EUROPEAN COLLABORATIVE ACTION

URBAN AIR, INDOOR ENVIRONMENT AND HUMAN EXPOSURE

Environment and Quality of Life

Report No 22

Risk Assessment In Relation To Indoor Air Quality

EUROPEAN COMMISSION
JOINT RESEARCH CENTRE - ENVIRONMENT INSTITUTE - AIR QUALITY UNIT

2000

EUR 19529 EN

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Risk Assessment In Relation To Indoor Air Quality

Dedicated to late professor Christos C. Lefas

prepared by

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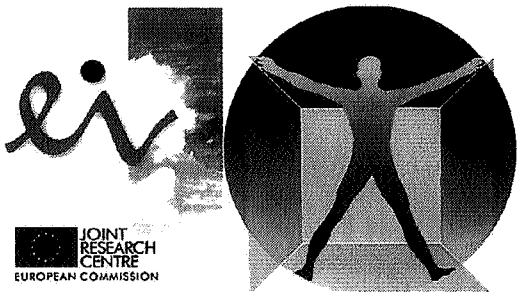

MANDATE: European Collaborative Action **"Urban Air, Indoor Environment and Human Exposure"** (formerly "Indoor Air Quality & it's Impact on Man")

For more than 12 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 has dealt 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 may all 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 will be hosted by and supporting the activities of the Joint Research Centre's Environment Institute in Ispra (Italy) dealing with Air Quality.

This focussed activity will proceed 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) *
- Report No. 2: Formaldehyde emission from wood-based materials: guideline for the determination of steady state concentrations in test chambers. (EUR 12196 EN) *
- Report No. 3: Indoor pollution by NO_2 in European countries. (EUR 12219 EN)
- Report No. 4: Sick building syndrome - a practical guide. (EUR 12294 EN) *
- Report No. 5: Project inventory. (S.P.I. 89.33) *
- Report No. 6: Strategy for sampling chemical substances in indoor air. (EUR 12617 EN)
- Report No. 7: Indoor air pollution by formaldehyde in European countries. (EUR 13216 EN) *
- Report No. 8: Guideline for the characterization of volatile organic compounds emitted from indoor materials and products using small test chambers. (EUR 13593 EN)
- Report No. 9: Project inventory - 2nd updated edition. (EUR 13838 EN) *
- Report No. 10: Effects of indoor air pollution on human health. (EUR 14086 EN)
- Report No. 11: Guidelines for ventilation requirements in buildings. (EUR 14449 EN)
- Report No. 12: Biological particles in indoor environments. (EUR 14988 EN)
- Report No. 13: Determination of VOCs emitted from indoor materials and products. Interlaboratory comparison of small chamber measurements. (EUR 15054 EN)
- Report No. 14: Sampling strategies for volatile organic compounds (VOCs) in indoor air. (EUR 16051 EN)
- Report No. 15: Radon in indoor air. (EUR 16123 EN)
- Report No. 16: Determination of VOCs emitted from indoor materials and products; second interlaboratory comparison of small chamber measurements. (EUR 16284 EN)
- Report No. 17: Indoor Air Quality and the use of Energy in Buildings. (EUR 16367 EN)
- Report No. 18: Evaluation of VOC emissions from building products: solid flooring materials. (EUR 17334 EN)
- Report No. 19: Total volatile organic compounds (TVOC) in indoor air quality investigations. (EUR 17675 EN)
- 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.

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Abstract

ECA (European Collaborative Action on, "Urban Air, Indoor Environment and Human Exposure"), 2000 Risk Assessment In Relation To Indoor Air Quality, Report No 22. EUR 19529 EN. Luxembourg: Office for Official Publications of the European Communities

People will never live in a risk free environment. Still we must aim at minimising all risks and most importantly risks that are imposed on without their consent or even knowledge. A building is built for and perceived as shelter – against weather and unwanted intruders, for thermal comfort, privacy and property. Health threatening risks that the dwellers of a building cannot sense or expect contradict directly the whole concept of a building.

Risk assessment is a scientific multidisciplinary paradigm to identify, quantify, describe and compare risks. Risk management is an administrative paradigm to develop and compare risks reduction priorities and alternatives, to organise and manage risk-controlling practices and to evaluate the achievements. Risk assessment and management have existed always. The general formal paradigms that are being applied in today's societies, however, are quite recent, and still under continuous development.

This main body of this report presents the state of the art of modern risk assessment and risk management paradigms, highlighting also the historical development that has lead to the present practices, and applies them specifically into building environments.

The examples section in the end of this report applies the formal risk assessment protocol of the EC (and similarly USEPA), to a variety of building related health risk. These examples are not intended as recommendations, instead they are selected to highlight the level of success (or failure) in applying one strict protocol to multiple extremely different problems.

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0 SCOPE OF THIS REPORT

This report has been prepared for those individuals who need to be involved in assessing and managing risks in buildings; professionals, teachers and administrators, in the fields of public health, building design, and regulation/inspection of buildings, individuals who need to assess and manage potential, suspected and materialised health risks related to indoor environments. The scope of the main body of this report is methodological and contains: (i) introduction to the modern methods – and vocabulary - of systematic risk assessment that have evolved since the late 1970's; (ii) assessment of how these methods may be applied for risk assessment in indoor environments; (iii) some discussion on how the risk assessment framework may still need to be developed to better meet the existing risk assessment needs in indoor environments; and (iv) description of the links of risk assessment to risk management and risk communication. The annexed nine examples are included to highlight on one hand the very diverse types of risk assessment needs in buildings, and on the other hand the degree to which the concepts of the Commission Directive 93/67/EEC on Risk Assessment (CEC 1993c) are applicable for these needs.

Introduction

The goal of this report is to assist professionals and administrators who are not risk assessment experts in their tasks of identifying, assessing and managing health risks in building environments. This report focuses on health risks related to poor indoor air quality. Scientific *risk assessments* should follow rigorous predefined protocols. Their outcomes allow comparison of various risks present in buildings with each other, as well as with other risks, including those of outdoor air risk factors. They also form a sound basis for many - though not yet all - practical risk management decisions that building designers and managers must make.

People spend usually well over 90% of their time in indoor spaces, where a number of hazards may generate risks to their health and reduce quality of life. Major sources of contaminants are the occupants themselves (e.g. bioeffluents), building materials and furnishings (e.g. VOCs, formaldehyde), the processes that occur within buildings (e.g. smoking, photocopying) and heating, ventilation and air conditioning (HVAC) components. Outdoor contaminants are added to this endogenous pollution through the ventilation system or by infiltration.

At this stage the most obvious open needs for advanced indoor environment risk assessment and management rationale are for non-cancer endpoints, complex and variable exposure mixtures, and general populations in common indoor environments.

Risk management is an organised effort to collect information about and to control risks. The public cannot and will never achieve risk-free air, water, or food. Risk management is fundamentally a question of values. In a democratic society, there is no acceptable way to make these choices without involving the citizens who will be affected by them.

Due to the huge numbers of indoor environments and the wide variety of potential risks in them, most real life risks are identified, "assessed" and decided upon by occupants, maintenance personnel and building managers. Only a small minority of practical cases will

ever be dealt with by professionally trained risk assessors, whose role is therefore much more important in providing examples, guiding regulation and providing educational materials for the public.

The goals of ECA for this discussion of risk assessment related to non-industrial indoor air quality, are the following:

1. *Assessing risks linked to indoor systems performance, indoor exposures and indoor related health effects in the general public, including sensitive groups.*
2. *Comparing risks (ranking) within the same type of indoor related effect (cancer, sensitisation, irritation, odour, annoyance), and between different types of indoor related effects.*
3. *Assessing the joint risks of combinations of several indoor system performances, sources and/or exposures.*
4. *Assessing cost-effectiveness of alternative choices or countermeasures (e.g., setting source control priorities).*
5. *Incorporating risk assessment into the building design, construction, operation and maintenance processes by developing risk assessment procedures which combine expert panel judgements, occupants' perceptions of risk, and encouraging producers of systems and materials to better use systematic analyses of risks, benefits and costs, incl. life-cycle assessments.*
6. *Educating occupants to enlightened risk perception and behaviour.*

The Report presents an indoor air oriented review of the existing risk assessment, risk management and risk communication principles. It also recommends a framework for the conduct of building related risk assessments. Finally it presents a set of practical risk assessment examples, which are selected to highlight the many different settings and needs for risk assessment in buildings, but do not necessarily present the views of EC, ECA or WG-14.

1 EXISTING EC DOCUMENTS AND REGULATIONS

EU and Member States have already produced a number of documents that are useful in assessing indoor air quality risks. Commission Directive 93/67/EEC (CEC 1993c) lays down the principles for assessing risks to man and the environment of substances notified in accordance with Council Directive 67/548/EEC (CCEE 1967). It also covers classification, packaging and labelling of dangerous substances.

The principles for assessment of risks to man in the directive are pertinent to the assessment of human risks due to individual chemicals in the indoor environment. These principles follow closely the principles defined by the U.S. National Academy of Science (NAS) and the National Research Council (NRC), and applied by the U.S. Environmental Protection Agency (EPA).

The European Community adopted Council Regulation (EEC) 793/93 (CEC 1993a) on the evaluation and control of the risks of existing substances (The Existing Substances Regulation) on March 1993. Article 10(3) of the Regulation requires a member state (or "rapporteur") designated by the Community to carry out a risk assessment of selected substances and, in appropriate cases, to recommend a strategy for limiting the risks posed by each substance. Article 11(1) requires the European Commission to present recommendations for control measures and/or surveillance programmes to the Committee established under Article 15.

Commission Regulation (EEC) 1488/94 (CEC 1994), adopted on June 1994, lays down how a risk assessment must be carried out. This regulation and proposed accompanying technical guidance do not say how the rapporteur should draw up a risk reduction strategy.

2 WHAT IS RISK?

Even in a framework limited to professional health risk assessment, risk has had many definitions. The following are found in relatively recent literature:

- A function determined by three variables: (a) the likelihood of a particular hazard causing harms to the exposed individuals, (b) the magnitude (severity) of the harms or their consequences, and (c) the number of people exposed to the hazard (e.g. Cox and Tait 1991).
- The probability that an event will occur, e.g. that an individual will become ill or die within a stated period of time or age (Last 1995).
- The magnitude of an unwanted consequence related to some activity multiplied by its probability (Morgan 1993).
- A characteristic of a situation or action wherein two or more outcomes are possible, the particular outcome that will occur is unknown, and at least one of the possibilities is unwanted (Covello and Merkhover 1993).
- ...incidence and severity of the adverse effects likely to occur in a human population ... (Commission Directive 93/67/EEC definition of 'risk characterisation')

These definitions do not differ only in wording but also in substance. Sometimes the type of application accounts for the differences in definitions. For example if only fatal outcomes are considered, the severity element can be dropped. Often the differences between the definitions are even more fundamental. Webster's Unabridged Dictionary defines risk as it is defined in mathematical risk analysis, but also as a synonym to danger, peril, jeopardy, hazard. Lay people have other, yet different definitions of risk, which must be taken into account in risk communication. Their perceptions of risk depend on previous knowledge of risks, dread of its consequences (e.g. asbestos insulation in a building), and whether the risk has been imposed by an anonymous body outside of one's control, or it has been voluntarily taken for a practical or emotional purpose. In ICON-magazine (October 1997) 40 individuals - public figures in arts, athletics, business, politics, media and public service, none from science - were interviewed about 'What Is Risk' in their own lives. Not one of those interviewed was talking about the risk as a professional risk assessor defines it. They were discussing much more about active risk taking than passive risk acceptance, mostly about calculated risks, risks that were searched for, and of the personal emotional rewards related to taking and surviving those risks. No wonder, there are considerable differences between the risk assessments by many scientific assessors, let alone between the risk perceptions of the public and scientists. These may simply be often due to the fact that the two sides mean two or more different things with the same word - risk.

It must be acknowledged that in assessing, managing and communicating risks, the word "risk" can refer to both risk as probability (when the outcome is predefined, e.g. cancer or death), or risk as probability multiplied by severity (when the outcome(s) as well as its probability are both subjects of the assessment). In addition "risk" can be expressed as risk of an individual (per year or lifetime), or risk in a population (per 1,000,000 individuals, or a specified population e.g. occupants of an office tower). This terminological flexibility should be accepted, because it rarely leads to real confusion, but allows the use of common risk assessment vocabulary and expression of risks for different purposes. The rest of the terminology for risk assessment and risk management has been adopted from the Commission Directive 93/67/EEC (see chapter 4).

3 INTRODUCTION TO RISK ASSESSMENT

Risk assessment is a science based systematic approach for evaluating the risk associated with an agent, a planned action or an existing condition. Risk assessment has also been described as a way of examining risks so that they can be better avoided, reduced, or otherwise managed (Wilson and Crouch 1987).

The work of deciding policies affecting indoor environments have become more dependent on formal, quantitative risk assessment because of increasing attention to the prevention of human health damage on one hand, and increasing demands for energy and cost effectiveness of buildings on the other. Risk assessment helps set priorities for regulation of the very large numbers of chemicals that are of potential concern and helps direct limited social and governmental resources against the most significant risks. (Russel and Gruber 1987).

What is special about indoor risk assessment? A demonstration of the difficulty in applying traditional single chemical based risk assessment to indoor environmental problems is the fact that the starting point which determines the risk assessment approach in a specific case can be a

- *substance* (e.g. formaldehyde or radon in indoor air),
- *source* (e.g. building materials or smoking),
- *environment* (e.g. "sick building" or mouldy kindergarten),
- *target group* (e.g. atopic students in water damaged schools),
- *effect* (e.g. allergy or "sick building syndrome" outbreak),
- *or pathway* (e.g. ventilation system).

While the starting point defines the main emphasis of a risk assessment, the whole chain from emissions to effects needs to be covered in a comprehensive risk assessment of any indoor air hazard.

3.1 Risk Assessment Tools

Much of our present knowledge on the health effects of indoor contaminants is based on occupational health studies, both epidemiological and toxicological. While this forms an invaluable database for indoor risk assessment, caution should be applied in interpreting it. On one hand occupational exposure levels are usually much higher than levels in other indoor settings, but on the other also less complex, better controlled, limited to the working hours/days and to healthy adult subjects.

In practice epidemiological and toxicological research both serve important and different purposes in risk assessment. Toxicological animal experiments or controlled human exposure studies provide pharmacokinetic and mechanistic information on causal links from controlled exposures to effects. Epidemiological studies provide quantitative associations for heterogenous human populations in variable, complex and dynamic - i.e. realistic - exposure settings.

The risk assessment process usually involves numerical modelling of the exposures from specific surces, relationship between the estimated exposure (dose) to the harmful agent and the observed health outcome. This process has two types of built-in error. The first is practical, based on possible errors in the selection of the model or random errors and biases in measurement of exposure and health effect, which are the necessary input values for the model. The other type of error is fundamental. *Verification and validation of numerical models in natural systems* - including the relationship between exposure and risk - *is [in principle] impossible because natural systems are never closed and model results are always non-unique. Such models need to be confirmed case by.* The most appropriate use of models is *for sensitivity analysis.* (Oreskes et al.1994).

There are numerous methods for analysing the uncertainties in risk assessment. Uncertainty propagation calculates the uncertainty in the risk induced by the uncertainties in the inputs. Sensitivity analysis compares the importance of the input values' uncertainties in terms of their relative contribution to the resulting uncertainty in the risk estimate.

Models can provide policy-makers with risk estimates which, combined with professional and political judgement, should serve as a basis for setting priorities, balancing risks and benefits, and establishing degrees of urgency for public health problems.

3.1.1 Exposure Models

Two types of models used in exposure assessment are considered here; deterministic and probabilistic exposure assessment models.

Deterministic models use single-value best estimates for each input variable to produce single-value estimate of the exposure. Deterministic exposure models are useful when the processes are well understood and uncertainties small. An example is the indoor air concentration caused by a known source, which can be solved by ventilation engineer's dilution equations when the source emissions, compartment volumes, air exchange rates and concentrations in diluting air are known.

When trying to capture the high end of exposure (and risk) in a deterministic model by using conservative instead of most probable input values, conservatism accumulates multiplicatively to the output value, and the assessor ends up with a poor understanding of the relevance of the model output.

In reality, risk processes are seldom understood well enough for deterministic treatment, which treats exposure conditions as if no variability and uncertainty exists. Usually input variables and processes contain variability and uncertainty. *Variability* represents the heterogeneity in a population, for which there is no single true or correct value, but a range. It is irreducible trough further research. *Uncertainty* represents ignorance about poorly characterised phenomena or model. It can in principle be reduced trough further measurement or research. Because of both variability and uncertainty most of the input values are really variables with a probability distribution of values. (Burmaster and Bloomfield 1996).

Interest in probabilistic exposure assessment methods has increased because they incorporate variability/uncertainty into the risk assessment. Morgan and Henrion (1990)

and Burmaster (1996) present good introductions to probabilistic methods. One simple example is the estimation of the distribution of exposure within a subpopulation visiting a room as a product of the distribution of concentrations in the room, and distribution of times spent in the same room by different members of the subpopulation.

Including also the distributions of individual susceptibilities and dose response models allows expansion from probabilistic exposure assessment to probabilistic risk assessment.

3.1.2 Dose/Response Models

Dose response assessment for exposure to a given contamination aims at establishing a no-observed-adverse-effect-level (NOAEL) or, if such is not available, the lowest-observed-adverse-effect-level (LOAEL). Yet, for acute toxicity and irritation it is in many cases sufficient to derive LD-50 and LC-50 values from animal tests specified in the directive. For mutagenicity, carcinogenicity, skin and respiratory sensitization it is often considered sufficient to determine whether a substance has an inherent capacity to cause such effects.

Epidemiological Models

Sometimes risks (probabilities) in indoor environments can be quite high – examples could be carbon monoxide poisoning from a faulty hot water heater or sensitization to allergens from a contaminated humidifier in a ventilation system. However, more often indoor environmental risks are low, statistical associations between the hazard and the health effects may be weak (yet statistically significant), but still - due to large exposed populations and long exposure times - the accumulated total health effects can be considerable and involve thousands of individuals. Evaluating such weak associations of often nonspecific health effects with low levels of complex mixtures of indoor contaminants commonly found in indoor environment involves a number of methodological difficulties. To detect a weak association reliably one needs large and comparable populations, a large gradient in exposure, and good exposure and health data. In a prospective design, a long follow-up time may be necessary, and with a case-control design a retrospective assessment of exposure may be needed. However, even if the detected associations between the exposure and health are weak (symptom increased by less than 20% in the exposed group) the effects should not be ignored. When millions of individuals are exposed, the health of a large proportion of this group may be affected for a lifetime.

The epidemiological risk assessment process faces two types of error: Random errors, which widen the band of uncertainty and biases, which shift the observed association towards some - often unknown - direction from the truth. In principle the errors are due to the fact that an environmental epidemiologist has very limited control over and imperfect information about the study subjects. The advantage of epidemiological surveys is that they assess real populations in their normal daily activities exposed to real environmental mixtures. An association, observed in a well designed environmental epidemiology study, may contain large uncertainties, but it is definitely relevant for man in his environment.

Usually the measurements are available only from a subgroup selected (randomly) from the studied population, and the inference on the studied association (risk) in all population

must be based on the extrapolation of the estimates derived in the sample. Appropriate design of the epidemiological study and application of statistical techniques allows assessing the magnitude of possible (random) error of the extrapolation of the risk estimate from the studied group to the target population.

An important task of the models used in epidemiology is to control for a possible confounding or effect modification by factors associated with both exposure and the studied health outcome. An obvious requirement for such effects to be controlled is availability of information on the confounders collected together with the data on exposure and health. If that information is available before the study, the design of data collection can take the possible confounding into consideration through an appropriate sample design (e.g. by using stratified sample design).

Risk estimates obtained from epidemiological studies are often used for assessment of risks in populations not covered by such studies. Usually, relative risk estimates, or exposure – response associations, used in such assessment are based on observations and estimates from more than one epidemiological study.

Toxicological Models

Experimental studies are crucial in collecting mechanistic information about the cause and effect, i.e. to establish biological plausibility for an observed association. In principle the issues of uncertainty and bias apply also to experimental toxicological research on laboratory animals. However, such research is conducted in significantly better controlled, closed systems than epidemiological research, making experimental results more precise, repeatable and assignable to the assumed cause for cause - effect conclusions. These benefits are, however, compromised by the facts that experimental research is usually conducted applying artificial exposures (usually short, high and isolated) of homogenous (age, strain, gender) groups of "wrong" species (rats, hamsters). Extrapolation of these experimental results employs a number of uncertainty factors, which are well suited for certain preventive risk assessments, but are rarely precise enough when a dose-response relationship is needed for quantification of risk in a general population. An uncertainty factor of 10 is usually applied for interspecies extrapolation. Controlled experimental studies on humans, when ethically appropriate, are therefore important final steps in risk assessment. However, such studies are not possible when the effects are expected to occur after a long exposure, with a long lag time, or in a selected group of sensitive subjects.

Approaches to establish NOEL or LOEL make use of only one point in the dose-response curve. More modern methodologies have been developed which ensure the use of all dose response information (e.g. benchmark approach) or to incorporate mechanistic data to reduce uncertainties and account variability (biologically based modelling).

The development of biologically-based or physiologically based pharmacokinetic (PBPK) dose-response models is a relatively new area. These models incorporate mechanistic information about chemical disposition, tissue-toxicant interactions, and tissue response into an overall model of pathogenesis (Jarabek 1995a, b) for a given chemical in a given species. The advantage of these models to risk assessment is that as more data on mechanisms become incorporated into an assessment, the need to compensate for lack of knowledge by using large uncertainty factors decreases.

WHO has produced a practical Criteria Document for derivation of health based exposure limits (WHO/IPCS 1994).

Perception Models

Exposure-effect assessments for sensory stimuli do not primarily aim to determine a NOAEL or LOAEL but to describe the full curve of association. As in modern toxicological methodologies, the perception models make use of all exposure effect information. For acute sensory effects such as odour and sensory irritation, exposure-response curves are commonly used for assessing ED50 (effective dose 50) for detection or recognition (ECA 1999).

The effect of exposure can refer to either sensory stimuli or cognitive stimuli. For sensory stimuli perceived intensity increases as a power function of concentration (Stevens 1975) (The general exposure-effect equation for intensity of perceived sensory stimuli is $R=c \cdot C^a$ when "R" denotes perceived intensity, "C" concentration and "c" and "a" are constants).

The perceived intensity-exposure-function is different for different stimuli, including air contaminants. For odours the exponent "a" is smaller than one, which means that the olfactory system attenuates the stimulus at high concentrations. For sensory irritation the exponent "a" is typically larger than one, indicating exponential gain of stimulus with increasing concentration.

Also for cognitive stimuli the exposure-effect relationships follow exponential gain. Cognitive stimuli can be, for example, perceived duration of an adverse event and perceived time lap to a specific event. Also perceptual aspects such as familiarity and predictability of exposure may be used as stress-related cognitive stimuli (Evans 1982).

Perceptual interaction models are well established and models are available for some environmental agents. As with toxicological models the perceptual models incorporate mechanistic data to reduce uncertainty and account for variability. For odours, the interaction of constituent compounds in mixtures is less than additive. This attenuation surpasses that of the power function for single compounds (Berglund and Olsson 1993a, 1993b).

Indoor air is usually perceived in its entirity, and people cannot pinpoint the particular constituents in an air sample by the chemical senses alone. Pattern recognition models present a possibility to classify, from a sensory point of wiev, new intruding chemical constellations in indoor air exposures. Pattern recognition analysis of indoor air samples point out the joint impacts of large numbers of chemical and sensory components that characterise indoor air quality. It is probable that the chemical senses perform a pattern analysis on exposure to a complex mixture of air pollutants (Berglund and Lindvall 1986).

In real life the exposure-effect relationship is complicated because perceptions, like symptoms, may be attributed to irrelevant environmental exposures. Therefore, it is important to distinguishing carefully between symptom attribution and environmental perception (Berglund and Gidlöf Gunnarsson 2000). For environmental perceptions the models mentioned above are pertinent, but for symptom reports there are no models to control for false symptom attributions.

Perception models are especially suited for exposure-effect assessments of individuals.

3.2 RISK COMPARISONS

The purpose of risk assessment is to help in making decisions about how to deal with a variety of hazards. While we are not born with an instinct about what a risk of "one in a million per lifetime" means, simple hazard ranking or more detailed risk comparison is often easier to achieve and more practical than full quantitative risk assessments. It is particularly helpful if we can compare risks that are calculated in a similar way and measured with the same "yardstick". Another common procedure is to compare exposures only. In some cases it is also useful to contrast risks to highlight the different ways in which they are treated in society. (Wilson and Crouch 1987)

More than two risks can be compared to each other through risk ranking. Risks may be ranked by using risk statistics (only available for common clear-cut risks like home accidents or cigarette smoking), by using animal toxicity or carcinogenicity data (which may not provide appropriate ranking for humans (Ames *et al.* 1987)), or by professional judgement of a panel of experts (Slovic *et al.* 1980).

One example of hazard ranking, based on professional and lay persons' judgements for inputs into a predefined logical construct, is presented in Raw *et al.* (1995), which produced the following rank order of hazards in homes in the U.K. (only physical, chemical and biological listed here):

1.	Hygrothermal conditions
2.	Radon
7.	House dust mites
8.	Environmental tobacco smoke
12.	Carbon monoxide
14.	Fungal growth
	etc.

"Risk ladders", often based on a combination of statistics and professional judgement, compare quantitative probabilities of similar risks, usually premature death. Such risk ladders have been presented for some transportation and environmental hazards, including also indoor hazards.

Risk comparison can be relatively straightforward when comparing well-known risks and similar outcomes, such as lung cancer risk from ETS, radon and asbestos exposure. Such a risk comparison is, however, rare luxury. Risk comparison is most needed and also often most controversial when comparing different risks of competing or alternative products or techniques.

Risk comparison - to be meaningful - should also use background data of comparable quality, or at least clarify the major differences and their possible consequences for the risk assessment. The universal "yardsticks" that have been used in such comparisons include cases of death or disease, money and lost workdays. The most straightforward looking, yet most problematic of these units are cases of death and money. The raw cases of death do not make a difference between the death of a paralysed 90-year-old, a 30 year old working

mother, and a 10 year old schoolboy. DALY (Disability Adjusted Life Years), and QALY (Quality ...) units correct for this discrepancy, but being less straightforward than simple death counts they are rarely applied. The most obvious costs and benefits (e.g. in buying a floor covering) may be easy to estimate, but they may be marginal compared to longer term costs (e.g. ventilation needs and absenteeism due to irritation symptoms) or indirect costs (e.g. injuries due to slippery floor) and benefits (e.g. cleaning benefits).

Comparison of dissimilar risks (like accidents from one slippery carpet vs. sensory stress from irritating VOCs of another carpet) should make the best in utilising any relevant and straightforward comparison units. Comparisons should also provide the risk managers and lay people with additional information - such as uncertainties, weights of evidence, conditions of analysis, and other factors restricting comparability - in a form which allows them to make as informed and responsible selections as possible. In risk assessment simply incomparable qualities should be described but not compared. It is the task of risk manager to do the decisions based on best available information.

