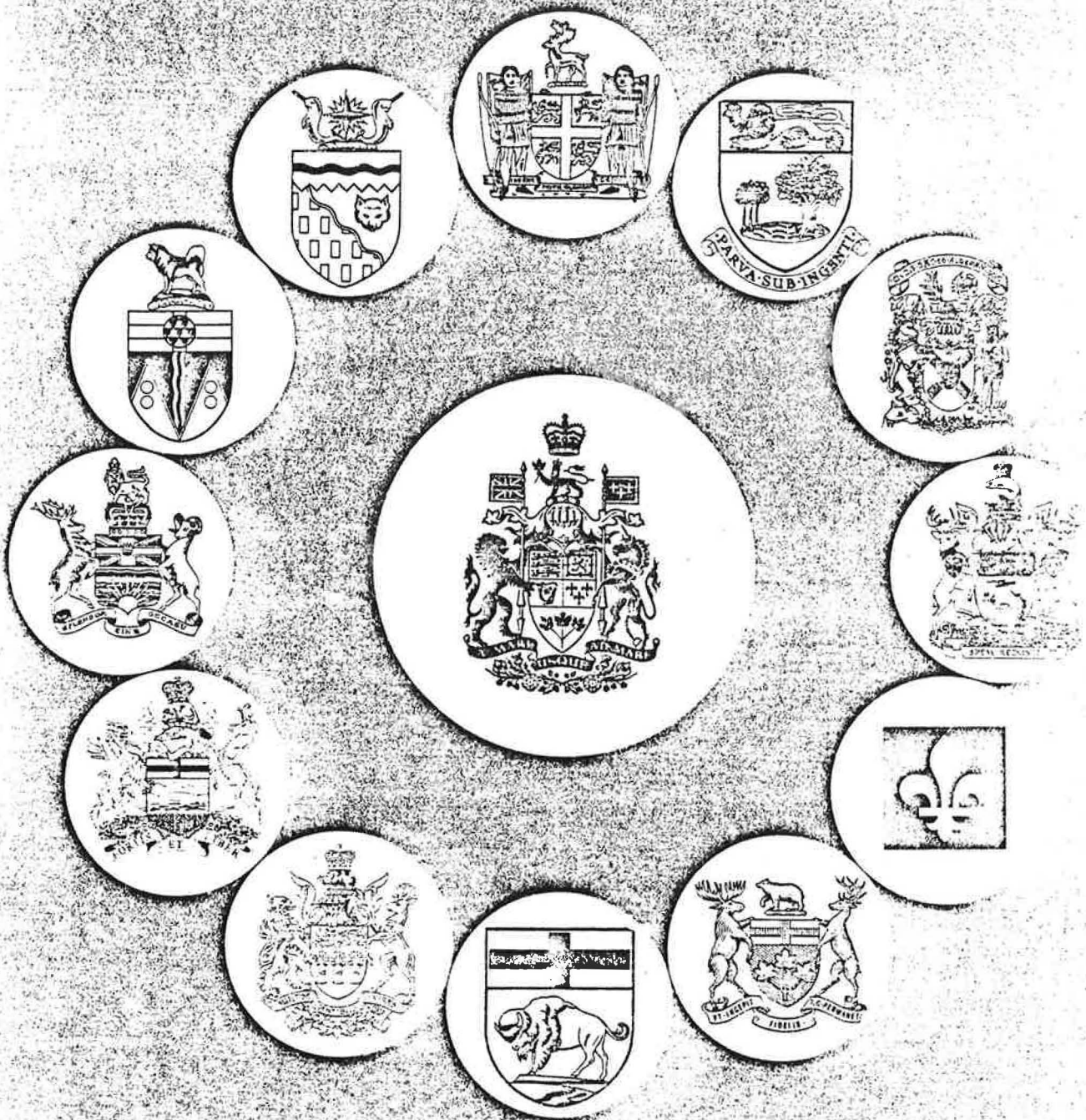




Exposure Guidelines for Residential Indoor Air Quality

The Federal-Provincial Advisory Committee On Environmental and Occupational Health



EXPOSURE GUIDELINES FOR RESIDENTIAL
INDOOR AIR QUALITY

A Report of the
Federal-Provincial Advisory Committee
on Environmental and Occupational Health

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PREFACE

It is now recognized that non-industrial indoor environments can contribute significantly to human exposure to airborne pollutants. This is particularly true in countries such as Canada where long cold winters and hot summers result in considerable periods of time being spent in climate-controlled buildings. Also, within the past few years, incentives have been given to reduce the consumption of energy, particularly oil, in buildings by reducing the rate of air exchange, installing additional insulation and using alternative sources of energy. Such measures are likely to raise the concentrations of some contaminants in indoor air above those found in outdoor air and so have aroused the concerns of public health authorities, those concerned with building standards and home construction, and the general public. In April 1979, the World Health Organization convened a meeting of experts to discuss the health aspects of indoor air quality. This group recommended that health authorities draw up guidelines for indoor concentrations of air contaminants to protect the health of occupants of homes.

In June 1980, the Ontario Deputy Minister of Health requested that a federal-provincial study be undertaken to establish standards for air quality in new housing. A proposal to convene a Working Group on Indoor Air Quality was formally tabled at the October 1980 meeting of the Federal-Provincial Advisory Committee on Environmental and Occupational Health and subsequently agreed to by the Deputy Ministers of Health.

The Federal-Provincial Working Group on Indoor Air Quality was requested by the Advisory Committee to consider "a definition of acceptable air quality", the need for "objective and/or maximum acceptable concentrations for specified substances" and a "specification of ventilation rates, or recirculation criteria". The scope of the work was to be restricted to "domestic premises" and recommendations were to be developed to protect the general public assuming exposure for 24 hours per day (i.e., continuous).

The Working Group met for the first time in September 1981. During the subsequent four years, the scientific literature on 18 substances, or groups of substances, was collected and reviewed to arrive at the guidelines and recommendations made in this document. While the list originally included radon, the Working Group on Indoor Air Quality did not develop a recommendation for this substance. Because of the specialized knowledge required to review radioactive

substances, the Advisory Committee referred radon and its decay products to the Federal-Provincial Subcommittee on Radiation Surveillance. A guideline for radon will be published separately.

It is anticipated that these guidelines will provide a reference point against which the adequacy of residential indoor air quality and/or remedial measures can be judged.

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* Deceased.

1.0 INTRODUCTION

1.1 Background

Criteria for acceptable air quality have existed for many years for the industrial workplace and outdoor environments. Only recently, however, has widespread attention been drawn to potential hazards posed by the presence of airborne contaminants in non-industrial indoor environments. Several factors have contributed to this awareness:

- a trend towards tighter building construction, as incentives are given to conserve fuels;
- the presence of an increasingly complex array of synthetic chemicals used in building and insulating products, furnishings, consumer products and hobby and craft materials;
- the use of alternative heating systems which, if not properly installed or designed, can release combustion by-products;
- a recognition that prolonged exposure even to very low concentrations of chemical contaminants may result in delayed toxic effects; and
- the realization that a very high proportion of the average individual's time is spent indoors.

1.2 Sources of Indoor Air Contaminants

The quality of indoor air is influenced both by the quality of outdoor air and by the emission characteristics of indoor sources.

1.2.1 Outdoor Sources

In almost all inhabited enclosed spaces, there is a continuous exchange of air with the outside. Therefore, all contaminants of outdoor air are likely to be present indoors. Important pollutants in this category include carbon monoxide, oxides of nitrogen, oxides of sulphur, particulate matter, ozone (and other photochemical oxidants) and lead.

These pollutants originate, to a large extent, from automobile and factory emissions and other combustion processes. Generally, in the absence of indoor sources of these contaminants, concentrations indoors will be close to or lower than those outdoors.

A natural pollutant is radon-222, a radioactive gas that decays to non-gaseous radioactive species which can be adsorbed onto suspended particulate matter and hence be deposited in the lung. Domestic water, natural gas, underlying soil and groundwater can be significant sources of radon in dwellings.

1.2.2 Indoor Sources

Internally generated airborne pollutants fall into one of three categories:

- those formed in combustion processes for heating and cooking;
- those derived from construction materials and furnishings;
- those related to human activity or presence.

Concentrations of contaminants in the first and last categories tend to vary with time; those in the second are likely to be more constant, assuming that air exchange rates remain constant.

- (i) Combustion Processes. Furnaces and other combustion appliances can be sources of indoor pollutants, notably carbon monoxide, especially if they are not properly vented or routinely serviced. Since combustion in wood-burning stoves is much less complete than in oil and gas furnaces, pollutant emissions from them can be greater. Though by-products should be vented to the outside, leaks and improper operation of these appliances can cause emissions to the indoors. Contaminants associated with wood-burning stoves and fireplaces include carbon monoxide, oxides of nitrogen and sulphur, aldehydes and polycyclic aromatic hydrocarbons.

A major potential source of combustion by-products is gas-fired stoves. Emissions from the oven and pilot light are not always vented and can contribute to indoor levels of carbon monoxide, nitrogen oxides and formaldehyde.

Kerosene heaters are becoming increasingly popular for space heating. Since these systems are often unvented, the potential for high contaminant levels again exists. In particular, the improper use of kerosenes with high sulphur contents or of poorly designed units could result in emission of oxides of sulphur as well as some of the other combustion by-products previously mentioned.

- (ii) Building Products and Furnishings. Synthetic polymers used in furnishings and decorative materials can slowly degrade, releasing small quantities of the original constituents or reaction by-products. Draperies, rugs and fabrics, the great majority of which contain man-made fibres, are sources of a variety of organic and, potentially, microbiological contaminants.

Formaldehyde is released from wood laminates and particleboard in which formaldehyde-containing resins have been used. Urea-formaldehyde foam insulation is a significant source of formaldehyde and possibly other gaseous products.

