Health Effects of Indoor Air Pollutants

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1 Introduction

Much attention is paid to outdoor air quality and its impact on health, but individuals may spend 90% or more of their time indoors. For many people, especially potentially vulnerable groups such as the very young, the elderly and the sick, this means at home. Hence the quality of air inside the home environment is extremely important, and although a good deal of public interest and concern continues to be directed at the effects of outdoor air pollution, there is a growing tide of scientific opinion that the quality of air in the home environment is of equal or greater significance to human health and well-being. This growing interest has resulted in increasing research activities and, importantly, heightened awareness among regulators and policy makers.

This article reviews current information on levels and the risks to health and well-being of some major indoor air pollutants in dwellings. Because indoor air quality is very much dependent on prevailing climate, day-length, building construction, use of different fuels and heating/cooking methods, *etc*., assessments of this kind tend, by necessity, to have a national or regional focus and to make most use of 'local' exposure measurements. Therefore this paper concentrates particularly on the UK situation, whilst acknowledging and drawing information from important studies and developments elsewhere.[1] Many of the considerations and conclusions will be relevant to other countries, especially those with similar climate and building stock.

For a number of pollutants found indoors, the main sources are outside. Where significant indoor sources exist, these will tend to dominate personal exposure. Certainly it is known that the behaviour of individuals and their activity patterns (reflecting the time spent in various different 'micro-environments') can markedly affect their exposure to a range of air pollutants. Increasingly, therefore, the need is being recognized to take much better account of indoor exposures and to understand the importance of personal behaviour patterns. Only in this way can adequate assessments be made of the true impact of air pollution on health.

There are other important factors which affect how indoor air pollution is

[1] IEH, *Assessment on Indoor Air Quality in the Home*, Assessment A2, Institute for Environment and Health, Leicester, 1996.

considered in relation to outdoor air pollution. One of these is risk perception. For many individuals, for whatever reason, the perception of risk from outdoor air is substantially higher than for indoor air; indeed, it is unusual for the home environment to be considered 'hazardous' in this sense. Another important factor is 'controllability'. The principal outdoor sources of pollution (vehicles, factories, *etc.*) lend themselves to formal legislative control procedures, whereas an individual's exposure in the home is very much dominated by personal choice and behaviour with respect to ventilation, use of personal and consumer products, *etc.* Nevertheless, there is scope for control of emissions, especially from appliances and building products, and this is one area where efforts are currently under way (see below).

2 The Pollutants

A large number of natural and man-made substances can be identified in the air inside a typical home, many of which arise from sources within the home. The impetus over recent years to conserve energy has resulted in warmer, 'tighter' buildings with much reduced air exchange and therefore a greater propensity for indoor pollutants to build up. The combination of reduced ventilation rates (especially in winter), warmer and more humid conditions indoors, together with the greater use and diversity of materials, furnishings and consumer products, has resulted in concentrations of a wide range of pollutants occurring indoors at levels exceeding those outdoors. The following sections review exposure and health data for some of the most important indoor pollutants.

Nitrogen Dioxide (NO_2)

Nitrogen dioxide, a product of fossil fuel combustion, is one of the most ubiquitous indoor pollutants, especially in homes with gas cookers and other unflued combustion appliances. Because of this widespread exposure it has been the focus of much attention with respect to possible health effects. It is of particular importance in the UK, where almost 50% of homes are equipped with gas cookers and thus approximately 30 million people are potentially exposed indoors to NO_2 and related products of combustion.

Long-term average outdoor NO_2 levels in the UK fluctuate with season and degree of urbanization. They are typically well below WHO guideline values, although short-term peaks (*e.g.* one hour) can reach high levels, particularly in areas with heavy road traffic.[2] Outdoor levels are important determinants of indoor levels, but the latter are normally lower unless there is an indoor source.[3]

[2] MAAPE, *Oxides of Nitrogen*, Advisory Group on the Medical Aspects of Air Pollution Episodes, Third Report, HMSO, London, 1993.

[3] S.K.D. Coward and G.J. Raw, in *Indoor Air Quality in Homes: Part 1, The Building Research Establishment Indoor Environment Study,* ed. R.W. Berry, S.K.D. Coward, D.R. Crump, M. Gavin, C.P. Grimes, D.F. Higham, A.V. Hull, C.A. Hunter, I.G. Jeffrey, R.G. Lea, J.W. Llewellyn and G.J. Raw, Construction Research Communications, London, 1996, p. 67.

Recent studies in UK homes with and without gas cookers[3–6] have shown one- or two-week averages ranging from 25 to 70 $\mu g\,m^{-3}$ and from 13 to 40 $\mu g\,m^{-3}$, respectively. Continuous monitoring in kitchens with gas cookers[6] has shown one-hour average levels of up to 1115 $\mu g\,m^{-3}$; this compares with the 1987 WHO one-hour guideline value of 400 $\mu g\,m^{-3}$ (soon to be reduced to 200 $\mu g\,m^{-3}$). These limited data suggest that in many homes using gas for cooking, levels in the kitchen (and possibly in other rooms) approach or exceed this guideline value.

The most frequent end-points in studies looking at health effects from indoor NO_2 have been respiratory illness and/or symptoms in children. These outcomes have been defined differently in different investigations, and may not all represent the same disease process. For example, symptoms such as wheeze and cough may indicate a chronic disorder such as asthma or an acute infection in an otherwise normal person. However, many of the published reports do not discriminate clearly between infections and other types of respiratory disease, which must therefore be considered together. Eleven of the epidemiological studies looking at respiratory illness in children were included in a meta-analysis carried out by Hasselblad *et al.*[7] Four different statistical methods were used to combine the results of the studies and calculate summary odds ratios. All four methods produced the same estimate for the effects of NO_2 exposure on respiratory illness, and there was little or no change in the odds ratio when the analysis was limited to studies of children aged 5–12 years, with studies analysed separately according to whether NO_2 was measured directly or inferred from the presence of a gas cooker. The authors concluded that children exposed to a long-term increase of 30 $\mu g\,m^{-3}$ NO_2 (equivalent to having a gas cooker) suffer a 20% increase in their risk of respiratory illness. Other studies, not included in Hasselblad's analysis, have produced inconsistent results. Two of these were part of the US Six Cities studies,[8,9] and only showed associations of gas cooking with previous respiratory disease (before age two). Also in the US, an early study showed an association of gas cookers with cough, but associations with other symptoms, although positive, were not statistically significant.[10] In the Netherlands, one study found that use of unvented water heaters and increased personal exposure to NO_2 were associated with a higher prevalence of respiratory symptoms,[11] while in another investigation, indoor levels of NO_2 did not differ between children with respiratory problems and asymptomatic controls.[12] A Canadian case-control study found higher levels of NO_2 personal exposure among asthmatic children

[4] R. J. W. Melia, S. Chinn and R. J. Rona, *Atmos. Environ.*, 1990, **24B**, 177.

[5] G. J. Raw and S. K. D. Coward, in *Proceedings of Unhealthy Housing: The Public Health Response*, University of Warwick, Coventry, 1992.

[6] D. Ross, *Continuous and Passive Monitoring of Nitrogen Dioxide in UK Homes*, BRE Note N109/94, Buildings Research Establishment, Watford, 1994.

[7] V. Hasselblad, D. M. Eddy and D. J. Kotchman, *J. Air Waste Manage. Assoc.*, 1992, **42**, 662.

[8] F. E. Speizer, B. Ferris, Y. M. M. Bishop and J. Spengler, *Am. Rev. Respir. Dis.*, 1980, **121**, 3.

[9] D. W. Dockery, J. D. Spengler, L. M. Neas, F. E. Speizer, B. G. Ferris, J. H. Ware and B. Brunekreef, in *Air Waste Management Transaction Series: TR-15,* ed. J. Harper, Air & Waste Management Assoc., Pittsburgh, 1989, p. 262.

[10] R. Dodge, *Arch. Environ. Health*, 1982, **37**, 151.

[11] D. Houthuijs, B. Remijn, B. Brunekreef and R. de Konig, in *Proceedings of Indoor Air '87*, Institute for Water, Soil and Air Hygiene, Berlin, 1987, p. 463.

[12] G. Hoek, B. Brunekreef, R. Meijer, A. Scholten and J. Boleij, *Int. Arch. Occup. Health*, 1984, **55**, 79.

than among non-asthmatics.[13] Of the studies that have looked specifically at health effects in infants or neonates, no relationship has been shown between respiratory illness and the presence of a gas cooker[14] or NO_2 levels in the bedroom or kitchen.[15] In adults, studies of the relation between the use of gas cookers and the occurrence of respiratory illness have provided conflicting results. Two studies, for example, showed no significant association; indeed, the subjects using gas had slightly less respiratory illness than those who used electricity for cooking.[16,17] In a third study there was some association between gas cookers and breathlessness in non-smoking men but not in women, whose exposure to NO_2 might be expected to be higher.[18] In another study, prevalence of respiratory symptoms in women was associated with the frequency with which the kitchen was filled with heavy cooking fumes but not with gas cooking *per se*.[19] However, a more recent paper has shown an apparent connection between gas cooking and ill-health in women.[20] Thus there is little consistent evidence at present to suggest that the use of gas cookers has any important effect on the incidence of respiratory illness in adults, although further work is needed.