Even qualitatively similar risks can be difficult to compare. While the range of uncertainty in risk assessment is relatively small for well known risks like cigarette smoking or high levels of carbon monoxide or radon in indoor air, the range is much broader for less understood human risks like those from low level exposures to VOCs in indoor air. Risk comparisons based on the high ends of the probability distribution (of exposure variability, recipient susceptibility and data uncertainty) may overestimate a little known risk by orders of magnitude in relation to a well known risk, making such a comparison outright misleading. Paucity or abundance of data about a hazard should clearly not in itself be a reason for assigning a low or high priority to that hazard.

Because different chemicals exhibit different dose-response curves (e.g. sigmoid vs. linear, with and without threshold, etc.) it is essential that risk comparison is based on realistic and probable exposures. Just the simple looking task of adding lifetime risks from compounds in a mixture can be very complicated when the harms of the respective compounds are different and their risks have different dose-response curves.

Crawford and Wilson (1996) suggest that in the final analysis comparing and adding low level risks may not turn out to be so complicated, after all. If a pollutant produces cancer via the same mechanism by which background cancers occur, then there results a linear incremental response to the incremental dose (Crump *et al.* 1976 and Guess *et al.* 1977). Even if the biological dose response mechanism has a threshold, or is non-linear, the existence of background cancers shows that the threshold is already exceeded by some background agent. Then, when a small amount of another agent operating in the same way as the first, is added, an incremental response linear with the incremental dose can result almost independently of the particular biological mechanism relating dose and response. This may also be true for non-cancer endpoints. The requirements are that (i) there exists a reasonably large background of the biological effect under consideration, and (ii) the pollutant acts in the same way as the background. It is evident that this might be satisfied by a large number of biological effects and pollutants. The generality of the argument suggests that linear dose-response relationship may be the rule, rather than the exception, at the low doses typical of exposures to indoor pollutants.

3.3 COMMON DENOMINATORS OF EFFECT

To reduce the difficulties of comparing very different risks, common denominators of effect have been searched for and defined when possible. The first such effect denominator is cases of death, or more accurately DALYs or QALYs. Others include cases of cancer (practised) or sensitisation (suggested), which would allow bringing all carcinogenic or allergenic substances and exposures on the same scale. For exposure assessment development of common denominators would facilitate comparisons of dissimilar load and risk factors. At present such denominators only exist for mutagenecity (poor qualitative value), and intensity of odour and mucosal irritation. Thus it is not yet possible with respect to the majority of the non-carcinogenic substances in the indoor air to express and measure their exposures in easily comparable ways.

However, an issue that needs to be discussed first is:

Individuals and Populations

Increasing requirements for energy conservation, safety of indoor environments, and economy in construction and building operation lead to narrow operating margins. This raises the need for more careful definition of the risks to be consider as well as the target population. This holds for buildings in general and for specific buildings, such as schools, offices, etc.

The target population in designing industrial occupational environments is usually non-pregnant, healthy adults between 20 and 65 years of age, not exposed outside of the working hours. The reference population for indoor air quality guidelines have not been specifically defined, because it has been assumed to be the general population with uninterrupted exposure. In practical considerations excessively sensitive individuals have been excluded from this general population. This exclusion may need to be reconsidered or at least defined more clearly, because - for example in Sweden - the fraction of the young population, which is atopic and predisposed to allergies and asthma, is already 5-7% and appears to be increasing about 0.2% per year. Equally exposed to indoor environment, individuals show large differences in susceptibility. In indoor air risk assessment, since no "standard human" exists, the difficulty is to deal with a juxtaposition of exceptions and particular cases. The roles of children, elderly, sensitive, health compromised, special social groups etc. need to be highlighted, when defining the reference populations.

Most specific risk assessments deal with population risks, i.e. risks that would materialise in a sufficiently large population consisting of individuals of different ages, predispositions, socioeconomic and health status, and exposed to other unspecified risks in a way which is comparable to the general population. The risks are expressed as expected excess cases of disease or death within this population, or as probabilities, e.g. $3 * 10^{-4}$ per year. In principle such probabilities have a meaning - i.e. the validity of the statement can be tested - only at population level. Individual risk depends strongly on other exposures and host factors such as genetic predisposition, atopy, immune status, and personal behaviour.

Absolute and Relative Risk Models

In principle a health risk due to an exposure may be expressed as absolute or relative to some background condition. A relative risk expression is applicable when the added risk is

known or assumed to be proportional to pre-existing background risk, (e.g. the added risk of lung cancer from radon or asbestos exposure, which is much higher for smokers than non-smokers). A relative risk is presented as a percent (25%) or fractional (1.25) increase over background. An absolute risk expression is applicable to risks, where either no background risk exists (e.g. mesothelioma risk from asbestos exposure), or the added risk is assumed to be independent of this background risk. An absolute risk is presented as a number of cases per year (or sometimes lifetime) per number of individuals caused by a unit exposure (e.g. 100 (#/year) /10^6 • (mg/m^3)). To complicate the matters further, results of a relative risk model may, for a defined population, be expressed in absolute risk units for easier comprehension (e.g. 150 – 450 cases of lung cancer per year from radon in Finland).

3.3.1 Cancer

The International Agency for Research on Cancer (IARC) developed in the 1970s a systematic approach for cancer risk assessment of (mostly) individual compounds. This approach is based on a systematic compilation of existing data about sources of exposure, measured exposure levels, epidemiological and toxicological human data, experimental animal data, bioassay genotoxicity data and any other relevant data. These compilations are then critically reviewed and analysed by a panel of selected experts, who come up with a panel judgement as to the carcinogenicity class of the compound (or a mixture). This classification is not based on carcinogenic potency or quantitative estimation of risk to any population, but instead on the strength of evidence - i.e. IARC has evaluated toxicological properties of chemicals, not health risks of exposures.

IARC has until 1999 classified 836 agents, mixtures and exposures for carcinogenicity in the following way:

- Group 1. Proven human carcinogens 75
- Group 2A. Probable human carcinogens 59
- Group 2B. Possible human carcinogens 227

Group 3 is for agents, which are not classifiable, and group 4 for agents which are probably not carcinogenic to humans. (IARC 1999)

EU classification of carcinogens (CEC 1993b) has similarities with that of IARC. Category 1 contains substances known to be carcinogenic to man, category 2 substances which should be regarded as if they were carcinogenic to man, and category 3 substances which cause concern for man for possible carcinogenic effect, but the available information does not allow satisfactory assessment (ECA 1997). It is interesting to note that while the IARC approach keeps a clear distance to practical decision making, EU classification is more oriented towards the needs of administration.

The risk associated with lifetime exposure to a certain concentration of a carcinogen in the air – the unit risk - has generally been estimated by linear extrapolation from an observed risk at high exposure. The carcinogenic potency is usually expressed as an incremental unit risk estimate, i.e. as additional lifetime cancer risk occurring in a hypothetical population in which all individuals are exposed through life to a concentration of 1 $\mu g/m^3$ of the agent in the air they breathe.

Unit risk estimates allow comparison of carcinogenic potencies of different agents, and avoid of any reference to the acceptability of risk. The decision about the acceptability of a risk should be made by national authorities in the framework of risk management.

3.3.2 Allergy

The European Commission has developed criteria for classification of skin and respiratory sensitisers on the basis of the properties of the chemicals (European Commission Directive 96/54/EEC).

Respiratory sensitisers are classified on the basis of

- evidence that the substance can induce specific respiratory hypersensitivity
- or positive results from an appropriate animal test(s)
- specific provisions for isocyanates.

Skin sensitisers are classified on the basis of

- practical experience showing the substance or preparation to be capable of inducing sensitisation by skin contact in a substantial number of persons
- or positive results from an appropriate animal test.

WHO has developed a classification of skin and airway sensitisers in testing substances that may be used e.g. in indoor environments (Flyvholm *et al.* 1997). The scheme is similar to the one IARC has developed for cancer.

Specific hypersensitivity risk assessment involves a qualitative (classification) assessment, based on the weight of evidence of how sure we are that the substance is a human sensitiser. By sensitiser is here meant a substance or preparation which, if it is inhaled or penetrates the skin, is capable of inducing specific hypersensitivity such that on further exposure characteristic adverse effects are produced. Specific hypersensitivity may be caused by allergic sensitisation or by some uncertain mechanism.

The decision to consider a substance as a sensitiser is based on the qualitative evaluation of all available information on sensitisation, ensuring that the association is unlikely to be due to chance alone.

For airway-sensitising substances:

Class I includes substances which are classified as inducers of specific airway hypersensitivity because there is sufficient human evidence present. Animal or other evidence may be present or absent. In principle, sufficient human evidence can be obtained only by specific provocation testing.

Class II includes substances deemed to be probable inducers of specific airway hypersensitivity for which, at one extreme, the evidence of sensitisation in humans is almost sufficient (limited evidence), as well as substances for which, at the other extreme, there are no human data, but for which there is experimental evidence of sensitisation.

Class III includes non classifiable substances for which there are no interpretable data available.

To be classified as not an inducer of specific airway hypersensitivity, data shall exist which suggest that the chemical substance does not cause specific hypersensitivity.

A classification is also given for the potency of substances to induce airway hypersensitivity. A material has;

1. a **very strong risk potential** when it can cause airway hypersensitivity in a substantial part of the exposed population (e.g, 30%),

2. a **strong risk potential** when it more than occasionally can cause airway hypersensitivity in an exposed population (e.g. 5%), and

3. a **weak risk potential** when it happens just occasionally.

This classification is based on the strength of evidence - i.e. WHO is aiming at evaluating sensitising properties of chemicals, not health risks of exposures. Prevention of allergy/hypersensitivity caused by chemicals is the primary objective. The classification system provides a scientific basis for compiling accepted lists of substances causing allergy/specific hypersensitivity in order to facilitate risk management and assist in priority setting. However, additional information is also required on potency and the nature and extent of exposure.

However, no substances have been systematically classified according to this scheme. Consequently no lists of classified allergens can be provided at the time of this report.

3.4 Systematic approaches to Risk Assessment

The conceptual structures, both unintentional and carefully developed, used in assessing risks, are logical models that help in unifying risk assessment procedures, improving comparability and repeatability of risk assessments, and linking risk assessment to risk management and risk communication.

In order to produce repeatable and comparable risk assessments, selection of a systematic approach or paradigm is needed. The first such approaches were used in assessing risks in and around nuclear facilities, and they were based on the most exposed individual or worst case scenarios.

A modern risk assessment paradigm is based on either the organisation of information compilation and management in the *administrative risk assessment procedure* (i.e. NAS/NRC 1983) or it may be based on modelling *the flow of molecules from the emission source to the environment, exposure and target organ* (Covello and Merkhover 1993). These approaches are in no way contradictory, they are complementary.

3.4.1 NAS/NRC 1983

The U.S.EPA announced its first cancer risk assessment guidelines in 1976, but "they lacked the intellectual construct that could serve to frame the thinking and discussion about risks" (Barnes 1994). A new and comprehensive conceptual model or scheme for risk assessment was published by the U.S. National Academy of Sciences in 1983, known generally as the "NAS (1983) Paradigm". 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. The risk assessment process was divided into four steps, namely:

* **Hazard identification**	- Is this chemical harmful?
* **Dose-response Assessment**	- How bad is it?
* **Exposure Assessment**	- Who is exposed? What levels? How long?
* **Risk Characterisation**	- How does the matter now look like?

Originally NAS 1983 paradigm set a strict order (above) for these steps, and also separates risk assessment from risk management, so that risk management would follow from a completed risk assessment.

The NAS Paradigm has finally succeeded in setting a widely agreed framework for risk assessment / risk management, and it has had broad applications from carcinogenic compounds to noncarcinogens, mixtures, radiations and other situations. **Figure 2** presents the logical framework of the NAS 1983 risk assessment model and a comparison to another model by Covello and Merkhover (1993) (see next section).

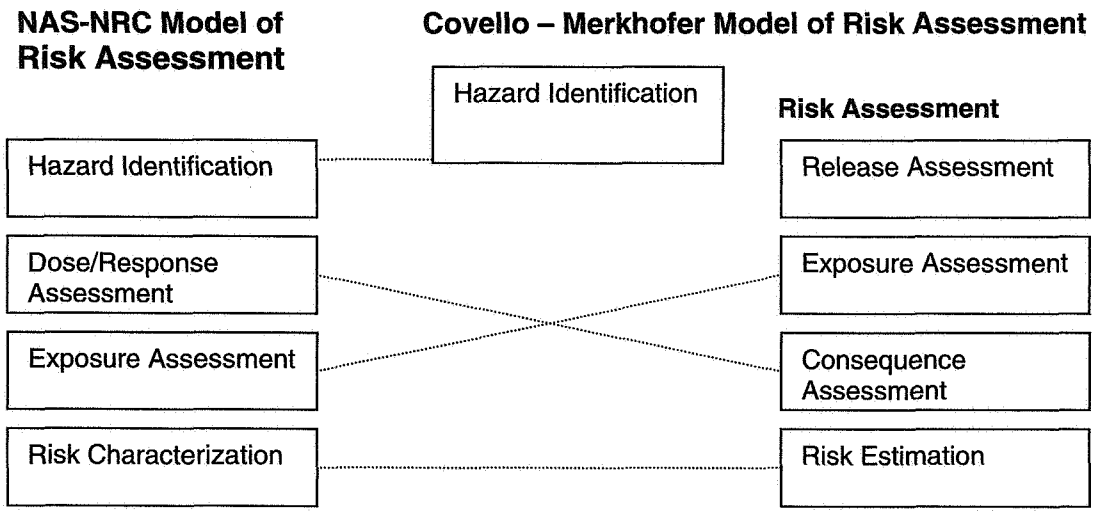

Figure 2. *The structure of the NAS/NRC 1983 risk assessment model compared to another model by Covello and Merkhover, 1993. The structure of the NAS model is defined by policy needs, while the Covello-Merkhover model is more scientifically constructed.*

An example of risk assessment based on this approach is ECA (1997) Report "Evaluation of VOC Emissions from Building Materials (Solid Flooring Materials)", which presents a thoroughly evaluated risk assessment case and highlights the complexity and amount of work which is needed for such an assessment.

However, the wide application of the NAS paradigm has also exposed some basic weaknesses in it. Total separation of risk assessment and risk management does not exist in real life, because it is not possible or even desirable in many cases. Instead of risk management following from risk assessments, risk assessment has been often explicitly guided by the gaps of information identified in the needs of risk management. The NAS paradigm also favours the more manageable single chemical (e.g. asbestos or dioxin) approach over consideration of more realistic exposures to dynamic mixtures (e.g. traffic exhaust, mouldy building). The NAS approach does not address adversity of the effect, and therefore helps little in comparison of multiple effects and different effects. While science (Risk Assessment) refuses to compare apples and oranges - real life (Risk Management) has to base some tough decisions on such comparisons.

3.4.2 Covello-Merkhover 1993

The Covello and Merkhover (1993) risk assessment framework addresses a problem in the NAS paradigm, namely the role of hazard identification. Should hazard identification relate to effects observed in the experimental setups in the laboratory, or to exposures likely in the field? While the flow diagram of the NAS paradigm follows from an administrative viewpoint, the flow diagram of the Covello-Merkhover approach for risk assessment is based on the causal flow of events from sources of pollutant to exposure, health consequences and scientific conclusions. Hazard identification can happen at any point of this flow or outside of it in the laboratory, and it is considered as external in the Covello-Merkhover approach.

The Covello-Merkhover approach is particularly suitable for risk assessment where the development of risk is followed according to a scientific causal model from source to health outcome, such as diesel exhaust from tailpipe to ambient air, lungs and target organs. It does not apply as directly into cases where the risk assessment focus is a sensitive subgroup (e.g. asthmatics) a particular environment (e.g. a sick building), or disease (e.g. hypersensitivity).

3.4.3 The European "Commission Directive 93/67/EEC"

This directive "lays 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)". These principles as well as the terminology of this Directive are quite similar to those of NAS/NRC 1983. The directive defines that "the assessment of risks should be based on a comparison of the potential adverse effects of a substance with the reasonably foreseeable exposure of man", and "the assessment of risks to man should take account of the physico-chemical and toxicological properties of a substance."

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 characterisation' as the summary. Their roles and relations to each other and also to 'risk management' are presented in **Figure 3.**

Unlike the original NAS/NRC 1983, in this EC framework hazard identification, dose-response assessment and exposure assessment may all be connected with each other and with risk characterisation. Also the connection between risk assessment and risk management can be bidirectional. In practice these changes allow for risk assessment to follow from specific needs of risk management. It also allows risk management to be based on e.g. hazard identification alone. Such a case could be the prohibition of the use of an identified carcinogen (e.g. asbestos) in building products without requiring comprehensive dose-response and exposure assessments.

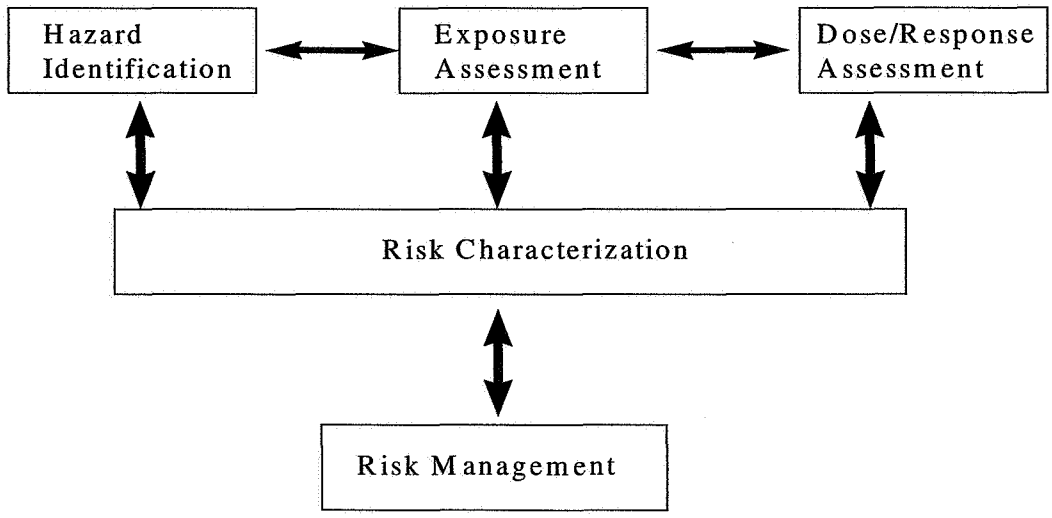

Figure 3. *The EU risk assessment framework (Commission Directive 93/67/EEC).*

3.4.4 Comparison of the three approaches

The above mentioned most commonly accepted and used risk assessment frameworks are compared and summarised in **Table 2.** The framework set by Commission Directive 93/67/EEC follows closely the NAS/NRC 1983 principles. The Covello-Merkhover framework differs from the two in that it separates hazard identification from the risk assessment. It is considered as a independent step that must precede risk assessment.

3.4.5 Beyond the traditional Risk Pathway

All the above mentioned risk assessment paradigms and frameworks are strong in analysing physico-chemical and toxicological pathways for contaminant specific health outcomes when information is available on exposure and dose response relationships and

the effect is clear and measurable. (e.g. lung cancer from radon, aspergillosis from mould). They are much weaker in dealing with essentially non-specific irritation and stress symptoms with low severity and high probability, which are caused by irritating chemicals (e.g. a mixture of VOC_s) in previously sensitised (e.g. dust mite exposure) tissues and the perception of risk caused by sensory perception of potentially harmful exposures (ETS) or alarming information. Both irritation and risk perception can cause health consequences, which, although not specific to any chemical (but instead to irritation and stress), are causally linked to emissions of harmful pollutants. Such effects are often more host than agent dependent, but this fact in no way negates the causal role of the agent or source.

Table 2. *The main steps of the three most common risk assessment frameworks.*

	Commission Directive 93/67/EEC 1993	**NAS/NRC 1983**	**Covello-Merkhover 1993**
Hazard Identification	identification of the adverse effects which a substance has an inherent capacity to cause	determination of whether a chemical adversely affects human health	*actually external to the model* identifying the risk agents and the conditions under which they potentially produce adverse consequences
Release Assessment	External	External	quantifying the potential of a risk source to introduce risk agents into the environment
Dose-response Assessment	estimation of the relationship between dose, or level of exposure to a substance, and the incidence and severity of an effect	determination of the relationship between the level of exposure and the probability of occurrence of adverse effects	**Consequence Assessment** quantifying the relationship between exposures to risk agents and health consequences
Exposure Assessment	determination of the emissions, pathways and rates of movement of a substance and its transformation or degradation in order to estimate the con-centrations/doses to which human populations are or may be exposed	determination of the extent of exposure	quantifying the exposures to risk agents resulting under specified release conditions
Risk Characterization	estimation of the incidence and severity of the adverse effects likely to occur in a human population or environ-mental compartment due to actual or predicted exposure to a substance, and may include risk estimation, i.e. the quantification of that likelihood	description of the nature and often the magnitude of risk, including the accompanying uncertainty	**Risk Estimation** estimating the likelihood, timing, nature, and magnitude of adverse consequences

In order to cover all relevant exposure - health sequences in complex environmental settings a more holistic approach is needed, where all the different causal chains from pollution sources to different health outcomes are considered, described, analysed and discussed, and summing of the results is done with caution, see **Figure 4.**

Constructing a risk assessment model - that will combine the toxicological, sensory-irritation and psycho-social causal chains leading from a source of contamination to the agent, irritation and stress specific health effects - is not as difficult as one might think. **Figure 4** presents such a multidisciplinary construct of all causal links from (indoor) pollution source to various health outcomes, where all links can be subjected to scientific analysis.

This approach has, however, not yet been tested in practice. For such a test a problem where many of these links would be active and significant would be most useful. A multidisciplinary research team is needed to carry out the assessment.

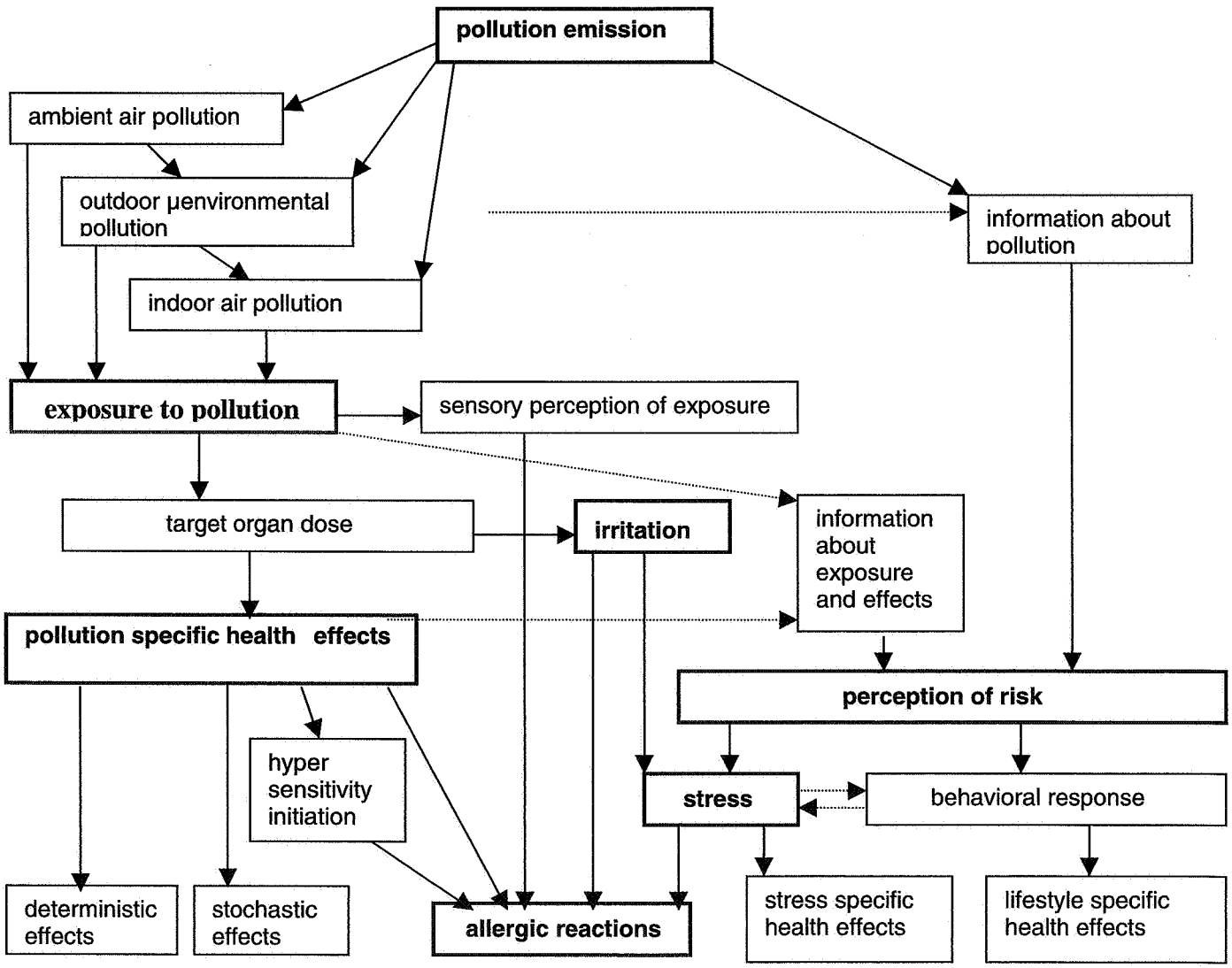

Figure 4 *A multidisciplinary construct of the different types of causal links from pollution sources to various health outcomes (Jantunen 1998, unpublished).*

4 IAQ RISK ASSESSMENT FRAMEWORK

The selected framework (illustrated in **Figure 3**) considers hazard identification, exposure assessment and exposure/dose-response assessment as parallel steps. Important modification is the interaction between stages. Steps do not necessarily follow each other in a time sequence, but can be done simultaneously or findings may direct back to stage that has been carried out earlier. Furthermore, the selected framework accepts a close connection between risk assessment and risk management. There is or should not only be one-way flow of information from risk assessment to risk management but risk management can and should drive the risk assessment process.

The following definitions will be adopted for the purpose of this report (Commission Directive 93/67/EEC).

Hazard identification - Identification of the adverse effects which a substance has an inherent capacity to cause.

Dose/Response assessment - Estimation of the relationship between the dose or level of exposure, and the incidence and severity of an effect.

Exposure assessment - Determination of the emissions, pathways and transformations in order to estimate the concentrations/doses to which humans are or may be exposed

Risk characterisation - Estimation of the incidence and severity of the adverse effects. This may be accompanied with quantification of the likelihood of the detriment occurring and qualitative or quantitative estimation of uncertainties.

Risk Management - Identification, selection and implementation of risk management alternatives.

The following discussion covers these risk assessment and risk management terms in the contents of indoor air risk assessment.