Fibrous materials such as asbestos and fibrous glass are present in some building materials and may be released to the indoor environment especially when such products are disturbed during building-alterations.

(iii) Human Activity. The variety of contaminants that result from human activity is extremely broad.

Tobacco smoking is a major source of indoor air pollution. While smokers subject themselves to mainstream smoke, bystanders can be involuntarily exposed to significant amounts of respirable particles, carbon monoxide and oxides of nitrogen, as well as numerous harmful organic contaminants, in sidestream smoke. Over fifty components of cigarette smoke are known to cause adverse health effects, and twelve of these are known or suspected carcinogens.

Human metabolic activity itself influences air quality by reducing the concentration of oxygen and increasing the level of carbon dioxide. Respiration, perspiration and food preparation add water vapour as well as odour-producing substances to the indoor atmosphere. A large variety of biological agents may be present in the home, for example, micro-organisms from occupants, pets and insects; microbial growths may also occur on moist surfaces or in stagnant water. Pollens, spores, cell debris and insects are present in dust originating both indoors and outdoors.

Air fresheners, furniture waxes, polishes, cleansers, paints, pesticidal formulations, fabric protectors, deodorants and other products frequently used in the home are sources of various inorganic and organic chemicals. Many substances found in the workplace may also occur in the home as a result of hobby or craft activities. Moreover, workers exposed to chemicals in the workplace may bring these contaminants into the home on their clothing. In some circumstances this may be a means by which significant amounts of potentially harmful substances are introduced into indoor air.

2.0 PURPOSE AND SCOPE

2.1 Objectives

Generally, people can be at home for as much as 70% of their time and, for some segments of the population (the very young, the old and the infirm), this percentage can be much higher. In developing recommendations for the domestic environment, the Working Group considered it prudent to assume that certain segments of the population are at home on a continuous basis. Furthermore, some individuals may be at special risk from indoor air pollution. Such individuals comprise those whose physiological processes are either not fully developed or are deteriorating, or for whom pathological or physiological changes impair the ability to surmount the adverse effects of exposure to a pollutant. Therefore, a primary objective was:

"to develop guidelines for the concentrations of selected contaminants of residential indoor air, taking into account such factors as the sensitivity of groups at special risk and the sources and mechanisms of action of contaminants".

These guidelines may not provide complete protection for the hypersensitive portion of the population which requires extraordinary measures to achieve such protection.

The need to specify ventilation rates for domestic premises that reflect the indoor air quality guidelines was also considered but was deemed to be beyond the expertise of members of the Working Group. Moreover, a prescription for ventilation requirements is only one of several strategies that might be adopted for controlling the presence of airborne contaminants in the home. Other possibilities include specifications for building design and materials and consumer products. In many instances, measures to minimize exposure to chemical contaminants can be taken by the occupants. Public education is an important strategy for attaining an acceptable quality of indoor air, and the Working Group therefore established as its second objective:

"to develop, where practicable, other guidelines or recommendations for measures that will preserve or improve air quality in domestic premises".

2.2 Definitions of Indoor Air Quality

As a guiding principle, air within domestic premises should be sufficiently free from biological, physical and chemical contaminants to ensure that there is a negligible risk to the health and safety of the occupants.

Following an examination of the available information, 17* substances, or groups of substances, were selected for detailed review because of their potential to cause adverse health effects and their possible presence indoors. Guidelines expressed in terms of concentration ranges were developed for nine of these. For the others it was not possible to express the guidelines in quantitative terms, either because the data base was inadequate or because human exposure limits were deemed inappropriate; where possible, however, recommendations are made in this document on measures that can be taken to control indoor exposure to contaminants.

The guidelines in this document therefore encompass two categories of contaminants:

- those for which recommendations are expressed in terms of ranges of concentrations;
- those for which recommendations are specified as practical measures that can be taken to reduce exposure.

Section 4, Part A of this document deals with those contaminants for which exposure limits could be derived exclusively on the basis of health considerations (see Section 3.2.1). These are specified for the following substances:

- aldehydes (total)
- carbon dioxide
- carbon monoxide
- nitrogen dioxide
- ozone
- particulate matter
- sulphur dioxide
- water vapour

The potential for adverse effects from long-term exposures as well as from shorter-term higher-level exposures has been considered. Two kinds of exposure limits are therefore specified.

* Information on radon was also reviewed but no recommendations were developed by the Working Group on Indoor Air Quality.

The Acceptable Long-Term Exposure Range (ALTER) is that concentration range to which it is believed from existing information that a person may be exposed over a lifetime without undue risk to health.

The Acceptable Short-Term Exposure Range (ASTER) is that concentration range to which it is believed from existing information that a person may be exposed over the specified time period without undue risk to health.

An important consideration in deriving acceptable exposure ranges is the possibility of interactive effects, since many contaminants are likely to be simultaneously present in the home environment. Where possible, due account was taken of the potential for synergistic and additive effects. However, in most cases there were insufficient or inadequate data to completely resolve this problem.

Section 4, Part B contains recommendations for long-term exposure to formaldehyde, on the basis that it is a possible human carcinogen. For carcinogenic effects, guidelines were not established exclusively on the basis of health considerations; rather they were set as low as possible taking into account the cost and technical feasibility of attainment, as well as the associated health risks (see Section 3.2.2).

Section 4, Part C of these guidelines deals with those contaminants for which the formulation of exposure ranges was deemed inappropriate or was not feasible. This group comprises:

- biological agents
- chlorinated hydrocarbons
- fibrous materials
- lead
- pest control products
- polycyclic aromatic hydrocarbons
- product aerosols
- tobacco smoke

Recommendations for procedures that would reduce or eliminate exposure in the home are provided for these substances.

In developing the recommendations for these 17 substances or groups, the Working Group critically reviewed the available literature. The criteria and rationale on which these guidelines are based are briefly outlined in this

document. A more detailed description of the scientific criteria and reference material is available in a separate publication entitled "Exposure Guidelines for Residential Indoor Air Quality: Supporting Documentation".

It is recognized that the above lists do not fully represent the range of compounds found in the home environment. As new data become available, the need for developing exposure guidelines for additional contaminants or groups and revising the current guidelines, should also be considered.

2.3 General "Indicators" of Indoor Air Quality

2.3.1 Carbon Dioxide

Increasingly, complaints made by occupants of some large buildings are being linked with poor indoor air quality. The occurrence of some of these complaints (headache, fatigue, unpleasant odours, stuffiness and undue warmth) has been associated with elevated concentrations of carbon dioxide. Whether a concentration range could be prescribed for carbon dioxide that would be indicative of acceptable air quality from the viewpoint of comfort or aesthetic considerations was therefore considered.

Carbon dioxide is produced by human respiration and can be reduced significantly only by ventilation of the building. The concentration of carbon dioxide in indoor air is therefore closely related to the ventilation rate. The degree of ventilation needed to maintain carbon dioxide at a low level within a building also helps to reduce the levels of other indoor pollutants and to improve overall indoor air quality. Carbon dioxide is useful as an indicator of general air quality only in buildings where there are significant metabolic and/or combustion sources of carbon dioxide. Otherwise, carbon dioxide levels will be low over a wide range of ventilation rates (for example, in large houses with only one or two occupants and no unvented combustion appliances).

The trend in recent years towards minimizing ventilation within houses in order to reduce energy consumption for heating and cooling can lead to increases in carbon dioxide levels within residences and to a general deterioration in indoor air quality.

In several studies, comfort factors have been correlated with carbon dioxide concentrations. Collectively, these studies suggest that carbon dioxide concentrations above 1800 mg/m³ (1000 ppm) are indicative that there is an inadequate supply of fresh air, although complaints have been documented at

concentrations as low as 1100 mg/m³ (600 ppm). However, from a review of the direct physiological effects of exposure to carbon dioxide, as opposed to subjective symptoms, a higher maximum exposure concentration is recommended (Section 4.A.2).

It must be noted that these studies were conducted in buildings with mechanical ventilation systems and with occupancy rates quite different from those of residences. Moreover, the effects observed are probably not attributable to the presence of elevated concentrations of carbon dioxide, but rather to undesirable concentrations of other substances that result from inadequate ventilation, and for which carbon dioxide provides a suitable surrogate parameter. Therefore caution must be used in interpreting carbon dioxide concentrations as a general indication of residential indoor air quality.

2.3.2 Water Vapour

Relative humidity has also been considered as an indicator of indoor air quality. Air-to-air heat exchangers which are designed to bring in outside air and to turn on and off at pre-set relative humidity levels, have been developed. The control of indoor air pollutants other than excessive water vapour by heat exchangers operating in this way may not be satisfactory for the following reasons:

- in a large house with a low occupancy, relative humidity may not rise sufficiently to trigger air exchange, although other pollutants may be present at unacceptable concentrations:
- changes in occupancy throughout the day affect the rate at which water vapour is generated, whereas other pollutants may be emitted continuously:
- the variation in indoor relative humidity with geographical area and season necessitates different control settings for each location and season.

Therefore, relative humidity is not suitable as a general indicator of residential indoor air quality.

3.0 DERIVATION OF GUIDELINES AND RECOMMENDATIONS

3.1 Data Base for Derivation of Exposure Guidelines

Since research into the health effects of residential indoor air quality is at an early stage, there is a dearth of reliable information on the health effects that result from exposure to the low levels and mixtures of contaminants likely to be found. In most cases, therefore, the Working Group relied upon the results of laboratory experiments using animals, clinical studies with human volunteers, and epidemiological investigations of urban air pollution and the occupational environment. The results of epidemiological and clinical studies are the most relevant for establishing acceptable levels of exposure of humans to air pollutants. Nevertheless, the application of each of these types of study involves a number of assumptions and hence uncertainty in the derived dose-response relationships.

