

Controversies regarding dampness and mold growth in homes

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Whether it is in wine or cheese or rotten tomatoes, humans depend on their noses to guide them to what is good and keep them away from what is bad for them. The substances that waft through the air and leave their indelible impression on our noses can be grouped into the category of volatile organic compounds (VOC). The association of these odor-producing chemicals with fungi goes back to the very first recognition that fungi existed. Pasteur devised his germ theory of disease based upon his experience working in the wine industry.

The review by Hope and Simon¹ covers the four basic components of fungal related disease: allergy, infection, irritation, and intoxication. They reviewed studies containing over 14,000 adults and children that produced conclusions consistent with the proposition that humans have a very poor toleration for fungi and fungal products in the respiratory tract. This broad statement would be disputed by few practicing allergists and researchers in the field. However, the exact nature of the offending agent and the pathogenesis are areas of heated argument.

This review of health effects associated with damp indoor spaces finds evidence of a relationship between exposure and aero-irritant health effects for both adults and children residing in damp indoor spaces. It also indicates that many studies have been performed that indicate the presence of airborne irritant exposure in nonoccupational settings. This would appear to be in some disagreement with the position paper of the AAAAI on the medical effects of mold exposure.² The primary areas of disagreement include: The occurrence of mold-related toxicity (mycotoxicosis) from exposure to inhaled mycotoxins and the occurrence of mold-related irritant reactions from exposure to fungal irritants (MVOC) in nonoccupational settings

The disagreements result primarily from the nature of the evidence and the rules of evidence followed by the two groups of authors. The authors of the position

paper insist on very stringent rules of evidence. For a causal relationship between fungal exposure and health effects to exist, a series of hypotheses must be proven. To conclude that an individual has experienced harm from indoor airborne exposure to mold, it is necessary for the following hypotheses to be tested and shown to be correct: 1) the suspected mold is present in the building, 2) there is a pathway from the source to where the harmed person lives, 3) fungal-derived substances such as spores, mycelia, MVOC, ergosterol, etc., travel from the source to the harmed person, 4) the person experiences sufficient airborne exposure over a long enough time to suffer harm, 5) the person is sensitive to the measured exposure, and 6) the harm is caused by the exposure and not by other factors. If these series of hypotheses are not tested, it is not possible to positively conclude that an individual has experienced harm from mold exposure. Therefore a strict causal relationship is very difficult to demonstrate.³

The authors of the current review only aim to survey the current literature and determine whether currently held beliefs can be accepted or whether additional work is necessary. They need merely to show that there is a trend in the recent literature that provides support for a new idea (airborne intoxication or irritation related to fungi in domestic settings). It is perfectly reasonable for them to examine the current evidence, state a new hypothesis, and call for studies designed to test this hypothesis. They can also call for sensible actions that might be taken to alleviate the problems they describe. They can suggest that questions about long-term exposure to indoor environmental dampness or the presence of visible mold should be included by physicians when taking a history. They might give advice as simple as buying a \$20 moisture monitor to constantly monitor indoor humidity levels. It is very appropriate for a review article to put forward a new hypothesis or propose very practical actions even in the absence of a proven causal relationship.

The calls in this review for additional investigations into mycotoxins and MVOC are perfectly logical. For the past 30 years, since the discovery of IgE, allergy has been focused on proteins. From the immunoglobulin protein that dominates the field to the protein allergens that are continuing to be discovered to the small pro-

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tein cytokines that control the cellular interactions of the immune system, research on proteins of immunological importance has been a gold mine of knowledge discovery. Even though this focus on proteins has produced great strides forward, the importance of smaller molecules to the area of respiratory disease has languished. The Hope and Simon review in this issue of the journal brings the focus around to these small but potent molecules. These authors point out that VOC have proven to be important agents of respiratory symptoms in occupational medicine. Occupational researchers have developed a great deal of information concerning human sensitivity and irritant threshold levels of VOC. Hope and Simon point out that preliminary measurements of free and particulate-bound VOC levels in damp environments indicate the potential that irritant thresholds might be reached.

The authors point out that much work remains to be done in defining a pathophysiological mechanism for the role of VOC in irritant respiratory effects. This biologic mechanism would permit allergists to deal

with their patients using a rational explanation for their symptoms. Additional studies to determine MVOC levels in categories of damp homes, and to identify fungal contribution to indoor VOC levels are necessary. Threshold levels for irritant effects resulting from chronic exposures need to be determined for many additional VOC molecules. Much work remains to be done before we can declare that one man's perfume may be another man's asthma trigger, but the review by Hope and Simon certainly makes the case for the urgency of such work. However, calling for additional work is a long way from defining a causal relationship.

References

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