4.1 Hazard Identification

Indoor air hazard identification may be based on any finding or observation, within or outside of the building under concern, which raises a concern about potential risk of indoor air on human health. (i) Presence of an indoor source (e.g. unvented gas heater), which is capable of producing harmful releases, (ii) detection of a chemical/agent in the air, which is known to be hazardous to health at least at high concentrations (benzene vapour, asbestos fibres, *stachybotrys* spores), (iii) detection of construction defects (water pool against the wall after rain) or a malfunctioning building system (failing ventilation air blower), or (iv) observation of risky occupant activities (leaving a car engine running in an attached garage) may all identify a risk that warrants further investigation and possibly remedial action.

A strong case for hazard identification is any observation of uncommonly high and persistent levels of symptoms and complaints or disease within the occupants. This is especially pertinent when these are consistent across the occupants, timing (onset after entering the building and offset after leaving the building) links the symptoms to exposure in a specific building or space within it, and a biologically plausible and technically logical link between a source or agent and the symptoms exists.

A different viewpoint for hazard identification is the presence in a building of special host factors, such as individuals with immunosuppressive medication or disease, newly born babies or asthmatics. Such a hazard identification does not need to stem from the presence of any identified pollution sources but from the presence of highly susceptible individuals (e.g. allergics) or groups (e.g. immunosuppressed patients) who may not be protected by otherwise sufficient air quality.

4.2 Dose /Response Assessment

The title "dose/response assessment" relates to experimental studies, where doses are administered or otherwise accurately known. Exposure/response assessment is often used as a proxy for this, because most of the human data are based on estimation of exposure rather than dose.

The needs and sources of exposure/dose-response assessments for indoor air pollutants are not significantly different from those of other health risk assessments. Most of the dose/response data come from extrapolations from experimental studies on single chemicals/agents in laboratory settings. Human exposure/response data can be obtained from extrapolations from epidemiological and toxicological studies in occupational medicine, and interpretations from outdoor air epidemiological and building epidemiological studies

Biomarkers of exposure (e.g. cotinine in urine resulting from tobacco smoke exposure) or effects (e.g. elevated IgE due to immune system reaction to a specific microbial exposure) may sometimes be useful in establishing dose-response relationships. However it is then necessary to understand that biomarkers provide only quite limited information about the time of exposure and often no information at all about the exposure medium, route or location.

While dose-response data are often available from experimental animals or from healthy workers, we would really needed dose-response data from susceptible subgroups, such as babies, asthmatics or individuals allergic to e.g. cat dander, because they may suffer from symptoms at orders of magnitude lower exposure levels than the general population. Due to ethical restrictions, such data can almost only be generated in carefully focused and implemented epidemiological studies.

Indoor air is a mixture of outdoor air and pollutants from indoor sources. Exposures indoors consist therefore even more than outdoors from mixtures of interacting compounds, including specific indoor combinations of ETS, CO, gas burner effluents, microbiologicals, radon, etc. Understanding the dose response behaviour of such mixtures proportions is poorly developed - and at the same time quite important for quantitative indoor air risk assessment. (Jantunen *et al.* 1997)

4.3 Exposure Assessment

Direct exposure measurement is invasive, expensive and consequently a rare luxury in indoor air exposure assessment. Because most individuals spend much of their time in the same indoor space (home, office), which is usually well mixed within less than an hour,

microenvironmental measurement in this space approximates closely personal exposure in the same space as long as the individual and his activity is not the source.

When the source of concern is known and the source strength and ventilation rate are also known, deterministic dilution models can be used to compute indoor air levels for a room. The advantage of exposure modelling vs. measurement is that past and future concentrations for different scenarios can be computed, and that with sufficient background information from sources, ventilation and time activity patterns, exposures within a building can be computed for large numbers of individuals. Such models, however, are simplifications of reality. Usually they do not take into account the variable air exchange effects of wind pressure, open and closed doors or varying air pressure gradients around the building, the unaccounted sources, the sink effects of many building materials and furnishings and the memory effects from absorption/resorption phenomena. Therefore the models need to be validated against measured data, and their best application is expanding the information from a limited short-time/small-sample data to cover past, future time and a large population. Instead of measured or modelled exposure data, different levels of exposure proxies are much simpler and cheaper to apply - but also much less accurate. An advanced exposure proxy is the use of data found in literature for a similar setting, preferably same city, e.g. to assess the impact of the presence or absence of gas stove in the kitchen (from questionnaire) on NO_2 exposures of children. The simplest NO_2 exposure proxy is to use the presence or absence of a source (gas stove) without assigning any concentration values. A yet further simplification is to use a proxy of indoor air pollution without assigning either concentration or compounds, like when using damp building or presence of a smoker as a proxy (or label) of exposure in an epidemiological study. Yet, it is important to realise that the cruder the exposure assessment, the more overlap of the true exposure distributions of the "exposed" and "non-exposed", and the smaller the probability of detecting an effect.

The stable and uniform indoor temperatures, rather constant and stationary indoor sources and ventilation systems, and virtual absence of indoor meteorology cause fundamental differences between pollution concentration distributions indoors vs. outdoors.

4.4 Risk Characterization

The most significant differences between indoor and outdoor air risk characterisation are (i) the multitude and great variability of the indoor environments and (ii) the fact that most people spend in excess of 90% of their time in mostly same indoor environments. Consequently exposures to air pollutants from indoor sources have higher between individual and smaller within individual differences than exposures to air pollutants from outdoor sources. Also, air pollution exposures - also for outdoor air pollutants - are almost always dominated by exposures indoors.

Special emphasis should be given to identifying the settings where highly susceptible recipients and high exposures overlap, and to characterising the exposures and risks of this subgroup.

While most indoor environments are occupied by the same individuals for the same hours every day, some indoor environments (ice arenas, barbershops) are only visited voluntarily, irregularly and for short periods. Indoor air risk characterisations for resurfacing machine

operators in an ice arena and for ice hockey spectators are two completely different issues, although the source, the building and the indoor air are the same.

Indoor air risk characterisation for a building, its occupants and even a specified agent should involve very different considerations if the exposure is limited to the time when the chemical is applied, as in painting or gluing, compared to longer term exposure commitment, like when selecting wall and insulation materials.

Risk characterisation should provide risk management carefully evaluated information about the voluntary/involuntary nature of the exposure, about the possibilities of the exposed to identify exposure and protect themselves, about the frequencies and time allocations of different population groups to exposures and future exposure time commitments linked to different alternatives.

5 AFTER RISK ASSESSMENT

5.1 Risk Management

Risk assessment disentangles an identified risk complex (e.g. cancer associated with radon in buildings) into its details (e.g. the transport of radon from soil into the building, deposition of radon daughters in the human respiratory tract, epidemiological determination of the dose response, etc.), and analyses these details one by one and in relation to each other. Risk management must entangle these details back again into an efficient and enforceable policy to reduce the risk (e.g. integrating radon safety requirements in the building code, or radon survey and mitigation programme for the existing building stock)

While risk assessment should be a scientific exercise, risk management by necessity involves also technical, economic, social and legal realities. Risk management decisions concerning a specific risk are based on one hand on risk characterisation and on the other hand on the process that generates and evaluates the policy options to reduce the risk.

The issue most specific for indoor air risk management becomes obvious when one visualises the millions of indoor environments in just one city, and the multitudes of potentially hazardous situations in them. A vast majority of both indoor air risk observations (assessments) as well as decisions (management) will remain to be made by occupants themselves. No imaginable public service or professional expert resources can ever cover but a small fraction of all hazardous IAQ situations. At best these are regulations that protect the population at large, or representative and generalisable examples for the rest of the cases. From this follows that at a national or European level risk communication is the most crucial single component of indoor air risk management, much more so than for ambient air risk management. Most of the day to day risks can only be prevented or managed by occupants themselves (See 5.2 Risk Communication).

The largest investment of most individuals is their home, and buildings also represent in the order of 2/3 of national property in most European countries. This fact sets very specific requirements for any interventions to the building stock, as well as for the allocation of financial burdens and benefits. The fact that most indoor environments are private property and protected by privacy of the individuals significantly limits the options of public indoor air risk management. On the other hand, when the same individual both pays for and benefits from managing of the IAQ risks, properly informed *market forces* should produce optimal protection. The role of the public service and experts is then to provide the owners and occupants, buyers and sellers correct, reliable and relevant information about the costs and benefits of alternative technologies and materials.

The capabilities of public services and experts to manage most indoor air risks lie crucially on their abilities to establish and maintain public *confidence,* to explain the risks in a way that the public is willing and able to comprehend, and to present practical solutions which are technically, financially and legally feasible, and which sufficiently acknowledge the diversity of the buildings, the occupants and the risks.

The public services have quite different responsibilities when managing risks of rental apartments, hotels, public buildings, workplaces, schools, hospitals, shopping malls etc. These

buildings should be ensured to be safe for a great majority of the population (excluding only exceptionally sensitive individuals) by public health, occupational health and building inspection authorities as the last resort. When credible concerns arise they should be addressed promptly and properly for the sake of public health, confidence and liability. One of the most important special tasks for the public services and experts is identification, assessment and management of the situations where susceptible population groups and risky indoor environments coincide.

The core of ambient air risk management is in the ambient air quality guidelines and standards. The ambient air concentration limits have questionable applicability for indoor environments, because they are derived for different pollution mixtures and exposure patterns, comparable monitoring and implementation alternatives do not exist indoors, and besides indoor air quality for an individual may depend strongly on the behaviour of the same individual. When ambient air quality standards are applied for indoor air quality, short term (24 h - 1 wk) measurements of indoor pollutants should usually be related to long term (annual) outdoor air guidelines.

The occupational exposure limits (OEL, TLV, MAK, etc.), although derived for indoor environments, are not directly applicable either, because they have been developed for healthy adult populations and controlled exposures.

Risk managers can intervene in many points (that lead from the primary source of risk to its materialisation). They can prevent the hazardous process, reduce exposures, modify effects, alter perceptions or valuations through education and public relations or compensate for damage after the fact. (Morgan 1993). Specific indoor air risk management options are source removal or modification, and dilution by ventilation. General options are administrative regulations and voluntary measures for approval or labelling of building materials and chemicals, ventilation systems, building practices, building maintenance and inspections, and indoor air quality.

Risk-benefit Analysis: In managing risks from indoor air pollutants, there is a case for erring on the side of caution and taking some action to reduce risks if there is any suspicion that a hazard exists. This gives human health and environmental quality the benefit of any doubt that exists in the scientific evidence. However:

- any chemical can be hazardous at sufficient concentration, and yet may not be desirable to reduce all concentrations to zero;
- precaution may divert resources into too many actions, and lead to sub-optimal protection of health and/or the environment. It may also unjustifiably deny the benefits of using a chemical, product or technique; and
- limiting the use of one chemical may entail the application of substitutes that have less well-characterised risks.

Hence, legislation in Europe has moved towards approaches based on overall risk rather than mere hazard identification. Article 10 of the Council Regulation (EEC) 793/93 (Existing Substances Regulation) requires that: "Where such control measures include recommendations for restrictions on the marketing and use of the substance in question the rapporteur" [on behalf of the body carrying out the risk assessment] "shall submit an *analysis of the advantages and drawbacks* and the availability and replacement substances".

5.2 Risk Communication

Buildings are built for shelters from the cold (or heat), wind and rain of the outdoor air. As long as people feel that they are in control of their own indoor air quality there is usually a higher tolerance to poor indoor air quality compared to outdoor air quality - after all one can always go outdoors or open a door or window. This tolerance is greatly decreased if these elements of self control are removed. Typically indoor air also contains higher levels of most air pollutants than outdoor air.

Risk communication is closely linked to risk management. Successful communication will not raise undue concerns, but suggests changes in the acts of the decision makers and/or behariour of vulnerable individuals towards a direction that effectively reduces avoidable risks, yet avoids nonessential interference into the lives of the individuals or to the choices of the enterprises. Poor risk communication may not only fail to produce the wanted risk reduction, it may also unnecessarily limit individuals' options, add public and private costs, and generate new risks due to nonproductive public concerns and potentially harmful behavioural changes.

The essence of good risk communication is simple: Learn what people already believe, tailor the language of your communication to this knowledge and to the decisions people face and then subject the resulting message to careful empirical evaluation and editing before distribution to the public at large.

The background information that needs to be collected prior to any indoor air risk communication campaign includes (in addition to risk assessment conclusions and risk management decisions) assessment of public's perceptions and understanding of the problem, consideration of the implications of intervention into private properties and lives, legal liabilities, and feasibility of the solutions.

With this information at hand, the message(s) must be developed and its practical necessity, scientific correctness, general comprehensibility and implications for field assessments and solutions need to be carefully tested before further distribution.

Increased (decreased) *trust* in the managing organisation of the activity under concern will lead to lower (higher) levels of perceived risk and thus increase public support (opposition) for the activity. Understanding the dimensions of trust is essential for developing policy strategies that will gain public acceptance. (Flynn *et al.* 1992)

Economically and technically sound policies may be doomed if people believe that they distribute benefits and burdens unfairly. Three concepts emerge from the philosophical and cultural basis of risk sharing: *parity,* in which each individual, group or country is treated equally; *priority,* or giving the burden to those most deserving of it; and *proportionality*, or the sharing of burdens according to need or contribution. (IIASA 1993)

The selection of the means of communicating to and with the affected public then depends on the estimated size of the target population and the availability of expert resources. The ideal situation of two way face to face communication must usually be limited to problems involving single buildings (sick office tower) or small areas ("Love Canal"). Such communication allows for *in situ* evaluation of the correct transmission of the message and therefore reduces the requirements for message preparation and enables rapid response to

local concerns. Nationwide indoor air risk communication campaigns usually need to maximise the use of public media and information material distribution combined with short term training of local responsible authorities and professional groups. Correcting, rewording or amending a message that has already been so distributed is difficult and effectively erodes public confidence. Careful development and testing of the message and its distribution is therefore essential for the success of any such campaign, and by necessity increases the time lag between problem identification and public authorities' response.

For different reasons both face to face and media communication requires almost always more working hours than is expected and allocated.

6 DISCUSSION and CONCLUSIONS

The goal of a risk assessment is to provide measures of risks, which contain the probabilities and severities of the possible adverse outcomes, the uncertainties involved, and which allow a maximum degree of comparability between different hazards and risks. Risk assessment is an essential input to risk management of IAQ, which is an organized effort to collect information about and to control risks related to indoor air quality.

The following **Discussion** addressees separately the six goals that were set in the **Scope of this Report.**

6.1 Why specific IAQ risk assessment are needed.

The sensitivities to hazards are not the same for all citizens. They relate to different ages and common conditions like those of infants, elderly and allergic people. Equally exposed individuals show large differences in susceptibility to air pollutants. In the field of IAQ, more than in another, it is clear that no "standard human" exists. The target population for air quality guidelines has not been specifically defined but has been assumed to be the general population. However, in practice sensitive individuals have been excluded. The difficulty has been to deal with a juxtaposition of exceptions and particular cases. Furthermore, compared to outdoor air pollution, pollutants from indoor sources are likely to exhibit significantly larger between-individuals and smaller within-individual exposure differences.

The target population for indoor air risk management needs to be defined. A crucial reason is the growing fraction of the general population, which is atopic and therefore likely to develop respiratory, eye and skin symptoms on exposure to irritating pollutants. After the target population definition the most obvious open needs towards an adequate rationale for IAQ risk assessment and risk management are for

- non-cancer health endpoints,
- complex and variable exposure mixtures, and
- heterogenous populations in different indoor environments.
- quantifying exposures to substances with respect to respiratory irritation and inflammation, and neurobehavioral symptoms (e.g. headaches)

6.2 Comparing risks.

In rare cases risk comparison can be relatively straightforward, such as when comparing well known risks with similar outcomes. The most useful universal yardsticks of effect are disability or quality adjusted life years. Risk calculations expressed in unit risk estimates can be used to compare the carcinogenic potency of different agents and can help set priorities in control of such pollutants. However, risk comparison is most needed and also often most controversial when comparing risks of competing or alternative products or techniques which present less obvious, subtle or subjective adverse effects. Furthermore, comparison of indoor air risks with each other and with other more commonly known risks

in life, using lay people's language and yardsticks, could provide an efficient way of communicating these risks.

Incomparable qualities of risk determinants may well be described but they should not be compared quantitatively. Also qualitatively similar risks can be difficult to compare. Risk comparisons based on the high ends of the probability distribution may overestimate a little known risk by orders of magnitude in relation to a well known risk, making such a comparison misleading. Direct and short term costs and benefits are sometimes easy to estimate, but they may be marginal compared to unestimated longer term and/or indirect costs and benefits.

Common yardsticks for exposures could help in comparing otherwise different risks. At present such yardsticks only exist for odor intensity and mucosal irritation (and mutagenecity). For the majority of the non-carcinogenic substances in the indoor air it is presently not possible to express exposures in easily comparable and biologically meaningful ways.

Further methodological developments are needed to

- compare risks with different magnitudes of uncertainty, and
- select and apply a common scale for qualitatively different risks.

6.3 Joint risks of combinations of sources and exposures.

In indoor air, the rule is not that a single agent from a single source is causing a single effect which can be managed by a single action. Rather the rule is the complex pollutant mixture originating from multiple sources, which are not easily managed jointly. Synergism of effects and risks may occur. Furthermore, not only adverse health effects need to be considered but also odor and sensory irritation.

To a limited number of air pollutants risk assessment has been successfully applied (Jantunen *et al.* 1997, WHO 2000). However, for most indoor air pollutants, and especially for combinations of exposures, even basic information is still missing. The principles laid down in the Commission Directive 93/67/EEC do not consider simultaneous exposures to several pollutants.

Risk assessment based on no-observed-effect-levels (NOEL) needs additional assumptions on the effects of mixtures. Additivity of effects or of complex exposures are not addressed at present in air quality standards or guidelines perhaps with the exception of PM_{10}.

The total human exposure to air pollutants is made up of the sum of exposures from different pathways, and incurred in different locations at different times. The summation ideally should be based on good understanding of the quantitative relationship between exposure and the adverse effect on human health. The present state of knowledge is usually inadequate to take full account of the absence or presence of thresholds, specific effect of peak concentrations, or the combined effect of exposure to multiple pollutants.

Furthermore, health effects typically involve respiratory and mucosal irritation, headache and malaise etc., which are mostly nonspecific and may be linked to several environmental

or other conditions. When they combine moderate severity with high probability, they may be difficult to assess, but yet posses high public health significance.

Further methodological development is needed to

- Assess the health effects of complex mixtures within heterogenous populations including sensitive individuals.

6.4 Risks and benefits of alternative selections.

The building sector is facing increasing demands for prevention of adverse health effects, but also for energy– and cost-effectiveness, and promotion of sustainable development. These requirements lead to narrow operating margins and necessitate good understanding and precise control of the indoor environments by building designers, constructors, owners and operators. Given these conditions, IAQ policy and management can no longer be based on common sense and rules of thumb, but have become dependent on rigorous, quantitative and scientific risk assessments. Failure to identify, assess and manage one indoor hazard (e.g. large water damage or exposed friable asbestos) in a single large office building could cost in lost leases, health claims, renovating and/or reconstruction, as much or even more than all money spent on indoor air risk assessment.

The risk-benefit analyst needs reliable and relevant input data (qualitative and quantitative with uncertainty estimates) about exposures related to health risks, costs (investment and operating) of risk reduction alternatives, and achievable risk reductions. Also, assessments of possible indirect and longer term consequences are needed.

Limiting the use of one building product or technique may entail the application of substitutes that have less well characterised (but possibly greater overall) risks or which may be less familiar to users (thus creating greater risks in practice). Excessive precautions may unjustifiably deny the benefits of using the chemical or technique, either the benefits to the user or the producer. Hence, legislation has moved towards approaches based on overall risk assessment rather than mere hazard identification.

In order to quantify and compare the risks, costs and benefits of different building construction and maintenance alternatives,

- common yardsticks are needed to bring the different health, environmental and societal risks and benefits to the same scale with monetary costs and benefits.

6.5 Risk assessment and management procedures in building design, construction, operation and maintenance.

Risk assessment and risk management in buildings are intimately linked together and should feed to each other. Risk management may become urgent because of complaints from occupants, inspection of the building or new research findings. For certain risks, a very detailed risk assessment is needed in order to generate rational management alternatives. For others, a simple expert judgement is sufficient for selecting the appropriate management alternative. For yet others, normal market forces of supply and demand can

do the work provided that relevant information is made easy to access, comprehend and judge by the consumer.

Occupant perception of odours, noise or mucosal irritation can be a useful source of information for the risk assessor. Occupant perception of risk may itself generate health risks due to stress, and should be seen as a reason for more objective risk assessment, or, in clear cases, prompt risk management actions.

The basic risk-control objectives include primarily

- a focussed reduction of risk among the high risk groups, experiencing either high pollutant exposures and/or high personal susceptibilities,
- a general reduction of the total estimated population risk, which may be achieved by reducing the average pollutant exposure.

If the high risk group is clearly defined, quite limited, and the risk is specific, risk reduction should focus on these occupants. Otherwise, the risk control method of choice should be the general reduction, because it steers the whole state of the art technology towards safer practices. In the long run this protects all individuals regardless of where they live and work.

Legal liability should be a strong argument to architects, building constructors and managers to make use of systematic risk assessments. Risk assessment in buildings should develop from being reactive to proactive. A stepwise procedure is suggested comprising of

- risk assessment protocols for certified building risk experts,
- building codes (by governmental and/or other bodies) that reduce and prevent building related health risks for the general population,
- clearly defined and certified "Good Design, Construction and Management Practices" (by professional organisations) for more ambitious risk management, and
- professional case by case risk assessment and management expertise into the design, construction and management of very demanding buildings, e.g. medical facilities.

On the national and EU levels, a systematic process is needed for setting up a IAQ risk management program which may include:

- surveys for determining the distributions and determinants of indoor pollutant levels (e.g. radon) in the building stock of interest;
- use of the survey results and other information for identification of the high exposure buildings and areas as well as the high risk groups, and
- development of a risk management strategy, through a cost–benefit optimisation and risk communication process that considers associated social interests and priorities. The tools include building codes and guidance, ventilation requirements, IAQ guidelines, restrictions on building materials, and certified procedures for building inspections.

6.6 Enlightened risk perception and behaviour through education.

Most of the daily risks are managed by occupants themselves, hopefully guided by sound risk communication. The essence of good risk communication is simple: learn what people already believe, tailor the language of your communication to this knowledge and to the

decisions people face, subject the resulting message to careful empirical evaluation, and then develop your risk communication materials and procedures. It is also essential to acknowledge that lay people have different definitions for the term, risk, which must be taken into account in risk communication by the experts and administrators.

Occupants should not only be seen as sources of complaints and subjects of expert analysis and actions. The major determinants of high perceived risk include inability to act against imposed exposures, lack of trust on the responsible authorities, lack of previous knowledge of the risk, and dread of consequences. Educating the occupants about risks, which are relevant for them, providing them with the information that they need, and in the form that they can comprehend will reduce

- real risks,
- misplaced concerns,
- harmful health effects, and
- time allocation of the administrators.

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Annex 1. EXAMPLES

to: ECA Report No. 22
Risk Assessment in Relation to Indoor Air Quality

To illustrate how risk assessment has been and can be carried out to answer very different building related needs, these examples are selected to describe what actually has been (examples; 1, 3, 4, 5, 6, 8, 9) or could have been (2, 7) done by different local (6), national (2, 7, 8, 9) and international (1, 3, 4, 5) authorities about some specific cases (6) and general issues (others). These examples have not been selected as recommendations of what or how indoor risk assessments should be done.

The purpose of these nine examples is to highlight two issues:

1. The great variety of the very different risk assessments needed for the building environments including individual buildings, building materials and techniques and the building stock.
2. The degree to which the concepts and structure of the European Commission Directive 93/67/EEC (Laying down the principles for assessing risks to man and the environment of substances) is applicable in assessing these nine different indoor environment risks.

And to bring some examples of

3. how the very different types of risk assessment needs that may be encountered within the millions of buildings of Europe have been approached in different situations.

In the table - that fills the next page - are extracted the risk management options that have been suggested in or for each of the 9 EXAMPLE cases. This also highlights the multitude of alternative risk management options for the multitude of indoor environment risks.

Possible options in risk managem	Radon	Glycol Ethers	Partic. Matters	ETS	VOC Floor	Damp Mould	Vent.Syst	Dust Mite	Risk Rank
Restrictions									
– materials	X								
– systems									
– behaviours			X	X					
Permissable levels	X								
Substitution		X							
International standards							X		
Commissioning							X		
ALARA			X						
Subsidies	X					X			
Taxation									
Labelling									
– environmental									
– energy									
– special IAQ					X				
Def. of risk areas	X								
Education of									
– designers							X	X	
– public	X			X			X	X	
Expert evaluat.									X
Building codes	X		X	X			X	X	

CONTENTS

1 RADON

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1.1 Introduction

The scope of this example is to illustrate the risk assessment of a carcinogen for which many quantitative estimates have been made and extensive data of exposure in dwellings are available.

Radon is one of a very small number of substances, among those present in indoor air, which have been established to be human carcinogens on the basis of human studies. As such it is a Group 1 and Group A carcinogen, according to the classification used by the World Health Organisation (WHO/IARC 1988) and by the US Environmental Protection Agency (EPA 1987), respectively. The established adverse health effect arising from the inhalation of radon and its decay products is lung cancer. Other health effects have been studied but there is no conclusive evidence at present concerning radon-induced health effects other than lung cancer (Darby et al. 1995, WHO-ROE 1996).

Radon (Rn) is a naturally occurring radioactive noble gas which exists in several isotopic forms. Only two of these isotopes occur in significant concentration in the general environment: Rn-222 (usually and henceforth here referred to as "radon"), a member of the radioactive decay chain of uranium-238, and Rn-220 (often referred to as "thoron"), a member of the decay chain of thorium-232. Radon is the first and only gaseous and inert element of the radioactive chains, so that it can easily leave the place of production (soil, rock and building material) and may finally enter the indoor air. The presence of thoron in indoor environments is usually small compared with that due to radon, due to its much shorter half-life (55 seconds vs. 3.82 days), therefore also its contribution to human exposures is relatively small, with some exceptions (e.g. Guo et al. 1992, Mjönes et al. 1996, Bochicchio et al. 1996). Hereafter we will refer only to radon.

Radon produces a series of decay or daughter products (see simplified decay schemes in Figure 1.1). From a health perspective the most significant are the four short-lived daughter products of polonium-218 to polonium-214 inclusive, which are referred to in various ways: radon daughters, radon progeny, radon decay products. These elements, unlike radon, shortly after their formation attach themselves to aerosol particles; only a small fraction of them remain in unattached form, depending on aerosol size and concentration and on ventilation (Nazaroff and Nero, 1988). When radon and its short-lived decay products are inhaled the radiation dose to lung tissue is dominated by the alpha particles emitted by the deposited decay products, which cause, especially those ones attached to small size aerosols or in unattached form, damage to sensitive lung cells, thereby increasing the probability of cancer developing. It must be emphasised that the contribution to lung dose arising from the radon gas itself is small in comparison, as very little radon is absorbed by lung tissue because it is an inert gas and for the same reason, unlike its decay products, it cannot be adsorbed onto lung airway surfaces. Therefore radon acts mainly as the source of its decay products, which actually deliver the dose to the lungs; however, as a convenient abbreviation, health effects of radon decay products are often referred to as health effects of radon.