3.1.1 Epidemiological Studies

Most of the relevant epidemiological studies of populations are observational (non-experimental) in nature; that is, the allocation of individuals into study groups on the basis of exposure is not under the control of the investigator. Such observational studies can be further classified as descriptive (cross-sectional) or analytical (cohort or case-control) studies. In cohort studies, exposure and outcome are monitored over time; as a result, the quality of evidence obtained from such longitudinal investigations is generally considered to be superior to that from cross-sectional studies, in which populations are monitored at one time. However, the results of all observational studies must be evaluated against the following features of study design;

- a) Estimation of Exposure - In most observational studies conducted to date, pollution data are usually obtained from one or several outdoor monitoring stations; however, the exposure burden can vary greatly between individuals living in the same neighbourhood because of local climatic conditions and special features of the indoor environment. For example, for pollutants generated mainly in the outdoor environment (oxidants, sulphur dioxide), indoor concentrations will normally be less than those outdoors; however, indoor levels (from indoor sources) may have greater temporal peaks with concomitant effects on health. Exposures to some pollutants (nitrogen dioxide, carbon monoxide) are not well represented

by ambient air measurements if there are significant indoor sources of these pollutants. Only in very recent studies have investigators attempted to take such factors into account in estimating exposure.

- b) Role of Confounding Variables - In observational studies of populations exposed to air pollutants, a host of confounding variables (e.g., socioeconomic status, smoking, occupational exposure, meteorological factors), many of which have greater effects than air pollution, must be considered.
- c) Measurement of Outcome - There is substantial variation in the method of measurement of many health-state indicators in the studies conducted to date; such indicators include lung function, hospital admissions and frequency of symptoms. In many of the studies, outcome is determined by questionnaire, and responses may be biased by the way and conditions under which the questions are asked.

Even in those studies where the design is acceptable, interpretation of the results is complicated. For example, it is often difficult to attribute effects observed in populations exposed to air pollution to a single contaminant. It is possible that the contaminant investigated may serve as an indicator, or surrogate, for the effects of another contaminant or a combination of contaminants. Also, since exposures are not subject to the manipulation of the investigator, it is difficult to determine whether mean or peak concentrations, variability, or some other aspect of air pollution is the most important determinant of health effects.

In summary, those epidemiological studies considered most relevant for developing the guidelines have the following features:

- longitudinal study design;
- adequate control of appropriate confounding factors;
- some attempt to take individual variations in exposure into account, (e.g., area of residence, gas stove use).

Epidemiological investigations of the effects in the general population are considered to be the most relevant. Studies of persons exposed to airborne pollutants in the workplace may not reflect potential problems of the general population, since the young, the elderly and other high-risk groups are not accounted for. Moreover, exposure periods and the mixture of pollutants will be different from those in the home.

3.1.2 Clinical Studies

Clinical studies are generally, though not always, conducted in controlled laboratory environments. These studies probably provide the most reliable data from which to derive exposure-response relationships that form the basis for air quality standards. However, clinical studies are restricted for ethical reasons to the examination of mild, temporary effects of short-term exposures in a limited number of subjects. As such, they are most suitable for developing short-term exposure limits.

A good clinical study should control for extraneous variables and experimental bias. In order to satisfy this need, most clinical studies employ a control group. Comparison with this group provides an indication of the effects that the experimental conditions exert on exposed individuals. The inclusion of such a group is very important if the effect of the experimental intervention is to be isolated.

In order to further reduce experimental bias in clinical studies, the allocation of subjects into experimental and control groups should be a random process. That is, all subjects should have an equal chance to be assigned to either the experimental group or the control group. Through this process the study will have a good chance of obtaining comparable groups, such that the measured and unknown characteristics of the subjects at the time of group-allocation will be, on the average, evenly balanced between groups. Also, since most statistical procedures are based on normal distributions, the randomization procedure is necessary in order to meet the assumptions of the statistical design.

In order to reduce experimental bias to a minimum, some studies employ designs in which the subjects, and sometimes the investigators, are not aware of the particular intervention to which a subject has been assigned. The advantage of these "blind" designs is that they reduce the possibility that the subjects and/or investigators will favour certain outcomes based on group status.

Only through the employment of blind designs can the investigator be reasonably assured that extraneous variables and experimental bias have been controlled as much as possible. However, it is sometimes impossible to employ such a design for practical reasons. If, for example, the investigators are testing the pulmonary effects of ozone, and the subjects

and investigators can detect the treatment condition by the smell of ozone, it is clearly not practical to attempt a blind study. This is a limitation of many clinical studies of airborne pollutants.

3.1.3 Animal Studies

Although numerous studies of the effect of airborne pollutants in animal species have been conducted, levels of exposure have, in general, been much higher than those in ambient air. In addition, extrapolation of the results to ambient levels is complicated by the distinct anatomical differences between the respiratory tracts of animals and man. Also, studies are frequently confined to unusually high concentrations of no more than one or two pollutants, rather than to the low concentrations and mixtures of substances generally found in the home. However, the results of such studies are useful in the identification of target organs and systems, in the clarification of mechanisms of toxicity and in the assessment of carcinogenicity.

The reliability of carcinogenesis bioassays in animal species is evaluated on the basis of several features of the design and the results of the study. These features include the size of the experiment (i.e., the numbers of exposed and control animals); the influence of environmental factors (e.g., diet); the route and method of exposure; the doses administered; the species, strain and sex of the animals; the types, site, incidence and time to the development of tumours; and the nature of the exposure-response relationship. Information concerning the kinetics, metabolism and mechanism of action, and the results of epidemiological studies in human populations are also considered when the relevance of the results of carcinogenesis bioassays for man is assessed.

3.2 Approach Used in Deriving Exposure Guidelines

In order to establish accurate, defensible exposure guidelines it is essential to determine the quantitative relation between a given pollutant and its effects. The terms "exposure-response" and "exposure-effect" denote this quantitative relationship. Owing to ethical considerations, such quantitative relationships are difficult to determine with precision in human populations. Nevertheless clinical and epidemiological studies combined with laboratory animal studies can provide a substantial amount of quantitative information concerning the effects of exposure to a given pollutant.

3.2.1 Non-carcinogenic Substances

Regulatory agencies have traditionally attempted to determine a level of exposure below which there are no apparent detrimental effects. This so-called "threshold level" is closely related to the lowest level at which minimal, or reversible, effects can be observed - the "lowest-observable-adverse-effect level" (LOAEL). A safety factor may be incorporated into the derivation of a regulatory standard or guideline depending upon the number and quality of studies upon which the LOAEL is based. This approach has been used to establish guidelines for a number of indoor air pollutants considered in this document.

The size of the safety factor depends to a large degree on whether human rather than animal data are available, whether studies have been conducted directly on those segments of the population believed to be at high risk, and the quality of the studies themselves. Ultimately the choice is based on a consensus decision by experts, but strictly has no scientifically defensible basis.

Owing to uncertainty concerning data obtained in observational studies, the World Health Organization has used a safety factor of two in recommending guidelines for daily and annual exposure to air pollutants; this value has been adopted in deriving some of the guidelines specified in this document.

In instances where there are sufficient data from reliable clinical studies of transient changes in groups at risk (for example, changes in pulmonary function in exercising asthmatics), no safety factor is incorporated in the derivation of a short-term exposure guideline.

Because of the wide variation in individual susceptibility to irritants, notably aldehydes, short-term exposure guidelines have been derived by applying a factor of five to the lowest value reported to cause a significant increase in symptoms of irritation.

It has been suggested that occupational hygiene limits could be adapted for the residential indoor environment by applying a safety factor to accommodate differences such as exposure times, pollutant mixtures and population sensitivities. Without a thorough knowledge of the scientific basis for the occupational limits, however, the Working Group considered such an approach to be scientifically indefensible.

3.2.2 Carcinogenic Substances

There is evidence that for carcinogenic substances threshold levels may not exist. It follows in such cases that there is no level of exposure at which a hazard does not exist, although at very low concentrations the health risks may be so small as to be undetectable. For carcinogens, the derivation of acceptable exposure limits using experimentally derived LOAELs and safety factors is considered inappropriate.

Ideally, exposure to known or suspected carcinogens should be avoided. However, it is not possible to eliminate certain carcinogens from the environment. The maximum concentrations in the exposure ranges listed in this document for formaldehyde, a suspected carcinogenic substance, are the lowest levels that it is practical to achieve and at which there is no undue public health risk.

To ascertain the level of risk, or probability of adverse response at the low concentrations likely to be encountered in the environment, several statistical procedures have been developed to predict the shape of the dose-response curve at doses below those administered in experimental studies. The shape of the extrapolated dose-response curve can influence considerably the value of the exposure concentrations at which there appears to be a negligible human health risk with estimates sometimes ranging over several orders of magnitude, depending on the mathematical model employed.

In deriving the long-term exposure guidelines for formaldehyde, the mathematical procedure for determining cancer risk was selected taking into account as far as possible information concerning the mechanisms of action. Nevertheless, it must be recognized that the calculated risk at low levels of exposure is probably overestimated owing to the conservative assumptions upon which the mathematical model is based.

3.3 Monitoring Procedures

Methods for monitoring indoor air quality have not been standardized to this day. Many studies have been carried out with a combination of older methods and instruments (used also for ambient air monitoring) together with those that have only recently been developed. There is now a trend towards developing smaller portable instruments which can serve both as personal monitors and as stationary (area) monitors. Passive and active personal monitors that measure particulate matter (filters) and gases (absorbers and

adsorbers) have been developed. The small size and quiet operation of this newer equipment render it suitable for use in indoor environments. Such monitors also offer the possibility of determining pollutant levels near the breathing zone of individuals, thus enabling direct estimates of personal exposure to be made.