A number of studies have looked at the potential influence of gas cooking and/or indoor NO_2 levels on measures of pulmonary function rather than clinical illness. In children, some studies have reported small negative effects of gas over electric cooking on spirometric indices, but between the different indices the effects are generally inconsistent. In adults, small detrimental effects of gas cooking have been reported on one or more measures of pulmonary function,[20] but again there are inconsistencies between indices. While some of these studies included NO_2 measurements, because of possible confounding it is not possible to attribute any differences in lung function to indoor NO_2 alone, even where associations were found. In support of this, single and repeated chamber studies of various durations have failed to show any effect of NO_2 up to 1880 $\mu g\,m^{-3}$ on indices of lung function.[2] A few studies have examined the effect of cooking or NO_2 levels on pulmonary function in patients with asthma, but the numbers are generally too small to permit firm conclusions to be drawn.

Conclusions. Overall, the published evidence on health effects of NO_2 points most to a hazard of respiratory illness in children, perhaps resulting from increased susceptibility to infection. However, in interpreting this evidence, several sources of uncertainty should be taken into account, including publication bias, reporting bias, multiple testing errors, confounding, pollutant interactions and use of proxy measures of NO_2 exposure. Also there is a dearth of studies on asthmatics, bronchitics and other potentially susceptible groups. A number of

[13] C. Infante-Rivard, *Am. J. Epidemiol.*, 1993, **137**, 834.
[14] S.A. Ogston, C.V. du Florey and C.H.M. Walker, *Br. Med. J.*, 1985, **290**, 957.
[15] J.M. Samet, W.E. Lambert, B.J. Skipper, B.J. Cushing, W.C. Hunt, S.A. Young, L.C. McLaren, M. Schwab and J.D. Spengler, *Nitrogen Dioxide and Respiratory Illness in Children. Part I: Health Outcomes*, Research Report Number 58, Health Effects Institute, Cambridge, 1993.
[16] M.D. Keller, R.R. Lanese, R.I. Mitchell and R.W. Cote, *Environ. Res.*, 1979, **19**, 495.
[17] M.D. Keller, R.R. Lanese, R.I. Mitchell and R.W. Cote, *Environ. Res.*, 1979, **19**, 504.
[18] G.W. Comstock, M.B. Meyer, K.J. Helsing and M.S. Tockman, *Am. Rev. Respir. Dis.*, 1981, **124**, 143.
[19] T.P. Ng, K.P. Hui and W.C. Tan, *J. Epidemiol. Community Health*, 1993, **47**, 454.
[20] D. Jarvis, S. Chinn, C. Luczynska and P. Burney, *Lancet*, 1996, **347**, 426.

research needs are apparent, including: the identification of homes with high indoor levels of NO_2 to establish whether such levels are associated with detectable health effects; further work on health effects of NO_2 and gas cooking in potentially susceptible groups; identification of reasons why some homes have high levels of NO_2; and further information on the pathogenic mechanisms of NO_2 toxicity in the lung. While it does seem clear that any risk of respiratory illness from the levels of NO_2 currently found in most homes is small, it would seem prudent to encourage any measure that will minimize indoor NO_2 levels. This is especially relevant because of the large number of people potentially exposed and because of the uncertainties regarding effects on susceptible groups such as asthmatics and bronchitics and people who spend a particularly large proportion of time indoors such as very young infants and the elderly. Further work is needed on exposure to gas combustion products (the full mixture) and their effects on health, including the postulated potentiation of responses to indoor allergens, such as those from house dust mites, by concomitant exposure to irritant gases such as NO_2.

Formaldehyde and Other Volatile Organic Compounds (VOCs)

VOCs originate from a number of sources within the indoor environment, including building materials, paints, furnishings, furniture, adhesives, cleaning agents, tobacco smoke and the occupants themselves. Numerous VOCs, representing most organic families (typically aliphatic and aromatic hydrocarbons, halogenated compounds and aldehydes) have been measured in indoor air. Formaldehyde occurs ubiquitously in the environment. It is produced naturally and by many industrial processes, and is found in vehicle exhausts and cigarette smoke. It also occurs naturally in fruits and vegetables and other foods. In residential indoor air, the principal source of formaldehyde is off-gassing from urea formaldehyde foam insulation (UFFI) and particle board used in construction; other sources are furniture, furnishing and household cleaning agents. For both formaldehyde and VOCs, airborne concentrations depend on the age of the source material and ventilation, temperature and humidity. Active and passive cigarette smoking also contribute to total exposure. Exposure to these substances is therefore widespread and the potential consequences to health are significant.

Formaldehyde. Within the UK, the most extensive investigation of formaldehyde exposure in the home has been that conducted by the Building Research Establishment (BRE) in 180 homes mainly within the Avon area.[21] These studies demonstrated somewhat increased formaldehyde levels in newer homes, homes with integral garages, homes with new furnishings and recently decorated homes. Outdoor levels in the BRE study were around one tenth of those found indoors; most other investigations have similarly shown outdoor levels of formaldehyde to be lower than indoor levels. The mean annual indoor formaldehyde levels

[21] V. M. Brown, D. R. Crump and M. Gavin, in *Indoor Air Quality in Homes: Part 1, The Building Research Establishment Indoor Environment Study*, ed. R. W. Berry, S. K. D. Coward, D. R. Crump, M. Gavin, C. P. Grimes, D. F. Higham, A. V. Hull, C. A. Hunter, I. G. Jeffrey, R. G. Lea, J. W. Llewellyn and G. J. Raw, Construction Research Communications, London, 1996, p. 18.

found (0.020–0.025 mg m^{-3}, according to room sampled) were at the lower end of the 0.01–0.1 mg m^{-3} range reported in a European survey of formaldehyde concentrations in residential homes and schools[22] (some with UFFI, others without), and were similar to or less than those reported in contemporary US studies. Overall, the homes in the UK BRE survey do not appear to differ greatly, in terms of formaldehyde levels, from residential dwellings elsewhere in the world; certainly there is no evidence that concentrations are greater. Many studies on formaldehyde levels in homes were carried out in the USA in the 1980s, in both conventional and mobile homes. The latter contained many formaldehyde-emitting materials such as UFFI and were considered to pose potential health problems. A review on indoor air pollution by Samet *et al.*[23] indicated that in homes with UFFI, formaldehyde concentrations were about twice those in homes without UFFI (0.02–0.16 and 0.04–0.08 mg m^{-3}, respectively). North American studies have, like the UK BRE investigations, demonstrated higher formaldehyde concentrations in newer compared with older dwellings. In a study of Tennessee homes,[24] for example, those less than five years old had mean formaldehyde levels of around 0.1 mg m^{-3}, whereas in homes between five and fifteen years old the mean was 0.05 mg m^{-3} and in older homes the mean concentration was 0.038 mg m^{-3}. The half-life of formaldehyde for new homes appears to be around four to five years.[25] Energy conservation measures in dwellings have been shown to cause an increase in exposure to formaldehyde. In one US study,[26] conventional houses had a mean formaldehyde level of 0.05 mg m^{-3} compared with 0.08 mg m^{-3} in energy-efficient houses; for normal *versus* energy-efficient condominiums, the level was 0.11 mg m^{-3} compared with 0.22 mg m^{-3}.

Numerous studies, including those in occupational settings, have investigated and reported health effects related to exposure to formaldehyde by inhalation. Effects range from subtle neuropsychological changes, mucous membrane irritation of the eyes, nose and throat, and airway irritation, to asthma and cancer. The odour threshold for formaldehyde is in the range 0.06–1.2 mg m^{-3} and for throat irritation is in the range 0.12–3.0 mg m^{-3} for most individuals. Eye irritation has been reported at levels as low as 0.01 mg m^{-3}. In a review of a number of US studies in which symptoms among residents of mobile homes or homes with UFFI had been investigated,[27] exposures ranged from 0 to 9.6 mg m^{-3}. Although the studies are suggestive of some irritant effects, the limited exposure reporting and inconsistent symptoms reporting in these studies do not allow conclusions regarding specific effects at particular levels of exposure. It is, however, clear that mucous membrane irritation to the eyes and throat can

[22] ECA-IAQ, *Indoor Air Pollution by Formaldehyde in European Countries*, European Collaborative Action 'Indoor Air Quality and its Impact on Man', Report No 7. EUR 13216 EN, Office of Publications for the European Communities, Luxembourg, 1990.

[23] J. M. Samet, M. C. Marbury and J. D. Spengler, *Am. Rev. Respir. Dis.*, 1987, **136**, 1486.

[24] A. R. Hawthorne, R. B. Gammage, C. S. Dudney, D. R. Womack, S. A. Morris, R. R. Westley and K. C. Gupta, in *Specialty Conference on Measurement and Monitoring of Non-Criteria (Toxic) Contaminants in Air*, Chicago, Illinois, March 1984, *Environ. Int.*, 1986, **12**, 221.

[25] P. W. Preuss, R. L. Dailey and E. S. Lehan, in *Formaldehyde: Analytical Chemistry and Toxicology*, ed. V. Turoski, American Chemical Society, Washington, 1985, p. 247.

[26] T. H. Stock and S. R. Mendez, *Am. Ind. Hyg. Assoc.*, 1985, **46**, 313.