1.2 Identification of hazard and sources

Identification of hazard: It should be noted that exposure to radon is not a new phenomenon and documentary evidence from as far back as the 16th century indicates that elevated radon exposure was probably responsible for excess lung cancer mortality of miners in some Central European mines, such as the silver mines in Bohemia (see Jacobi 1993 and Samet 1994 for more detailed historical notes). At the end of last century, lung cancers were first reported in autopsies of Schneeberg (Saxony) miners, although they were identified as primary cancer of the lung only early in this century. The hypothesis of a relation between lung cancers and radon exposure was suggested after systematic radon measurements carried out in these mines in 1936-1940, but was not generally accepted at that time. After the Second World War, an extensive mining and processing of uranium for military purposes started, and little attention was paid to the radiological protection of workers. Only in the 1950s did systematic measurements of radon in mines commence, and dosimetric studies established the dominant role of radon decay products with respect to radon itself. The first epidemiological studies in uranium mines started in the sixties, and lead to the first guidelines for control of radiation hazards (FRC 1967). Many other studies followed and evidence is now available from about 20 cohort of underground miners, including non uranium miners. In the most recent pooled analysis of 11 such studies, 40% of all lung cancers among miners are attributed to exposure to radon decay products (Lubin *et al.* 1994). In dwellings, attention to the radon problem was paid later than in mines. The first measurements were made in Sweden in the fifties, but only twenty years later extensive national and regional surveys were carried out (see section 6.1.4).

Sources. The main source of indoor radon is its immediate parent radium-226 in the ground of the site and in the building materials (e.g. Nero 1988, 1989). Outdoor air usually acts as a diluting factor, due to its normally low radon concentration, but in some cases, as in high rise apartments built with materials having very low radium content, it can act as the principal contributor to indoor radon. Tap-water and the domestic gas supply are usually radon sources of minor importance, with a few exceptions. In most situations it appears that elevated indoor radon levels originate from radon in the underlying rocks and soils (e.g. Castrén *et al.* 1985). This radon may enter living spaces in dwellings by diffusion or pressure driven flow if suitable pathways between the soil and living spaces are present. It should be noted, however, that in a minority of cases elevated indoor radon levels may arise due to the use of building materials containing high levels of radium-226. Examples of such materials, used in some buildings, are by-product gypsum, alum shale and volcanic tuffs (UNSCEAR 1982, Sciocchetti *et al.* 1983, Swedjemark and Mjönes 1984).

1.3 Exposure/Response Assessment

For radon, the assessment of the exposure/response relationship is made on a quantitative basis. There are three different approaches (see Fig 1.2) used to estimate the lung cancer risk arising from exposure to radon decay products in indoor air (ECA-IAQ 1995).

In the *dosimetric approach,* which is the most indirect one, the radiation dose to lung tissues is estimated through complex dosimetric models (see UNSCEAR 1988 and 1993 for reviews), which take into account both physical parameters of the inhaled air (such as radon progeny concentration, fraction of progeny attached to aerosols, aerosol size distribution,

etc.) and physiological parameters of the human respiratory tract (such as respiratory rate, thickness of bronchial epithelium, location of target cells, etc). The risk associated with this calculated dose is evaluated using a risk/dose factor, obtained mainly from epidemiological studies on Hiroshima and Nagasaki survivors (ICRP 1991), corrected with "weighting factors" to take into account that survivors received instantaneous whole body exposures to gamma and (to a lessor extent) neutron radiation, while radon in dwellings mainly causes a continuous lifetime exposure of the lungs alone to alpha radiation.

In the *miner epidemiology approach,* the risk estimates obtained from epidemiological studies on underground miners exposed to radon are applied to the general population, corrected for differences in exposure conditions and in exposed populations (NRC 1991). A recent pooled analysis of 11 miner cohorts (68000 miners and 2700 lung cancers) has been used to model the lung cancer risk (Lubin et al. 1994). The main results of this analysis are: there is a linear relation between Excess Relative Risk (ERR) and radon exposure expressed in Working Level Month (WLM); ERR/WLM decreases with attained age, with time since exposure, and with time after cessation of exposure; adjustment for arsenic exposure reduces the estimated risk; the interaction between radon and smoking seems to be somewhat submultiplicative, but not compatible with simple additivity (see 6.1.5); the ERR is lower for exposures received at high rates. Concentration of radon and its decay products in dwellings is generally much lower than in underground mines. However, significant excess risk was found (Lubin *et al.* 1997) also in miners exposed to 50–100 WLM (the exposure unit for radon decay products). These values are equivalent to a lifetime exposure in dwellings with radon concentration of ~200–400 Bq/m^3, which, based on the findings of many national surveys, may be present in up to 10% of dwellings in many countries (ECA-IAQ 1995).

The *residential epidemiology approach* is more recent and is aimed to reduce the uncertainties of extrapolation from miners to the general population and from mines to dwellings. It consists mainly of case-control studies to directly estimate the risk due to exposure to radon in dwellings. The statistical power of residential epidemiological studies is generally low, due to the low radon concentration in most dwellings and other reasons (Lubin *et al.* 1995), and as a consequence the estimated risk from these studies are not as statistically robust as those from the miner studies. In contrast to such case-control studies, some controversial ecological radon studies have been reported suggesting that the radon risk diminishes as the concentration increase (Cohen 1995), but such studies are generally considered to have severe methodologic limits (e.g. Stidley and Samet 1993). To overcome this problem, the published results from eight of the largest studies have recently been analysed together (Lubin and Boice 1997). This meta-analysis is based on over 4000 lung cancer cases and 6000 matched controls, and shows a slightly significant increase in lung cancer risk with increasing indoor radon exposure levels: a relative risk of 1.14 (95% confidence interval: 1.01–1.30) for 25 years exposure at 150 Bq/m^3 (the action level in the U.S.), which corresponds to 1.5 for a lifetime exposure at 200 Bq/m^3 (the action level in many EU countries). More information will come from the ongoing pooled analysis of the results of the case-control studies that used similar protocols (Samet 1995). Some of these studies have already been concluded while some are still ongoing, both in Europe and in North America, involving over 10000 cases and 15000 controls.

The risk factors obtained using the three different approaches are reasonably well in agreement (ECA-IAQ 1995). In particular, there is a difference of a factor 2-3 between the dosimetric and the miner epidemiological approaches, which is probably related to the

chosen values of some weighting factors used (Birchall and James 1994), while the present risk estimate from residential epidemiological approach is very close to that from miner epidemiology (Lubin and Boice 1997).

In conclusion, the estimated lifetime risk for a lifetime exposure to a radon concentration of 100 Bq/m^3 is considered to be ~1%, with an overall uncertainty probably less than a factor 3 (ECA-IAQ 1995). Uncertainties of this magnitude are common in estimates of the risk due to radiation or other causes, such as chemical substances and so on, where often the uncertainties are much higher. Applying these risk/exposure factors to the typical average indoor radon concentrations in European and North American countries (see 1.4), a significant fraction (typically of the order of 10%) of total lung cancers can be attributed to exposure to radon and its decay products. For example, in a country of 50 millions with a lung cancer lifetime risk of 3% (which is the value assumed by ICRP for its "reference" population), we can estimate that 3 persons per 1000, that is about 2000 each year, may die because of lung cancer due to radon exposure.

It should be strongly emphasised that the majority of the total lung cancers are due to smoking. Moreover, a synergistic effect seems to occur, in a greater or lesser degree, between radon and cigarette smoking both in mines and dwellings, so that smokers exposed to radon have probably a higher risk (6-10 times) than non-smokers (ICRP 1991, Pershagen *et al.* 1994). However the numerical estimates of this synergism are still very uncertain (see also 1.5).

1.4 Exposure Assessment

Radon concentration in indoor air in not constant but depends on time of day, season, weather conditions, ventilation habits, *etc.* The best estimate of the average value, useful for risk assessment, is the annual average value. The preferred method in such long term measurements is to use passive alpha track detectors which record the alpha activity from radon and its decay products. However other techniques are also used to measure radon concentration for short or long periods (e.g. ECA-IAQ 1995).

Since the 1980s, in many countries surveys of radon levels in dwellings have been carried out (e.g. McGregor *et al.* 1980, Swedjemark and Mjönes 1984, Put *et al.* 1985, Schmier and Wicke 1985, McLaughlin and Wasiolek 1988, Wrixon *et al.* 1988, Langroo *et al.* 1991, Swedjemark *et al.* 1993, Castrén 1994, Bochicchio *et al.* 1996,b). The first surveys were mainly small localised short term screening surveys, often followed by national surveys in which year long average indoor radon concentrations have been determined in *representative* samples of national housing stock, which is the recommended methodology (UNSCEAR 1993). In several countries measurements have been intensified in high radon areas in order to find the dwellings with radon concentrations over the action levels of the national regulations.

A summary of the principal results from national surveys carried out in EU Member States, other European countries, North America, Japan and Australia can be found in many review papers (e.g. UNSCEAR 1993, ECA-IAQ 1995, WHO-ROE 1996). The measuring technique in these surveys was generally based on the use of some form of passive alpha track detector. The size of these surveys usually ranged from a few hundreds to several thousands. However, in some countries (e.g. Czech Republic, Sweden, UK and USA) the up-to-date total radon dwelling data that have been acquired are much more extensive and in most cases these measurements were addressed to find high radon concentration values.

National surveys to date have shown that the average indoor radon concentration is in the 10 to 140 Bq/m^3 range. Examples of low, medium, or high average value countries are reported in Table 1.1. Regional average values above this range have been found in some countries. A good example of this is the UK which has a national average value of 21 Bq/m^3 while Cornwall in south-west England has an average value of about 170 Bq/m^3. Moreover, a small percentage of measured radon concentrations are considerably above this range, e.g. in many national surveys the percentage of dwellings in excess of 400 Bq/m^3 ranges from about 0.5 to 3%. As far as the maximum indoor radon concentration likely to be present in any country is concerned it is impossible to estimate its value. Concentrations greater than 100 000 Bq/m3 have already been detected in individual dwellings in some countries. In most situations it appears that elevated indoor radon levels originate from radon in the underlying rocks and soils. Finally, indoor radon levels generally appear to be approximately log-normally distributed; a number of surveys have shown, however, that the log-normal approximation may significantly underestimate the percentage of dwellings at the highest radon levels (e.g. Goble and Socolow 1990, Bochicchio *et al.* 1994, Castrén 1994).

Most of the radon concentration measurements have been made in dwellings, because people usually spend most of their time there. However in the last years, in some countries, there has been an increase in the number of measurements being carried out in normal workplaces, mainly in schools, but also in offices, *etc.* (e.g. Gooding and Dixon 1992, Poffijn *et al.* 1992).

In case-control studies, radon exposure is usually assessed by a contemporary measurement of the radon concentration in all dwellings used by cases and controls in the period under study. However present radon levels may differ significantly from those in the past, when subjects received most of their exposure, especially if structural changes have taken place in the dwelling. This bias can be tentatively limited if a strict protocol for case and controls selection is used, i.e. excluding all subjects who lived in houses that have had any significant structural change that could affect radon concentration. A complementary technique for retrospective assessment of radon exposure based on the build-up of Polonium-210 on glass surfaces in dwellings is now available (Falk *et al.* 1996, Fitzgerald and McLaughlin 1996). A similar approach is also now under development in which the build-up of Po-210 in porous materials (volume traps) in dwellings is measured as an aid to retrospective assessment of radon exposure (Oberstedt and Vanmarcke 1996).

1.5 Risk Characterisation

Some of the most relevant aspects of the estimated risk due to radon exposure are the following:

i) The radon risk assessment is carried out on the basis of human data, both of miners exposed at a range of values that includes exposures found in dwellings (e.g. Lubin *et al.* 1997), and of the general population exposed in dwellings (e.g. Lubin and Boice 1997).

ii) The risk–exposure relationship appears essentially linear, both in miner and residential epidemiological studies. In some miner studies, for very high exposure values, a lower risk has been observed for higher exposure rates (Lubin *et al.* 1994), however such effect is not expected for exposure values generally found in dwellings (Brenner 1994).

iii) There is synergism, or interaction, between radon and tobacco smoke. This interaction can be described by the following formula (e.g. Lubin and Steindorf 1995):

$$RR_{Rn,s} = \alpha\ (RR_{Rn}\ RR_{s}) + (1- \alpha)\ (RR_{Rn} + RR_{s} -1)$$

where $RR_{Rn,s}$ is the relative risk for exposure to both radon and smoking, RR_s is the relative risk for exposure to smoking (= 1 for non smokers), RR_{Rn} is the relative risk for exposure to radon (= 1 for no exposure to radon). The interaction depends on the value of " parameter:

α	> 1	supra-multiplicative interaction
α	$= 1$	multiplicative interaction
$0 < \alpha < 1$		intermediate interaction
α	$= 0$	additive interaction
α	< 0	sub-additive interaction.

The most relevant cases for radon-smoke interaction are the multiplicative and intermediate interactions.

In the *multiplicative interaction,* assumed by NRC (1988) and EPA (1992), smokers and non-smokers exposed to radon have the same relative risk compared with those not exposed. Therefore the absolute risk of smokers exposed to radon is much higher than the absolute risk of non-smokers exposed to radon, and the largest fraction of the lung cancers attributable to radon are expected among smokers.

In the *intermediate interaction,* estimated by Lubin *et al.* (1994) and BEIR VI Report (NRC 1998), smokers exposed to radon have a lower relative risk (compared with smokers not exposed to radon) than non-smokers exposed to radon (compared with non-smokers not exposed to radon). However, due to the high relative risk of smokers not exposed compared with non-smokers not exposed, the absolute risk of smokers exposed to radon is still much higher than the absolute risk of non-smokers exposed to radon. Therefore, most of the lung cancers attributable to radon are still expected among smokers.

iv) The distribution of individual lifetime risk is different from the distribution of radon concentration in dwellings, depending on the mobility of the population under study (e.g. Liu *et al.* 1992). Actually, this difference is common for all the indoor risk sources.

1.6 Relevance for Risk Management and Risk Communication

(The following text is largely based on the WHO document "Indoor Air Quality: A risk-based approach to health criteria for radon indoors", EUR/ICP/CEH 108(A), 1996)

Management of the radon risk proceeds both from radiation protection and from indoor health questions. A health risk is considered severe when the annual risk for individuals exceeds 10^{-3}. In a modern industrial society risks above this level are not considered acceptable even for workers. In the case of such a risk, it is required that action should be taken. This was one of the components that ICRP used to reduce the dose limits for workers to 20 mSv/year. At present knowledge health risks of this order of magnitude correspond to radon concentrations in dwellings of about 1000 Bq/m^3. Radon concentrations above this limit should be avoided whenever possible. Annual risks for individuals below 10^{-3} can still be significant and further risk reduction procedures may be needed.

In general, a systematic process, from risk identification to choosing implementation approaches and options, is needed for selecting a risk management program. These ordinarily should include:

a) radon surveys for determining the frequency distribution of indoor radon levels in the building class of interest;

b) using the radon survey results and other information, for specification of the highest risk groups and if possible, the areas where they occur;

c) development of a risk management strategy, through an optimization and decision process that considers the issues noted above and associated social interests and priorities.

Several basic risk-control objectives may be identified as possibilities. These include primarily:

i) substantial reduction of risk among high-risk groups, ordinarily defined as those experiencing high radon exposures;

ii) reducing the total estimated population risk, which may also be interpreted to be equivalent to reducing the average risk from radon.

Risk-control criteria may also involve other aspects of exposures and risks. One is that a distinction may be made between smokers and nonsmokers. This could lead to an emphasis of risks among smokers, and a targeting of them, because the added risk associated with radon among smokers is greater than among nonsmokers, if the risk is positively synergistic (e.g., multiplicative, rather than additive).

As a means to achieve indoor radon control objectives a number of regulatory instruments may be used. These include the following:

Restrictions on Building Materials. Many restrictions and norms apply to building materials, including the radioactive characteristics of the material. Some countries and international organizations have established controls of radon exposure from building materials.

Building Codes and Guidance. Building codes exist in most countries but nevertheless mechanisms may differ, as well as the rules for compliance. Radon control through the use of building codes can be effective for new buildings (e.g. U.S., Sweden, Ireland,United Kingdom).

It should be noted that such codes may not be enforced in some countries and this may jeopardize the effectiveness of this type of instrument. In the case of schools there are a number of regulatory instruments utilized by various countries to assure a healthy and safe environment in schools. These can used for radon control purposes in schools.

Radon affected areas. Definition of a radon affected area is an instrument that is being used in some countries. It provides a framework for defining specific prescriptions in building codes, provisions for governmental financial support for measurement and remedial actions.

The experience of a number of countries illustrates a wide range of approaches for controlling radon health risks, including:

- public education campaigns to inform the public about radon to encourage voluntary risk reduction;
- use of government assistance or other incentives (such as grants for remedial actions or a free testing program for high risk areas) to encourage radon risk reduction;
- regulations (such as new construction codes) which require action to reduce radon, especially in high-risk areas.

Glossary

See ECA-IAQ Report No. 15 (1995).

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Table 1.1 *Examples of countries with low, medium, and high concentration values in dwellings.*

	Average (Bq/m³)	% > 200 Bq/m³	% > 400 Bq/m³
Low-Rn countries			
United Kingdom	21	0.5 %	0.2 %
Medium-Rn countries			
Germany	50	1.5 – 2.5 %	0.5 – 1.0 %
Ireland	60	3.8 %	1.6 %
Italy	75	4.7 %	1.0 %
High-Rn countries			
Finland	123	12.3 %	3.6 %

Radionuclide	Historical name	Half Life	Energies (MeV) and intensities (%) of emitted alpha particles *
Rn-222	Radium emanation	3.82 d	5.490 (100%)
↓ α			
Po-218	Radium A	3.04 min	6.003 (~100%)
↓ α			
Pb-214	Radium B	26.9 min	–
↓ β,γ			
Bi-214	Radium C	19.7 min	–
↓ β,γ			
Po-214	Radium C '	164 µs	7.687 (100%)
↓ α			

* Energies of emitted beta and gamma radiation are not shown here.

Figure 1.1 *Simplified decay scheme of radon-222 and its short-lived decay products.*

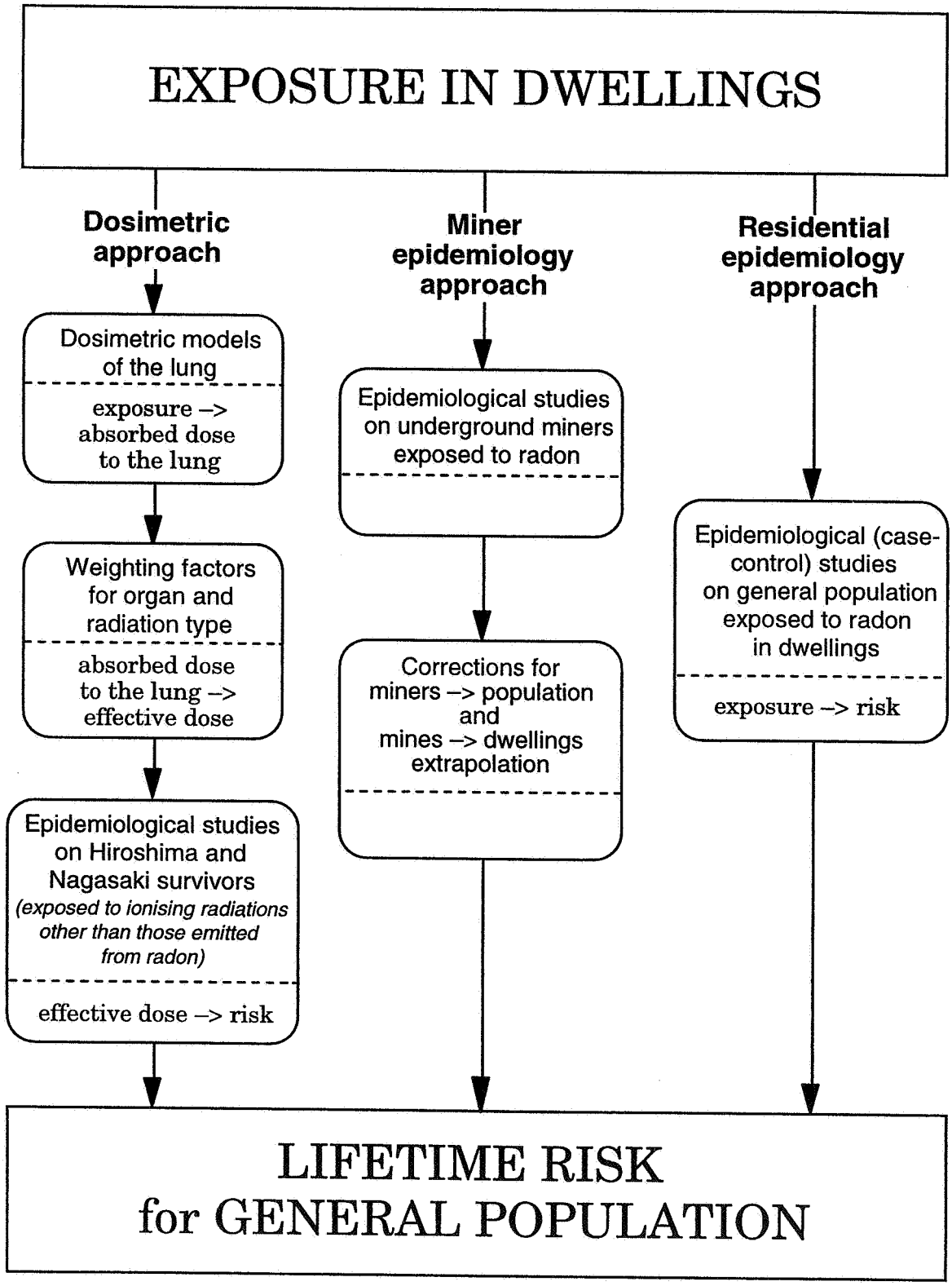

Figure 1.2 *Outline of the three different approaches for lifetime risk assessment for a chronic exposure of general public to radon indoors.*

2 INDOOR AIR QUALITY AND GLYCOL ETHERS

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2.1 Introduction

Glycol ethers (GE) are a family of chemicals widely used in domestic and industrial products (see appendix for chemical formula and abbreviation). There are 2 main groups of GE: E series formed from ethylene glycol and generally the most toxic, and P series formed from propylene glycol. GE are either ethers or acetates of ethers. Both groups are similar in toxicity, since acetates of ethers are quickly converted in ethers as soon as they enter the body by the esterases enzymes.

GE can be found more especially in water-based products, due to their technological property of being quite soluble in water. The main products in which GE are found are paints, inks, varnishes, cleaning agents... They can also be found in indoor air from the use of domestic products and in the emission from flooring materials and building materials such as paints, varnishes and lacquers.

2.2 Hazard Identification

Systemic effects

Reproductive and hematologic toxicity

GEs can be classified in three groups (Cicolella 1997):

Group 1: 2 subgroups are considered in regard to the quality of data.

GEs belonging to the subgroup 1a are toxic for reproduction :

- both male and female genital systems are impaired: infertility, testicular atrophy and sperm quality decrease.
- development of embryo and foetus is disturbed in various ways : malformation, growth retardation, functional deficits and death.

They are also toxic for hematopoiesis, through an action on bone marrow cells which causes a decrease of the amount of white and red cells along with thrombocyte cells. Reproductive toxicity occurs at a lower level than hematotoxicity.

These outcomes have been shown on various kinds of animals and, for 10 years, in humans. Sperm quality decrease has been observed among shipyard painters (along with white and red cells decrease) (Welch, 1988) and foundry workers (Ratcliffe, 1989). In a fertility clinic, Veulemans (1993) has found a significant relationship between the exposure to 2-ethoxyacetic acid (2-EAA), the common acid metabolite of 2-ethoxyethanol (2-EE) and 2-ethoxyethyl acetate (2-EEA), and a decrease of sperm quality. This decrease is about three times higher in the exposed group than in those unexposed.

Spontaneous abortions and hypofertility have been observed in the semi-conductor industry in relation with GE exposure (Gray, 1996, Schenker, 1996). Recently, Cordier (1997) has found a significantly higher proportion of malformation in children whose

mothers had been exposed to GE during the first three months of their pregnancy. Saavedra (1996) has described 44 cases of malformation among children whose mothers had been occupationally exposed to a mixture of 2-ME and ethylene glycol.

For GE from subgroup 1b, data available are related to developmental toxicity, and only in some cases to testicular and blood toxicity. No human data are available.

For the moment, only Group 1a is classified as reprotoxic under European Union regulation.

Group 2: For GE of this group, data about male reproductive system toxicity are conflicting, but there is no doubt about female reproductive system toxicity (infertility) and developmental toxicity. The effects are not malformations, but mainly skeletal variations and post natal mortality. It occurs at the same level of a slight maternal toxicity. Due to the toxicity of GE in general, it can be speculated, as recommended by US EPA guidelines, that this developmental effect might be related to the intrinsic toxicity of these molecules, not merely a consequence of maternal toxicity.

Hematologic outcomes are different too: the effect is peripheral (hemolysis) not central. Recently, 2-butoxyethanol (2-BE), the major GE in Group 2 and its acid metabolite (2-butoxyacetic acid, 2-BAA), have been found to be intrinsically teratogenic in the rat embryo culture test from 0,4 mM concentration (Giavini,1993). One can speculate therefore they can be teratogenic in humans too, because they stay much more in humans than in rodents (Biological half-life of 2-BAA is 6 h in humans against less than 1 h in rat).

Group 3: GE belonging to this group are not toxic for reproduction. Among GE of this group are GE from the E series with a higher molecular weight, called Group 3a e.g. 2-BEE, 2-BEEA, ...and the major part of the P series , called Group 3b (1M 2P, 1M 2PA...). The subgroup differences are related to differences in metabolic pathways, through aldehyde and acid steps for Group 3a and through propylene glycol for Group 3b.

Other effects

Limited data among Swedish painters using water-based paints have suggested that some GE used in these paints (2-BE, 2-BEE, 2-BEEA, 2-EEE, 2-MEE) could be involved in the occurrence of bronchial hyperresponsiveness, asthma, eye symptoms and increased concentrations of blood eosinophils suggesting possible immunological and inflammatory effects (Wieslander 1994, Norbäck 1996). Berlin (1995) has described the case of a woman reacting as soon as she entered into a freshly painted room. According to the patch tests, 2-(2-butoxyethoxyethanol) (2-BEE) was the cause of this skin hypersensitivity.

Genotoxicity

Genotoxicity can be suspected from positive in vitro tests with the intermediary metabolites of some E series GE (Elias 1996). Methoxyacetaldehyde, metabolite of 2-methoxyethanol can be classified as mutagen of class 3 according to the European Union classification. Genotoxicity hypothesis is supported by several in vivo tests (Oudiz, 1993, Anderson, 1996). No animal chronic data and no epidemiologic data are available.

2.3 Dose-Effect Relationship

The developmental effects can be chosen as critical effects i.e. those occurring at the lowest dose. Data on hypersensitivity or genotoxicity are insufficient to set up NOAELs.