Monitoring to ascertain compliance with the short-term exposure guidelines should be conducted when "worst case" situations are anticipated, and procedures should be designed to provide an accurate assessment of occupants' actual exposure. It is recommended that samples be taken when and where maximum concentrations are expected and that the averaging times specified along with the ASTER be used.

In the case of the long-term exposure it is more difficult to specify appropriate monitoring procedures. Sampling should be conducted over periods long enough to encompass any diurnal, seasonal or other temporal fluctuations. The magnitude and frequency of these fluctuations may vary considerably from contaminant to contaminant and be a function of dwelling type, location and the activity of the occupants. In many cases annual averages have served as a basis for formulating long-term air quality standards. Frequently, annual averages are based on random sampling of 24-hour average readings. Monthly or weekly averages are typically determined using daily averages, although passive monitors now available may have collection periods of 7 days to 90 days.

It is recommended that a minimum sampling period of 24 hours be used and that samples be taken when concentrations are expected to be at their highest. Should concentrations be outside the specified long-term exposure range, additional samples should be taken to ascertain whether levels are likely to remain elevated, or whether fluctuations will result in the long-term (often annual) average concentrations falling within the guideline value.

If contaminant levels are found to be outside any of the recommended exposure ranges, the source of the problem should be identified. Any possible corrective measures should be taken or advice sought from health authorities.

4.0 GUIDELINES AND RECOMMENDATIONS

PART A: SUBSTANCES WITH EXPOSURE GUIDELINES - NON-CARCINOGENIC EFFECTS

4.A.1 Aldehydes*

In cases where more than one aldehyde is detected in indoor air, the sum

$\frac{c_1}{C_1} + \frac{c_2}{C_2} + \frac{c_3}{C_3}$ should not exceed 1, where c_1 , c_2 , and c_3 are the concentrations

of formaldehyde, acrolein and acetaldehyde, respectively, as measured over a 5-minute period, and C_1 , C_2 and C_3 are as follows:

- C_1 (formaldehyde) - 120 $\mu\text{g}/\text{m}^3$ (0.10 ppm)
- C_2 (acrolein) - 50 $\mu\text{g}/\text{m}^3$ (0.02 ppm)
- C_3 (acetaldehyde) - 9000 $\mu\text{g}/\text{m}^3$ (5.0 ppm).

Aldehyde concentrations in indoor air generally exceed those outdoors. The primary sources include gas stoves, space heaters and tobacco smoke.

Identities of all the aldehydes produced during incomplete combustion of organic fuels are not yet known, but measurements in indoor locations have shown that formaldehyde, acetaldehyde and acrolein are the major aldehydes present.

Concentrations of acrolein in indoor air range from 2 to 50 $\mu\text{g}/\text{m}^3$ (0.001 to 0.02 ppm); limited data available indicate that levels of acetaldehyde average about 17 $\mu\text{g}/\text{m}^3$ and range from 1 to 48 $\mu\text{g}/\text{m}^3$.

The major effect on human health of airborne aldehydes is irritation of the eyes, nose and throat. In recently conducted clinical studies, significant increases in symptoms of irritation have been observed at levels of formaldehyde greater than 1200 $\mu\text{g}/\text{m}^3$ (1 ppm) (exposure periods 1.5 to 30 minutes). Data derived from observational studies of populations exposed to formaldehyde in the occupational environment or in public or residential buildings are less reliable owing to limitations of the investigations conducted to date. In the best-conducted studies, symptoms of irritation have not been associated with exposure to levels less than 600 $\mu\text{g}/\text{m}^3$ (0.5 ppm).

* See also Section 4.B.1, "Formaldehyde"

There are few reliable data available concerning levels of the other aldehydes that induce symptoms. Acrolein is one of the most irritating of the aldehydes identified in indoor air, with most people reporting eye irritation at levels of less than 1 mg/m^3 . A significant increase in symptoms of eye irritation has been associated with exposure to levels as low as $210 \text{ } \mu\text{g/m}^3$ (0.09 ppm): in the same study, however, eye irritation at a concentration of $800 \text{ } \mu\text{g/m}^3$ was only slight. Severe irritation results from exposure to concentrations of $1900 \text{ } \mu\text{g/m}^3$ (0.8 ppm). Chronic effects following exposure to acrolein have not been reported and there has been no evidence of carcinogenicity in long-term bioassays with laboratory animals.

Acetaldehyde is considerably less irritating than acrolein; symptoms of irritation have been associated only with exposure to levels greater than 46 mg/m^3 (25 ppm). In a long-term bioassay in rats, significant increases in the incidence of nasal adenocarcinomas and squamous cell carcinomas were observed following inhalation of acetaldehyde; however, the administered dose levels (1400 , 2700 and 500 mg/m^3 ; 750 , 1500 and 3000 ppm) and mortality rates during the study were extremely high. Moreover, data given in the published report of the investigation were insufficient to permit meaningful quantitative risk estimation.

The recommended values for C_1 , C_2 and C_3 are 5 to 10 times less than the concentrations reported to induce significant increases in symptoms of irritation. Concentrations satisfying the relationship given above should be low enough to minimize additive irritant effects of the specified aldehydes in the general population.

4.A.2 Carbon Dioxide

Based on health considerations, the acceptable long-term exposure range (ALTER) for carbon dioxide in residential indoor air is $< 6300 \text{ mg/m}^3$ (< 3500 ppm).

Carbon dioxide is a colourless, odourless and non-flammable gas, which is produced by metabolic processes and by the combustion of fossil fuels. The average concentration of carbon dioxide in the atmosphere is about 620 mg/m^3 (~ 340 ppm), but levels vary widely with time and location. Indoor levels tend to be higher than outdoor levels. Gas stoves and unvented kerosene heaters are major sources of carbon dioxide indoors, but in poorly ventilated rooms, levels may exceed 5400 mg/m^3 (3000 ppm) from human metabolism alone.

An increase in the ambient level of carbon dioxide brings about a rise in the acidity of the blood and an increase in the rate and depth of breathing. Over prolonged periods, of the order of days, regulation of blood carbon dioxide levels occurs by kidney action and the metabolism of bone calcium. The latter process leads to some demineralization of the bone. Exposure to levels of 27 000 mg/m³ (15 000 ppm) or more for several days has induced reversible changes in the lung membrane of guinea pigs. In humans, exposures to carbon dioxide levels of over 90 000 mg/m³ (50 000 ppm) have produced effects on the central nervous system such as headache and dizziness and visual distortions: there is some evidence of cardiovascular effects at similar concentrations. Subjective symptoms such as fatigue, headaches and an increased perception of warmth and unpleasant odours have been associated with carbon dioxide levels of 900 mg/m³ to 5800 mg/m³ (500 to 3200 ppm). In some of these studies the symptoms may have been caused by other substances, with the carbon dioxide acting as a surrogate measure of air quality (Section 2.3.1).

The lowest concentration at which adverse health effects have been observed in humans is 12 600 mg/m³ (7000 ppm), at which level increased blood acidity has been observed after several weeks of continuous exposure. A maximum exposure level of 6300 mg/m³ (3500 ppm) should provide a sufficient margin to protect against undesirable changes in the acid-base balance and subsequent adaptive changes such as the release of calcium from the bones. This level should also provide an adequate safety margin for sensitive groups. At such a level, the effect of carbon dioxide as a ventilatory stimulant is likely to be small and so would not greatly increase the dose received of other pollutants present in the air.

Changes in the acid-base balance and release of calcium from bones occur in response to chronic carbon dioxide exposure rather than to brief excursions in concentration. Thus, a short-term exposure range is not required for this substance.

4.A.3 Carbon Monoxide

The acceptable short-term exposure ranges (ASTER) for carbon monoxide in residential indoor air are:

< 11 ppm - 8 hour average concentration

< 25 ppm - 1 hour average concentration.

Carbon monoxide is a colourless, odourless gas that is produced in the combustion of carbonaceous materials and also in human metabolism. It combines with haemoglobin to form carboxyhaemoglobin (COHb), which reduces the oxygen supply to body tissues. Endogenous levels of carboxyhaemoglobin are approximately 0.5% of the total haemoglobin (written, 0.5 COHb%).

Sources of carbon monoxide in indoor air include gas and oil appliances, tobacco smoke, and the infiltration of carbon monoxide in polluted outdoor air. Outdoor levels of 0.05 to 0.9 mg/m³ (0.04 to 0.8 ppm) have been measured in rural areas, and levels as high as 57 mg/m³ (50 ppm) have been found in urban areas, although levels of 1.1 to 11 mg/m³ (1 to 10 ppm) are more typical. Indoor levels generally follow outdoor levels except in houses with unvented or poorly vented combustion appliances or where there is tobacco smoking; carbon monoxide levels of approximately 115 mg/m³ (100 ppm) have been found in the kitchens of some houses immediately after gas stoves were used for cooking.

Exposure to carbon monoxide levels leading to carboxyhaemoglobin concentrations of approximately 2.5% to 10% has been shown to cause adverse effects on the cardiovascular system, to decrease exercise capacity and to impair psychomotor performance. Elevated carboxyhaemoglobin levels in women who smoked during pregnancy have been associated with low birthweight and educational retardation of their children. Groups that may be at particular risk from the effects of carbon monoxide exposure include those with cardiovascular, cerebrovascular and peripheral vascular diseases, fetuses, the newborn, pregnant women and individuals living at high altitude.

Experimental results suggest that, in general, such sensitive individuals can tolerate increases in carboxyhaemoglobin levels of up to 1.5 COHb%: the guidelines are intended to ensure that increases due to ambient carbon monoxide remain below this limit. Since carboxyhaemoglobin levels depend on the concentrations of both carbon monoxide and oxygen, levels are expressed only as ratios (parts per million by volume) so that the guidelines will be independent of ambient pressure.