[27] J. M. Samet, M. C. Marbury and J. D. Spengler, *Am. Rev. Respir. Dis.*, 1988, **137**, 221.

occur at the higher formaldehyde levels which arise due to UFFI or new furnishings and carpets. A range of effects reported among residents of US mobile homes[28] included burning eyes, cough, fatigue, dizziness, sore throat and wheeziness. The weekly average exposure was $11.9\,mg\,m^{-3}\,h^{-1}$. Some of the reported symptoms are known to be smoking related. A Canadian study[29] of respiratory symptoms, respiratory function and other effects in residents of UFFI-containing and control homes and a group of formaldehyde-exposed technicians found a higher prevalence of some non-specific symptoms in the UFFI home resident group compared with the two other groups, but no increase in prevalence of respiratory symptoms. The former finding is somewhat surprising, as the highest exposed group were the laboratory technicians, among whom there was no decrease in lung function. In a two-part study, also in Canada, comparing the health characteristics and respiratory function of occupants of homes containing UFFI and control homes both before and after remedial work or removal of UFFI, although some subjective measures of health appeared to be associated with formaldehyde or UFFI, there was no association between formaldehyde or UFFI and objective measures of lung function.[30–32] Some investigations have attempted to see if there are any specific effects among groups at extra risk from formaldehyde in the home. One study[33] demonstrated that asthma and bronchitis, but not other respiratory symptoms, were more prevalent among children in houses with higher (above $0.07\,mg\,m^{-3}$) formaldehyde concentrations; among adults, chronic cough in non-smokers was related to elevated formaldehyde levels, but respiratory symptoms and disease were not. Chamber studies showed no lung function changes when healthy volunteers were exposed to formaldehyde concentrations of 2.5 or $3.6\,mg\,m^{-3}$ for 40 or 180 minutes, respectively.[34,35] Similar studies, in which volunteers with a history of asthma and hyperactive airways were exposed to $3.6\,mg\,m^{-3}$ formaldehyde for 180 minutes, also demonstrated no effect on lung function.[36] In a further group of asthmatics exposed to 0, 0.12 or $0.85\,mg\,m^{-3}$ formaldehyde for approximately 90 minute periods in an exposure chamber, no exposure-related effects on lung function or bronchial reactivity were reported.[37]

Other Volatile Organic Compounds. Owing to differences in definitions of VOCs and TVOCs ('Total VOCs')[38] and in the efficiency with which individual

[28] K. S. Liu, F. Y. Huang, S. B. Hayward, J. Wesolowski and K. Sexton, *Environ. Health Perspect.*, 1991, **94**, 91.
[29] M. J. Bracken, D. J. Leasa and W. K. Morgan, *Can. J. Public Health*, 1985, **76**, 312.
[30] I. Broder, P. Corey, P. Cole, M. Lipa, S. Mintz and J. R. Nethercott, *Environ. Res.*, 1988, **45**, 141.
[31] I. Broder, P. Corey, P. Cole, M. Lipa, S. Mintz and J. R. Nethercott, *Environ. Res.*, 1988, **45**, 156.
[32] I. Broder, P. Corey, P. Brasher, M. Lipa and P. Cole, *Environ. Res.*, 1988, **45**, 179.
[33] M. Krzyanoski, J. J. Quackenboss and M. D. Lebowitz, *Environ. Res.*, 1990, **52**, 117.
[34] E. N. Schachter, T. J. Witek, T. Tosun and G. J. Beck, *Arch. Environ. Health*, 1986, **41**, 229.
[35] E. N. Schachter, T. J. Witek, D. J. Brody, T. Tosun, G. J. Beck and B. P. Leaderer, *Environ. Res.*, 1987, **44**, 188.
[36] L. R. Sauder, M. D. Chatham, D. J. Green and T. J. Kulle, *J. Occup. Med.*, 1986, **28**, 420.
[37] H. Harving, J. Korsgard, O. F. Pederson, L. Mølhave and R. Dahl, *Lung*, 1990, **168**, 15.
[38] ECA-IAQ, *Total Volatile Organic Compounds* (*TVOC*) *in Indoor Air Quality Investigations*, European Collaborative Action 'Indoor Air Quality and its Impact on Man', Report No 19. EUR 17675 EN, Office for Official Publications of the European Community, Luxembourg, 1995.

compounds, particularly very volatile organic compounds, are collected on absorbent materials, comparison of exposure data between studies is difficult. The most informative studies on exposure to VOCs in the home are the BRE study in the UK and the US EPA Total Personal Exposure Methodology (TEAM) Studies. Both of these large-scale investigations, although studying other exposures in the home and with different overall aims, included comprehensive elements to assess exposure to VOCs over a long period of time. In the BRE study,[39] over 200 individual VOCs were identified. The study found mean TVOC concentrations in different rooms were similar (0.2–0.4 mg m^{-3}) and indoor levels were ten times higher than those measured outdoors, which is broadly consistent with the US TEAM studies (see below). There was a significant relationship between increased TVOC concentrations and painting and decorating, the highest exposure to VOCs occurring during these activities. A number of guideline values for TVOCs from 5000 $\mu g\, m^{-3}$ down to 200 $\mu g\, m^{-3}$ are reported in the literature; the current BRE mean figure falls towards the lower end of this range. In the first part of the US TEAM study, conducted in New Jersey,[40] indoor levels of VOCs were found to be consistently higher than outdoor levels. There was a wide variation in individual exposure to specific compounds, breath levels of chloroform were related to levels in drinking water, and the strongest association was in breath analysis of benzene and styrene for smokers. For benzene, combining data from a number of elements of the TEAM study[41,42] it was estimated that 6 $\mu g\, m^{-3}$ of an average personal exposure of 16 $\mu g\, m^{-3}$ could be accounted for by outdoor air and the remaining 10 $\mu g\, m^{-3}$ was due to personal activities (including smoking, which represented 50% of the exposure). Later analysis of the TEAM study[43] suggested that personal indoor air TVOC samples exceeded 1 mg m^{-3} in about 60% of all samples and 5 mg m^{-3} in about 10% of samples. A further study in North Carolina,[44] which was not part of the TEAM study, confirmed that peak exposures to VOCs were associated with painting and decorating activities and house cleaning.

Although large numbers of VOCs can be measured in indoor environments, most are present at levels that are orders of magnitude below those at which toxicological or even sensory effects would be expected in humans. However, they occur in variable and complex mixtures to which individuals are exposed for perhaps 80–90% of their time. Probably the most informative studies on health effects, albeit acute effects, of VOCs are gained from controlled chamber studies using defined concentrations of mixtures and defined endpoints. Studies by Otto

[39] V. M. Brown and D. R. Crump, in *Indoor Air Quality in Homes: Part 1, The Building Research Establishment Indoor Environment Study*, ed. R. W. Berry, V. M. Brown, D. R. Crump, M. Gavin, C. P. Grimes, D. F. Higham, A. V. Hull, C. A. Hunter, I. G. Jeffery, R. G. Lea, J. W. Llewellyn and G. J. Raw, Construction Research Communications, London, 1996, p. 38.

[40] L. A. Wallace, E. D. Pellizzari, T. D. Hartwell, R. Whitmore, C. Sparino and H. Zelon, *Environ. Int.*, 1986, **12**, 369.

[41] L. A. Wallace, *J. Am. Coll. Toxicol.*, 1989, **8**, 883.

[42] L. A. Wallace, *Risk Anal.*, 1990, **10**, 59.

[43] L. A. Wallace, *Ann. N. Y. Acad. Sci.*, 1992, **641**, 7.

[44] L. A. Wallace, E. D. Pellizzari, T. D. Hartwell, V. Davis, L. C. Michael and R. W. Whitmore, *Environ. Res.*, 1989, **50**, 37.

et al.,[45] using a mixture of 22 VOCs at 25 mg m^{-3} (total), evoked irritancy symptoms, as measured by questionnaire response, but no neurobehavioural changes. Other studies with VOC mixtures, also in chamber situations, have evoked positive subjective responses to air quality in a group reporting 'sick building syndrome' (SBS) symptoms at work, but not in a control group,[46] and possible lung function changes among a group of non-smoking volunteers.[47] However, in neither study was a clear association between VOC exposure and effect found. VOCs may also have a role in the perception of air quality, but this is not easy to separate from other factors contributing to the overall odour of indoor air. Apart from odour recognition itself, the perception of unpleasant odour may signify poor air quality and lead to or trigger secondary effects.[48] Various methods, including the use of trained panels to make subjective evaluations of perceived air quality in relation to occupant comfort using quantitative descriptions of pollution emissions and air quality, have been developed over the last few years,[49] although the methods are not universally accepted. Moreover, there appears to be no consistent association between occupant dissatisfaction with air quality and odour perceptions, or between perceived air quality and TVOC level. Factors such as temperature and humidity have also been reported to be important determinants of perceived air quality and of SBS symptoms[50,51] (see below). The subject of sensory perception of air quality is under current review by the European Concerted Action on Indoor Air Quality and its Impact on Man.