Animal data are sufficient to establish NOAELs (No Observed Adverse Effect Level) for 19 reprotoxic GE in groups 1 and 2. For developmental toxicity, following the recommendations of WHO (1994) a safety factor of 1000 has been chosen:

- 10: to extrapolate from animals to humans
- 10: to take into account the variations within the human species
- 10: to take into account the teratogenicity of these chemicals.

Experimental data with 2-methoxyacetic acid (2-MAA), the acid metabolite of 2-ME have shown that the teratogenicity of 2-ME is directly related to the whole burden of 2-MAA. Biological half-time of acid metabolites being much higher in humans than in rodents, humans are, for that reason likely to be more sensitive to GE than rodents, in terms of reproductive toxicity. Epidemiological data have shown, among shipyard painters and foundry workers, sperm quality to decrease and hematologic disorders to appear at mean exposure levels of about 1/20 of NOAEL in animals, which confirms that statement of higher sensibility of humans, and supports therefore, the need for high safety factors.

According to US EPA guidelines for developmental toxicity risk assessment (1991), it is assumed that, in most cases, a single exposure at any of several developmental stages may be sufficient to produce an adverse developmental effect. Thus human exposure estimates used to compare with the RfD are usually based on a daily dose that is not adjusted for duration or pattern of exposure

The table below gives the reference doses (RfD) for developmental toxicity for GE measured in indoor air, as derived by Cicolella (1997) from NOAELs in animals:

	2-ME	**2-MEA**	**2-EE**	**2-EEA**	**2-BE**	**2-MEE**	**2-EEE**	**2-EEEA**	**2-PhE**
RfD (µg/kg/d)	3	48	18	27	23	50	2200	2500	400

2.4 Exposure Assessment

Occupational exposure or consumer exposure during the application of the products have not been considered. Only exposure after application has been taken into account.

Saarela (1997) has analyzed 27 flooring materials. Four GEs have been found:

Glycol ether	Emission factor (µg/m2/h)		number of materials in which GE was found
	Mean	Maximum	
2-ME		56	1
2-MEA	38	59	3
2-EE	6	13	8
2-BEE	151	666	10

The same author has found GEs in emissions from cushion vinyl (3,9 mm and 2,5 mm). Traces were still found up to 8 weeks after application (see the following table):

Glycol ethers	Emission factor (µg/m²/h) after			
	3 days	2 weeks	30 days	8 weeks
2-EE (CV 3,9 mm)	9	7	6	4
2-EEA (CV 2,5 mm)	24	7	4	0
2-EEA (CV 3,9mm)	24	19	17	13

Emissions from other materials used in homes have been analysed. Dietert (1996) has reviewed data about emissions from new carpets. 2-BE has been detected in 9 of 19 carpet samples, the highest level being 0,326 mg/m².

In a general review of indoor air concentration, Shah and Singh (1988) reported a mean concentration for 2-BE of 2 ppb (10 µg/m³). In a survey of VOCs in 12 California offices, Daisey (1994) has detected 2-BE with a range of concentration between < 0,4 to 27 ppb. Zellweger (1997) has measured VOC emissions from 33 building materials including paints, primers and sealants. Various GE have been found in 23 of them, especially in water-based materials: Group 1: 2-MEEE (3), 2-EEE (1) 2-EEEA (1), Group 2: 2-BE (5), 2-PhE (1), Group 3: 2-BEE (11), 2-BEEA (3), 1M2P (4), others P series (7).

2.5 Risk Characterization

The calculation of a daily dose for an average adult, breathing 20 m^3 per day and weighing 70 kg, has been made on the basis of 24 h emissions, between 24 h and 48 h after application, from a 16 m2 surface in a 40 m^3 room. The critical effect being developmental toxicity, the dose has to be expressed on a daily basis and therefore the risk is expressed as a ratio: daily dose/RfD. If this ratio exceeds 1, it is considered that there is a risk for people living in such an environment. This ratio is a rough estimate and only the order of magnitude has to be considered.

Using data from Saarela and Zellberger, the table below gives the ratio daily dose/RfD to be expected from the presence of different flooring and building materials emitting GE from Groups 1 and 2, in a standard room, during 24 h, 24 h after application, if no other indication is mentioned.

Material	2-ME	2-MEA	2-EE	2-EEA	2-MEEE	2-BE	2-EEE	2-EEEA	2-PhE	Total
Flooring	50	30	2							90
Flooring (Cushion vinyl)			3 (3d)	2 (3d)						5
Sealant					800	200				1000
Parquet Sealing A					200		20	7		200
Parquet Sealing B						3000				3000
Parquet Sealing C						300				300
Parquet Sealing D						1000				1000
Lacquer						400				400
Acrylic monomer Paint					3000					3000
Parquet varnish									100	100

In interpreting the table above, one should take into account that this refers only two the second day after application - the exposure decays in time. One should also consider that actual application of sealants is only a minimal fraction of the modelled 16 m^2 in a 30 m^3 room.

2.6 Conclusions

Emissions of some reprotoxic glycol ethers right after application of flooring and building materials may pose problems for the health of the embryo and the foetus. Major problems are due to the high toxicity of certain GE, like 2-ME and 2-MEA or to the high level of emissions of GE like 2-MEEE or 2-BE in uses like parquet sealants. Daily dose/Reference dose ratios in these cases go over the unity from a long shot. Even if a possible overestimate still remains possible, in such an assessment, the order of magnitude is high enough to

suspect a possible health problem and to promote therefore substitution in favour of less toxic GE or other solvents in those materials.

The current analysis is based on embryo- and foeto-toxicity. Other health effects on blood or spermatogenesis need a more permanent exposure to be induced. More epidemiologic and experimental data are needed to know whether hypersensitivity and genotoxicity have to be considered.

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Appendix: Formulas and abbreviations of main glycol ethers

Name	Abbreviation	CAS number	Group
2-methoxyethanol	**2-ME**	**109-86-4**	**1a**
2-methoxyethyl acetate	**2-MEA**	**110-49-6**	**1a**
2-ethoxyethanol	**2-EE**	**110-80-5**	**1a**
2-ethoxyethyl acetate	**2-EEA**	**111-15-9**	**1a**
2-(2-methoxyethoxy) ethanol	**2-MEEE**	**111-77-3**	**1b**
2-(2-ethoxyethoxy) ethanol	**2-EEE**	**111-90-0**	**1b**
2-(2-ethoxyethoxy) ethyl acetate	**2-EEEA**	**112-15-2**	**1b**
2-butoxyethanol	**2-BE**	**111-76-2**	**2**
2-phenoxyethanol	**2-PhE**	**122-99-6**	**2**
2-(2-butoxyethoxy) ethanol	**2-BEE**	**112-34-5**	**3a**
2-(2-butoxyethoxy) ethanol acetate	**2-BEEA**	**124-17-4**	**3a**
2-(2-(2-ethoxyethoxy) ethoxy)ethanol	**2-EEEE**	**112-50-5**	**3a**
1-methoxy 2-propanol	**1M2P**	**107-98-2**	**3b**
1-methoxy 2-propanol acetate	**1M2PA**	**108-65-6**	**3b**

3 HEALTH RISK OF INDOOR AIR FINE PARTICULATE MATTER

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3.1 Introduction

The scope of this example is to present a general risk assessment for particulate matter (PM) in indoor air. Specific risk assessment for environmental tobacco smoke is presented in a separate example in this report.

The reason why PM has become a major interest and even battleground of environmental health risk assessment is the observed considerable excess mortality associated with the levels of ambient air PM in the absence of a plausible scientific explanation. On one hand many recent epidemiological findings show that the differences in present levels of urban outdoor air PM in West Europe and North America, although lower than 30...50 years ago, are still associated with significant differences in mortality and morbidity. Yet, these findings are difficult to explain because the observed acute and long term mortality increase appears to be caused by extremely small doses to relatively non-toxic compounds, and occur more as cardiovascular and less as respiratory mortality. Although there are suggestions for the mechanisms that could explain the obvious discrepancy, these are far from proven.

TSP is abbreviated from Total Particulate Matter as collected by a high volume sampler, PM_{10}, $PM_{3.5}$ or $PM_{2.5}$ refer to particulate matter, where particles larger than 10, 3.5 or 2.5 μm in aerodynamic diameter have been separated out - usually by impactor or cyclone type preseparators (in fact, the separation curve is smooth, and a cut size of, e.g., 10 μm means that the separation is 50% at this aerodynamic particle size).

3.2 Identification of hazard and sources

The hazard identification of PM is based on outdoor air studies only. It is based on a number of short term time series studies and three long term cohort studies, which have linked significant mortality differences to different levels of outdoor PM between different days in one city (time series studies) and between different cities (cohort studies). There is sufficient consistency in both the causes of excess mortality (mostly cardiovascular, less respiratory and no other) and the level of mortality increase caused by an increase in ambient PM level to identify a health hazard that needs to be investigated in great detail, and possibly to be acted on by both exposure reducing and indoor and outdoor source reducing measures.

Indoor air PM has two different origins.

Coarse particles (>1.0 μm 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 reentrained. Outdoor air coarse particles are generated by mechanical erosion; wind, traffic, and materials handling and they penetrate poorly into indoor environments.

Fine particles (< 1.0 μm) are produced by tobacco smoking, cooking, unvented kerosene heaters and wood burning. They contain mostly sulphates, nitrates, ammonia, semivolatile

organics, polyaromatic hydrocarbons and elemental carbon (soot). In outdoor air the precursors of fine particles are gaseous sulfur, nitrogen and organic compounds. They are also emitted directly by incomplete combustion processes such as diesel and petrol engines, wood burning and barbecuing. Fine particles do not settle out of indoor air. They move freely with air currents and stick to any surface they touch. From outdoor air they penetrate effectively indoors through most ventilation systems.

3.3 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 not exhaled, 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).

Epidemiological Data

Time Series Studies on Short Term Health Effects

Our knowledge about the health effects of the particulate matter has improved considerably since extended time series of outdoor air quality data - mostly U.S. - from PM_{10} and $PM_{2.5}$ samplers have become available for epidemiological analyses. In a study on particulate air pollution and daily death rate in Steubenville OH, Schwartz and Dockery (1992) found a 6% increase in daily deaths when daily TSP levels increased from 36 μg/m³ to 209 μg/m³. This result has later been confirmed in new time series studies in the U.S., China (Xu *et al.* 1994), and in the European APHEA study by Katsouyanni *et al.* (1995); in Lyon (Zmirou et al. 1996), Paris (Dab *et al.* 1996), Athens (Touloumi et al. 1996), Köln (Spix and Wichmann 1996), and Milan (Vigotti *et al.* 1996). Combined analysis of the APHEA data from 5 West European cities indicates a 2% increase in daily deaths resulting from a 50 μg/m³ increase in daily PM_{10} level (Katsouyanni et al. 1997).

The APHEA study has also produced disease and hospitalisation data (Anderson *et al.* 1997) which support the findings of the death rate data, namely that existing levels of particulate air pollutants in West European cities have a significant impact on the cardiovascular and respiratory health of the urban populations.

Based on extensive review of the literature, WHO Air Quality Guidelines (WHO 2000) concludes that a daily outdoor air $PM_{2.5}$ increase of 25 μg/m³ increases daily total mortality by 15%, and daily outdoor air PM_{10} increase of 50 μg/m³ increases total mortality by 15 (± 4%).

Cohort Studies on Long Term Health Effects

In the first cohort study on the relationship between annual average pollution levels and adjusted mortality-rate ratios in a cohort of 8.000 adults in six cities followed over 14-16 years, Dockery *et al.* (1993) found that although many pollutants were associated with increasing mortality, the association was strongest for $PM_{2.5}$. An increase in the annual average $PM_{2.5}$ level from 10 to 30 μg/m³ was associated with a mortality increase of 26% in total and 37 % in lung and heart disease.

In a larger cohort study on the associations of $PM_{2.5}$ levels and adjusted mortality rate ratios in 50 cities in cohorts of 295.000 individuals, Pope *et al.* (1995) found that an increase of the annual average $PM_{2.5}$ by 24.5 $\mu g/m^3$ was associated with a 17% increase in total mortality and 31% increase in lung and heart disease mortality.

In another U.S. cohort study the association of the death rate among 3.8 million babies 1..12 months of age with outdoor air PM10 levels during the first 2 months after birth shows that compared to the low exposure group ($PM_{10} < 31 \pm 8$ $\mu g/m^3$), in the high exposure group ($PM_{10} > 45 \pm 5$ $\mu g/m^3$) 10 % more babies died, 26 % more from sudden infant death syndrome, and 40 % more from respiratory causes (Woodruff *et al.* 1997).

Concluding from the three cohort studies, typical urban outdoor air levels of PM_{10} and $PM_{2.5}$ appear to increase long term death rate, i.e. reduce life expectancy. This increase is consistent between different studies, and seems to affect at least babies and adults.

Toxicological Data

Currently understood toxic mechanisms of individual harmful compounds or their combinations in the particulate matter can hardly explain the observed mortality increases. The total mass of $PM_{2.5}$ particles inhaled into the lung during a full year, assuming 30 $\mu g/m^3$, is in the order of 1 mg. Indeed, this fact indicates that the observed health effects of the atmospheric particulate matter may not be caused by the toxicity of any chemical component of the particulate matter. However, there are new, yet unpublished experimental data, which support the epidemiological findings (Godleski 1998). Healthy dogs and compromised dogs with induced bronchitis and induced coronary heart disease, were exposed to relatively clean urban air, in which the fine particulate matter fraction has been concentrated by an order of magnitude. Healthy dogs were not harmed, but exposure of dogs with a cardiovascular or/and bronchial precondition resulted in significant and prompt mortality.

For the considerable fraction of urban dwellers, who live with asthma, chronic bronchitis or coronary heart disease, the experimental results indicate that the safety margin in the present day urban air fine particle levels and air quality guidelines is small or nonexistent. The epidemiological studies point to exactly same conclusion.

Indoor Air Particles?

All the available epidemiological and toxicological data are based on relating PM levels measured at fixed urban ambient air monitoring sites to long or short term mortality and morbidity of populations or cohorts. How would these findings relate to the health risks of indoor air particles? Individual exposures to PM can be divided into outdoor exposures in ambient air (PM measured at urban monitoring sites), in outdoor microenvironments (e.g. traffic exhaust particles on busy streets), indoor exposures to PM from outdoors and exposures to PM generated indoors (ETS, cooking, *etc.*). In average, individuals in non-smoking environments acquire roughly one half of their fine PM exposures from outdoor air particles - note, mostly in indoor environments - and the other half from indoor and personal sources. The average exposure of individuals in smoking environments is two times higher, with half of the total exposure coming from ETS.

The indoor and personal PM sources have typically much stronger immediate impact on personal PM exposures than the ambient air PM levels. However, the ambient air PM, especially the long lived and effectively penetrating $PM_{2.5}$, forms the large scale exposure baseline on which the impacts of the more variable near field, indoor and personal PM sources are superimposed.

The question - 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 - remains unanswered. If the health effects of fine PM are mostly independent of the origin/composition of the particles, as epidemiological studies seem to indicate, then indeed the risks of the indoor PM should be assessable on the basis of the ambient air based epidemiological studies. If the health effects of PM do depend on their origin/composition, the health effects indoor PM cannot be directly assessed from the ambient air based epidemiological studies. This problem, however, is lager in principle than in the practice: When 50 - 60% (in the absence of smoking) of indoor air fine PM is of outdoor origin, and people spend 80 - 90% of their time indoors, most of the PM exposure is to outdoor air particles. Tobacco smoke, the most significant indoor PM source, is also a well known health hazard. Consequently the overall uncertainty as to the health effects of indoor air PM relative to the better known health effects of outdoor air PM is hardly larger than a factor of 2.

3.4 Exposure Assessment

PM Exposure Studies

On one hand the recent epidemiological findings about the public health impacts of atmospheric PM and on the other hand the tremendous costs involved in significant reduction of the present PM levels in most regions of the industrialized world lead to increasing demand for better information about;

- what chemical and physical characteristics of the PM are most significant for the health consequences observed,
- what 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 measures.

Personal exposure studies are needed to answer these questions.

Personal PM exposure studies using volunteers have been reported by Dockery and Spengler (1981), Sexton *et al.* (1984), Spengler *et al.* (1985), and Lioy *et al.* (1990). Their main findings are that outdoor PM_{10} level was not an important determinant of personal exposure (except during high pollution episode), personal exposure levels were systematically higher than outdoor air levels, and that PM_{10} exposures of tobacco smoke-exposed people are twice as high as those of others.

A much larger evaluation of personal PM_{10} exposures of the entire population was conducted in Riverside, CA in the PTEAM study (Wallace *et al.* 1991, Wallace *et al.* 1993, Özkaynak *et al.* 1993, Thomas *et al.* 1993, Clayton *et al.* 1993, Özkaynak *et al.* 1996).

178 people carried personal monitors 24 h at the time. Also the PM concentrations inside and outside of the home of each of the 178 participants were monitored with stationary PM_{10} and $PM_{2.5}$ monitors, and outdoor air levels at central sites were monitored with high volume PM_{10} samplers. Following each monitoring period the participants answered a time/activity questionnaire. Daytime personal PM_{10} exposure levels, as well as the levels of nearly all particle bound elements were elevated relative to both indoor and outdoor levels. Nighttime personal exposure levels were lower than outdoor but higher than indoor levels. Smoking, cooking, dusting and vacuuming were dominant sources for high indoor particle loads. PM_{10} and $PM_{2.5}$ concentrations in smoking homes were considerably higher than, typically twice as high as, those measured in non-smoking homes.

Variations of the outdoor central site PM_{10} levels could explain only 37% of the variations in daytime and 54% in nighttime personal PM_{10} exposures. This means that about 1/2 to 2/3 of the personal PM_{10} exposures are explained by indoor, microenvironmental (e.g. in traffic) and/or personal (e.g. barbecuing) sources. Indoor $PM_{2.5}$ concentrations follow outdoor levels closer than PM_{10} concentrations.

Results of PM Exposure and Microenvironmental Studies

A comprehensive review of the indoor air PM studies has been 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 3.1.

Table 3.1. *The observed median(* levels for $PM_{2.5\cdots3.5}$ and PM_{10} in American personal and microenvironmental PM exposure studies.*

	$PM_{2.5\cdots3.5}$ (µg/m³)	**PM_{10} (µg/m³)**
Personal exposures	**22 - 44**	**33 - 129**
Home indoor levels	**11 - 42**	**22 - 78**
Home outdoor levels	**10 - 38**	**18 - 83**
Central monitoring site	**18 - 33**	**38 - 76**

*) 50 % of the measured values are higher

As a summary of the impacts of certain indoor activities on personal PM exposures and indoor concentrations, the most significant is, of course, smoking. An average $PM_{2.5\cdots10}$ level increase in smoking v.s. non smoking environments is 30 - 40 µg/m³ or doubling of the non smoking level. Cooking increases PM exposures 7 - 26 µg/m³, unvented kerosene heaters 5 - 30 µg/m³ and wood stoves 0 - 10 µg/m³.

So far no source contributions have been published for personal PM exposures. Table 3.2 presents a summary of the contributions of different sources to indoor and ambient air PM.

Table 3.2. *Contributions of different sources to indoor and outdoor air PM combined from a body of literature, mostly American. Note that, due to the data availability, the contributions to indoor air levels are expressed 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. See also chapter **SOURCES**.*

Source category	Indoor air $PM_{2.5...10}$	Outdoor air $PM_{2.5...3.5}$
smoking	**24 - 71 % (*)**	**negligible**
cooking	**- 25 - % (*)**	**2 - 3 (µg/m³) (not fire, but frying fumes)**
wood burning	**3 - 21 %**	**1 - 4 (µg/m³)**
soil dust	**4 - 50 %**	**1 - 23 (µg/m³)**
industry, heating	**10 - 38 %**	**4 - 6 (µg/m³)**
traffic emissions	**5 - 30 %**	**5 - 17 (µg/m³) (mostly diesel)**
secondary PM	**not available**	**11 - 22 (µg/m³) ($SO_4^=$, NO_3^-, NH_4^+)**

*) where named activity takes place

It is important to realise that in the cities of developing countries both indoor and ambient fine PM levels are usually very much higher due to dispersed heating with solid fuels, uncontrolled industrial emissions and the large quantities of non-catalyst twin stroke engine vehicles.

3.5 Risk Characterization

There are sufficient reasons to assume that the fine PM in outdoor (and indoor) air is hazardous to public health even at the presently common relatively low concentrations. We do not know yet (i) what characteristics make the particles harmful, although combustion generated particles are the most suspected, (ii) what characteristics make individuals more susceptible, although individuals compromised by cardiovascular or respiratory diseases are the most likely targets, or (iii) what biological mechanisms are responsible for the observed acute and long term mortality increase.

The U.S. cohort studies on long term health effects suggest that a 10 µg/m³ increase in the long term mean $PM_{2.5}$ level increases total death rate by 7-13%. U.S.EPA (1996) concludes in its new Air Quality Criteria for Particulate Matter that;

- There is very much evidence that daily outdoor air fine PM ($PM_{2.5}$) is significantly increasing daily deaths and cases of disease at present concentrations in (West European) and North American cities.
- There is less, but still convincing evidence, that fine PM also reduces life expectancy.
- There is no clear evidence about what physical or chemical fine PM characteristics make it hazardous.

- There is no indication of a threshold level, below which fine PM is harmless - if one exists, it is below today's cleanest cities PM levels.
- Elderly individuals with cardiopulmonary diseases appear to be at highest risk, asthmatic children may also form a susceptible group.

All present epidemiological evidence is based on outdoor ambient air pollution data. The observed health risks of outdoor air PM result mostly from exposure to these particles in indoor environments. However, although the risks of fine PM from indoor sources (except smoking) are not known, this does not much affect risk assessment for indoor fine PM, because 50 - 65% of it comes from outdoor air/sources. Therefore, if we can accept an maximum additional uncertainty factor of 2, we can apply the risk estimates from outdoor air PM to indoor exposures as well.

Also the WHO expert group responsible for preparing material for the new WHO Air Quality Guidelines (WHO 2000) concludes that the epidemiological studies do not support any threshold level, below which particulate matter exposure could be considered safe to the general public. Instead of an air quality guideline value, the group suggests a unit risk estimate for particulate matter:

- A 100 $\mu g/m^3$ increase in 24 h average PM_{10} exposure results in a 6...8 %, $PM_{2.5}$ exposure in a 12...19 % increase in daily deaths within a population,
- a 50 $\mu g/m^3$ increase in 24 h average PM_{10} exposure results in a 3...6 %, $PM_{2.5}$ exposure in about 25 % increase in total hospital admissions, and
- among asthmatics a 25 $\mu g/m^3$ increase in 24 h average PM_{10} exposure results in a 8% increase in symptom exacerbation and bronchodilator use, and a 12 % increase in cough.

The epidemiological evidence and occupational hygiene experience suggest that the short term acute death risk of a 24 h PM_{10} level of 100 $\mu g/m^3$ is probably negligible for the healthy majority of the population, but amplified for babies in their first year(s) of life, for ashmatics, and for the large numbers of adult and elderly individuals with underlying cardiovascular or respiratory diseases.

3.6 Relevance for Risk Management and Risk Communication

In a situation, where a safe threshold level for fine PM exposure cannot be established, and where mechanistic information or information about the chemical characteristics that define the observed PM toxicity is missing or very uncertain, a risk manager would normally ask for more research, facts and time before making any decisions. However, when at the same time 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, even in the absence of complete mechanistic explanations, strong reasons to search for effective, "no regrets", exposure reducing policy alternatives.

Because all fine PM exposures seem to be harmful, like we assume to be the case for ionising radiation, the RM policy should aim at reducing all exposures, and not just searching and reducing the highest levels, or levels that exceed a certain level. Consequently ALARA (as low as reasonably achivable) should be the obvious policy choice.

In indoor environments the requirements for effectiveness, no regrets and ALARA 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 halves the PM exposures of all affected non-smoking individuals. It is difficult to imagine any exposure reduction measures with comparable cost/effectiveness. The other significant indoor PM sources are any unvented or leaking combustion devices, kerosene heaters, fireplaces, and to a lesser extent gas stoves and hot water heaters (geysers).

The entry of outdoor PM into indoor environments can be reduced by sealing the buildings and equipping the two way mechanical ventilation systems with EU6 or EU7 class filters. Such actions must, however be balanced against the risks from sealed buildings and ventilation systems.

Building codes, good maintenance of the ventilation system, education and training of the building operators and elimination of smoking indoors can reduce PM exposures (and presumably also the related health risks) of the affected populations more than any realistically achievable urban outdoor air quality management measures in many years.

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4 ENVIRONMENTAL TOBACCO SMOKE

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4.1 Introduction

Health effects of environmental tobacco smoke (ETS) have been a subject of several reviews, including the WHO evaluation held in the framework of the update and revision of WHO Air Quality Guidelines (WHO 1996). ETS has been shown to increase the risk of a variety of diseases and no safe level of exposure to ETS can be recommended. Among the most serious health effects of prolonged exposure to ETS well supported by scientific evidence is increased risk of lung cancer. The WHO AQG review has also noticed that there has been a growing evidence on the impacts of ETS on cardiovascular diseases. Since the review, results of new studies were published confirming the previously observed associations (Law *et al. 1997*). Health effects of parental exposure have been also observed in children and include increase in the risk of lower respiratory tract infections such as bronchitis and pneumonia, upper respiratory tract irritation, middle ear effusion, reduction in lung function, and both symptoms and new cases of asthma.

In this example, the relative risk estimates are combined with the information on smoking prevalence and mortality data in 15 EU countries to estimate the proportion of deaths due to lung cancer (LCa) and ischaemic heart disease (IHD), as well as estimated annual number of deaths attributable to ETS exposure in nonsmokers married to smokers.

4.2 Identification of Hazard and Sources

Environmental tobacco smoke is produced by smoking tobacco. It is a combination of the smoke exhaled by the smoker and the smoke released directly from the burning cigarette between and during the puffs. ETS is a complex mixture containing over 4000 compounds, including more than 40 known or suspected human carcinogens as well as other toxic agents. The known human carcinogenic compounds are benzene, 2-naphthylamine, 4-aminobiphenyl, nickel and polonium-210, and the toxic substances include carbon monoxide, nitrogen oxides, ammonia, and hydrogen cyanide.

4.3 Dose-Response Assessment

Due to the complexity of the ETS composition and variation of the exposure in time and space, as well as the delayed nature of most of the health outcomes, for the determination of the dose-response relationship mostly indirect indicators of exposure have been used. The most consistent measures of association have been obtained through the assessment of the prolonged presence of smokers in the indoor environment of the studied subjects, e.g. spouses, co-workers or parents.

Most studies indicate a rather small increase in the health effects but the results are consistent and allow to combine larger number of studies for estimation of the association. Recent meta-analysis of close to 50 epidemiological studies on the association between lung cancer and exposure to ETS from spousal smoking confirms this association and provides quantitative relative risk estimates for nonsmokers who live with smokers (*Hackshaw et al. 1997*). Relative risk (RR) estimate(and 95% CI) based on the results of this meta-analyses,

are 1.24 (1.13 - 1.36) for females, and 1.34 (0.97 - 1.84) for males (Hackshaw *et al. 1997*). The less precise estimate for males results from a scarcity of relevant data. While there were 37 studies in nonsmoking women, involving 4626 LCa cases in close to 1/2 million population at risk, only 9 studies included nonsmoking men, with 274 LCa cases and 117 thousand population at risk.