4.A.4 Nitrogen Dioxide

The acceptable exposure ranges for nitrogen dioxide in residential indoor air are:

ALTER: <100 µg/m³ (< 0.052 ppm)

ASTER: <480 µg/m³ (< 0.25 ppm) - 1 hour average concentration.

Nitrogen dioxide (NO_2) is the only oxide of nitrogen which has been shown to be detrimental to human health at concentrations that may be encountered in indoor air.

The primary outdoor sources of nitrogen dioxide are vehicular and industrial emissions. In general, nitrogen dioxide concentrations in urban atmospheres are higher than those in rural atmospheres, reflecting the large contribution of nitrogen dioxide from technological sources. In North America, the background level of nitrogen dioxide in rural areas is less than $19 \mu\text{g}/\text{m}^3$ (0.010 ppm). In urban centres, nitrogen dioxide levels are at least double this value. During the period 1977 to 1981 the average of nitrogen dioxide annual means for Canadian urban centres decreased from 60 to $44 \mu\text{g}/\text{m}^3$ (0.031 ppm to 0.023 ppm). The highest annual mean reported in Canada for nitrogen dioxide ($80 \mu\text{g}/\text{m}^3$; 0.042 ppm) occurred at a commercial site in 1981.

Gas stoves and unvented combustion appliances are major sources of nitrogen dioxide indoors. The indoor/outdoor ratio of nitrogen dioxide concentrations is generally less than unity in dwellings in which there are no major indoor sources, and greater than unity in dwellings with gas stoves and/or other combustion appliances. Families living in rural or low-pollution areas and who use gas for cooking are exposed to indoor nitrogen dioxide levels of roughly $30 \mu\text{g}/\text{m}^3$ (0.015 ppm), although average concentrations of $100 \mu\text{g}/\text{m}^3$ (0.050 ppm) have been recorded in some homes.

Interpretation of the results of available epidemiological studies on health effects associated with nitrogen dioxide exposure is rendered difficult by the lack of accurate exposure data and by confounding factors such as exposure to other pollutants. Despite these limitations, the epidemiological studies have provided some useful data concerning exposure-effect relationships. In these studies an increased prevalence of respiratory illness in adults and children chronically exposed to mean levels of near $200 \mu\text{g}/\text{m}^3$ (0.10 ppm) nitrogen dioxide was observed.

The results of clinical studies indicate that both normal and asthmatic subjects can experience detrimental respiratory effects when exposed for brief periods to concentrations of approximately $960 \mu\text{g}/\text{m}^3$ (0.5 ppm). The short-term effects of nitrogen dioxide exposure below $960 \mu\text{g}/\text{m}^3$ (0.5 ppm) have been examined in only a few studies. A "no-adverse-effect level" cannot be clearly identified from the results of these studies; therefore a safety factor of two was applied to arrive at the recommended short-term exposure range.

4.A.5 Ozone (Oxidants)

The acceptable short term exposure range (ASTER) for ozone in residential indoor air is $< 240 \mu\text{g}/\text{m}^3$ (< 0.12 ppm) - 1 hour average concentration.

Infiltration of outdoor air is the principal source of oxidants in indoor air. Ozone, nitrogen dioxide, hydrogen peroxide and peroxyacylnitrates are photochemical oxidants that may be present in indoor air. Nitrogen dioxide is examined in Section 4.A.4. Of the remaining oxidants, ozone is the most prevalent. Concentrations of ozone indoors are generally much lower than those outdoors, but may approach outdoor levels if windows are open. Indoor concentrations of ozone follow outdoor fluctuations with a time lag of one hour or less. The average annual outdoor concentration for urban centres in Canada was $30 \mu\text{g}/\text{m}^3$ (0.015 ppm) in 1979. Indoor ozone concentrations are typically less than $40 \mu\text{g}/\text{m}^3$ (0.02 ppm), although peak levels of 200 to $400 \mu\text{g}/\text{m}^3$ (0.1 to 0.2 ppm) have been reported. Ozone can be generated in the home by arcing of electric motors and by improperly installed or maintained electrostatic air cleaners.

Ozone is an irritant that can cause coughs, chest discomfort, and irritation of the nose, throat and trachea. Ozone consistently causes detrimental effects on the lung function of healthy subjects at concentrations at or above $600 \mu\text{g}/\text{m}^3$ (0.30 ppm). Furthermore, ozone causes detrimental effects on the lung function of healthy subjects engaged in strenuous physical activity at concentrations lower than $600 \mu\text{g}/\text{m}^3$ (0.30 ppm), possibly as low as $240 \mu\text{g}/\text{m}^3$ (0.12 ppm). Results of epidemiological studies conducted to date support this finding. The available epidemiological data are, however, insufficient to serve as a basis for establishing an acceptable long term exposure range.

Individuals exposed to concentrations of ozone between 200 and $800 \mu\text{g}/\text{m}^3$ (0.10 and 0.40 ppm) have exhibited an adaptive response, at least in terms of lung function. At present, it is not known if this adaptation is beneficial or detrimental in the long term. Available data are insufficient to serve as a basis for establishing an acceptable long-term exposure range for ozone.

4.A.6 Particulate Matter

The acceptable exposure ranges for fine particulate matter ($< 2.5 \mu\text{m}$ mass-median aerodynamic diameter - MMAD) in residential indoor air are:

ALTER: $< 40 \mu\text{g}/\text{m}^3$

ASTER: $< 100 \mu\text{g}/\text{m}^3$ - 1 hour average concentration.

Airborne particulate matter is a mixture of physically and chemically diverse substances, present in air as suspensions of solids and/or liquid droplets, varying in size from about 0.005 μm to 100 μm . The size range of concern when human health effects and indoor air quality are considered is from 0.1 to 10 μm in aerodynamic diameter, particles smaller than this generally being exhaled. Above 15 μm , most particles are too large to be inhaled. Virtually all particles between 10 and 15 μm are deposited in the nasopharyngeal region of the respiratory tract; health effects are associated primarily with the deposition of particles in the thoracic (tracheobronchial and pulmonary) regions. Particles have been further divided into a coarse fraction, normally around 2.5 μm and above, and a fine fraction under this size. It is this latter fraction that can reach the lung alveoli.

Indoor particles come from both indoor and outdoor sources, but the indoor matter differs in both size and chemical composition from that originating outdoors. Indoors, particles occur primarily in the fine fraction, because indoor sources such as combustion appliances and cigarettes tend to produce fine particles, and the building envelope acts as a partial filter to screen out larger particles. Indoor particulate matter contains a much higher fraction of organic matter than that of outdoor air, largely because of household activities such as cooking, cleaning and use of consumer products.

Indoor concentrations of fine particulate matter tend to be higher than those outdoors. Average concentrations of particles under 3.5 μm (respirable suspended particulates or RSP) range between 20 and 30 $\mu\text{g}/\text{m}^3$. Higher concentrations have been noted in "dirty" cities with high outdoor levels, and in homes with smokers or wood stoves. Cigarette smoke appears to be the most significant indoor source of particulate matter, and the presence of resident smokers has been shown to raise levels of fine particles in homes by between 12 and 40 $\mu\text{g}/\text{m}^3$ per smoker.

Numerous epidemiological studies indicate that human health has improved as concentrations of airborne particulate matter have decreased. Despite the many uncertainties in these studies, they provide some useful information on levels at which adverse health effects might be expected. Increases in

Increases in mortality have been observed especially among the elderly and those with pre-existing respiratory or cardiovascular disorders, when they were exposed to concentrations of particles (including coarse particles) above $500 \mu\text{g}/\text{m}^3$ accompanied by high sulphur dioxide levels for periods of one to four days. Increases in hospital admissions and in respiratory clinic visits were also noted at about the same levels, while increased prevalence of respiratory symptoms and discomfort in persons at increased risk because of pre-existing respiratory conditions were first observed at levels in the range 250 to $350 \mu\text{g}/\text{m}^3$. In children, marginal decrements in lung function lasting several weeks were also associated with short exposures at about these levels, which were correlated with outdoor and indoor RSP levels estimated to be about $80 \mu\text{g}/\text{m}^3$. Clinical studies, while not necessarily representing usual exposure conditions, also indicated that short exposures to concentrations of fine particulates (expressed as sulphuric acid) above $100 \mu\text{g}/\text{m}^3$ could result in irritation and alterations in respiratory function in asthmatic subjects and in slowing of bronchial clearance in normal individuals.

Chronic exposures for periods of several years to moderate levels of airborne particles estimated to be around $180 \mu\text{g}/\text{m}^3$ total suspended particulates or $80 \mu\text{g}/\text{m}^3$ fine particles (respirable suspended particulates or RSP) appear to be correlated with increased prevalence of respiratory symptoms and chronic respiratory disease, accompanied by reduced respiratory-function measurements, in adults and children.

4.A.7 Sulphur Dioxide

The acceptable exposure ranges for sulphur dioxide in residential indoor air are:

ALTER: $< 50 \mu\text{g}/\text{m}^3$ (< 0.019 ppm)

ASTER: $< 1000 \mu\text{g}/\text{m}^3$ (< 0.38 ppm) - 5 minute average concentration.

Sulphur dioxide is the main oxide of sulphur found in indoor air. Indoor concentrations are generally lower than those outdoors by a factor of about two, primarily because most sources are outdoors, and sulphur dioxide is readily absorbed by furnishings and fabrics.

Interpretation of the results of available epidemiological studies on health effects associated with exposure to sulphur dioxide is complicated by a paucity of representative exposure data and by confounding factors such as

exposure to other air pollutants. However, such studies have provided some useful albeit uncertain data concerning exposure-effect relationships. Excess mortality, particularly among the elderly and those with pre-existing cardiopulmonary disease, has been observed in populations exposed to 24-hour pollution episodes in which sulphur dioxide concentrations exceeded 300 to 400 $\mu\text{g}/\text{m}^3$ (0.12 to 0.15 ppm). Increases in hospital admissions and emergency room visits have also been associated with exposure to these levels. Increased prevalence of acute and chronic respiratory symptoms and impaired pulmonary function have been observed in adults and children exposed for extended periods (>1 year) to mean levels of 100 $\mu\text{g}/\text{m}^3$ (0.038 ppm) sulphur dioxide.