Conclusions. With regard to formaldehyde, chamber studies with normal adults or those with pre-existing asthma have not demonstrated any dysfunction at mean formaldehyde levels typically found in homes, or even at levels several times higher. Moreover, epidemiological studies have not demonstrated any increase in respiratory symptoms or lung function at estimated current domestic levels. It is concluded that most people would fail to show any sensory effects at an ambient maximum concentration of 0.1 mg m^{-3} averaged over 0.5 h, although some individuals might show transient effects at or below this level. For the protection of health, exposure to formaldehyde in the domestic environment should remain at or below current levels; significantly higher levels should be avoided. There are a number of research needs on the health effects of domestic formaldehyde exposure. For example, the incidence and nature of hyper-reactivity to formaldehyde should be studied across a wide range of concentrations, and the effects of combined exposures to formaldehyde and other common household substances

[45] D. Otto, L. Mølhave, G. Rose, H. K. Hudnell and D. House, *Neurotoxicol. Teratol.*, 1990, **12**, 649.
[46] S. K. Kjærgaard, L. Mølhave and O. F. Pederson, *Atmos. Environ.*, 1991, **25A**, 1417.
[47] H. Harving, R. Dahl and L. Mølhave, *Am. Rev. Respir. Dis.*, 1991, **143**, 751.
[48] WHO, *Indoor Air Quality: Organic Pollutants*, Euro Reports and Studies No. 111, World Health Organization, Copenhagen, 1989.
[49] P. O. Fanger, *Energy Build.*, 1988, **12**, 1.
[50] L. Mølhave, S. K. Kjærgaard, O. F. Pederson, A. H. Jorgenson and T. Pedersen, in *Proceedings of Indoor Air '93*, Helsinki, 1993, p. 555.
[51] L. Berglund and W. S. Cain, in *Proceedings of Indoor Air Quality '89: The Human Health Equation,* ASHRAE, Atlanta, 1989, p. 93.

should be investigated. There are many formaldehyde sources in the home, so any control strategy has to take account of this multiplicity of sources.

Regarding other VOCs, there is no evidence to suggest that current typical indoor (domestic) exposure to VOCs—either individually or as a total—poses a health risk. Based on chamber studies, TVOCs at concentrations greater than 25 mg m^{-3} may cause acute irritancy and other transient effects; although such concentrations are unlikely to be encountered under normal domestic conditions, they could occur during painting/decorating or excessive solvent usage. The composition of TVOCs is complex and variable and health effects resulting from exposure are generally poorly characterized. It is, therefore, prudent to minimize exposure to TVOCs, particularly genotoxic and carcinogenic substances. For certain specific VOCs, such as benzene, appropriate guidelines may be applied. In the case of benzene, the UK Expert Panel on Air Quality Standards (EPAQS) recommended standard of 16.2 μg m^{-3} (5 ppb) running average, together with the recommendation to reduce overall levels of exposure to benzene such that (for non-smokers) ambient air pollution is no longer the main source of individual exposure,[52] are to be encouraged. Information should be available to people about the most important sources of VOCs in the home, including activities that lead to exposure, so that they may consider how to minimize their exposure and any associated effects. Also, consideration should be given to monitoring indoor air quality in homes in order to assess the effectiveness of control measures applied, for example, to building materials and techniques, and to consumer products. With respect to outstanding research needs on VOCs, further toxicological data should be collected on individual VOCs, and on their sensory thresholds, and methods should be improved for the evaluation of the sensory and neuropsychological effects of VOCs.

Fungi and Bacteria

Damp and mould are relatively common conditions in European housing, and there is a history of concern regarding the possible effects on health of exposure to fungal spores (and other fungal-derived material) and, to a lesser extent, bacteria. Many different species of bacteria and fungi can be found in homes, associated with various forms of organic matter such as surface coating of walls, wood, fabrics and foodstuffs. Some species are particularly associated with dampness in buildings and several health effects (other than infections) have been attributed to the saprotrophic bacterial and fungal flora of the indoor environment.

In the recent BRE study,[53] two different methods were used to sample airborne fungi and bacteria: a filter technique and a multi-stage Andersen sampler. The data from these two sampling methods were comparable, both in total numbers and species present, to other studies reported from the UK and elsewhere. The

[52] Expert Panel on Air Quality Standards, *Benzene*, HMSO, London, 1994.

[53] C.A. Hunter, A.V. Hull, D.F. Higham, C.P. Grimes and R.G. Lea, in *Indoor Air Quality in Homes: Part 1, The Building Research Establishment Indoor Environment Study*, ed. R.W. Berry, S.K.D. Coward, D.R. Crump, M. Gavin, C.P. Grimes, D.F. Higham, A.V. Hull, C.A. Hunter, I.G. Jeffrey, R.G. Lea, J.W. Llewellyn and G.J. Raw, Construction Research Communications, London, 1996, p. 99.

isolates identified were not atypical, with *Penicillium*, *Cladosporium*, *Aspergillus* and *Mycelia sterilia* predominating. It should be noted, however, that minor species, which could pose a biological hazard, might not be detected and the amount of cultivable organisms may only represent a small and variable fraction of the total airborne flora. Longer sampling periods associated with the filter method might give a more representative estimate of levels than the shorter sampling period used with the Andersen sampler, but like all other sampling methods, there are certain intractable technical deficiencies. As with fungi, the concentrations of bacteria obtained with the two sampling methods were of the same order of magnitude as found in other studies. In addition, Gram-positive bacteria predominated over Gram-negative types, which is compatible with previous findings. However, the rank order of the bacterial genera differed slightly between the two methods and this may be due to the desiccation effect on the non-spore-forming bacteria seen with the filter collection method. From other studies, it can be concluded that the numbers of viable bacteria recorded would form an even smaller proportion of the total count than is the case for fungi.

A number of epidemiological studies conducted in Europe and North America have investigated the relation between home dampness and respiratory morbidity in children and adults. In most of these studies, information about the exposure variables (home dampness and mould) and the outcome variables (respiratory symptoms) was obtained by questionnaires. Only in a few of the studies was further information about exposure to moulds obtained by actually measuring the number of airborne propagules. Two such studies reported a positive association between airborne fungal counts and some respiratory symptoms in children,[54,55] whereas a third found no such association.[56] A fourth study found no association between fungal counts in house dust samples and respiratory symptoms in children.[57] The limited number of studies linking the measured levels of airborne organisms in the home with adverse health outcomes all relate to fungi; there appear to be none concerning bacteria.[58]

Conclusions. There is consistent evidence of an association between damp and mouldy housing and reports of respiratory symptoms in children. However, the causal interpretation of these findings remains uncertain. Numerous fungal and bacterial species are present in homes and the health effects of many species and their products are unknown or poorly understood. Epidemiological studies relating measurements of indoor airborne fungi to respiratory disease generally have not shown convincing associations. It is not clear whether this is due to the recognized limitations of current mycological methods in providing an index of relevant exposure, or to the true absence of a health effect. The literature relating domestic mould growth to non-respiratory disease is extremely sparse and

[54] S. D. Platt, C. J. Martin, S. M. Hunt and C. W. Lewis, *Br. Med. J.*, 1989, **298**, 1673.
[55] M. Waegemaekers, N. Van Wageningen, B. Brunekreef and J. S. M. Boliej, *Allergy*, 1989, **44**, 192.
[56] D. P. Strachan, B. Flannigan, E. M. McCabe and F. McGarry, *Thorax*, 1990, **45**, 382.
[57] A. P. Verhoeff, J. H. Van Wijnen, E. S. Van Reene-Hockstra, R. A. Samson, R. T. Van Strien and B. Brunekreef, *Allergy*, 1994, **49**, 540.
[58] B. Flannigan, in *Clean Air at Work*, ed. R. H. Brown, M. Curtis, K. J. Saunders and S. Vandendriessche, The Royal Society of Chemistry, Cambridge, 1992, p. 366.

although there are recognized health hazards, no epidemiological data exist to quantitate exposure–response relationships. A number of studies have drawn attention to a relationship between dampness and mould growth in houses and symptoms of respiratory disease in their occupants, but these relationships cannot at present be attributed to specific fungi or bacteria in the air. Mould and dampness are often associated with poor ventilation, which tends to increase exposure to other contaminants as well as microbiological products. Improved ventilation could be expected to reduce indoor dampness and mould growth.

There appear to be complicated inter-relationships between dampness and other building factors, heating (type and degree), the presence of mould and socio-economic factors in the association with occupants' ill-health. There is a dearth of information on the toxicity of fungi and bacteria and their metabolites, which needs to be addressed, and the general issue of damp housing and health similarly requires further study.

House Dust Mites

It is well established that house dust mites are ubiquitous in homes in warm temperature regions and that their relative abundance is largely determined by the internal microclimate, since they tend to prefer warm damp conditions. There is clear evidence that antigen derived largely from house mite faeces is one of the major causes of allergic sensitization and that people who have been sensitized to mites are more likely than those not sensitized to manifest symptoms of asthma and other allergies.

Temperature and humidity are important factors affecting the distribution and abundance of house dust mites, influencing the quantity of mite allergens, the species of mites found and the distribution of mites within a house. Increased ventilation and air conditioning is associated with lower levels of mite allergens and has been shown to reduce seasonal increases of mite allergens in the US.[59] A recent study by the BRE,[60] looking at homes in the county of Avon, UK, found high numbers of mites in both living room and bedroom carpets; 95% of mites sampled were *Dermatophagoides pteronyssinus* (from which the allergen Der p1 is derived). A correlation between relative humidity and mite numbers was confirmed. Mite counts typically used in earlier studies are technically demanding, time consuming and may underestimate the number of live mites.[61] The problems with the methodology relate to several stages in the analysis. Sampling strategy has not been standardized and the actual methodology seems to vary from study to study. Extraction of mites from dust is not necessarily quantitative. Nevertheless, this method does allow species identification and can be useful in intervention studies, as decline in mite numbers may precede changes in antigen levels.[62] More recently, with the development of sensitive and specific monoclonal

[59] C. M. Luczynska, *Respir. Med.*, 1994, **88**, 723.