Also for IHD relative risk estimate was derived from the meta-analysis of 19 studies (Law *et al. 1997*). The risk of IHD is estimated to increase 1.30 times (95% CI: 1.22 - 1.38) in persons exposed to spousal smoking in comparison to the risk of people whose spouse has been a non-smoker. There is no major differences between sex-specific estimates, or for the estimates for fatal and non-fatal IHD cases.

4.4 Exposure Assessment

Several methods can be use to assess exposure to ETS (WHO 1997). They range from use of questionnaires, through microenvironmental monitoring for substances contained by ETS (nicotine in vapour phase or respirable particulate matter) to testing for biomarkers of exposure (nicotine or cotinine in blood, saliva or urine for assessment of the exposure in the recent 24 hours or for nicotine in hair for an assessment over the recent few months). Due to their widespread use in epidemiological studies deriving exposure - response associations and relative availability of population data on frequency of active smoking in the population, the most feasible exposure indicator for risk assessment is the proportion of non-smoking population having long term contact with smokers, e.g. non-smokers married to a smoker.

Also time of exposure is important for the risk assessment. For LCa, exposure as long as 20 years before the disease is believed the most relevant as a risk factor and, therefore, the smoking prevalence data from early 1970s were used in analysis of LCa mortality in 1990, similarly to that of Tredaniel (1997). For IHD, the epidemiological studies have used contemporary (passive) smoking data as an exposure indicator. In this analysis, national estimates of smoking frequency in 1990 (or in the closest available year) were used.

Since there are no data on proportion of non-smokers married to a smoker (Pe) in each of the countries, the national sex-specific data on proportion of smokers in adult population from the WHO Health for All data base were used for the present analysis. It was assumed that the frequency smokers is independent of marital status, and that there is a concordance of smoking habits within the married couple. The calculations were made assuming that the odds for a smoker to be married to a smoker are 3 times higher than the odds for a smoker to be married to a non-smoker (concordance ratio CR = 3). (CR=2 and 4 were used as well in sensitivity testing). Estimates of Pe, separate for males and females, and for 1970s and 1990, are presented in Table 4.1.

4.5 Risk Characterization

Methods

The method of estimation is based on calculation of attributable proportion AP and attributed number of cases, routinely applied in epidemiology (Last 1995). Since a number

of parameters necessary for calculation of these estimates (such as proportion of non-smokers married to smokers, or number of disease cases in this group) are not readily available in statistical data bases, the methods used by Tredaniel *et al.* (1997) were followed here. This example repeats calculations of the latter report using the updated relative risk estimates, and adds similar calculations for IHD.

Proportion of disease cases attributed to the exposure can be calculated as attributable proportion APe:

$$APe = (RRe-1) / (RRe-1+1/Pe)$$

where:

RRe is the risk of disease in non-smoker married to a smoker relative to the risk in non-smoker married to a non-smoker

Pe is the proportion of non smokers married to a smoker (our proxy indicator of exposure)

The number of cases attributed to the ETS exposure can be calculated, for each sex separately, as:

$$Ne = Na * APe$$

Na is the number of cases occurring in married non-smokers:

$$Na = Nt * (1 - APs) * Pm * (1 - Ps)$$

where:

Nt is total number of cases in 1990. Number of LCa deaths was taken from Tredaniel *et al.* (1997), and the number of IHD deaths is based data reported by the Member states for the project "Atlas of mortality in Europe" (WHO 1997b).

Pm proportion of married adults (data for early 1970s were used for both LCa and IHD estimation; more recent statistics were not readily available, and the rates are fairly constant).

Ps proportion of active smokers (in 1970 or 1990, respectively)

APs proportion of cases attributable to active smoking,

$$APs = (RRs - 1) / (RRs - 1 + 1/Ps)$$

RRs is relative risk of the disease due to active smoking.

For LCa, the RRs =10 was used.

For IHD, RRs = 2, with RRs = 1.8 or 2.5 used in sensitivity analysis.

Besides the APe and Ne estimates for the central RRe value, the calculations were repeated for the lower and upper limits of 95% confidence interval of the RRe provided by the meta-analyses. The sensitivity analysis involved central RRe only.

Results

Between 8% and 14% of LCa deaths in non-smoking women can be attributed to husband's smoking, with upper limits of estimates reaching 18% (Figure 4.1). For non-smoking men, the best estimate of the proportion of LCa attributable to wife's smoking varies between 1% and 9%. Upper limits of estimates reach 20%. Since the 95% confidence interval of RRe estimate from the meta-analysis contains 1.00, a possibility for zero increase in risk of LCa in males can not be rejected based on the available data.

In non-smoking women, the proportion of IHD deaths attributable to spousal smoking was estimated to be between 6% and 14% in individual countries of EU (Figure 4.2). For non-smoking males, this proportion varied between 2% and 8% of IHD deaths. Since the RRe estimates reported by epidemiological studies were similar for fatal and non-fatal cases, it can be assumed that the similar proportions of non-fatal IHD cases can be attributed to ETS in the EU countries.

In 1990, the total number of LCa deaths reached some 138 thousand in males and 34 thousand in females of the 15 EU countries. Active smoking was responsible for 78% - 85% of the cases in males and 45% - 81% in females (assuming RRs = 10 for LCa). The number of LCa deaths in non-smokers attributed to the spousal exposure to ETS was estimated to be 657 females and 328 males.

The total number of fatal IHD cases was close to 310 thousand in males and 271 thousand in females in the 15 EU countries in 1990. Proportion of the deaths attributable to active smoking was much smaller than that for LCa, and ranged from 21% to 32% in males and from 11% to 27% in females (assuming RRs = 2.00 for IHD). The number of IHD deaths in non-smokers was, therefore, considerably greater than that due to LCa. In the result, the number of IHD deaths in non-smokers attributed to spousal exposure to ETS exceed 11 thousand deaths in women and 5 thousand deaths in men (Figure 4.3).

Sensitivity analysis, assuming various values of CR (2, 3 or 4) and RRs indicates that the estimates of attributable number of cases, both for LCa and IHD, may vary within +-20% from the central values presented here. However, the overall impact of ETS on health in the form of increased number of IHD cases remains by order of magnitude higher than that of increased number of LCa deaths.

Table 4.1 *Exposure to spousal ETS in different countries of Europe.*

	1970		1990	
Country	Women married to smoking men	Men married to smoking women	Women married to smoking men	Men married to smoking women
Austria	36.3%	6.0%	31.4%	15.2%
Belgium	44.7%	14.3%	31.1%	17.8%
Denmark	52.9%	29.3%	27.8%	27.2%
Finland	40.9%	11.4%	26.9%	14.0%
France	56.1%	19.5%	39.2%	21.6%
FRG	58.3%	16.5%	26.7%	11.8%
GDR	58.3%	16.5%	26.7%	11.8%
Greece	47.4%	5.4%	52.2%	18.7%
Ireland	37.4%	24.4%	23.9%	21.7%
Italy	49.0%	8.8%	30.8%	10.8%
Luxemb	44.7%	14.3%	34.3%	16.5%
Netherlands	60.5%	26.2%	30.8%	21.7%
Portugal	38.6%	5.3%	42.8%	6.8%
Spain	64.0%	4.6%	38.4%	13.1%
Sweden	46.2%	21.1%	19.9%	20.0%
UK	56.3%	25.4%	23.9%	21.7%

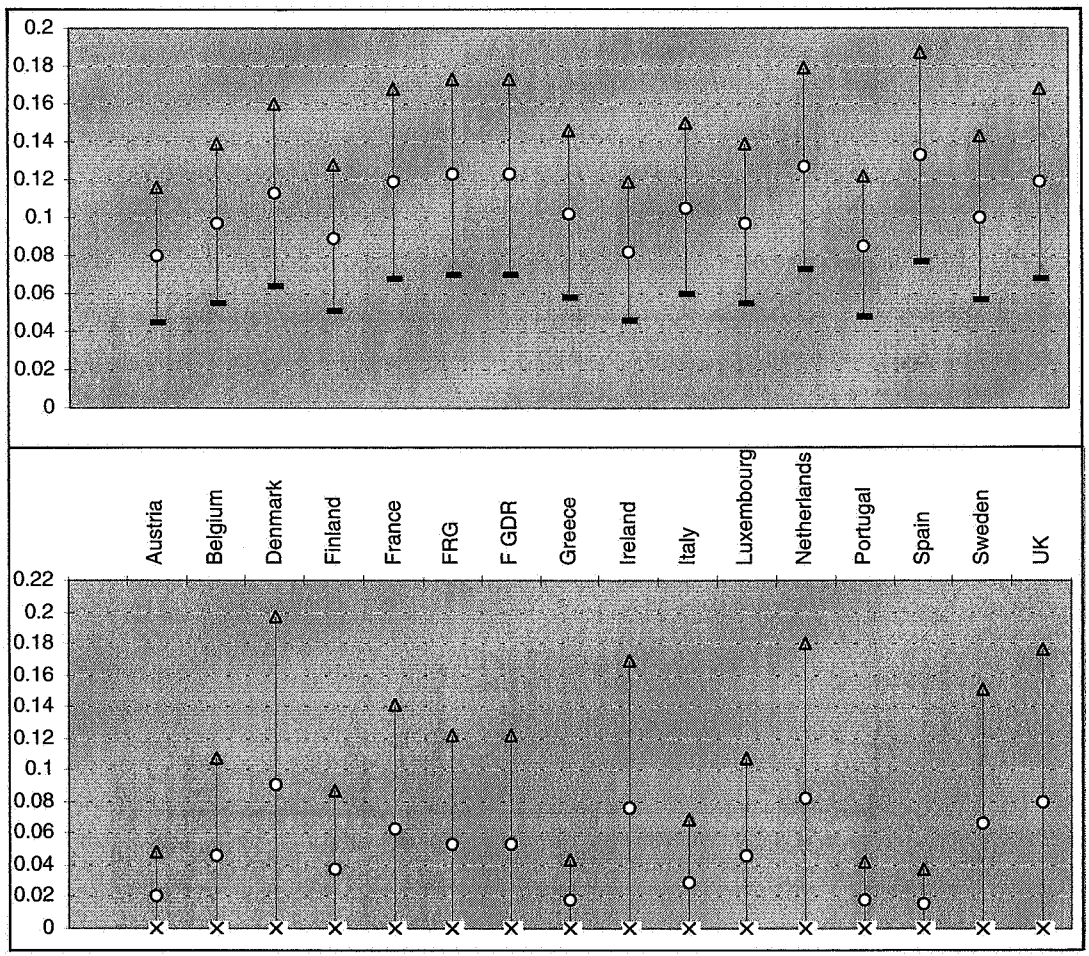

Figure 4.1 *Proportion of lung cancer deaths in non-smoking women (above) and men (below) attributable to spousal smoking.*

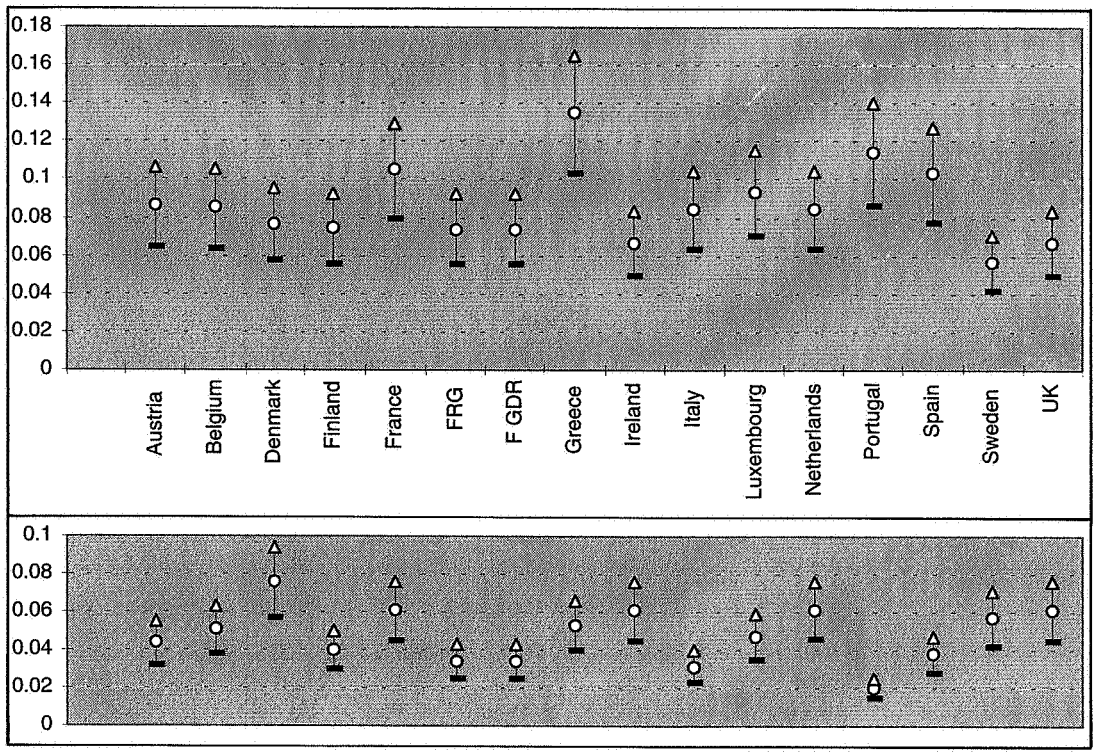

Figure 4.2 *Proportion of ischaemic heart disease deaths in non-smoking women (above) and men (below) attributable to spousal smoking.*

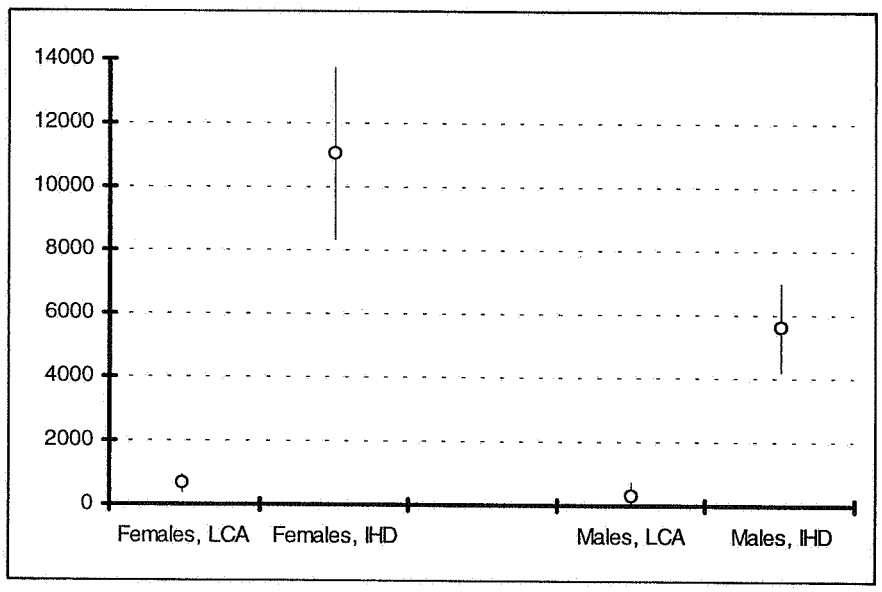

Figure 4.3 *Number of lung cancer (LCA) and ischaemic heart disease (IHD) deaths in non-smokers attributable to smoking spouse in the 15 EU countries, 1990.*

4.6 Relevance for Risk Management and Risk Communication

The estimates of effect contribute to the understanding of the magnitude of health impacts in Europe and allow to appreciate in a more complete way the contribution of ETS to the disease burden. While the relatively few cases of LCa may be acceptable for some individuals, the several times more frequent heart disease may be sufficient to prevent the exposure through individual behaviour of non-smokers or through their influence on smokers aimed at their smoking cessation (leading, in addition to the reduction of risk from ETS exposure, to reduction of even more severe health risks from active smoking).

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5 VOC EMISSIONS FROM FLOORING MATERIALS

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5.1 Introduction

This example deals with the assessment of risks associated with emissions of volatile organic compounds (VOCs) from new flooring materials.

Human exposure to VOCs is a public health issue (WHO 1989). In the European Community, population exposure to air pollution by VOCs inside buildings is much larger than outdoor exposure, because (a) the concentrations of most VOCs are higher indoors than outdoors, (b) on average the time spent indoors is about tenfold greater than the time spent outdoors and (c) susceptible sub-populations such as infants, elderly and people of poor health spend most of their time indoors.

New flooring materials are an important source of VOCs in indoor air. Every year, large quantities of new flooring materials are introduced into buildings. Between 1990 and 1995 on average about 1.7 billion m2 of flooring materials have been consumed annually in the European Union (see Table 5.1). For the textile flooring materials only, this corresponds to a wholesale price of about 5 billion Euro per year. Hence, flooring materials are also economically important.

Table 5.1. *Annual consumption of flooring materials in the European Union. Average values calculated from statistical data for the period 1990 -1995 *)*

Flooring material	**Consumption [mill. m²]**	**Calculated share [%]**
textile	938	56
stone and ceramic tiles	329	20
plastics and cushioned vinyls	247	15
parquet and wood	67	4
linoleum	32	2
rubber (excluding mats.)	15	1
laminate	24	1
cork	12	1
total consumption	**1 663**	**100**

**) Source: TFI, Deutsches Teppich-Forschungs Institut e.V., Aachen*

This example deals with the potential hazards of VOC emissions from new flooring materials after their installation in indoor environments. VOC emissions from new flooring materials are called 'primary' emissions in contrast to 'secondary' emissions, i.e. emissions of VOCs which have been adsorbed on a flooring materials after their installation in the indoor environment and which have been emitted by other sources. Secondary emissions are not included in the risk assessment presented here.

Flooring materials may also be the origin of other hazards. Examples are the release of low or non volatile organic compounds (e.g. plasticisers or biocides) or of microbiological pollutants (e.g. in case of non-appropriate cleaning or water damage), or accidents due to sliding or stumbling. In addition, flooring materials may cause hazards during their production and disposal. All these hazards are not considered here.

The risk of VOC emissions from new flooring materials is an example of the situation where a potential hazard is due to a well identified source of pollution and raises considerable concern but where the risk cannot be quantified because of a nearly complete lack of exposure/response data at the exposures of interest. Nevertheless, because of the public concern about VOC emissions, the potentially severe health and economic consequences of the emissions (see following section) and a growing interest of manufacturers in low emitting products and their certification, the reduction of VOC emissions from flooring materials to values as low as reasonably achievable (ALARA) appears highly desirable and possible.

In order to provide a rational basis for this type of risk management, a risk assessment approach as proposed by Wilson and Crouch (1987) is needed that provides a way of examining risks so that they can be better avoided or reduced. Report number 18 of this series (ECA-IAQ 1997) has recently proposed a RA procedure for flooring materials which - using expert judgement and the very limited knowledge available - provides a basis for risk management and is briefly described in this example.

5.2 Identification of Hazards and Sources

Many VOCs found in flooring material emissions are known to have short-term and long-term adverse effects on human health and comfort. With respect to comfort VOCs are associated with the perception of odours. Adverse health reactions include irritation of mucous membranes, mostly of the eyes, nose and throat, CNS mediated effects such as headache, tiredness or nauea and long-term toxic reactions of various kinds (ECA-IAQ 1991).

The first two types of health reactions are typical for the sick building syndrom (SBS) and therefore, VOCs are often suspected to be a cause of SBS. A few VOCs that may be emitted by flooring materials are human carcinogens and some VOCs are potential sensitizers (Flyvholm and Bakke 1994).

The sources considered in this example include all new solid flooring materials such as carpets, vinyl flooring, wood parquet or linoleum. Not included are materials used for *in situ* installation and/or surface treatment of flooring materials such as glues or lacquers.

5.3 Exposure/Response Assessment

Dose/response data for mixtures of VOCs as they are typically emitted from flooring materials are not available for any health effect. Also for individual VOCs dose-response data down to concentrations resulting from flooring material emissions (see below) are nearly completely lacking. Only for carcinogens, the no-threshold hypothesis combined with linear extrapolation allows a risk estimate based on lifetime inhalation unit risks.

Therefore, exposure/response assessment for effects of VOC emissions from flooring materials other than cancer requires drastic simplifications. The evaluation procedure adopted by ECA-IAQ (1997) uses two different approaches for toxic and sensory effects respectively.

Toxic effects. The ECA-IAQ procedure substitutes dose-response data for VOC mixtures by estimates of VOC concentrations below which there are no indications that VOCs may cause health effects. The estimates are based on the following simplifying assumptions:

1. For individual compounds, all effects (except carcinogenicity, see above) are assumed to have a threshold which can be derived from no observed effect levels (NOELs) or data based on these levels such as air quality guidelines (AQGs) or occupational exposure limits (OELs).
2. OELs are divided by an uncertainty factor in order to account for the longer exposure duration in the indoor environment compared to occupational exposures and the higher sensitivity of the general population compared to workers and the obtained values are rounded down to one significant digit. Thereby 'lowest concentrations of interest' (LCIs) are determined.
3. Only VOC concentrations[3] 5 $\mu g\ m^{-3}$ are taken into consideraton and are called 'indoor relevant concentrations'. This concentration value is only half the lowest AQG (for formaldehyde) and more than a thousand times smaller than the lowest OEL value used for determining LCIs. There may be chemicals with toxic effects even at lower concentrations. However, for the time being no such chemicals are known to be emitted from flooring materials.
4. Effects of individual compounds are assumed to be additive.

The indoor relevant concentrations have to be determined by small chamber emission tests using appropriate test conditions and a simplified exposure model (see below). For RA of the emissions, the concentrations are divided by the respective LCIs and summed up. The sum must not exceed the value 1.

Sensory effects. At indoor concentrations of VOCs typically resulting from flooring material emissions (see below), sensory effects (odours, sensory irritation of eyes, nose and throat) are most likely to occur (see also ECA-IAQ 1999). By their very nature, these effects can only be measured by human test panels. No models exist which would allow to determine these effects for mixtures based on data obtained for individual compounds.

For these reasons, the ECA-IAQ procedure prescribes panel tests of sensory irritation and of odour of VOC emissions. Material emissions have to be tested under indoor relevant conditions, i.e. applying realistic low area specific ventilation rates (see below). Not more than 10 % of the test panle members must perceive irritation.

TVOC. The ECA-IAQ procedure sets also limits for the emission of total volatile organic compounds (TVOC) after 3 and 28 days of testing in order to prevent both strong sensory irritation of people installing flooring materials and, independently from specific toxicological considerations but following the ALARA principle, longer term TVOC concentrations that exceed typically occurring lower range indoor levels.

5.4 Exposure Assessment

As exposure/response assessment, also exposure assessment is based on simplifying assumptions, the most important of which are:

1. The new flooring material makes the predominant or at least a major contribution to indoor VOC exposure.
2. The indoor concentrations resulting from the VOC emissions depend only on the emission factor E [$\mu g\ m^{-2}\ h^{-1}$] and the area specific ventilation rate q [$m^3\ m^{-2}\ h^{-1}$]. This presumes that VOCs are not adsorbed to indoor surfaces and do not react and that the air in the room under consideration is well mixed.

These two assumptions provide a very simple steady state model that links concentrations C [$\mu g\ m^{-3}$] with emission factors E and the area specific ventilation rate q by the equation:

$$C = E / q .$$

Using emission factors of 27 flooring materials determined in small test chambers by Saarela *et al.* (1994) and an area specific ventilation rate q = 1.25 $m^3\ m^{-2}\ h^{-1}$ (corresponding to 0.5 air exchanges per hour for rooms with 2.5 m height), average concentrations of 61 individual VOCs range from 0.8 to 290 $\mu g\ m^{-3}$ with a 50%'ile of 13 $\mu g\ m^{-3}$, a 75%'ile of 34 $\mu g\ m^{-3}$, a 90%'ile of 121 $\mu g\ m^{-3}$ and a 95%'ile of 255 $\mu g\ m^{-3}$. The corresponding maximum concentrations range from 0.8 to 1145 $\mu g\ m^{-3}$. Because most individuals in Europe spend in the order of 90% or more of their time indoors, they are assumed to be exposed continuously to this concentration estimate.

The above described exposure and exposure-response assessments provide the basis of an evaluation procedure intended for labelling of flooring materials that are supposed not to present a hazard with respect to their VOC emissions. The evaluation procedure is summarized schematically in Figure 5.1.

Emissions from nine flooring materials, including two cushion vinyl products, a wax and an oil treated beech parquet, dried spruce, birch and pinewood planks and varnished oak and pinewood planks were subjected to the toxicological (not the sensory) part of the evaluation procedure shown in Figure 5.1 (ECA-IAQ 1997a, appendix 6). Out of these, three materials did not fulfil the conditions for labelling, one material fulfilled the conditions only at an area specific ventilation rate $\geq 1.25\ m^3\ h^{-1}\ m^{-2}$ and one material only at $\geq 2.5\ m^3\ h^{-1}\ m^{-2}$.

5.5 Risk characterization

The potential risks of VOC emissions from flooring materials that are taken in consideration by this RA example are cancer, health effects that are reflected in AQGs and OELs and, in addition,

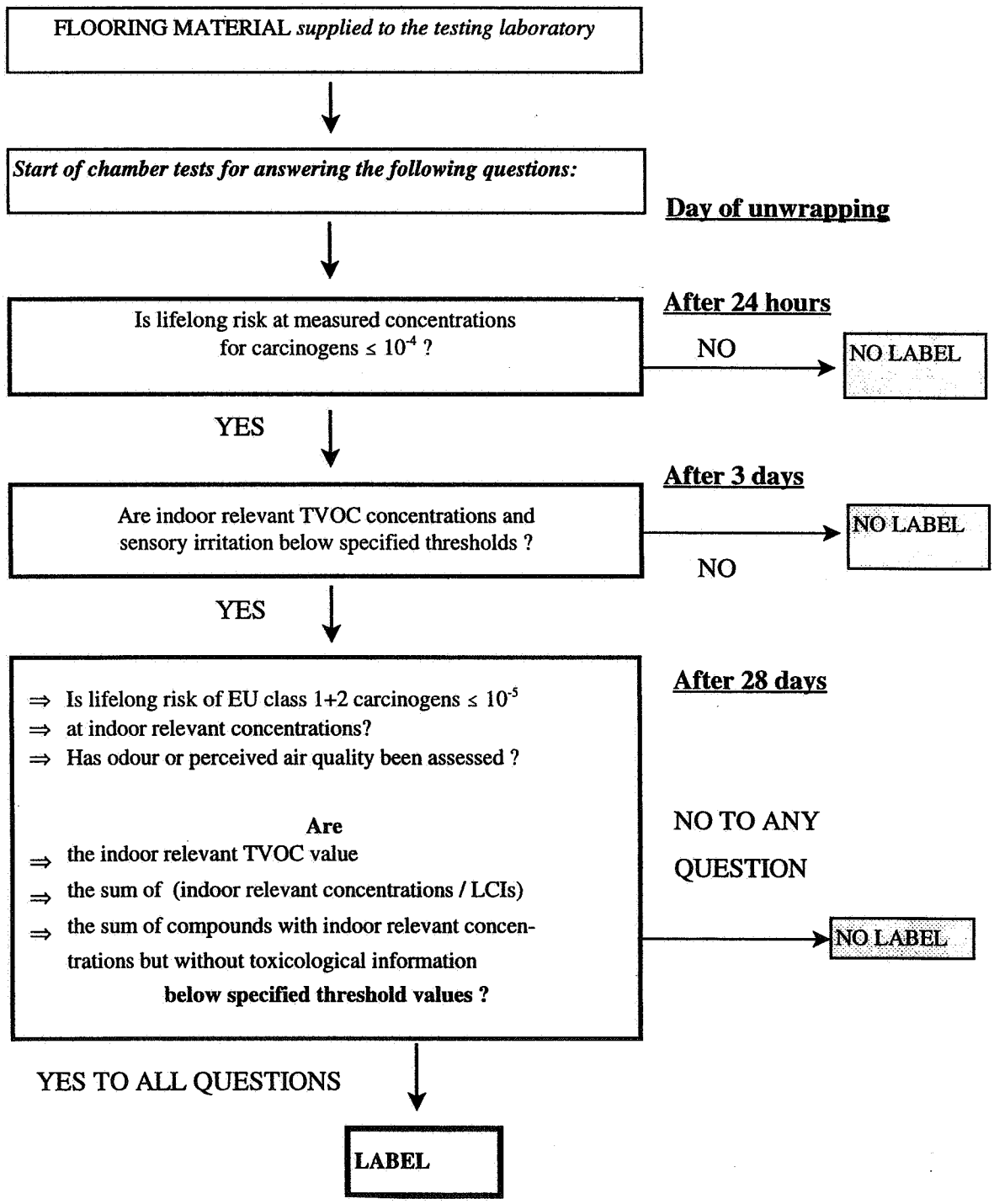

Figure 5.1 *Scheme of the evaluation/labelling procedure for VOC emissions from carpets.*

sensory irritation. AGQs and OELs are mostly based on acute, reversible effects but sensitizing properties are also taken into account.