Relevant data have also been obtained from clinical studies; however, exposures in such investigations are short and do not necessarily represent usual exposure conditions. In normal subjects, increased airway and nasal flow resistance and a change in the mucociliary flow rate have been observed following exposure to 2600 $\mu\text{g}/\text{m}^3$ (1.0 ppm) sulphur dioxide; reversible increases in specific airway resistance have been observed in asthmatics exposed by natural breathing for brief periods to concentrations exceeding 1000 $\mu\text{g}/\text{m}^3$ (0.38 ppm).

4.A.8 Water Vapour

Based on health considerations, the acceptable short-term exposure ranges (ASTER) for water vapour in residential indoor air are:

30% to 80% relative humidity - summer

30% to 55% relative humidity - winter*

* unless constrained by window condensation.

For purposes of indoor air quality, the most useful measure of water vapour levels is relative humidity, the ratio of the concentration of water vapour present to the concentration needed to saturate air at that temperature. Indoor humidity is determined by the humidity and temperature of outdoor air as well as by indoor sources and sinks of water vapour. The main indoor sources are human and animal metabolism, and such activities as bathing, cooking, and the washing and drying of clothes. Small amounts of water vapour are also produced by combustion.

The moisture content of indoor air is reduced by dilution with drier outdoor air, by condensation on cold surfaces, and by absorption or adsorption of water by materials in the home. Homes heated electrically are likely to have higher indoor relative humidities in winter than comparable homes heated by combustion furnaces, since the latter tend to increase the infiltration of dry outside air. Relative humidities in Canadian homes have been found to range from 21% to 68%.

In conjunction with temperature and air flow, relative humidity affects comfort, conditions of 20% to 60% relative humidity at temperatures between 20° and 25°C usually being judged comfortable. Long periods of low relative humidity are believed to cause dryness of the skin and mucous membranes, which may lead to chapping and irritation. High humidity at high temperatures leads to increased sweating and a loss of electrolytes from the blood; prolonged exposures may lead to heat exhaustion or heat stroke. Groups that may be at particular risk from high humidity are those suffering from cardiovascular disease, infants born two or three weeks before term, and the elderly. Arthritis sufferers have been found to experience increased symptoms when a rise in humidity accompanies a drop in atmospheric pressure. Sufferers from asthma develop symptoms of bronchoconstriction after exercise more readily when breathing air at low humidity.

Several species of bacteria and viruses survive best at low or high, rather than intermediate, humidities. Humidity levels above 50% have been found to increase the population size of moulds, fungi and mites that may cause allergies. The evidence suggests that humidity levels should be maintained between 40% and 50% to reduce the incidence of upper respiratory infections and to minimize adverse effects on people suffering from asthma or allergies. Such a range would be hard to maintain, however, and exposure to higher or lower levels is unlikely to affect the health of most people.

PART B: SUBSTANCES WITH EXPOSURE GUIDELINES - CARCINOGENIC EFFECTS

For substances designated as human, or potential human carcinogens, a continuing effort should be made to reduce exposure to the lowest possible level. It is recognized that attainment of this goal must be considered in light of the cost and feasibility of remedial measures and technological changes. It was, therefore, considered desirable to specify exposure guidelines both in terms of what can be attained now (action level) and what should be striven for as a longer term objective (target level).

4.B.1 Formaldehyde*

The exposure guidelines for formaldehyde in residential indoor air are:

ACTION LEVEL: 120 $\mu\text{g}/\text{m}^3$ (0.10 ppm)

TARGET LEVEL: 60 $\mu\text{g}/\text{m}^3$ (0.05 ppm)

Formaldehyde is a colourless gas with a pungent odour. It combines readily with water to form a non-volatile compound and has a tendency to be absorbed on to surfaces and textiles, such as carpets and curtains. An equilibrium is established between formaldehyde in air and that adsorbed on surfaces and within wood products such as particleboard.

Formaldehyde is formed naturally in the environment; outdoor concentrations in remote regions are about 7 $\mu\text{g}/\text{m}^3$ (0.006 ppm). Outdoor levels in Canadian locations are about 10 $\mu\text{g}/\text{m}^3$ (0.008 ppm). Man-made sources of formaldehyde include combustion and the decomposition of formaldehyde resins used in wood, paper, textiles or urea-formaldehyde foam insulation (UFFI). In two large Canadian surveys, average formaldehyde concentrations of 14 and 42 $\mu\text{g}/\text{m}^3$ (0.011 and 0.034 ppm) were found in homes that did not have UFFI; higher average levels were detected in homes with UFFI (66 $\mu\text{g}/\text{m}^3$; 0.054 ppm). Levels in mobile homes which generally contain a high proportion of urea-formaldehyde resin pressed wood products tend to be even higher than the concentrations in homes with UFFI.

Formaldehyde is a vitally important intermediate in the normal metabolism of cells. It serves as a building-block for the synthesis of purines, pyrimidines and many amino acids and lipids and is a key molecule in one-carbon metabolism. It is present at low levels in body fluids, and exposure to exogenous sources does not lead to any appreciable increase in these levels.

* See also Section 4.A.1, "Aldehydes".

Formaldehyde gas is a sensory irritant, primarily affecting the nasal passages, respiration and the eyes (see Section 4.A.1). In addition, in two well-conducted bioassays and a more limited study, formaldehyde has been carcinogenic in two strains of rats, producing a high incidence of nasal squamous cell carcinomas (38% to 50%) following administration of approximately 18 mg/m^3 (15 ppm). Formaldehyde is also genotoxic in a number of assays and is weakly mutagenic in cultured human cells as well as in other mammalian cells, *Drosophila*, fungi and bacteria. Although the epidemiological studies conducted to date provide little convincing evidence that formaldehyde is carcinogenic in human populations, the possibility cannot be excluded owing to limitations of the available data.

Because of the possible carcinogenicity of formaldehyde, it would be prudent to reduce indoor levels as much as possible. The action level of $120 \text{ } \mu\text{g/m}^3$ is the lowest concentration considered to be feasible at the present time. However, it is recommended that in future, and where remedial measures are taken, every effort be made to reduce concentrations to below the target value ($60 \text{ } \mu\text{g/m}^3$).

PART C: SUBSTANCES WITH RECOMMENDATIONS FOR CONTROLLING EXPOSURE

In examining the need for guidelines for pollutants listed in this part of the document, the scientific literature was reviewed as it was for pollutants listed in Parts A and B of this Section. The development of quantitative exposure guidelines was, however, considered inappropriate for a number of reasons:

- for some groups of substances the individual components may have widely differing toxicological properties; the complexity of the mixtures precluded establishing a guideline for each constituent or for the group as a whole;
- establishment of air quality guidelines may not be the appropriate strategy for control, especially where inhalation is not the most significant route of exposure;
- there are deficiencies in the scientific data base.

For these substances or groups, information on potential adverse health effects and possible sources has been provided, and recommendations that should help to eliminate or reduce exposure to them developed.

4.C.1 Biological Agents

In order to prevent many of the common indoor problems due to biological agents, measures should be taken to ensure that:

- excess humidity and condensation are not present;
- surfaces are kept free of dust;
- stagnant water sources, such as humidifier tanks, are kept clean and occasionally disinfected;
- a high standard of appropriate personal hygiene is maintained.

The indoor environment can present the potential for illness due to exposure to biological agents. These agents may include microorganisms from humans, pets or insects within the home, or from growth on surfaces or in stagnant water. Dust from outside and inside the home includes pollens, spores, cells, cell debris and insects. Such materials in the air may cause infectious diseases or allergic illnesses in sensitized persons in the home. Illnesses must be systematically investigated in order to reveal the cause and to determine possible means of control. Control measures include disinfection and physical removal of the agent where possible and various means of controlling particulate matter and excess humidity. Due

to differential individual sensitivity, however, in some cases extraordinary measures may be required to prevent symptoms from occurring.

The variety of biological agents that may occur in air is immense, and their potential for effects on susceptible individuals is unpredictable. The complexity of the problem and the lack of data from which contamination levels can be related to disease incidence mean that it is not possible to recommend limits for biological agents in general.

4.C.2 Consumer Products

It is recommended that exposures resulting from the use of consumer products be kept to a minimum by ensuring adequate ventilation and observing any other precautionary measures described on the product label, and in any accompanying information.

Pest control products should be used only when absolutely necessary.

4.C.2.1 Chlorinated Hydrocarbons

Large quantities of chlorinated hydrocarbons are produced and used annually, worldwide. They are present in the home environment principally as solvents, cleansers and aerosol propellants, and some individuals may be exposed to relatively high levels of them in the pursuit of hobbies. In some cases, chlorinated hydrocarbons may be released continuously from household products; they have also been detected in drinking water.

Chlorinated hydrocarbons are absorbed into the body principally by inhalation, but also through the skin and gastrointestinal tract; they tend to accumulate in fatty tissues such as the brain, bone marrow and body fat. Recovery from the acute effects of exposure to the volatile chlorinated hydrocarbons is usually complete, but after repeated exposures, adverse health effects can include depression of, or permanent damage to, the central nervous system, irritation of the eyes and lungs, and damage to the skin, liver and kidneys. In the case of dichloromethane, a metabolite is carbon monoxide, which can cause cardiovascular stress (see Section 4.A.3).

In the home, exposures occur primarily through the use of consumer products and can be of short duration, but levels may be sufficiently high to have the potential for adverse health effects.