[60] C. A. Hunter, I. G. Jeffrey, R. W. Berry and R. G. Lea, in *Indoor Air Quality in Homes: Part 1, The Building Research Establishment Indoor Environment Study*, ed. R. W. Berry, S. K. D. Coward, D. R. Crump, M. Gavin, C. P. Grimes, D. F. Higham, A. V. Hull, C. A. Hunter, I. G. Jeffrey, R. G. Lea, J. W. Llewellyn and G. J. Raw, Construction Research Communications, London, 1996, p. 87.

[61] T. A. E. Platts-Mills and A. L. de Weck, *J. Allergy Clin. Immunol.*, 1989, **83**, 416.

immunoassays, it has been possible to quantify concentrations of mite antigens in dust,[63] but again there are problems of sampling strategy and technique. Occasional studies have used guanine as an indirect measure of allergen in dust.[64] This is a semi-quantitative technique that poorly correlates with allergen levels, but can be useful as a screening tool and has been used to monitor interventions. Measurements of allergen in air are critically dependent on sampling strategy. Domestic activity and the particle size associated with the allergen affect both the quantities of airborne allergen and the duration that allergens are airborne.[65] There is clear evidence from the studies that have been performed that antigen derived largely from mite faeces is one of the major causes of allergic sensitization.

Sensitization is much more likely to occur in people who are predisposed to the development of atopic disease on the basis of genetic predisposition and other as yet unknown environmental factors. People who have been sensitized to mites are more likely than those not sensitized to manifest symptoms of asthma and other allergies. Moreover, those sensitized to mites are likely to develop symptoms in response to exposure to the mites, either in the natural circumstances of house dust exposure or in the artificial circumstances of bronchial challenge. Furthermore, it has been shown in studies intended to reduce exposure of symptomatic individuals to mite antigen that a reduction in symptoms may occur. There is thus evidence that exposure to mites is a hazard with respect to development of sensitization, initiation of asthma and provocation of asthmatic symptoms.[61,62] However, it is far from clear whether mites are responsible, in whole or in part, for the general rise in the prevalence of asthma in the UK and elsewhere. There is no convincing evidence that there has been sufficient change in mite populations in houses to explain such a change,[66] and there are reasons to suppose that other factors relating to susceptibility are likely to be of additional importance. This is relevant, as a major effort to reduce mite populations in houses may not influence substantially the prevalence of asthma and allergic disease in the population, since susceptible people would still become sensitized to other common allergens. It is possible, however, that such measures would reduce the severity of symptoms in people already sensitized to mites.[67] Comparisons of disease prevalence in populations with different levels of mite allergen exposure are prone to confounding by other environmental exposures, or by differences in genetic constitution or lifestyle. For these reasons they are difficult to interpret as evidence either for or against an effect of mite allergen exposure on asthma prevalence, particularly when they are based on a comparison of only two study centres or populations. Similarly, changes in asthma prevalence over time may be due to factors other than changes in mite

[62] T. A. E. Platts-Mills, W. Thomas, R. C. Aalberse, D. Vervoet and M. D. Chapman, *J. Allergy Clin. Immunol.*, 1992, **89**, 1046.

[63] M. D. Chapman, P. W. Heymann, S. R. Wilkins, M. J. Brown and T. A. E. Platts-Mills, *J. Allergy Clin. Immunol.*, 1987, **80**, 184.

[64] J. E. M. H. Van Bronswijk, E. Bischoff, W. Schmiracher and F. M. Kniest, *J. Med. Entomol.*, 1989, **26**, 55.

[65] F. De Blay, P. W. Heymann, M. D. Chapman and T. A. E. Platts-Mills, *J. Allergy Clin. Immunol.*, 1991, **88**, 919.

[66] R. Sporik, S. T. Holgate, T. A. E. Platts-Mills and J. J. Cogswell, *New. Engl. J. Med.*, 1990, **323**, 502.

[67] M. J. Colloff, *Br. J. Dermatol.*, 1992, **127**, 322.

allergen exposure. A striking and widely quoted epidemic of asthma in the Fore region of Papua New Guinea[68] was attributed to the introduction of dust mite in blankets, but there were profound changes in many other aspects of the highlanders' lifestyle at the same time. Changes in mite allergen exposure at much higher levels do not appear to have contributed to recent increases in asthma prevalence (*e.g.*, in Australia[69] or the UK[66]). Most studies which have investigated the relationship between mite allergen exposure and mite sensitization within a population relate to children. A positive correlation has been reported from various centres with differing levels of allergen exposure, although the imprecision of a single cross-sectional exposure measurement leaves open the possibility that children apparently sensitized at very low levels of current exposure in such studies may have been exposed to high levels in the past. Thus it is not possible to determine whether there is a threshold exposure level below which sensitization does not occur. The importance of genetic predisposition in defining the position and shape of the exposure–sensitization relationship is recognized.[70,71] It is likely that there are some people who would not become sensitized even at very high levels of mite allergen exposure, but a plateau in the exposure–sensitization relationship at high levels has yet to be demonstrated by epidemiological studies.

For respiratory disease to develop, a series of steps must occur in a genetically predisposed individual. These are sensitization, the development of bronchial reactivity, and finally a response to continued exposure, producing symptoms or changes in lung function. A number of studies have attempted to assess the exposure–response relationship or threshold at which symptoms will occur when a sensitized individual is exposed. Most of these studies have been either birth cohort studies of asthma incidence, or cross-sectional studies of disease prevalence or severity, mainly in children. Two prospective studies of infants at higher risk of allergy have failed to offer conclusive evidence of a positive relationship. The widely cited paper by Sporik *et al.*[66] suggested that at higher levels of exposure in infancy (greater than 10 μg Der p1 g^{-1} dust), there is an increase in asthmatic symptoms up to age 11. However, this finding was of borderline statistical significance and the more convincing relationship was between early allergen exposure and an early age of onset of wheezing. A similarly designed study showed no association between allergen exposure in infancy and mite sensitization at age seven years.[72] The mite allergen concentration in the first and seventh years of life did not differ significantly between atopic children with and without a history of wheezing. A third cohort study of infants at high risk of allergy, which involved intervention, was also not suggestive of an

[68] G.K. Dowse, K.J. Turner, G.A. Stewart, M.P. Alpers and A.J. Woolcock, *J. Allergy Clin. Immunol.*, 1985, **75**, 75.

[69] J.K. Peat, R.H. Van den Berg, W.F. Green, C.M. Mellis and S.R. Leeder, *Br. Med. J.*, 1994, **308**, 1591.

[70] J. Kuehr, T. Frischer, R. Meinert, R. Barth, J. Forster, S. Schraub, R. Urbanek and W. Karmaus, *J. Allergy Clin. Immunol.*, 1994, **94**, 44.

[71] R.P. Young, B.J. Hart, T.G. Merrett, A.F. Read and J.M. Hopkin, *Clin. Exp. Allergy*, 1992, **22**, 205.

[72] M.L. Burr, E.S. Limb, M.J. Maguire, L. Amarah, B.A. Eldridge, J.C.M. Layzell and T.G. Merrett, *Arch. Dis. Child.*, 1993, **68**, 724.

exposure– response relationship for symptoms.[73] These findings are consistent with various cross-sectional studies[74,75] in which no significant or substantial association has emerged between mite allergen exposure and the prevalence of asthma symptoms in children, at exposures generally higher than $2\,\mu g\,g^{-1}$ dust.

Conclusions. Within the range of allergen exposures commonly encountered in homes (greater than $2\,\mu g\,g^{-1}$ dust), there is fairly consistent evidence of an increase in risk of mite sensitization with increasing allergen exposure. However, there is also evidence that allergen exposure may influence the risk of sensitization below the $2\,\mu g\,g^{-1}$ threshold. At all detectable levels of mite allergen exposure, a reduction may be expected to reduce the risk of mite sensitization.

Mite sensitization does not inevitably result in mite-sensitive asthma. The shape of the exposure–response relationship relating asthmatic symptoms (such as wheeze) to allergen exposure among sensitized subjects may be different from that relating allergen exposure to sensitization. The evidence from both cross-sectional and longitudinal studies is consistent with a saturation or plateau effect at levels of allergen exposure currently encountered in many homes. This implies that there may be little change in prevalence of asthma associated with a modest downward shift in allergen levels. The observational evidence relating prior allergen exposure to acute asthma attacks is inconsistent. Evidence from a large number of small controlled trials of diverse allergen reduction regimens suggests that there is little clinical benefit unless allergen levels are reduced substantially. However, each individual study lacks statistical power to demonstrate a small benefit which would nevertheless be of significance in public health terms. A formal meta-analysis is not possible because of the diversity of the outcome measures and exposure assessments in the different trials. Thus it is possible that reduction in the allergen exposure of asthmatic patients might result in a small reduction in morbidity, but the extent of the health gain (if any) cannot be quantified.

In conclusion, there is no convincing evidence of a strong exposure–response relationship between asthma symptoms and house dust mite allergen at the levels of exposure presently encountered. It may be that exposures are at the plateau of a non-linear exposure–response curve and would need to be significantly lower before a strong relationship is identified. Further studies are required to assess whether changes in the indoor environment, with and without changes in lifestyle, may lead to reduced exposure and decreases in symptomatic asthma. Further investigations are needed of the effectiveness of allergen reduction regimes, in terms of impact on allergen exposure and on incidence, prevalence and severity of symptoms. Studies should also be undertaken to clarify the exposure–response relationship between allergic sensitization, symptom prevalence and disease severity and exposure to house dust mites or mite allergens. Despite the uncertainties about the exposure–response relationship(s), a general reduction in mite allergen exposure in homes is encouraged. Lower indoor humidity could contribute to reducing mite numbers and therefore exposure to allergen.

