purifiers, while in reality they add a new direct source of toxic air pollution – ozone – and secondary source via highly irritative reaction products of ozone with some VOCs such as terpenes. It should be ensured that ozone emissions from devices commonly used indoors, such as printers and copy machines or other electrical devices are minimised.

It is recommended to minimise VOC emissions from the materials and products that are used indoors, because reactions of ozone with VOCs produce compounds that might be highly irritant. Therefore, it is recommended to develop harmonised European wide emission labelling systems to avoid unnecessary VOC emissions indoors.

## REFERENCES

Breysse PN, Buckley TJ, Williams D, Beck CM, Jo S-J, Merriman B, Kanchanaraksa S, Swartz LJ, Callahan KA, Butz AM, et al. (2005). Indoor exposures to air pollutants and allergens in the homes of asthmatic children in inner-city Baltimore. Environmental Research, Vol 98:2, 167-176.

Chao CYH (2001). Comparison between indoor and outdoor air contaminant levels in residential buildings from passive sampler study. Building and Environment, Vol 36:9, 999-1007.

EEA (European Environment Agency) 2003. Europe's environment. Environmental assessment report No 10. EEA Copenhagen.

EPA (2006). US Environmental Protection Agency, available in the internet at http://www.epa.gov/air/urbanair/ozone/index.html.

Kalabokas PD and Kotzias D (2004). Population exposure to atmospheric ozone in the European capital cities of Athens, Paris, Rome and their surroundings. Fresenius Environmental Bulletin, vol 13, no 5, pp. 465-471.

Monn C (2001). Exposure assessment of air pollutants: a review on spatial heterogeneity and indoor/outdoor/personal exposure to suspended particulate matter, nitrogen dioxide and ozone. Atmospheric Environment, Vol 35:1, 1-32.

Salmon LG, Cass GR, Bruckman K and Haber J (2000). Ozone exposure inside museums in the historic central district of Krakow, Poland. Atmospheric Environment, Vol 34:22, 3823-3832.

WHO (World Health Organization) 2000. Air quality guidelines for Europe 2000. WHO Regional Office for Europe, WHO Regional Publications, European Series, No. 91, Copenhagen, 2001.

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#### EUR 22503 EN - DG Joint Research Centre, Institute for Health and Consumer Protection

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Authors: Stylianos Kephalopoulos, Kimmo Koistinen, Dimitrios Kotzias, Christian Cochet, Eduardo de Oliveira Fernandes, Matti Jantunen, Thomas Lindvall, Marco Maroni†, James P. McLaughlin, Lars Mølhave, Bernd Seifert

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#### Abstract:

It is now well established that indoor air pollution contributes significantly to the global burden of disease of the population. Therefore, the knowledge of this contribution is essential in view of risk assessment and management. The ECA STRATEX 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 should be considered as a framework. This framework may have to be adapted to specific situations by policy makers, risk assessors, and risk managers.

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