4.C.2.2 Pest Control Products

Pest control products comprise a very large number of diverse chemicals. They are widely used in and around the home environment, both by residents and by professional pest control applicators. These products are employed to control insects in the home, to prevent insect damage to fabrics, to treat houseplants against both insects and fungi, to treat pets, and to disinfect the air, water and surfaces around the home. Occasional inadvertent exposure may occur because of impregnation of building products or household articles with pesticidal preservatives and subsequent release of these into the air. Surfaces in the home may be coated with pesticides after normal use, and food prepared or stored in the room during or after pesticide use can also be contaminated. Pesticides may also infiltrate homes after outdoor application. Many pest control products contain, in addition to the active ingredient(s), non-active ingredients such as solvents, wetting agents and stabilizers. Such ingredients may have much higher vapour pressures, and hence be present at higher airborne levels, than the active ingredients, and a few have biological activity of their own.

Exposure to pest control products in the home can occur both by inhalation and by absorption through the skin, for example, following contact with pesticide-treated surfaces. Pesticides may also be ingested following injudicious use in the vicinity of foods.

Available data on exposure levels in the home indicate that airborne levels of most pesticides are very low if products are used as directed. Misuse of pesticides, whether through failure to follow the instructions provided, through heavy use in areas where food is stored, prepared or eaten, through use in poorly ventilated spaces, or through misapplication of products designed for outdoor use, provides the greatest potential for exposure in the home, and levels can then be high enough to create a health hazard.

Adverse human health effects from exposure to low levels in the home tend to be non-specific, similar to ailments caused by many other chemical agents: respiratory effects, coughing, burning of eyes and nose, rhinitis, headache, dizziness, tiredness and general malaise. Two major groups of pesticides are anticholinesterase agents, causing augmentation of secretory activity in bronchial, lachrymal, salivary and other glands, and contraction of smooth muscles of the bronchioles. Allergic reactions to a few pesticides occur very occasionally. Most pesticides used in Canada today break down quickly in body tissues. No pesticides available for domestic use in Canada have been assessed as human carcinogens.

Pest control products are subject to extensive review and regulation by several federal and provincial departments before they are released to the market. This ensures that the consumer is provided only with products that, although potentially toxic, are safe if used as directed and that adequate instructions and warnings regarding the use of these products are also provided. Thus, the consumer is ultimately responsible for the proper use of pesticides in the home.

4.C.2.3 Product Aerosols

An aerosol can be a suspension of fine liquid or solid particles in air or other gases; in these guidelines, product aerosols are considered to be consumer products that are dispersed under pressure from disposable containers.

An aerosol product has three main components: the active ingredient or ingredients, the propellant, and miscellaneous additives used to improve the product, such as plasticizers, synthetic resins, surfactants, and emulsifiers. Some products consist of gaseous components only and therefore do not result in the production of an aerosol.

Aerosol propellants used currently in Canada include hydrocarbons, such as propane, butane and isobutane; nitrous oxide; dichloromethane (which is commonly called methylene chloride); carbon dioxide; dimethyl ether; and nitrogen. Nitrous oxide may also be used as the propellant for food products.

Product aerosols are formulated from a wide variety of chemicals, some of which are potentially toxic at high concentrations.

Certain components may be subjected to review, and hence control, under federal legislation such as the Hazardous Products Act and the Environmental Contaminants Act. For example, prior to 1980, fluorochlorohydrocarbons were used as propellants for most aerosol sprays. Fluorochlorohydrocarbons are still used as aerosol propellants in many products, including aerosol cooking sprays, but trichlorofluoromethane and dichlorodifluoromethane have been banned, under the Environmental Contaminants Act, for use as propellants in hair sprays, deodorants and antiperspirants.

Generally the use of product aerosols in the home is of short duration and intermittent, ranging from about once or twice a day for deodorant sprays to four times a year for oven cleaners.

4.C.3 Fibrous Materials

Precautions should be taken to minimize inhalation of, and skin contact with mineral fibres during home renovations and installation operations. Materials and products containing fibres should also be examined periodically for signs of deterioration. Advice should be sought before removing or damaging any materials thought to contain asbestos.

Asbestos is the general term for six fibrous silicate minerals that are useful because of their high tensile strength, durability, flexibility and resistance to heat and chemicals. A large proportion of total asbestos production is used in the construction industry in materials such as asbestos-cement sheeting and pipes. Under normal conditions of use, asbestos fibres are not expected to be released from such materials. Fibres may, however, be released from friable surfaces (such as sprayed asbestos-containing insulation or low-density insulation blocks), or from other construction materials in the course of renovation or maintenance. Available data indicate that, in general, asbestos levels in homes are not significantly greater than those in ambient air. Concentrations as much as three orders of magnitude higher have been measured during such operations as sanding of dry-wall taping compounds. Under the Hazardous Products Act, asbestos is now prohibited in most consumer products where respirable dusts are generated during normal use.

Prolonged exposure to elevated levels of asbestos fibres causes asbestosis, lung cancer, mesothelioma and possibly laryngeal cancer and malignancies of the gastro-intestinal tract. The risk of developing asbestosis associated with exposure to levels of asbestos in indoor or ambient air is probably negligible. It is difficult to quantify the risks of lung cancers and mesothelioma associated with exposure to indoor (and outdoor) levels owing, in part, to problems inherent in using data from historical epidemiological studies for extrapolation, but mainly owing to the complexity of asbestos itself (i.e., variations in risk associated with different sizes and properties of the fibres). Nevertheless, it is likely that these risks are small.

Man-made mineral fibres (MMMF) include fibrous glass, mineral wool and ceramic fibres. Fibrous glass accounts for approximately 80% of all MMMF produced and is used mainly as thermal or acoustical insulation. There are few data on levels of fibrous glass in residences; mean levels measured during the installation of glass-fibre insulation have ranged from 0 to 8 fibres/mL. Levels in homes are probably not significantly above ambient levels except during such installations or modifications. Levels in public buildings have been found to range from 0 to 0.008 fibres/mL.

Glass fibres cause transient irritation of skin and eyes in workers occupationally exposed to them. Long-term studies have provided only equivocal evidence of respiratory disease as a result of exposure to glass fibres; however, an excess of lung cancer deaths (not consistently related to dose or duration) has been found in mineral wool workers 20 or more years after first exposure. Smaller excesses have been observed in fibrous glass workers exposed for more than 30 years. The available data indicate that man-made mineral fibres are less pathogenic than asbestos, possibly because of their size distribution and lower tendency to fragment in the lung.

4.C.4 Lead

In order to minimize the exposure of people, and especially children, to lead of airborne origin, it is recommended that surfaces that may be contaminated be cleaned frequently and that a high standard of overall cleanliness be maintained.

Airborne lead is present mainly as inorganic lead compounds in dust particles. More than 90% of the global emissions of airborne lead are from man-made sources, principally the combustion of leaded gasoline, followed

by mining and smelting. Atmospheric levels of lead in remote areas are in the range 0.05 to 8 ng/m³. Levels in urban areas depend upon proximity to roadways and industrial sources, and upon such factors as traffic density, wind speed and height above the ground. Annual geometric mean lead concentrations measured in Canada declined steadily from 0.74 µg/m³ in 1973 to 0.27 µg/m³ in 1982.

The major indoor source of airborne lead is the outside air, and indoor levels tend to be lower than outdoor levels.

People are exposed to airborne lead both directly by inhalation and indirectly by ingestion of lead that has settled as dust. In adults, approximately 10% of the ingested lead is absorbed; for young children the figure may be as high as 53%. The amount of lead absorbed from the lungs is believed to range from 30% to 50% of the total inhaled lead.

Once lead is absorbed, it is distributed to the soft tissues and the skeleton. Lead in blood reflects current exposure to lead and has a biological half-life of about 16 days. Lead in the skeleton represents long-term accumulation and its half-life is several decades.

Lead can produce many toxic effects in the body. The main symptoms of lead poisoning include anaemia, abdominal cramps, constipation, renal damage and encephalopathy. Children are more sensitive than adults to the harmful effects of lead and may also experience irritability and loss of appetite. Learning impairment and alterations in neurobehavioural responses may occur at low exposure levels.

There is uncertainty in determining total exposure to airborne lead because of indirect exposure to lead of airborne origin that has settled as dust. Therefore it is not possible to derive an acceptable air lead level for the indoor environment.

Although lead is introduced into the domestic environment mainly as an airborne pollutant, the major pathway for exposure is through ingestion of dust once it has settled. Exposure to lead can be controlled to some extent in homes by frequent cleaning of surfaces, including food preparation areas.

4.C.5 Polycyclic Aromatic Hydrocarbons (PAHs)

Exposure to polycyclic aromatic hydrocarbons indoors should be kept to a minimum by:

- ensuring that any combustion systems, for example wood- and coal-burning stoves, are properly installed and maintained and operated under conditions of satisfactory ventilation;
- adhering to the guidelines and recommendations given in this document for particulate matter and tobacco smoke.

Polycyclic aromatic hydrocarbons (PAHs) are a large class of organic compounds, most of which are non-volatile solids that are very insoluble in water. They are frequently adsorbed onto the surfaces of particulates, and over 100 PAHs have been detected in airborne particulate matter.

Polycyclic aromatic hydrocarbons are produced when materials containing carbon and hydrogen are burned. Coal-burning and the use of internal combustion engines are reported to be major sources, although it has also been claimed that residential wood-burning is the major source of PAHs in the USA. Outdoor levels have been measured in the range 0.1 to 60 ng/m³ in urban areas and 0.001 to 2 ng/m³ in rural areas. Indoor levels are often dominated by levels in the outdoor air, but cooking (where charring of food occurs), improperly operating wood stoves and open fireplaces and tobacco smoking can add significantly to indoor exposures. There are few quantitative data on levels of airborne PAHs in houses, and limitations in the available methods for collecting and measuring PAHs may mean that the available data are unreliable.