Because of the non-availability of dose-response data and the scarceness of representative exposure data, meaningful risk estimates for health and comfort effects are not possible. Therefore, the approach presented here is of a preventive nature, i.e. it aims at reducing exposures below effect thresholds by source control.

Although effects usually attributed to indoor air pollution are reversible and not considered severe, a preventive approach appears justified because

- the entire population is at risk and
- the potential economic impact of poor indoor air quality is quite high and has been estimated to be in the order of tens of billions of ECU per year in Western Europe (ECA-IAQ 1996).

The procedure described here is supposed to provide flooring materials that do not present a hazard with respect to their VOC emissions. However, uncertainties of unknown magnitude remain with respect to potential effects of long term-low level exposures. Also, an unknown yet small percentage of the population that is hypersensitive to VOCs may not be protected.

5.6 Relevance for Risk Management and Risk Communication

As already mentioned in the introduction, this RA example is entirely aimed at risk management by reducing risks of VOC emissions from flooring materials to levels that, according to available knowledge, do not cause health or sensory effects to most people.

Risk communication can be effectively achieved by appropriate labelling of flooring materials (see Figure 5.1).

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6 TWO EXAMPLES ON ASSESSMENT OF HEALTH RISK IN DAMP/MOULD PROBLEM BUILDINGS

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6.1 Introduction

Mould, yeast, fungi and other microbes will easily grow in water damaged buildings. Buildings can be damaged by water from external sources, e.g. as a result of thawing ice and snow, flooding, seepage from the ground or by water from internal sources, e.g. leaking water pipes or sewers, or dampness from the kitchen, bathroom or sauna. Condensation of water to cold surfaces may also cause water damage.

Microbe growth caused by water damage or dampness in homes (Verhoeff and Burge 1997), offices (Lehtomäki *et al.* 1999), schools (Taskinen *et al.* 1997), day-care centres (Ruotsalainen *et al.* 1995), etc. has been associated with an increased prevalence of several symptoms and diseases in epidemiological studies. The health effects caused by indoor-air micro-organisms can be divided into irritating and non-specific symptoms, respiratory infections, allergic diseases, alveolitis and organic dust toxic syndrome and chronic bronchitis (Husman 1996). Inhabitants and workers of mouldy buildings are exposed not only to fungal spores but also to volatile chemicals (Pasanen *et al.* 1998), mycotoxins (Sorenson *et al.* 1987), glucans (Douwes *et al.*), and allergens of microbial origin (Menzies *et al.* 1998). Many of these exposures are inadequately known and no ways to measure the content of mycotoxins or specific mould allergens in the indoor-air exists.

EXAMPLE A: Company with an Indoor Air Problem

This company produces laboratory equipment in a building which has been built in four stages between 1955 and 1979. In 1990 major renovations were made before the production of laboratory equipment. The cellar consists of about 2 400 m^2 and the ground floor about 3 400 m^2. The number of employees had gradually risen from about 20 in 1991 to about 90 in 1996. Silicone containing lubricants and acryl containing glues are used in the assembly of the instruments and these chemicals were suspected for the irritation of eyes, upper respiratory tract and skin experienced by several employees. However, these symptoms were experienced also by employees not working with these chemicals and also in parts of the building where these chemicals were not used and which had a separate ventilation system. An industrial hygienist was called to assess the problem and in his preliminary walk through he smelled the typical mouldy smell inside the building.

6.a.2 Hazard identification

To find out if the indoor air problem was restricted to the use of lubricants and or due to some other indoor air problem, the symptoms of the employees were evaluated by a MM-40 questionnaire used in several countries in the evaluation of indoor problems (Andersson et al. 1998). High prevalence of work-place related eye and upper respiratory track symptoms were seen among the employees in all parts of the building including the cellar and the administrative section, where no lubricants nor glues were used. Thus, the exposure to these chemicals alone could not explain all the work-related symptoms.

The building itself was studied by several construction specialists, some of them working together with industrial hygienists and microbiological laboratories. Signs of water damages or dampness were found in large areas of the building. The foundation of the building was damp because the draining was lacking or blocked, and the ground, melting and rain water had free access to the foundation. The flat roof had several holes and cracks, and because of insufficient insulation the snow melted on the roof during the winter and water leaked into the ceiling structures through most of the year. Because of the insufficient insulation the walls and the ceilings were also wet by condensation. Moreover, three of the four ventilation units had condensation or leaking problems leading to mould growth.

Viable microbes were sampled only twice from the indoor air, but neither the count of colony forming units nor the identification of the micro-organisms suggested mould problem. However, in the material sampling of damaged flooring materials, paints, insulation etc. from different parts of the building abundant growth and micro-organisms - suggesting mould problems - were found. Samples were taken in several occasions. For instance in March 1997 21 material samples were taken and 16 of them contained micro-organisms indicating a mould problem. Thirty material samples from different parts of the building were analysed by SEM and 21 of them contained microbes more than ten-fold compared to reference values. From thirteen samples actinomycetes or some other micro-organisms - indicating moisture problems - were found.

Twenty-one material samples were analysed in two laboratories by SEM and by viable methods (Hagem-, DG18- and THG-agars). Using the laboratories' own criteria 15 samples were similarly classified (8 with no or minimal number of microbes or microbial growth and 7 samples indicating mould problem). Four samples suggested a mould problem by SEM but were considered non-damaged by viable methods. Two samples were damaged by viable methods but not by SEM. The predominant micro-organisms were *Aspergillus, Penicillium and Streptomyces.*

6.a.3 Exposure Assessment

To evaluate the exposure to moulds and yeasts the IgG antibodies against eight moulds and yeasts common in Finnish mould damaged buildings were measured from the serum samples of 70 employees. In this group the antibodies against all eight microbes were common and it suggested that the group had a common source of mould exposure. Antibodies against *Aspergillus fumigatus* were present in about 70% of the serum samples of the studied employees. The reference prevalence of the similar antibodies based on the samples analysed in the laboratory is 58% (Reiman *et al.* 1998).

6.a.4 Risk Characterisation

The adverse health effects caused by the mould exposure in this production building were evaluated by semi-structural health interviews and health check-ups done by two occupational physicians. Furthermore, the number and the reasons of contacting the occupational health unit during 1991-1996 were analysed. Most of the production had been moved to another building about six months before the medical check-ups and changes in the symptoms of employees were studied. One of the employees had asthma at the time he

was employed. However, additionally three employees developed asthma while working in the damaged building, which means that during 1991-1996 the incidence rate of asthma was 1 new case per 100 person-years, which is about 4 times higher ($p < 0.01$) than in the Finnish adult population in general (1996: 2,4 cases/1000 persons in the age group 16-64 years).

About 33 of the employees were estimated to have developed building related upper respiratory track or eye irritation symptoms and in 30 cases the symptoms disappeared or were relieved when the production was moved to another building. The corresponding figures for non-specific building related symptoms (including headache, chills, tiredness and dizziness) were 25 and 24 and for the reoccurring respiratory infections 11 and ten.

In 1996 the employees had an average of 4.8 contacts to the occupational health care unit, which is well above the national average 2.0. Most of the contacts had been made because of respiratory complaints. In the worst damaged areas an additional number of sick leaves and days lost because of respiratory diseases was seen while working in the damaged building. Soon after the production was transferred to another building, the number days lost because of respiratory diseases decreased (from 2.6 days/person-year to 1.3 days/person-year).

6.a.5 Risk Management

Most part of the production was removed to another building resulting in a noticeable improvement in the employees' health. The building itself has so severe damages that it is questionable if the repairs needed are economically reasonable.

EXAMPLE B. Building and the Symptoms of the Employees

This example deals with a 15-year-old office building with about 350 workers. The first complaints of respiratory and other symptoms, e.g. persistent cough, phlegm, wheezing, nasal congestion and excretion, airway irritation, headache and tiredness, appeared in 1992 among workers whose offices were located in the southern side of the building. Since then until 1997 about 15-20 workers around the building complained of similar symptoms.

6.b.2 Identification of Hazard and Sources

As symptoms seemed to be related to the indoor air quality of the building and as various water leaks through the roof and other moisture damages were known to have occurred in the building during the past years, dampness and possible microbial growth in the building structures and the ventilation system were suspected to be the most probable causes of the complaints. Technical surveys made by building engineers between March 1995 and May 1996 revealed that the main reasons for moisture damages included a lack of an airing slit and a wind-shelter board between wall insulation and brick cover, frost damages in foundation and faulty design of window sills. The relative humidity in the external brick walls measured in April 1996 and 1997 from 24 points were low, ranging from 15 to 32% at 17-23°C. However, the relative humidity in the brick cover and insulation material, especially below windows, was occasionally supposed to be sufficiently high to promote

microbial growth in window frames. No signs of moisture damages were detected in interior constructions and surfaces at that time. The ventilation system was also inspected in December 1995. Air leakages around the filter cassettes were detected and melting of snow had left smears on the bottom of filter chambers.

In 1997 the ducts and ventilation system were cleaned again in order to improve conditions. In addition, about half of the outer walls (only on the side of outer yard) were repaired by making the ventilation cavities within the walls broader and by increasing the slope of the window edge to decrease moisture content in the walls (Kokotti *et al.,* 1999).

6.b.3 Exposure Assessment

To evaluate the presence of microbial growth in constructions and the exposure of workers to micro-organisms, microbial concentrations and composition in the indoor air, building materials, and in dust accumulated on ventilation ducts were measured and IgG antibody levels against 8 or 14 indicator microbes were determined in serum of symptomatic as well as non-symptomatic workers.

Between 1993-1997, 36 air samples were collected in wintertime with a 6-stage impactor from 10 offices, and 30 material samples from window frames and insulation material as well as 17 dust samples from ventilation ducts were taken for microbiological analyses. Most of the samples were collected in areas of the building where the worst moisture damages were observed and workers complained of symptoms. Low levels of airborne microbial concentrations (fungal concentrations ranged from 2 to 80 cfu/m^3 (colony forming units) and bacterial concentrations from 10 to 360 cfu/m^3) were measured and only 5 of 30 material samples were detected to be contaminated by moulds. Melting of snow was also observed to have caused slight growth of yeasts in dust of ventilation ducts. The predominant micro-organisms in the air, material and dust samples were *Penicillium, Aspergillus* (*A. fumigatus, A. glaucus, A. versicolor, A. niger*), *Acremonium, Cladosporium, Oidiodendron, Phialophora, Polyscutalum, Phoma, Rhinocladiella, Exophiala, Aureobasidium, Paecilomyces, Trichoderma, Ulocladium, Wallemia sebi*, yeasts and actinomycetes, most of which are indicator organisms for moisture and mouldy damages.

The microbial measurements after the first phase of wall repair in 1998 showed that low concentrations of some microbes indicating moisture problem such as *Sphaeropsidales, Aureobasidium, Wallemia, Rhodotorula and Aspergillus versicolor* were still found mainly in indoor air but not in air of air handling systems (Kokotti *et al.*, 1999).

In 1995 and 1996, microbial exposure was also monitored by determining specific IgG antibody levels against *Aspergillus fumigatus, Acremonium curtipes, Cladosporium cladosporioides, Geotrichum candidum, Penicillium brevicompactum, Phialophora bubakii, Rhodotorula glutinis,* and *Sporobolomyces salmonicolor* in serum samples of 27 workers. In addition, IgG antibody levels against *Aspergillus niger, Humicola grisea, Paecilomyces variotii, Rhizopus nigricans, Streptomyces albus* and *Trichoderma viride* were measured in serum of 11 workers. Offices of 21 workers were situated in areas where the worst moisture damages were detected while offices of the remaining 6 workers were located in undamaged sides of the building, and, thus, IgG levels of those workers served as control

values in this assessment. The antibody assays were made from 10 exposed and 6 control workers in 1995 and from 11 exposed and 6 control workers in 1996. Moderate or high levels of IgG antibodies against *Aspergillus fumigatus, Rhodotorula glutinis, Sporobolomyces salmonicolor, Phialophora bubakii,* and *Acremonium curtipes* were detected among the workers. However, a statistical difference in IgG levels against yeasts (*Rhodotorula glutinis,* p=0.08 and *Sporobolomyces salminicolor,* p=0.03) was only found with the Fisher's exact test between the exposed and control workers whose serum samples were taken in 1996.

6.b.4 Risk Characterization

According to the technical survey, environmental measurements and exposure assessment, the exceptional exposure to micro-organisms was limited to the worst damaged areas of the building regarding about 20% of all the employees worked in the building. Building related symptoms and indoor environmental factors were evaluated by the MM-questionnaire. The results of the questionnaire were analysed in groups of 10 workers who worked at the same departments or departments next to each other in the same stock of the building and under the same air-handling unit. Only in one of the analyzed groups, the complaints of building related symptoms exceeded 20% and the environmental complaints exceeded 40%. Response rate to the questionnaire was 63%. According to these criteria, there seemed to be no generalized indoor air problem in the building, even if certain individuals in the worst damaged areas had daily symptoms, such as tiredness, headache, eye irritation and skin symptoms.

The first questionnaire survey by Kokotti *et al.* (1999) was conducted in spring 1997 using MM-40 questionnaire (Andersson *et al.,* 1993), which indicated that the employees were unsatisfied with the indoor air quality. The symptoms and complaints were asked again by the second survey in spring 1998 after the partial repair including only the outer walls in the side of outer yard. The data from remedied and unaltered areas were analyzed separately. In the summer 1998, the outer walls in the side of inner yards were also repaired and the third survey was conducted in spring 1999. Response rates of the three surveys were 80% in 1997 (222 persons) before partial repair, 65% in 1998 (157 persons) after the partial repair and 60% in 1999 (141 persons) after the complete repair.

No change was observed in the symptoms or in the complaints of the employees in these two areas before the total repair of all the walls. Stuffy air was still a common complaint. This could be due to the short time between the surveys because the third survey that was conducted after the complete repair resulted in less complaints about stuffy air in the areas repaired first. In addition, inadequate ventilation could cause inconveniences (Kokotti *et al.,* 1999).

The study by Kokotti *et al.* (1999) revealed that the partial repair of the suspected moisture problem was not enough and all the problems (constructional and ventilation technical) should be repaired at the same time in the whole building.

6.b.5 Risk Management

Many indoor problems, which were difficult to explain, were found in the study building. The time and money spent on this diagnostics was remarkable.

The problems connected to the process of improving the indoor air quality in the building was divided by Korhonen *et al.* (1999) into five phases: The first was the organization, which did not exist in any official form. The second was the problem identification concerning the responsibility of deciding whether there exist a real problem. The third phase, the survey of causes was even more difficult to divide according to the responsibility in examination of the causes of different concentrations and complaints and symptoms found by questionnaires. The fourth phase of the repair and mitigation was the most distinct area when the costs and responsibility were in consideration. However, there existed some problems in the choice of the different technical methods in repair work. The follow-up studies were found to be necessary, although, the responsibilities and the extent of the studies were not uncomplicated.

The conclusion of this example was, that in every large building there should be a research manager who understands technical competence of buildings (structures, ventilation and maintenance) and the health effects of the indoor air contaminants (e.g. microbes and volatile organic compounds). In addition, the occupational health and safety organization should take the initiative, and participate in the realization and the control of the survey of indoor air problems.

6.6 Comment on Both Cases

There are several possible causes of health effects related to damp and mouldy buildings including the number of viable microbes in indoor air, volatile organic compounds released by the microbes, mycotoxins, endotoxins and allergens. Since the exact cause is not known and in many cases there are no methods to measure the concentration of the possible causes (like measuring the concentrations of mycotoxins and allergens), the dose/response evaluation is in most cases impossible. Today, the only way to assess the exposure is by using the mould growth and IgG antibodies of the exposed workers and inhabitants as a rough surrogate of the true health hazard. However, the basic reason for moisture and possible mould problems is always connected to the technical competence of the building and this should be the main task in the indoor air quality risk management.

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7 RISK ASSESSMENT OF VENTILATION SYSTEMS

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7.1 Introduction

Heating, Ventilation and Air-Conditioning (HVAC) of buildings is becoming an ever more common amenity. Building occupants' comfort expectations have grown and so have building services, introducing complex new demands on HVAC systems. New challenges and new solutions, for reaching optimum overall system performance, make this topic a lot more demanding than it used to be. HVAC systems if properly designed, installed, operated and maintained can improve indoor thermal conditions and air quality. However, if all these stages are not carefully implemented, HVAC systems can constitute a source of problems (ranging from a simple source of noise to disturbing high indoor air flows) and in some extreme cases a health risk with even hazardous effects for humans, because of a negative impact on indoor air quality.

Regrettably, today too many HVAC-systems deteriorate rather than improve the indoor air quality (IAQ). This is a serious problem all involved disciplines must respond to in a respectful way rather than denying its existence in order to evade professional or economical responsibilities (Hanssen 1997). This example will therefore be based on a descriptive and qualitative approach to the case of risk assessment of a ventilation system with the main focus on hazard identification and sources.

7.2 Identification of Hazard and Sources

In order to identify hazard and sources related to air handling systems it is important, and necessary, to understand the wide diversity of HVAC-plants. Although the main principles for designing HVAC-systems are very similar, we have several options with regard to configuration. In each design case, the engineer has to combine components and equipment in order to assemble a HVAC-system that meets the demands required by the specific building.

Further, the mutual interactions between user and building implies that the performance of the HVAC-system also depends on the behaviour of the user; or in other words: whether or not the user works in harmony with the technical systems that are incorporated into the building. Until the present time, the effects of such interactions have been little studied (Lindvall and Valbjørn 1987).

Large spaces and other facilities which need considerable supply of fresh air are usually served by single zone or variable volume all-air systems. Separate air-handling units can serve each zone, although multizone, dual-duct or reheat types can also be applied with lower operating efficiency.

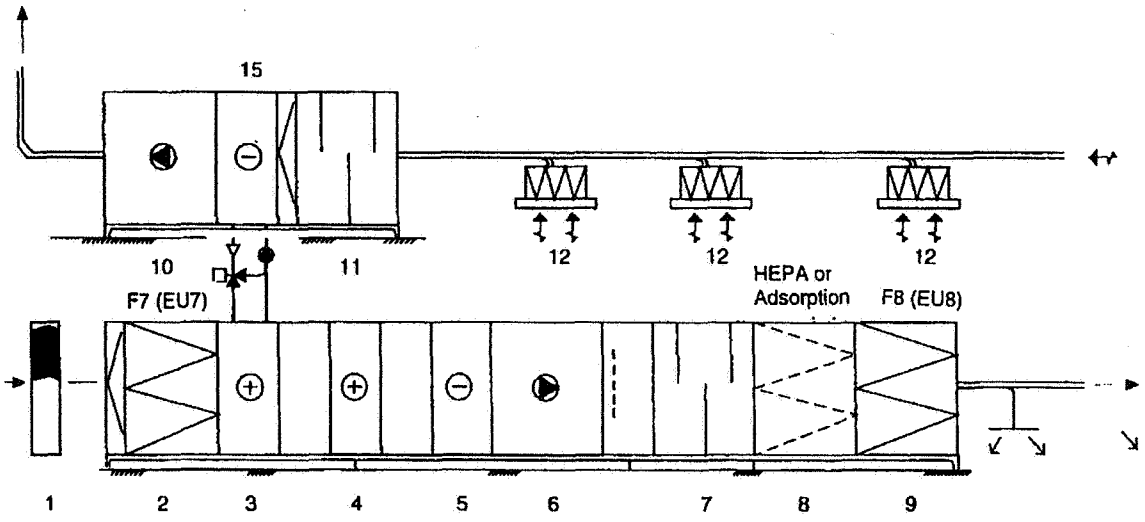

Figure 7.1 *Example of lay-out, all-air single zone system (Flatheim and Thomassen 1993): Outside air intake, louvre (1), primary air filter section (2), heat recovery unit (3), heating (4) and cooling (5) coil section, supply-air fan (6), sound attenuator (7) secondary filter(s) (8,9), sound attenuator (11), heat recovery unit (15), return-air fan(10).*

Centrally located air-handling units may include (see Figure 7.1): outside air intake with louvre and outside air damper, return air damper, mixing plenum, primary air filter section, preheat coil, heat recovery unit, heating and cooling coil section, supply-air fan section, sound attenuator, secondary filter(s) and humidifier. On the return-air side we may have: sound attenuator, filter section, heat recovery unit, return-air fan section, relief air damper, louvre and general exhaust (McQuiston and Parker 1988).

The air-distribution system comprises ductwork, fire and smoke dampers, thermal insulating materials, air flow control devices, noise reducing devices, sound attenuator, diffusers and grilles. The return air system consists of almost the same components.

Because of the great variety of components and equipment involved and the diversity of lay-outs of HVAC-systems, it is not possible to identify all hazards and sources on a general basis. However, some of the main risk factors can easily be identified.

Fungus is suspected to be among the worst problem sources with regard to microbiological pollution in indoor environments. There is considerable evidence suggesting that conditions favourable for fungus are found relatively frequently in ventilation systems. Fungi can lead to respiratory infections, allergic reactions and other health hazards, even if they are not normally adapted to temperatures as high as 37 °C.

In several "problem buildings" microbiologists from the research foundation SINTEF Unimed in Norway found microbes with worrisome qualities: mould that had adapted to temperatures high enough to allow growth in the human body (Ressem and Toenseth 1996). In an investigation of ventilation processed air from 21 office buildings located in Bergen, Trondheim and Lillehammer (Norway) over a period of 1 year they revealed that in air

samples from a total of six of these buildings there were mould that could easily be cultivated in a laboratory at 37 °C. The investigation verified that the troublesome mould grow inside the HVAC-system.

The same mould are to be found everywhere in ordinary outdoor air, but normally none of these fungi let themselves be cultivated in temperatures as high as 37 °C when they are collected out of doors in subarctic countries like Norway. The study provides strong indications that adaption to higher temperatures appears within ventilation systems. Nothing indicated that the buildings were damaged in such a way that the fungus would be given the chance to grow elsewhere in the building under temperatures as high as this the researchers emphasize. Aspergillus fumigatus was among the fungi that thrived at 37 °C in the tests carried out.

Whether people will or will not fall ill by working in indoor air containing mould that thrive at an atmosphere of 37 °C depends of several factors. In areas with a temperate or tropic climate, the mould flora of the air is adapted to increased temperatures. Exposure of mould that are able to grow at 37 °C is therefore not unfamiliar to people living in such areas.

In sub-arctic climate, however such exposure is unfamiliar and may be perceived by the immune system as antigenic. In these cases, long lasting specific stimuli, also in low doses, will stress the immune system. These reactions may turn out as allergies. In immunosuppressed individuals such exposure may turn out as respiratory infections the researchers points out (Ressem and Toenseth 1996).

If the findings are representative, modern ventilation systems may be potential breeding grounds for micro-organisms which in turn may represent a health risk for some people living and working in the air conditioned premises. Accumulated dirt combined with unwanted moisture or water inside the HVAC-system is therefore one of the main risk factors with regard to the ventilation system and indoor air quality.

7.3 Dose -Response Assessment

The general framework of dose-response assessment does not easily apply to indoor environment problems related to ventilation systems. In general, it is impossible to establish a dose-response relationship when dealing with ventilation as such. This is because ventilation is not the problem, pollution is the problem. In fact, ventilation is by necessity a part of the solution. By definition, ventilation is the process of supplying or removing air by natural means (including infiltration) or mechanical means, to or from a space, for the purpose of controlling air contaminant level, humidity, or temperature within the space.

Nevertheless, a ventilation system may be a risk factor if not properly designed, operated and maintained. It may also be a confounding factor. Characteristic dose-response curves related to ventilation systems and contaminants are illustrated in Figure 7.2. Nevertheless, a ventilation system may be a risk factor if not properly designed, operated and maintained. It may also be a confounding factor.

7.4 Exposure Assessment

In addition to removal of indoor generated pollutants, and thus reducing harmful exposures, a ventilation system may increase exposure. This because contaminated air ducts, filters and humidifiers may become significant sources of foul odours, dust and microbes. A ventilation system may also disperse locally generated indoor pollutants (e.g. ETS, renovation work dust and solvents) or outdoor contaminants (e.g. diesel exhaust, smoke, highway dust, mould and biocides like pesticides and insecticides) throughout a building. Air re-circulation, may be advantageous from an energy savings point of view, but introduces a risk of increased built-up of contaminants. The amount of recirculated air, depends on the function of the building (i.e. for hospitals 100% outdoor air is required). Partial re-circulation can allow the amount of fresh air supply to be adjusted, while maintaining the overall supply volume to the space and the necessary air distribution patterns. The outdoor fresh air inlets need to be carefully located, to minimize the intake of outdoor pollutants. For example, away from street level in heavy traffic areas and at a distance from the system's outlets of the same or neighbouring buildings.

