

Environmental and Workplace Health

Fungal Contamination in Public Buildings: Health Effects and Investigation Methods

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2. Health Effects of Indoor Molds (Continued)

2.4.2 Limitations

2.4.2.1 Exposure assessment

In most epidemiological studies on indoor mold and health, the exposure assessment was based on participants' self-reports. In the few studies where exposure to mold was assessed by a member of the research team, the exposure classification was based on dichotomous questions such as the presence or absence of dampness and/or mold; there was no quantitative exposure assessment, and therefore no determination of a dose-response relationship. Exceptions are the studies of Garrett *et al.* (1998) and Dales *et al.* (1999). Also, in most cross-sectional and case-control studies, the mold taxa present in homes were not identified. Mold species differ considerably, not only in their potential to cause adverse effects to human health, but also in the mechanisms by which they can affect health (i.e. through releasing volatile compounds, allergens or mycotoxins) and, therefore, in the nature of effects they can cause.

The difficulty of quantifying human exposure to mold is thus a major obstacle in ascertaining the existence of cause-and-effect relationships, as dose-response relationships cannot be assessed. This difficulty has led the Institute of Medicine (2000) to conclude that ". . . standardized methods for assessing exposure to fungal allergens are essential, preferably based on measurement of allergens rather than culturable or countable fungi . . ." in order to obtain a clear understanding of the effects of building-related fungi.

Quantitative measurement, rather than questionnaire-based assessment, of exposure to fungi may be a promising way to improve epidemiological studies. However, the traditional method of exposure measurement (i.e. air sampling and culture of fungal spores) shows several limitations that make their utility questionable. For example, airborne fungal spores can be sampled only over short periods of time, while air counts of fungal spores vary considerably over longer periods of time. Also, the culture medium used always favours some species over others, and some fungal taxa have the ability to inhibit the growth of other taxa in culture media.

For all the reasons mentioned above, the determination of surrogate markers of fungal growth, such as ergosterol and (1->3)- β -D-glucan, in house dust appear to be more promising (Dillon *et al.* 1999). Both ergosterol and (1->3)- β -D-glucan are cell membrane constituents in fungi (Li and Hsu 1996; Miller and Young 1997). (1->3)- β -D-glucan has been associated with increased peak expiratory flow (PEF) variability in asthmatic children (Douwes *et al.* 2000). There is, however, a need for further research to develop standardized protocols for the determination of (1->3)- β -D-glucan in the environment (Dillon *et al.* 1999). Determination of extracellular polysaccharides (EPS) of *Aspergillus* and *Penicillium* in house dust is another approach being developed for the assessment of exposure to mold. EPS is a fungi-specific marker but, unlike glucan, it is not suspected to be causally related to adverse effects on respiratory health (Chew *et al.* 2001).

Molecular approaches have been developed for assessing both qualitative and quantitative fungal exposure in buildings and other environments (Haugland *et al.* 1999). To date, there is little practical experience with this approach. Some research groups have proposed using animal-derived antibodies to provide quantitative and qualitative information on fungal exposure (Wijnands *et al.* 2000a, 2000b). Another approach to measuring fungal exposure, advocated by the US Institute of Medicine Committee on Asthma

(Institute of Medicine 2000), is to determine human fungal allergens or antigens. Research is under way on this in Canada.

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