Exposure to PAHs is possible through skin contact, inhalation and ingestion. Although it has been estimated that ingestion of foods accounts for most of the exposure to PAHs, exposures through the dermal and inhalation routes appear to have more significant effects on human health. In particular, in some instances elevated concentrations of PAHs have been found in air, and the concern over exposure to airborne PAHs centres on the potential of these compounds to cause lung cancer.

There is a paucity of toxicological data (especially inhalation data) for most individual PAHs and almost no data for PAH mixtures. Epidemiological studies of humans are limited by the fact that exposures are usually to low levels of PAH mixtures, and often in the presence of other pollutants. As a result it is impossible to identify the effects of any particular PAH and to reliably quantify the risks to human health.

The lack of reliable data on which to base dose-response relationships and the difficulty of distinguishing the effects of PAHs in the environment

from those of other pollutants prevent a guideline for exposure to PAHs in indoor air from being assigned. Since some PAHs are known to be carcinogenic, exposure to these substances should be minimized.

4.C.6 Tobacco Smoke

In view of the carcinogenic properties of tobacco smoke, it is recommended that any exposure to tobacco smoke in indoor environments be avoided.

Tobacco smoke is a complex mixture of substances, including carbon dioxide, carbon monoxide, oxides of nitrogen, and a large number of organic vapours and solids. Over fifty of the components are known to cause adverse health effects; twelve (including vinyl chloride, 2-naphthylamine, benzo(a)pyrene and formaldehyde) are known or suspected carcinogens. Carbon dioxide, carbon monoxide, oxides of nitrogen, formaldehyde and particulate matter are among the components of tobacco smoke for which individual air quality guidelines are recommended elsewhere in this document.

The largest amounts of most components are found in the smoke emitted into the environment directly from the burning end of the cigarette. Symptoms reported by non-smokers exposed to such "sidestream" smoke include eye, nose and throat irritation, headache, nausea, dizziness and loss of appetite. Furthermore, the lingering odour and reduced visibility from tobacco smoke are aesthetically unpleasant to many people.

Increased risks of lung cancer have been observed in non-smoking populations exposed to sidestream smoke. Other suspected health effects of tobacco smoke on non-smokers include the aggravation of such conditions as asthma and angina pectoris, increased risks of spontaneous abortion, congenital malformation or sudden infant death syndrome in the children of smoking mothers, and retarded development of children whose mothers were exposed to tobacco smoke during pregnancy. Increased risks of respiratory diseases have been observed among children whose parents were smokers, and non-smoking wives of smokers have been found to have increased risks of death from nasal sinus cancer and ischaemic heart disease.

Estimates have been made showing that non-smokers repeatedly exposed to tobacco smoke are at a significantly higher risk of contracting tobacco-smoke-induced lung cancer. While these calculations involve a number of assumptions that lead to uncertainty in the actual magnitude of the hazard, it is widely believed that there is no level of exposure to carcinogenic substances below which a risk does not exist.

GLOSSARY

Adenocarcinoma	malignant tumour of glandular origin.
Aerosol	particles dispersed in a gas.
Allergy	unusual sensitivity to particular food, particles, irritants, etc.
ALTER	acceptable long-term exposure range.
Alveolus	small cavity or socket (usually in lung where oxygen and carbon dioxide are exchanged).
Anaemia	deficiency of red blood cells or their haemoglobin.
Angina pectoris	thoracic (chest) pain with a feeling of suffocation.
Aromatic	compound having physical and chemical properties resembling those of benzene.
Asbestosis	chronic lung inflammation caused by inhalation of asbestos fibres.
ASTER	acceptable short-term exposure range.
Asthma	disease (especially allergic) of respiration, especially with paroxysms of difficult breathing.
Bronchiole	minute branch of bronchus.
Bronchus	either of the two main divisions of the trachea (windpipe).
Carcinogen	cancer-producing substance.
Carcinogenesis	production of a carcinoma.
Cardiovascular	pertaining to the heart and blood vessels.
Confounding factor	a variable that distorts the apparent magnitude of the effect of a study factor on risk.
Dermal	of the skin.
Dermatitis	inflammation of the skin.
Encephalopathy	any degenerative disease of the brain.
Endogenous	developing or originating within the organism.
Epidemic	prevalent among a community at a particular time (wide-spread).
Epidemiology	science of epidemics.

Exogenous	developed or originating outside the organism.
Genotoxic	toxic to the genome (i.e., DNA).
Haemoglobin	the oxygen-carrying pigment of the red blood cell.
Hydrocarbons	compounds composed of hydrogen and carbon.
Ischaemia	reduction of blood supply to part of the body.
Larynx (adj. Laryngeal)	the voice-box at the entrance to the windpipe in front of the neck.
Mesothelioma	malignant tumour derived from mesothelial tissue.
Morbidity	incidence of sickness in a given period.
Morphology	comparative anatomy.
Mortality	death rate in a given period.
Mucociliary	removal of inhaled particles from the lung by the clearance ciliated and mucus-coated epithelium of the conducting airways.
Mucosa	mucous membrane.
Mutagen	agent causing mutation.
Mutation	genetic change which when transmitted to offspring gives rise to heritable variation.
Pathogen	agent causing disease.
Pesticide	substance for destroying pests.
Photochemical oxidant	any chemical which enters into oxidation reactions in the presence of light or other radiant energy.
Pulmonary	pertaining to the lungs.
Renal	pertaining to the kidneys.
Rhinitis	inflammation of the lining of the nose.
Squamous	covered with, or composed of scales.
Surfactant	surface active agent, decreases surface tension.
Surrogate	something that acts for, or takes the place of another.
Synergism	combined effect of drugs, pollutants, etc. that exceeds the sum of their individual effects.

Teratogen an agent causing defects in a developing foetus.

Toxicological pertaining to toxicology, the study of the nature and effects of hazardous substances.

Trachea windpipe.

Summary of Exposure Guidelines

Contaminant	<u>Acceptable Exposure Ranges</u>		Page
	<u>ASTER</u>	<u>ALTER</u>	
Aldehydes (total)	$\sum c_i/C_i < 1^{(a)}$	-	16
Carbon Dioxide	-	$< 6\ 300\ \mu\text{g}/\text{m}^3$ ($< 3\ 500\ \text{ppm}$)	17
Carbon Monoxide	$< 11\ \text{ppm} - 8\ \text{h}^{(b)}$ $< 25\ \text{ppm} - 1\ \text{h}^{(b)}$	-	18
Formaldehyde	(c)	(d)	26
Nitrogen dioxide	$< 480\ \mu\text{g}/\text{m}^3$ ($< 0.25\ \text{ppm}$) - 1 h	$< 100\ \mu\text{g}/\text{m}^3$ ($< 0.05\ \text{ppm}$)	19
Ozone	$< 240\ \mu\text{g}/\text{m}^3$ ($< 0.12\ \text{ppm}$) - 1 h	-	21
Particulate Matter ^(e)	$< 100\ \mu\text{g}/\text{m}^3 - 1\ \text{h}$	$< 40\ \mu\text{g}/\text{m}^3$	21
Sulphur Dioxide	$< 1\ 000\ \mu\text{g}/\text{m}^3$ ($< 0.38\ \text{ppm}$) - 5 m	$< 50\ \mu\text{g}/\text{m}^3$ ($< 0.019\ \text{ppm}$)	23
Water Vapour	30-80% R.H. - summer 30-55% R.H. - winter ^(f)	-	24

^a $C_i = 120\ \mu\text{g}/\text{m}^3$ (formaldehyde); $50\ \mu\text{g}/\text{m}^3$ (acrolein); $9000\ \mu\text{g}/\text{m}^3$ (acetaldehyde), and c_i are respective concentrations measured over a 5 minute period.

^b Units given only in parts per million so that guidelines are independent of ambient pressure.

^c See Aldehydes (total).

^d See page 26.

^e $< 2.5\ \mu\text{m}$ mass median aerodynamic diameter - MMMD.

^f Unless constrained by window condensation.

APPENDIX B

Summary of Exposure Control Recommendations

<u>Contaminant</u>	<u>Recommendation</u>	<u>Page</u>
Biological Agents	In order to prevent many of the common indoor problems due to biological agents, measures should be taken to ensure that: <ul style="list-style-type: none"> - excess humidity and condensation are not present; - surfaces are kept clear of dust; - stagnant water sources, such as humidifier tanks, are kept clean and occasionally disinfected; - a high standard of appropriate personal hygiene is maintained. 	28
Consumer Products (chlorinated hydrocarbons, pest control aerosols)	It is recommended that exposures resulting from the use of consumer products be kept to a minimum by ensuring adequate ventilation and observing any other precautionary measures described on the product label and in any accompanying information. Pesticides should be used only when absolutely necessary.	29
Fibrous Materials	Precautions should be taken to minimize inhalation of, and skin contact with mineral fibres during home renovations and installation operations. Materials and products containing fibres should be examined periodically for signs of deterioration. Advice should be sought before removing or damaging any materials thought to contain asbestos.	32
Lead	In order to minimize the exposure of people, and especially children to lead of airborne origin, it is recommended that surfaces which may be contaminated be cleaned frequently and that a high standard of overall cleanliness be maintained.	33
Polycyclic Aromatic Hydrocarbons (PAHs)	Exposure to PAHs indoors should be kept to a minimum by: <ul style="list-style-type: none"> - ensuring that combustion systems, for example wood- and coal-burning stoves are properly installed and maintained and operated under conditions of satisfactory ventilation; - adhering to the guidelines and recommendations given in this document for particulate matter and tobacco smoke. 	34
Tobacco Smoke	- In view of the carcinogenic properties of tobacco smoke, any exposure to tobacco smoke in indoor environments should be avoided.	35