In some cases, the indoor air can be up to 10 times more polluted than the outdoor air (Pearson 1991) according to studies performed in the United States. Even in cases that the indoor air is not particularly

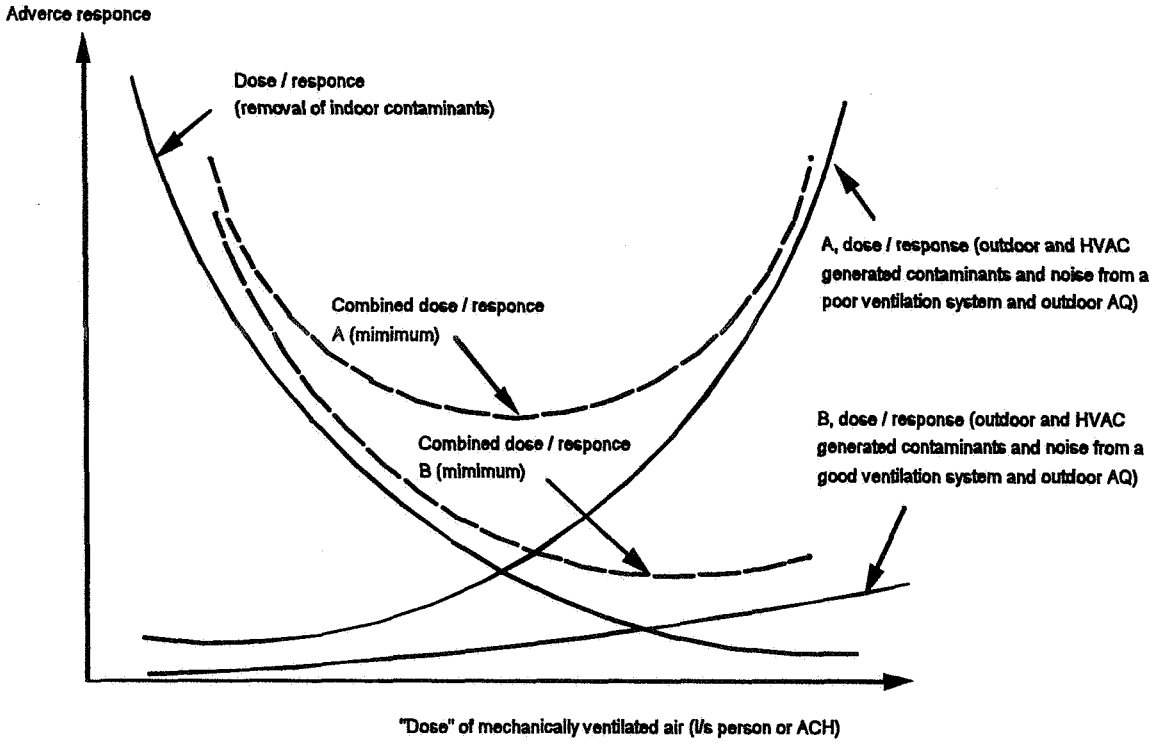

Figure 7.2 *Example of dose- response curves of a two ventilation systems, A and B.*

polluted, the fact that the occupants remain exposed to this air quality for long periods of time, in some cases, it may cause serious health problems. On the average, people remain indoors for about 22 hours every day, which means that the air quality of indoor environments is of great importance.

Furthermore; an incorrectly installed or operating ventilation system may fail to collect and exhaust local contaminants (e.g. fume hoods, dampness and moisture level in buildings), thus increasing occupant exposure. Inadequate ventilation of domestic bedrooms is a well-known problem. In office buildings, lack of commissioning of the ventilation system (e.g. insufficient balancing of the air flow rate) may increase the individual exposure.

Probably the most underestimated threat with regard to IAQ is contaminated ductwork or components. In a report from the National Energy Management Institute (NEMI) in USA (National Energy Management Institute 1993) it is emphasised that air-conditioning ductwork can become a serious source of pollution. The combined presence of moisture and deposits of dirt and organic matter allow growth of mould, fungi and bacteria that are carried in the air stream.

The ductwork system can also be the pollution pathway from the problem's point of origin to areas throughout the building. In some cases, a problem that started elsewhere in the mechanical system becomes magnified inside the ductwork (Hays *et al.* 1995).

7.5 Risk Characterisation

Many occupants express annoyance or even become ill in modern buildings. Terms like Sick Building Syndrome (SBS), Tight Building Syndrome (TBS), Building Related Illness (BRI), and Multiple Chemical Sensitivity (MCS), are introduced in order to define the problems and group the different characteristics. Symptoms commonly attributed to IAQ problems include headache, fatigue, shortness of breath, sinus congestion, cough, sneezing, eye, nose, and throat irritation, skin irritation, dizziness and nausea. Several of these symptoms may occur at the same time.

Often people suspect or blame the HVAC-system for creating this kind of problems and illnesses, but in real life it is often impossible to estimate the incidence of adverse health effects in a given population due to inadequate ventilation as such. Although the ventilation system obviously can be a part of the problem, individual probabilities, the magnitude of exposed population and uncertainties makes it impossible to generalize with regard to the effect of an insufficient ventilation system.

However, proper maintenance of HVAC systems can have a predominant influence on the system performance and its impact on indoor air quality. From related investigations in 147 buildings in the United States, it was found that about 70% of the indoor air quality problems were mainly attributed to poor operating conditions and maintenance of the systems (ASHRAE 1996). Based on the experience that has been accumulated over the years from similar investigations around the world, it is believed the improper maintenance and operation of the HVAC systems in a building, are the source for about 50% of the occupants complaints on the indoor air quality.

7.6 Relevance for Risk Management and Risk Communication

Ventilation by demand, individual control and satisfactory operation and maintenance is important to achieve good indoor air quality. Based on current knowledge we may conclude that existing IAQ standards, guidelines and recommendation combined with good

engineering practice should result in well designed HVAC-system. Unfortunately, the problems related to indoor air quality are not well known to building designers, engineers and professionals related to the construction and operation of buildings (Levin 1991). The main problem is that there has not been a proper dissemination of available knowledge and information that have been accumulated over several years of research in this area.

Extensive contamination control is probably the most important facet in risk management of a ventilation system. We have to prevent microbes, bacteria, viruses, fungi, spores and pollen from entering our HVAC-installations. Highway dust, mould and biocides like pesticides and insecticides, are examples of other unwanted elements that may be present in the outdoor air. In order to prevent the contaminants from entering the HVAC-system, air filters are installed.

To purify outdoor air and to keep the air treatment components and ducts free from dirt, an air filter of class G4 (EU 4) or better shall be installed close to the outdoor air inlet. A second filter, of class F7 (EU7) or better, shall be situated on the discharge side of the fan, where possible as the final component of the air handling unit. Care shall be taken to ensure that the joint between air filter and duct walls are airtight. All filters are to be protected against moisture penetration. The relative humidity (RH) in the filter should not exceed 90% (DIN 1946, Part 2). Where smoking is permitted, filters with a minimum rating of 80% efficiency (F7, EU 7) are required before any effective amount of tobacco smoke is removed.

Low efficiency pre-filters are usually included with high efficiency filters to extend their useful life. The combination of a filter with a pre-filter, can result to large differences in the development of bacteria. From research with various types of filters (Martiny, Moritz and Ruden 1994) it has been found that the average bacteria concentration was 910 cfu/cm^2, while the ratio of microorganism concentrations in the pre-filter and the main filter, was 24:1. Ionisation and chemically reactive filters should be considered where high concentrations of smoke or odours are present. The resistance to air flow through filters depends on filter construction, type of media, are of media/unit volume and the dirt-load condition, which is usually the main cause of excessive flow resistance and in some cases of serious indoor quality problems and health effects.

Unfortunately, air filters can be a source of contamination themselves. Under the wrong circumstances filters can become excellent reservoirs for microbial amplification. For this reason, special attention must be given to maintenance. Poor maintenance can cause rapid degeneration in filter performance and together with microbial activity on or inside the filter media it may have a serious effect on IAQ.

The purity of the air filter is, however, only one example of a potential risk factor in modern air condition systems. In practice, any component that leaves open the possibility of a retention of damp or pollution will represent a health risk factor. Noise attenuators and humidifier systems, as well as some regenerative heat recovery systems are perhaps equally subject to hygienic problems if they are not looked after and maintained properly.

Further, the result strongly depends on the quality of the components we use in our HVAC-installations. Investigations show that even if equipment is tested in accordance with relevant and accepted procedures, it may fail in a real situation after a short period of time.

This is probably an underestimated and neglected problem. It is a fundamental dilemma and this feature needs more attention in the future because it is decisive with regard to risk assessment and management of a ventilation system.

For successful system operation, control and prevention of potential problems, one should consider the following:

- The components of the HVAC-system must be so arranged and constructed that the system are easily accessible for purpose of operation, maintenance, inspection and repair. The materials used and the action taken to provide protection against fire, noise nuisance and corrosion must also be in compliance with the requirements relating to health (DIN 1946, Part 2 1994).
- The central plant and the air distribution system must be designed, manufactured and installed in such a way that cleaning of all internal surfaces and components is possible. Stiffeners and other equipment in the ductwork must be installed so that the cleaning of ducts is not obstructed (European prestandard CEN ENV 12097:1997 E).
- Establish a maintenance department, properly staffed. For small size buildings, authorize properly trained personnel to play this role or subcontract to outside professionals who provide this type of services. New technologies and systems introduce a need for well trained and qualified personnel, to monitor, operate and maintain them. Thus, keeping up to date with current advances and new technical information, is essential. In-house training of staff, hiring qualified personnel or a contractor, are necessary follow-up actions.
- Make the occupant users also aware of the problem, their involvement and role. Promote a campaign to increase general awareness and encourage participation. Encourage the cooperation of occupants in reporting problems at an early stage, like odours, air stuffiness and other equipment and system malfunctions, damages, *etc.* Unpleasant odours usually occur in places with indoor air quality problems and although the source may be difficult to identify, they also have a negative impact on people and strengthens the occupants complaints (Valbjoern, Hagen,, Kukkonen, and Sundell 1990). However, keep in mind that there are also several indoor pollutants that have or cause no distinct odours and consequently the final judgments should not be solely based on these indications.
- Preventive maintenance is a key factor for minimising the risks from potential problems. Establish a strict program for periodic maintenance checks for every piece of equipment and system component, in order to ensure proper operating conditions. Even visual checks of system components can be sufficient in many cases. Automatic control systems, sensors and other monitoring equipment, may drift in time and give unreliable readings and information. Periodic re-calibration and testing should be implemented on a regular basis. For example, regularly check the condition of ventilation filters, maintain them regularly and replace them if necessary.
- Develop and enforce a regular maintenance schedule of the whole system, according to the specific manufacturer recommendations and experience gained from the actual operation of the system. A well designed and implemented strategy can ensure proper system operation with mutual benefits in terms of optimum energy system performance

and healthy indoor conditions. For example, maintain a service file for all equipment and systems, including operation, inspection and service instructions; records and reports of the work performed, logs of the maintenance performed; operating data; and energy consumption (ASHRAE 1992). Keep all records, logs and service information current, complete and in order.

- For commissioning of new buildings, it is essential that an additional parameter on indoor air quality is also included in the overall procedures and methodologies usually being followed (Dols, Persily and Nabinger 1995).

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8 ALLERGY TO HOUSE DUST MITES

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8.1 Introduction

Sensitization to house dust mites may be viewed largely as a man-made health problem in temperate regions. It has strong links to deliberate changes in the built environment. Allergy to house dust mite presents a complex causal chain and physical, biological, immunological and technological factors are being used to characterize the risk. Allergy is also much dependent on the "host sensitivity" of the individual and this sensitivity is not evenly distributed in the general population; may be half or less seemingly are able to respond to allergens as indicated by the presence of antibodies in the blood.

8.2 Identification of Hazard and Sources

In childhood, asthma is mostly allergic and induced mainly by pollen, cats, dogs or house dust mites (Sears *et al.* 1989). The allergenicity of house dust is often related to its mite content (Voorhorst *et al.* 1967). Asthma is significantly more prevalent among house dust mite-sensitized children than among children sensitized to other allergens (Sundell 1994). The incidence and prevalence of childhood asthma is increasing, especially in industrialized countries (Fleming and Crombie 1987; Burr *et al.* 1989; Åberg 1989a; Gergen and Weiss 1992). As an example, the prevalence of asthma among school children in the Nordic countries has increased from about 2% in 1970 to 5-7% in 1990 (Formgren 1994). Since there has been no change in the human genotype during this time span, it is reasonable to search for causes in the environment and in lifestyle.

There is a marked difference in house dust mite infestation between cold and temperate regions (Wickman *et al.* 1993; Munir 1994). Infestation of house dust mites is associated with indoor air humidity (Hallas and Korsgaard 1983; Korsgaard 1983; Mosbech 1985; Murray *et al.* 1985; Andersen and Korsgaard 1986; Harving *et al.* 1993). Accordingly, house dust mites and sensitization to house dust mites should be rare in areas with long and cold winters, prolonged heating season and consequent dry indoor conditions but surprisingly in many cases this is not the fact (Turos 1979; Nordvall *et al.* 1988). Too low ventilation air rates indoors as well as changes in life style and hygienic maintenance have been proposed as explanations.

8.3 Exposure/Response Assessment

Many studies confirm that a main risk indicator of allergy towards house dust mites is house dust mite infestation. Respiratory symptoms, such as bronchial asthma/wheezing, have been found to be associated with sensitization to house dust mites (Shibasaki *et al.* 1988, Sears *et al.* 1989, Wickman *et al.* 1991). House dust mite allergens have been found in dust from 40% of mattresses of house dust mite sensitized children versus 19-23% of mattresses of control groups (Wickman *et al.* 1991). A threshold level of sensitization has been suggested ($\geq$ 2000 ng of mite allergen per gram of dust; Sporik *et al.* 1990). Furthermore, exposure to house dust mite allergen may play a causal role in the demonstrated association between damp homes and respiratory symptoms (Wickman *et al.* 1992, Brunekreef *et al.* 1993, Sundell 1994). Dust mite exposure may not be the significant

trigger of acute attacks of asthma but rather the crucial factor in the development of bronchial reactivity (Platts-Mills *et al.* 1991).

Both house dust mite infestation and adjuvant factors are more prevalent in damp homes than in others, so a multifactorial etiology should be suspected in many damp home-related health disorders (Sundell 1994). A damp home is associated with a low ventilation rate, and a low ventilation rate is associated with i.a. increased house dust mite infestation (Meyer 1983).

The rate of ventilation in the dwelling as a whole and especially in the bedroom is critical for moisture level and the degree of house dust mite infestation (Wickman *et al.* 1993, Harving *et al.* 1993, Sundell 1994). The infestation rate is low in homes exhibiting an indoor absolute humidity below 7 g/kg air during 1-2 months of the winter, corresponding to a relative humidity of 45% at 20-22 °C (Andersen and Korsgaard 1986). Increased ventilation during winter reduces the indoor air humidity, thus preventing major infestations of house dust mites (Harving *et al.* 1988, 1991, 1992, 1993).

8.4 Exposure Assessment

House dust mites can be found almost all over the world where there are human beings (Wickman *et al.* 1991). In humid and coastal areas the infestation and sensitization rates are higher (Korsgaard 1983, Murray *et al.* 1985, Yi-Chung and Kue-Hsiung 1989) than in dry areas (Vervloet *et al.* 1982, Linna 1983, Britton *et al.* 1986). In some parts of continental Europe residential infestation of house dust mites is very common (Sporik *et al.* 1990) but should be uncommon in regions which are cold several month a year and thus where indoor humidity drops because of heating. In the latter, elevated house dust mite concentration seems to be associated with either a low total ventilation of the home or a low infiltration of outdoor air into the bedroom. For Denmark it has been suggested that ventilation rates ≥ 1.0 air changes per hour would protect against mite infestation (Andersen and Korsgaard 1986).

Damp buildings typically are associated with high indoor air humidity and low ventilation rate (Wickman *et al.* 1993, Ekstrand-Tobin 1993, Sundell 1994). It is reasonable to assume that part of the association between damp homes and airway disorders may be due to inadequate ventilation. Indications of poor ventilation have been shown to be more common in the homes of house dust mite-sensitized children than in homes of control children (Sundell *et al.* 1994). Signs include elevated indoor air humidity, window water condensation on inner panes, extra insulation of the building envelope, ventilation without fans, no air-intake systems or air-intake systems but closed or partially closed. A large airflow within the home in total is seemingly not necessarily associated with a large reduction in humidity in the bedroom.

Single-storey single-family houses seem to be significantly more infested with house dust mites than multi-storey single-family houses and exhibit significantly more house dust mite allergen in mattress dust than homes in apartment blocks (Sundell 1994).

8.5 Risk characterization

The predisposing factors in airway hypersensitivity are only partially understood. There are great individual variations in susceptibility and predisposition. Exposure to house dust mite

allergen may play a causal role in the demonstrated association between damp homes and respiratory symptoms. However, it has been argued that dust mite exposure may not be the significant trigger of acute attacks of asthma but rather be the crucial factor in the development of bronchial reactivity. A threshold level of sensitization has been suggested (≥ 2000 ng of mite allergen per gram of dust).

Children´s environments are particularly important in view of small children´s underdeveloped immune systems and the proven correlation between early exposure and later allergic diseases (Swedish Commission on Environmental Health 1996).

For chemicals the European Union has developed criteria for classification of skin and respiratory sensitizing substances (EC Commission 1994). The classification principles as such may hold also for house dust mite allergens. Respiratory sensitizers are classified on the basis of, i.a., (a) evidence that the agent can induce specific respiratory hypersensitivity, or (b) positive results from an appropriate animal test. The World Health Organization has proposed criteria for classification of skin- and airway-sensitizing substances in the work and general environments (WHO 1997). In principle, sufficient human evidence that a substance is the inducer of specific airway hypersensitivity can be obtained only when it can be shown in more than one patient in more than one independent centre that there is a characteristic airway response after exposure to the substance and this can be provoked by a non-irritant exposure to the substance. Assuming that the WHO classification principle would hold also for house dust mite allergens, they should be classified at least as a probable inducer of specific airway hypersensitivity (Class II) since specific IgE antibodies can be demonstrated in skin and serological tests, and epidemiological evidence is present of an increased frequency of a marker in relation to exposure.

8.6 Relevance for Risk Management and Risk Communication

In the setting of priorities for actions against the most important environmental health risks in Sweden, dust mites, due to their allergic potential, were ranked by an expert panel as having the same (medium) priority as chemical pollutants from building materials, indoor particulates and microbially induced substances indoors. However, dust mites were ranked as having less priority than radon, environmental tobacco smoke, sick buildings and furred animals at home (Victorin *et al.* 1997).

In regions with a cold winter climate (low water content in outdoor air) and thus a potential to easily reduce the indoor air humidity during prolonged periods, ventilation of the home plays an obvious role as a means for reducing exposure to dust mite allergens.

Dwellings shall be ventilated so that there is no condensation on inner panes (more than a few cm at the bottom) at normal winter temperatures. Humidifiers should not be used in dwellings, schools or similar premises. Required ventilation air rates should be prescribed for specific regions taking into account climatological, building technological and behavioral characteristics of the region.

Good bedroom hygiene decreases the risk of house dust mite proliferation (wash bedclothes at > 60°C, airing of bedclothes in the window).

With respect to risk communication designers of buildings and systems and of building codes are prime targets. Either the competence levels of the designers have to be raised considerably, or building codes have to contain more relaxed criteria for energy conservation in residences and more stringent criteria for minimum ventilation air rate. Probably, a combination is needed. The residents need to be informed about the nowadays often forgotten hygienic rules of the past.

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9 INDOOR RISK RANKING AT NATIONAL LEVEL

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9.1 Introduction

Health and safety hazards can have outcomes ranging from effects on wellbeing, which are often transient (e.g. feeling uncomfortable because of odours), through illnesses requiring varying degrees of medical attention, to serious disablement and death. The probability of a particular outcome resulting from a particular hazard also varies widely, as does the strength of the evidence that links a particular hazard to a particular health outcome. This example represents an attempt to develop and apply a procedure that ranks hazards on the basis of these variables, so that action can be targeted effectively at national level.

Table 9.1 shows the two ends of a continuum from simple to complex procedures for risk assessment. Because the ranges of severity, probability and strength of evidence (quality of data) are great, the procedure described here has to be able to use relatively low quality data and hence does not take full advantage of the good data available in some areas. It is a simple procedure but relatively elaborate as a hazard ranking procedure, using expert panel judgements together with objective data as inputs to a numerical relative risk model.

The work is described by Cheyne *et al.* (1994) and Raw *et al.* (1995a,b); only a brief summary is given in the following paragraphs.

Table 9.1 *Contrasting approaches to risk assessment*

SIMPLE PROCEDURE	COMPLEX PROCEDURE
Limited precision	High precision
Averaging over many exposure levels and susceptibilities	Specified exposure and susceptibility
Low level data input requirement	High level data input required
Wide applicability	Narrow applicability
Assessing types of hazard/harm	Assessing specific buildings or rooms
Expert judgements of severity of harm	Severity of harm judged by specific metrics (e.g. useful days lost/economic cost)
Individual hazards/harms	Multifactorial exposures (hazardous situations) and multiple endpoints (harms)

9.2 Identification of Hazards and Sources

An expert group identified the hazards that are present in buildings in the UK and grouped them into broad categories (e.g. sources of infection, VOCs, particulates).

A series of rating exercises involving health and safety experts was then used to generate a list of harms that could result from these hazards, and to assign the harms to four classes according to their severity. Two independent approaches were used to derive the classes of harm and there was good agreement between the results obtained using these two approaches.

Class I harms are death and other 'extremely severe' outcomes (e.g. permanent paralysis below the neck, regular severe pneumonia or permanent loss of consciousness). Class II is applied to 'severe' outcomes, for example severe chronic confusion or dementia, mild strokes, regular severe fever and loss of consciousness for hours or days. Class III ('moderate/severe') outcomes include chronic severe stress, mild heart attack, regular severe dermatitis, malignant but treatable skin tumours and regular severe migraine. Class IV ('moderate') includes occasional severe discomfort, chronic/regular moderate skin irritation, benign tumours, occasional mild pneumonia and regular serious coughs or colds.

These outcomes are only examples; a more comprehensive list of harms under each class was available for comparison with the outcomes identified in the reviews. They represent harms as described to the subjects in the studies and do not always correspond to the exact medical terms that might be used.

Literature reviews were then used to complete a risk matrix for each hazard, having the form shown in Table 9.2. The matrices describe the seriousness of the harms that could befall an individual person, the total number of people affected annually in the UK, and the strength of evidence for the risk (high, medium or low, represented by ***, ** and * in the matrices). The number of people affected would in some cases be the number of occurrences (e.g. number of deaths) and in other cases the number of people affected (e.g. annoyed by odours) on a more continuous basis through the year. Since the population of the UK is constant between hazards, the number of people affected is proportional to the probability of the harm occurring in the UK.

Table 9.2 *Example of a matrix summary of buildingrelated health risks*

	NUMBER OF PEOPLE AFFECTED IN THE UK PER YEAR					
OUTCOME	100,000+	10,000+	1000+	100+	10+	1+
Class I					**	***
Class II				***		
Class III		*	**			
Class IV	***					

No attempt has been made to assign different weights to different lives. Thus, the death of an elderly person with a residual life expectancy of perhaps five years is assigned the same weight as the death of an infant.

For each hazard, a 'risk index' has been calculated. The index is based on the whole risk matrix, but weighted such that Class I harms have by far the greatest influence (the weightings given to Classes I to IV are 100,000, 2500, 50 and 1 respectively). A greater weighting is also given where the evidence is stronger (5 for high, 3 for medium and 1 for low strength of evidence). The indices are then based on the logarithm of the 'seriousness x number affected' values; this renders the process less sensitive to the exact values given to the weightings. Thus, risk was implicitly defined as a function of three variables: (i) the likelihood of a particular hazard causing harms to the exposed individuals, (ii) the severity of the harms or their consequences and (iii) the number of people exposed to the hazard.

The risk indices are not formal estimates of risk but a higher index should represent a higher risk. Although based, where possible, on actual numbers of deaths etc, the index is most useful for placing hazards in rank order, rather than stating the actual risks attributable to a particular hazard. The indices should therefore not be assigned an absolute value; they are useful only for placing the issues in a rank order. The hazards were thus ranked as shown in Table 9.3 for health hazards related to homes.

It is important to bear in mind that the risks associated with a particular hazard may be low because current controls are working successfully: this would not be an argument for relaxing controls. Alternatively the low rating might reflect simply a lack of evidence, and would therefore not indicate a cessation of research. There is also considerable variation among people in their susceptibility to the effects of each hazard; even hazards with a low overall risk may be significant for some individuals.

9.3 Relevance for Risk Management and Risk Communication

The purpose of this work was to compare the risks associated with various hazards in buildings, as a basis for risk management through development of standards and regulations in the UK. The procedure, although not the results, could be applied to any country or administrative area.

The output from the model is a rank order of hazards, based on a quantitative treatment of the data from the matrices. The model is relatively simple because it has to accept data at the population level from a wide range of sources and having a wide range of precision and certainty (Cox & O'Sullivan 1995, Raw & Hamilton 1995). It could, however, easily be made more detailed if the field to be covered, or the reference population, were more limited.

This procedure is, at present, used only to rank risks; this is because of the lack of precision and the high uncertainty in some of the input data. There is no reason in principle why ratios of risk should not be calculated by a simple adaptation of the procedure, if better input data were available for the field or population of interest.

By going through similar exercises involving members of the public rather than experts, the reasons for differences between expert and public estimation of risk are now being elucidated. This will provide a basis for targeting and phrasing risk communication to deal with the key issues.

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Table 9.3 *Health hazards in homes, grouped in rank order of risk*

Highest risk
Hygrothermal conditions
Radon[1]
House dust mites
Environmental tobacco smoke
Carbon monoxide
Second level of risk
Fungal growth
Security and the effects of crime
Noise
Lead[1]
Third level of risk
Sanitary accommodation[2]
Sources of infection other than sanitary accommodation[2]
Space
Volatile organic compounds[3]
Oxides of nitrogen[3]
Particulates[4]
Fourth level of risk
Sulphur dioxide and smoke
Landfill gas
Pesticides[2]
No clear basis for risk assessment
Lighting
Electromagnetic fields
NOTES
1. Geographically localised.
2. Important potential risk but largely controlled by current standards.
3. Low ranking may be due to insufficient research.
4. Recent research suggests this may get a higher ranking in future.

Appendix: Members of the ECA " Urban Air, Indoor Environment & Human Exposure" Steering Committee

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The Steering Committee

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People will never live in a risk free environment. Still we must aim at minimising all risks and most importantly risks that are imposed on without their consent or even knowledge. A building is built for and perceived as shelter – against weather and unwanted intruders, for thermal comfort, privacy and property. Health threatening risks that the dwellers of a building cannot sense or expect contradict directly the whole concept of a building.

Risk assessment is a scientific multidisciplinary paradigm to identify, quantify, describe and compare risks. Risk management is an administrative paradigm to develop and compare risks reduction priorities and alternatives, to organise and manage risk-controlling practices and to evaluate the achievements. Risk assessment and management have existed always. The general formal paradigms that are being applied in today's societies, however, are quite recent, and still under continuous development.

This main body of this report presents the state of the art of modern risk assessment and risk management paradigms, highlighting also the historical development that has lead to the present practices, and applies them specifically into building environments.

The examples section in the end of this report applies the formal risk assessment protocol of the EC (and similarly USEPA), to a variety of building related health risk. These examples are not intended as recommendations, instead they are selected to highlight the level of success (or failure) in applying one strict protocol to multiple extremely different problems.