[73] D. W. Hide, S. Matthews, L. Matthews, M. Stevens, S. Ridout, R. Twiselton, C. Gant and S. H. Arshad, *J. Allergy Clin. Immunol.*, 1994, **93**, 842.

[74] K. M. Strachan, B. K. Paine, B. K. Butland and H. R. Anderson, *Thorax*, 1993, **48**, 426.

[75] A. P. Verhoeff, R. T. Van Strien, J. H. Van Wijen and B. Brunekreef, *Clin. Exp. Allergy*, 1994, **24**, 1061.

Carbon Monoxide (CO)

Carbon monoxide is of particular interest and importance as many deaths and hospital admissions can be directly attributable to accidental domestic CO poisoning.[76] It is especially dangerous because it has no colour, smell or taste. Its toxic action is mostly through the displacement of oxygen in haemoglobin in the blood to form carboxyhaemoglobin, thus depriving the tissues of the body of their oxygen supply. Most fatal cases of carbon monoxide poisoning result from blockage of and/or leakage from flues of gas heating appliances.

There is a large body of literature concerning indoor concentrations and the health effects of CO (although very few studies have to date been conducted in the UK). Outdoor CO levels can be determinants of indoor levels but, where present, the major sources of CO in the home are gas cookers and certain types of heating systems that burn gas, wood, coal or paraffin. Environmental tobacco smoke, the presence of an attached garage and the proximity of heavily trafficked roads can also affect indoor CO levels.[77]

A recent UK study has shown typical 1-week average CO concentrations to reach 2.7 mg m^{-3} (2.4 ppm) in the kitchens of homes where there was gas cooking, compared with 0.9 mg m^{-3} (0.79 ppm) in kitchens where there was no gas cooking. Continuous monitoring indicated maximum 1-hour averages of 1.9–24.5 mg m^{-3} (1.7–21.4 ppm) in homes with gas cooking;[78] much higher peak levels of around 180 mg m^{-3} (160 ppm) for a 15-minute average have been associated with the use of a gas cooker grill.[79] Poorly installed, inadequately ventilated or malfunctioning appliances and accidentally blocked flues can also contribute to increased CO levels. Even in a sample of only 14 UK homes, a maximum 1-hour concentration of 57.0 mg m^{-3} (50 ppm) was recorded in the kitchen of one home in which the boiler was malfunctioning.[78] It is apparent that existing air quality guidelines[80]* are likely to be exceeded in a number of homes. While it is not statistically valid to extrapolate the data from the small study of 14 homes in the UK to the overall national (or international) situation, there is an obvious cause for concern.

Exposure to CO is normally evaluated in terms of percentage of carboxyhaemoglobin (COHb) in the blood, but the validity of COHb as a biomarker of health effect is open to question. Smokers have higher blood COHb levels and a higher threshold of effects. Although hypoxia, arising from preferential binding of CO to haemoglobin, is thought to be the main toxic mechanism by which CO acts, binding of CO to other blood components and enzymes may also play a part

* The current World Health Organization guidelines for CO are 100 mg m^{-3} for 15 minutes, 60 mg m^{-3} for 30 minutes, 30 mg m^{-3} for 1 hour and 10 mg m^{-3} for 8 hours.

[76] M. Burr, in *Building Regulation and Health*, ed. G.J. Raw and R.M. Hamilton, Construction Research Communications, London, 1995, p. 26.

[77] IEH, *Assessment on Indoor Air Quality in the Home 2: Carbon Monoxide*, Assessment A5, Institute for Environment and Health, Leicester, 1998, in press.

[78] D. Ross, in *Proceedings of the 7th International Conference on Indoor Air Quality and Climate*, ed. S. Yoshizawa, K.-i. Kimura, I. Ikeda, S.-i. Tanabe and T. Iwata, Institute for Public Health, Tokyo, 1996, p. 513.

[79] K.J. Stevenson, *Tokai J. Exp. Clin. Med.*, 1985, **10**, 295.

[80] WHO, *Update and Revision of the Air Quality Guidelines for Europe*, World Health Organization Regional Office for Europe, Copenhagen, 1994.

in its toxicity. A role in promoting atherosclerosis has been postulated for CO, although conclusive evidence is lacking, and immunological function and neurotransmission have also been investigated as possible targets for CO toxicity.[81]

Carbon monoxide is an important pollutant with respect to likely health effects following exposure in the home. While many of the published clinical investigations of CO intoxication in the home originate outside the UK, this does not limit their applicability. There may be differences in the types of cooking and heating appliances used but the health effects of the CO emitted from them will be broadly the same. Accidental exposures leading to acute, and sometimes fatal, health effects are well documented. Clinical reports of CO intoxication following exposure to high levels of CO have shown consistent symptoms such as headache, nausea and dizziness in the majority of patients. However, these symptoms are easily confused with those of other ailments, such as food poisoning or influenza, and missed or mis-diagnoses of CO intoxication can therefore occur.[82]

Numerous and varied observations have been made of the health effects of CO in controlled chamber studies.[81] These indicate that exposure to CO can cause performance decrements in certain neuropsychological tasks and that some people, primarily suffers of cardiovascular disease, may be more susceptible to low level exposure to CO associated with COHb levels as low as 2%.[83] However, the question of the COHb level at which cardiovascular indices do not differ from the norm has not been satisfactorily answered.[77]

Conclusions. The published evidence on health effects after domestic exposure points most to a hazard of acute CO intoxication from malfunctioning, unflued or poorly ventilated fuel burning appliances. It is also probable that in some homes CO levels routinely occur and persist that might possibly give rise to chronic health effects, particularly among sensitive groups (pregnant mothers, the foetus, children, the elderly and individuals suffering from anaemia and other diseases that restrict oxygen transport). Significant symptoms are generally experienced, even among normal healthy individuals, following exposure to CO concentrations high enough to produce COHb levels of about 20%. A great deal of importance would be attached to CO concentrations producing COHb levels above 10%, especially in sensitive individuals.

Although there is limited information from epidemiological studies on the health effects of CO at the low levels typically found in homes, the risk of adverse effects in healthy individuals as a result of exposure to CO in the home is thought to be low under normal circumstances (*i.e.* where appliances are installed and operated correctly). Nonetheless, it is prudent to continue to encourage measures which minimize CO levels, with particular attention being paid to gas combustion and other fuel-burning, especially unflued, appliances. It is also essential to increase awareness of the symptomatology of CO intoxication among health care professionals and others to whom the public look for advice and assistance.

[81] EPA, *Air Quality Criteria for Carbon Monoxide*, EPA/600/8-90/045F, US Environmental Protection Agency, Cincinnati, 1991.

[82] F. L. Lowe-Ponsford and J. A. Henry, *Advers. Drug React. Acute Poison. Rev.*, 1989, **8**, 217.

[83] E. N. Allred, E. R. Bleecker, B. R. Chaitman, T. E. Dahms, S. O. Gottlieb, J. D. Hackney, M. Pagano, R. H. Selvester, S. M. Walden and J. Waren, *Environ. Health Perspect.*, 1991, **91**, 89.

Leaving a patient in, or returning them to, a situation from which adverse health effects might develop is unacceptable and, with vigilance, need not occur.

There are a number of areas requiring further research.[77] In particular, more studies are required to determine the importance of indoor levels to overall personal exposure to CO, especially the significance of certain activities and situations which may lead to high exposures. It would also be of value to investigate exposure to CO in susceptible populations such as expectant mothers and those suffering from cardiovascular disease.

*Particles (PM_{10})**

Particulates continue to attract a good deal of attention as a possible major cause of early deaths in the population, as revealed for example by the US Six Cities study investigating the health impacts of outdoor PM_{10} levels.[84]

The major indoor sources of particles have been identified through studies performed in the US (*e.g.* the Harvard Six-City Study, the New York State Study and the EPA Particle Total Personal Exposure Assessment (PTEAM) Study).[85–88] Environmental tobacco smoke (ETS, considered separately below) has consistently been shown to be the most significant indoor particle source. Emissions from kerosene heaters and wood-burning stoves have also been shown to add to the indoor particle load, although these sources are of less significance in the UK and other countries. Some US studies have also identified cooking as a source of particles (from both the food itself and combustion of the cooking fuel).[87,89] Very fine particles are thought to readily enter buildings[90] so outdoor sources (*e.g.* traffic) are also important to personal exposure. The activity of occupants in the home has also been found to influence indoor particle levels. Vacuuming, sweeping and dusting have been shown to raise levels of particles.[87,89] Although these are not direct sources of particles, in that they represent re-entrainment of settled particles, such activities may affect the total indoor level to which occupants are exposed. Indeed, a 'personal cloud' effect has been described whereby personal exposure exceeds that expected from statically monitored indoor and outdoor levels.[91] This suggests that human behaviour and activity can markedly influence exposure.

Limited information suggests that indoor levels of particles are generally lower

* Particulate matter of 10 μm or less aerodynamic diameter.

[84] D. W. Dockery, C. A. Pope, III, X. Xu, J. D. Spengler, H. H. Ware, M. E. Fay, B. G. Ferris, Jr. and F. E. Speizer, *New Engl. J. Med.*, 1993, **329**, 1753.

