

EDITORIAL

Indoor fungal exposure — does it matter, and what can be done about it?

The tendency for fungi to grow inside dwellings has long presented a problem. The Old Testament describes the growth of mildew in a house, with instructions for dealing with it by removing the affected stones, scraping all the walls, and replastering; if the problem recurred, the house was demolished [1]. In modern houses the airtight design is liable to cause condensation of water vapour on external walls and unventilated crevices, which then tend to acquire patches of mould. Various species affect window frames, other woodwork, building materials, decorated surfaces, damp basements, the soil of houseplants, mattresses, and food [2–5]. The appearance and smell of mould are objectionable enough, but there is also a widespread belief, and some evidence, that it has adverse effects on health.

Ascertainment of indoor fungal exposure

In order to examine the possible health effects of indoor fungal exposure, it is obviously necessary to have some criteria for determining whether and to what degree it occurs. The simplest method is to ask the residents of a house if mould growth is visible indoors. Samples of the mould can then be taken so as to identify the organisms. The disadvantage of this approach is that it depends on the observational powers of the residents, who may be unaware of mould growing in less obvious places; it also gives little information about the degree of exposure. Inspection of the house by a surveyor (or other trained observer) gives more consistent findings, including some indication of severity [6]. The assessment of airborne exposure can take a variety of forms, the oldest of which is the use of settle plates. These show the relative prevalence of different fungi in the air, but have important limitations, including a tendency not to collect small spores [2]. Volumetric air samplers are commonly used now, and give more precise estimates of concentrations in air of different species. Airborne spores and other propagules are trapped; they are then either examined microscopically or incubated on agar media so that the resulting colonies can be identified and counted. Each of these sampling techniques ('non-viable' and 'viable', respectively) has its limitations [3]. The results are subject to wide fluctuations (both qualitative and quantitative) over short periods of time, so that the precision of measurement is offset by uncertainties about the extent to which the observations reflect habitual exposure. Furthermore, different fungi require different culture

media, and many airborne spores will not grow in culture and cannot be visually identified using currently available methods [4].

Another approach is to examine the concentrations in house dust of substances that are produced by fungi, thus giving an estimate of the biomass of airborne fungi over a period of time. Two such substances are β -glucan and ergosterol, both of which are characteristic components of fungal cells and have been measured as indicating total indoor exposure, although of course they do not give information about the species concerned [4].

In Britain, *Penicillium* is the fungus most frequently isolated indoors [5,7]; other common fungi are *mycelia sterilia* (non-sporulating fungi), *Cladosporium*, *Aspergillus* and yeasts. In Australian homes *Cladosporium* and *Penicillium* species make the largest contribution to viable indoor spore levels [8,9]. In the Melbourne survey reported in the present issue, indoor mould was ascertained in three ways: by questionnaire, air sampling with agar culture, and ergosterol concentrations in floor dust [9]. Visible mould was an independent predictor of both the total viable fungal propagules and the ergosterol levels, with a trend for each to increase with the number of mouldy surfaces. This suggests that information derived from questionnaires, with their advantages of cheapness and speed, is not necessarily to be despised. Ergosterol concentrations were weakly correlated with fungal levels only in those houses where the windows were closed at the time of sampling. The seasonal variation in propagule concentrations reflected variations that have been reported in outdoor air. These findings show the potential contribution of outdoor sources to indoor exposure, particularly in a climate where windows are likely to be kept open for much of the time.

Health effects

There are several pathological mechanisms that could provide a link between fungal exposure and ill-health [2]. Allergy to mould can cause asthma, allergic rhinitis and atopic dermatitis; *Alternaria* and *Penicillium* spores provoke immediate and delayed-onset asthma in sensitized persons [10]. Extrinsic allergic alveolitis is associated with a range of fungi, including *Cladosporium* and *Serpula* (dry rot). The spores of many indoor fungi contain a variety of mycotoxins, which may give rise to respiratory and other symptoms quite apart from allergic reactions; certain

fungi (e.g. *Aspergillus* species) produce carcinogenic aflatoxins. It is therefore possible that indoor moulds occasionally contribute to the risk of major disorders such as immunosuppression and leukaemia. Fungal cell walls contain β -glucan, which is known to reduce the number of alveolar macrophages and inflammatory cells and impair phagocytosis [4]. This could lead to increased susceptibility to infection. Furthermore, moulds emit volatile substances, giving a characteristic musty odour; some of these affect ciliary action or have irritant effects to which various symptoms have been ascribed [2,4].