[85] J. D. Spengler, D. W. Dockery, W. A. Turner, J. M. Wolfson and B. G. Ferris, *Atmos. Environ.*, 1981, **15**, 23.

[86] J. D. Spengler, R. D. Treitman, T. D. Toteson, D. T. Mage and M. L. Soczek, *Environ. Sci. Technol.*, 1985, **19**, 700.

[87] H. Özkaynak, J. Xue, J. Spengler, L. Wallace, E. Pellizzari and P. Jenkins, *J. Expos. Anal. Environ. Epidemiol.*, 1996, **6**, 57.

[88] B. P. Leaderer, P. Koutrakis, S. L. K. Briggs and J. Rizzuto, *Indoor Air Int. J. Indoor Air Qual. Clim.*, 1994, **4**, 23.

[89] R. Kamens, C.-T. Lee, R. Wiener and D. Leith, *Atmos. Environ., Part A*, 1991, **25**, 939.

[90] T. L. Thatcher and D. W. Layton, *Atmos. Environ.*, 1995, **29**, 1487.

[91] C. A. Clayton, R. L. Perritt, E. D. Pellizzari, K. W. Thomas, R. W. Whitmore, L. A. Wallace, H. Özkaynak and J. D. Spengler, *J. Expos. Anal. Environ. Epidemiol.*, 1993, **3**, 227.

than, but correlated to, outdoor levels unless there is a significant indoor source. Thus where there are no major indoor sources of particles, outdoor levels may be a reasonable proxy for indoor exposure, but they cannot accurately estimate *personal exposure* to particulates because of the 'personal cloud' effect.

Epidemiological studies have demonstrated a consistent and statistically significant association between ambient (outdoor) airborne particle level and the incidence of mortality or morbidity in human populations. The associations with death and hospital/emergency room admission rates are the most robust[92,93] and appear to operate at low exposure levels within the range frequently encountered in many developed countries.[94] Cardiopulmonary impairment appears to be the predominant effect, and the elderly or infirm appear to be at particular risk. In addition, irrespective of age, asthmatics appear to suffer increased symptomatology and increased risk of acute attack.[92,95,96] There is also evidence that ambient particle exposure is associated with falls in pulmonary function measures, especially for asthmatics.[97,98] Chronic effects on cardiopulmonary disease and, possibly, cancer have been noted,[84] while there are reports from China of small reductions in the duration of pregnancy and in birth weight.[99] There is also some evidence from the US for effects on postnatal mortality.[100] Despite the consistency of some of these findings, interpretation and comparison is difficult, not only because of the implicit limitations of epidemiological studies, but also because of the widely differing approaches taken to the classification and monitoring of the particulate fraction of ambient air. Because of this, the validity of the available epidemiology data on outdoor particle exposure has been questioned by some workers, and there is a need for better quantitative risk estimates of the long-term impact of PM_{10} exposure.[96,101] There is a dearth of studies investigating possible links between health effects and indoor or personal exposure to particles or other airborne pollutants such that, at present, these aspects cannot be confidently assessed.

In addition to the epidemiological evidence, there is some support for a causal link between non-ETS-derived particles and adverse health effects from human volunteer studies. In summarizing the status of knowledge, the UK Committee on the Medical Effects of Air Pollutants[95] noted that there had been few investigations, mostly focusing on sulfuric or other acid aerosols. There is evidence for effects on lung function and bronchial reactivity, although the effects

[92] D. W. Dockery and C. A. Pope, III, *Annu. Rev. Public Health*, 1994, **15**, 107.
[93] K. Katsouyanni, G. Touloumi, C. Spix, J. Schwartz, F. Balducci, S. Medina, G. Rossi, B. Wojtyniak, J. Sunyer, L. Bacharova, J. P. Schouten, A. Pönkä and H. R. Anderson, *Br. Med. J.*, 1997, **314**, 1658.
[94] B. Brunekreef, D. W. Dockery and M. Krzyzanowski, *Environ. Health Perspect.*, 1995, **103** (suppl. 2), 3.
[95] COMEAP, *Non-biological Particles and Health*, Committee on the Medical Effects of Air Pollution, HMSO, London, 1995.
[96] C. A. Pope, III, D. W. Dockery and J. Schwartz, *Inhal. Toxicol.*, 1995, **7**, 1.
[97] J. Q. Koenig, K. Dumler, V. Rebolledo, P. V. Williams and W. E. Pierson, *Arch. Environ. Health*, 1993, **48**, 171.
[98] L. M. Neas, D. W. Dockery, P. Koutrakis, D. J. Tollerud and F. E. Speizer, *Am. J. Epidemiol.*, 1995, **141**, 111.
[99] X. Xu, H. Ding and X. Wang, *Arch. Environ. Health*, 1995, **50**, 407.
[100] M. Bobak and D. A. Leon, *Lancet*, 1992, **340**, 1010.
[101] C. A. Pope, III, D. V. Bates and M. E. Raizenne, *Environ. Health Perspect.*, 1995, **103**, 472.

reported have been variable. In normal subjects, slight changes in lung function were noted at high concentrations ($100\,\mu g\,m^{-3}$) of sulfuric acid aerosol, while exposure to diesel exhaust at a concentration of $4.3 \times 10^6\,cm^{-3}$ was shown to elicit pulmonary inflammatory changes.[102,103] Overall, although human studies have confirmed that some particles can cause physiological change in healthy humans, this has only been demonstrated at exposures above those normally experienced in the environment. However, human volunteer studies have found differences between population sub-groups, for example regional deposition patterns within the respiratory tract differ between children and adults,[104] and this will result in different doses and sites of deposition at identical exposures. Also, as noted above, asthmatics seem to be more sensitive to the effects of particles. Non-human experimental work has confirmed the intrinsic toxicity of some particles and has suggested a number of possible toxic mechanisms. However, the relative importance of particle number, size, mass and composition is still not clear.

Other hypotheses (not assuming a causal relationship) have been put forward to explain the positive correlation between outdoor particle levels and mortality or morbidity rates observed in epidemiological studies. Valberg and Watson,[105] for example, support the view that there is no causal link between excess mortality or morbidity and exposure to particulate matter, but that particle levels are merely a marker for other causal factors (including increased exposure to indoor pollutants).

The role played by sources which are not of outdoor origin in determining personal exposure to particles needs to be elucidated. A key question concerns the contribution of indoor particulate sources to total personal exposure to particles, and hence the potential impact of indoor particle sources on health. If indoor particles significantly influence personal exposure levels, then the existing epidemiological evidence, which is based upon outdoor levels of particles, would need to be revisited and reinterpreted.

Conclusions. Epidemiological studies have shown that those at greatest risk from exposure to particles are people with existing respiratory or cardiovascular diseases. Many of these people may be elderly or infirm, and would be expected to spend the majority of their time indoors. Therefore, it may be reasonable to assume that personal exposure to particles in this group is most influenced by the level of particles in indoor air. Little is known about the level or composition of particles to which such susceptible groups are exposed, and whether this differs from the exposure of the general population. Whilst a link between particulates in outdoor air and ill health has been demonstrated through epidemiological studies, there have been no similar studies based on particle levels in indoor air.

Assuming that the relationship between particle exposure and effects on health is causal, remedial strategies should be aimed at reducing total personal exposure

[102] M. T. Newhouse, M. Dolovich, G. Obminski and R. K. Wolff, *Arch. Environ. Health*, 1978, **33**, 24.
[103] B. Rudell, T. Sandström, N. Stjerneberg and B. Kolmodin-Hedman, *J. Aerosol Sci.*, 1990, **21**, S411.
[104] W. D. Bennett, K. L. Zehman, C. W. Kang and M. S. Schechter, *Ann. Occup. Hyg.*, **41** (suppl. 1), 497.
[105] P. A. Valberg and A. Y. Watson, in *Proceedings of the Second Colloquium on Air Pollution and Human Health*, ed. J. Lee and R. Phalen, Utah, 1996, p. 4–573.

to particles. For an individual, personal exposure will comprise exposure from a variety of sources, which may be of outdoor or indoor origin. Where there is a significant indoor source of particles this may have a large influence on total personal exposure levels, and remedial action may be warranted. However, it may be appropriate to focus such action upon the susceptible groups in the population. For example, ventilation could be improved, or air filters fitted, in housing for the elderly and asthmatics. Alternatively, if certain types of particle are identified as being more harmful, controls could be put in place to limit exposure to these particles (*e.g.* fit extractor hoods to gas cookers).

3 Other Issues

For the particular pollutants considered here, there is a large variation in the amount of available information on levels in the home and in the degree of confidence in measuring and monitoring techniques and in the evaluation of likely health effects. Factors such as age, social class, ethnic group, geographical area and type of dwelling may also influence the likelihood and type of health effects brought about by exposure to these pollutants. Further studies are needed to clarify some of these issues.

There are a number of other specific indoor pollutants, not reviewed above, which are also important. These include radon (a naturally occurring radioactive gas suspected of causing many cases of lung cancer[106]), organochlorine compounds (used, for example, as domestic pesticides), other biological allergens (such as cat dander), fibres (both asbestos and man-made mineral fibres[107]) and environmental tobacco smoke. Although the health effects of tobacco smoke on adults are well known, the issue of environmental tobacco smoke (ETS) in the home tends to attract little attention because it seems so obviously under the direct control of the occupants. This is not, however, the case for children, who may be unwillingly and chronically subjected to tobacco smoke in the home (or *in utero*) at a critical time of life. Understanding the impact of ETS on the health of children is thus of particular importance. Tobacco smoke contains tar droplets and a cocktail of various other toxic chemicals including carbon monoxide, nitric oxide, ammonia, hydrogen cyanide and acrolein, together with proven animal carcinogens such as *N*-nitrosamines, polycyclic aromatic hydrocarbons and benzene. ETS is known to irritate the eyes, nose and throat, and exposed babies and children are more prone to chest, ear, nose and throat infections. Also, women exposed during pregnancy tend to have lower birthweight babies. In a recent review, the Californian EPA[108] concluded that causal links have been established between the exposure of non-smokers to ETS and the following adverse conditions, and estimated their associated relative risks (RR): death from

[106] ECA-IAQ, *Radon in Indoor Air*, European Collaborative Action 'Indoor Air Quality and its Impact on Man', Report No 15. EUR 16123 EN, Office for Official Publications of the European Community, Luxembourg, 1995.

[107] IEH, *Fibrous Materials in the Environment*, Report SR2, Institute for Environment and Health, Leicester, 1995.

[108] EPA, *Health Effects of Exposure to Environmental Tobacco Smoke*, Final Report, California Environmental Protection Agency, Sacramento, 1997.

heart disease (RR 1.3); lung cancer (RR 1.2); nasal sinus cancers (RR 1.7–3.0); and, in children, low birthweight (RR 1.2–1.4); sudden infant death syndrome (SIDS) (RR 3.5); asthma induction (RR 1.75–2.25) and exacerbation (RR 1.6–2); middle ear infection (RR 1.62); and lower respiratory disease (RR 1.5–2). Although the relative risk for some of the conditions was small, it was noted that the diseases are common and hence the overall impact on health is potentially quite large. A current series of articles by Strachan and others on a range of disease endpoints including, for example, middle ear disease,[109] has further emphasized that ETS, especially with regard to the exposure of children, is an important issue warranting close attention.

In addition to the particular pollutants mentioned above, there are other issues related to the theme of indoor air quality and health which require a rather different approach. The first is so-called 'sick building syndrome' (SBS), which comprises a range of disparate but common conditions and is associated with certain individual workplace buildings.[110] Various causes of SBS have been proposed, including volatile organic compounds (VOCs; see above), temperature and humidity, 'dustiness/cleanliness', dust mites, mechanical ventilation and various psychological factors, but none of these alone appears to explain the syndrome adequately. Also 'multiple chemical sensitivity', the condition whereby individuals appear to show exquisite sensitivity to very low concentrations of organic chemicals, is receiving further attention, having originally spawned much interest in the USA and Scandinavia but generally being ignored elsewhere. There is also continued interest with respect to increasing trends in asthma rates in developed counties and the possible role of the indoor environment.

Policy and Research Initiatives

In the UK, the publication in 1991 of the Select Committee Report on Indoor Pollution,[111] and the Government's subsequent response,[112] served to focus attention on the importance of the indoor environment, and this momentum has been maintained in recent years. Both the Department of the Environment, Transport and the Regions and the Department of Health are commissioning research on indoor air quality directly relevant to human health effects. More widely in Europe, a number of extensive multi-centre studies are underway or awaiting final analysis (*e.g.* APHEA and EXPOLIS*). These should provide useful information for the further assessment of exposure and health impact of a number of key indoor pollutants described here.

* APHEA: Air Pollution and Health: a European Approach; EXPOLIS: Air Pollution Exposure Distributions within Adult Populations in Europe.

[109] D. P. Strachan and D. G. Cook, *Thorax*, 1998, **53**, 50.

[110] ECA-IAQ, *Sick Building Syndrome—A Practical Guide*, European Collaborative Action 'Indoor Air Quality and its Impact on Man', Report No 4. EUR 12294 EN, Office for Official Publications of the European Community, Luxembourg, 1989.

[111] House of Commons Select Committee, *Indoor Pollution*, Sixth Report, HMSO, London, 1991.

[112] Cmnd 1633, *The Government's Response to the Sixth Report from the House of Commons Select Committee on the Environment, Indoor Pollution*, HMSO, London, 1991.

The UK National Environmental Health Action Plan (NEHAP),[113] which was published in 1996, identifies indoor air quality (IAQ) as a key area for action. This is particularly important because the UK was one of the 'pilot countries' chosen at the 1994 Helsinki inter-governmental conference on environment and health to produce the first NEHAPs, and it is thus likely that the considerations of indoor air quality contained in the UK document will carry through to other nations' NEHAPs. The UK plan provides a framework for actions by central and local government, industry and voluntary organizations to improve the environment for the benefit of health. The intention is for individuals to make informed decisions about their own homes, using appropriate, targeted information. Information is to be made available to people about the most important sources of pollutants in the home, including activities that lead to exposure, and actions which they can be taken to minimize exposure and any associated health effects. Such actions may include not smoking indoors, ensuring adequate ventilation when using a gas cooker, using water-based rather than solvent-based paints and choosing low-formaldehyde particle board or carpets. Better instructions for use of household products may also be warranted. In addition, manufacturers and suppliers of materials and furnishings are encouraged to reduce the levels of emissions from their products generally and to provide relevant product information so that people can choose to buy or specify the materials they require. The plan is to be revised and is likely to contain new initiatives relevant to indoor air quality. Targeted research in the area of indoor air quality and health is to continue in an effort to understand better the levels, sources and health effects of indoor pollutants, and to provide further knowledge about mechanisms of action and the role of mitigation procedures.

On the broader international scene, IAQ is continuing to receive attention through bodies such as the International Society for Indoor Air Quality and the major triennial 'Indoor Air' conferences. Also there is a NATO initiative on indoor air under its 'challenges of modern society' programme, and the World Health Organization is increasingly concerned about indoor air pollution in developing countries where exposures can be extremely high. Recognizing the real importance to public health of indoor air quality, the US Environmental Protection Agency recently launched a major policy and research initiative on the subject. The European Concerted Action on 'Indoor Air Quality and its Impact on Man' continues to add to its list of published reports on this theme, but with the exception of the Nordic countries, Germany and the Netherlands, policy on indoor air issues in Europe is generally poorly developed. To improve this situation, WHO Europe recently produced a document on indoor air pollution exposure assessment,[114] and is presently engaged in formulating a strategic approach to indoor air policy making. This latter publication is intended to inform and advise governments, public health authorities, and other policy makers and representatives of other sectors relevant to IAQ management on how

[113] DoE, *The United Kingdom National Environmental Health Action Plan*, CM3323, HMSO, London, 1996.

[114] M. Jantunen, J.J.K. Jaakkola and M. Krzyzanowski, *Assessment of Exposure to Indoor Air Pollutants*, European Series No. 78, World Health Organization Regional Office for Europe, Copenhagen, 1998.

to develop and strengthen IAQ policy in order to achieve health protection and promotion in the indoor environment. It recommends that a key strategy for the management of IAQ is the development of a comprehensive, scientifically sound and thoroughly considered 'action plan' (possibly part of a NEHAP) which should be targeted to new construction as well as existing buildings (and other indoor spaces), and should entail actions at both national and local levels. The quality of indoor air is determined by a large number of different factors and, consequently, different professions are involved in dealing with and solving indoor air problems. While central government may take the lead, industry and commerce also need to make appropriate contributions to the achievement of better indoor air quality. The role of the private sector in ensuring acceptable indoor air quality is therefore encouraged. The IAQ strategy needs also to include the assessment (and, where appropriate, the promotion or/and verification) of the 'safety' of building materials and equipment, furniture, consumer products and other materials used inside enclosed spaces. The European Concerted Action on Indoor Air and its Impact on Man is in the process of evaluating methods for the positive labelling of products which are low emitters of volatile organic compounds and has recently published a report on this topic.[115] Also there are likely in the near future to be European standards regarding emissions of formaldehyde from building materials.

One of the 'policy' issues frequently raised concerns specific standards or guideline levels for indoor air pollutants, but experiences in Germany led Seifert[116] to caution that such guideline values, if not introduced with sufficient care, may accelerate the already existing trend to solve air quality problems by litigation.

4 Conclusions

While the main focus of public concern may, for a while at least, remain on outdoor air quality, notably traffic pollution, it is clear that the indoor environment merits extra attention. This, after all, is where people spend the vast majority of their time, and the quality of the air in the home could have significant impacts in public health terms. Certainly the indoor environment has been shown to contain sources of various noxious substances. There is particular concern for potentially vulnerable or susceptible groups such as the very young, the sick (especially, perhaps, those with pre-existing cardiopulmonary disease) and the elderly, who spend a disproportionately large amount of time indoors at home. Within the scientific community there is the requirement to consider fully the role of indoor pollution in the context of total personal exposure in order to assess properly the impact of air pollution on the health and well-being of individuals and to facilitate the identification of effective control and remediation measures.

[115] ECA-IAQ, *Evaluation of VOC Emissions from Building Products—Solid Flooring Materials*, European Collaborative Action 'Indoor Air Quality and its Impact on Man', Report No 18. EUR 17334 EN, Office for Official Publications of the European Community, Luxembourg, 1997.

[116] B. Seifert, in *IEH Assessment on Indoor Air Quality in the Home*, *Assessment A1*, Institute for Environment and Health, Leicester, 1996, p. 324.

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