The question arises as to how much ill health is actually attributable to indoor exposure to fungi. A number of epidemiological studies in different countries have looked at the relationship between indoor moulds and symptoms. Several of these surveys showed associations between mould growth (ascertained in various ways) and respiratory symptoms [6,8,11–18]. Some studies have shown associations between mould growth and a wide range of symptoms in adults, not restricted to the respiratory tract [6,18]. The nature of some of these symptoms (such as backache, fainting, 'bad nerves', difficulties in concentration) raises the issue as to whether the observed associations are likely to be causal. There are several ways in which they could arise apart from a harmful effect of fungi on health. People who are aware of mould and damp in their houses may very well be alerted to minor symptoms that they would otherwise ignore, particularly if they hope to be rehoused on the grounds of ill health. Or the reporting bias may act in the opposite direction, in that patients with asthma (or their parents) have more reason to notice mould growth than do healthy people. This bias did not seem to occur in a Dutch study, where trained observers found no evidence of parental over-reporting of mould in the homes of children with chronic respiratory symptoms [16]. If there is a real association, it may be attributable to some other factor. People who live in substandard accommodation are likely to be disadvantaged in other ways that increase the risk of illness; even if socioeconomic status is allowed for, it is conceivable that people who neglect their houses are similarly prone to neglect their own or their children's health. The dampness that promotes mould growth also encourages infestation with house dust mites, so that mould growth could simply be a marker for mite allergen.

These issues have been addressed in various ways. Most studies try to allow for confounding variables, but it is impossible to be sure that the attempt is successful. Some have examined objective health indices so as to remove reporting bias in relation to health effects. A Dutch survey showed that mould growth was related to peak expiratory flow rate variability as well as respiratory symptoms [17]. In some studies [7,8,19], though not in others [20,21], sensitivity to mould (as evidenced by skin tests or serum

RAST) was associated with indoor mould growth. A Canadian study suggested that fungal exposure leads to chronic stimulation of lymphocytes, which could reflect the incidence of infections or autoimmune disorders [22]. For mould exposure, the use of objective measurements rather than reported observations removes the effect of biased reporting. The study by Platt and colleagues [6] found that some symptoms were associated both with reported mould and with air spore count. On the other hand, the study by Strachan [13] showed that childhood wheeze was strongly associated with reported mould but not with air concentrations, except for the heterogeneous group of non-sporing fungi, suggesting that questionnaire-based data may be biased and unreliable. Conversely, the study by Garrett [8] found childhood asthma to be associated with exposure to airborne *Penicillium* spore levels but not with visible mould. It is obviously desirable that both fungal exposure and health outcomes should be ascertained by objective as well as subjective methods.

Determinants of indoor fungal exposure

Assuming that indoor fungi have adverse effects on health, how can their presence be discouraged? The Melbourne investigators recorded various details about the houses in question, and certain factors emerge as positive or negative determinants of fungal exposure [9]. Ventilation is clearly important: spore or ergosterol levels were lower if the houses were frequently aired, or if there was an extractor fan in the kitchen or a ceiling fan in the bedroom. A solid fuel fire also seemed to reduce spore levels, presumably by drawing air through the house. The effect of opening windows is not straightforward, however; an open window at the time of sampling was associated with higher spore concentrations in air but lower ergosterol levels in floor dust. Garrett *et al.*'s study similarly showed spore concentrations to be higher at the time the window was open but lower if the windows were opened most months of the year [8]. Thus opening the window causes a temporary rise in fungal exposure (since concentrations are usually higher outside than inside [2]), but a long-term fall. People who are allergic to moulds may be well advised to close the windows of rooms that they are occupying and to open other windows in the house.

Fungus levels were higher in the presence of old fitted carpets, but lower if frequent vacuum cleaning was carried out, suggesting further ways in which exposure can be reduced. Not surprisingly, spore number was associated with humidity, as others have found [5,8]. Noticeable mould growth was associated with the objective measures of exposure, as in a British study [5]; in Garrett's study [8] this was the case only for *Cladosporium*, but spore concentrations were lower where mould growth had been removed

by the occupants. It seems that dealing with visible mould is likely to reduce indoor exposure. In Danish houses affected by mould, substantial reductions in spore counts were achieved by what were described as 'simple measures' designed 'to correct construction faults or living habits' [23]. What is really needed is a randomized controlled trial of such measures among patients with mould-sensitive asthma. The presence of cats or dogs in the house was another major determinant in Melbourne, but it is less easy to translate this observation into acceptable advice: getting rid of the pet would strike a pet lover as even more drastic than the Old Testament remedy of house demolition.

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M. L. BURR

*Centre for Applied Public Health Medicine,
Temple of Peace and Health,
Cathays Park,
Cardiff CF1 3NW,
UK*