

disburse biased opinions as facts to its membership. I believe this article does not meet the minimal standard for a position paper by the Academy. It should be withdrawn. The Academy would do well to sponsor an open forum in which to debate the issues of health effects from mold exposure in the Journal.

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Disclosure of potential conflict of interest: V. A. Marinkovich has served as an expert witness in mold litigation cases.

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The role of airborne mold in chronic rhinosinusitis

To the Editor:

Bush et al¹ confuse the role of molds in chronic rhinosinusitis (CRS). They accept the paradigm of allergic fungal rhinosinusitis and state that the criteria have been well delineated and that allergic fungal rhinosinusitis is readily distinguishable from typical CRS. They do not mention that these criteria have been established in patients who had been preselected to have these criteria present.^{2,3} Recent advances in the detection methods for the criteria have resulted in the demonstration of those criteria in the vast majority of CRS cases.^{4,5} The only exception is the presence of an IgE-mediated allergy to molds, which must be seen as a comorbid allergic rhinitis to molds.³⁻⁷

The presence of airborne molds in the mucus of patients with CRS has been established by means of culture, PCR, histology, and antigen detection, whereas healthy control subjects also had positive cultures.^{4,5,8-13} The presence of eosinophilic mucus with cluster formation has been found to be present in more than 95% of unselected patients undergoing surgery for CRS when the mucus was preserved during surgery and no presurgical systemic steroids were given.^{4,5,14}

It has been found that immune cells from patients with CRS (PBMCs) react to common airborne fungi, specifically

Alternaria species, with the production of cytokines that are crucial for eosinophilic inflammation, namely IL-13 and IL-5.¹¹ The increased fungus-specific IgG levels correlated directly with the production of IL-5. This immune response was independent from the allergy status of the patients and absent in healthy control subjects.¹¹

In addition, *Alternaria* species also induced a striking degranulation of eosinophils.¹⁵ The fraction from *Alternaria alternata* that induced the degranulation had a molecular weight of approximately 60 kd, was highly heat labile, and worked protease dependant through a G protein-coupled receptor.¹⁵ Other fungal antigens did not induce eosinophil degranulation, nor did neutrophils respond to *Alternaria* species extracts, suggesting the presence of a fungal species and a cell type-specific novel immune response in human subjects.¹⁵

Although one trial delivering antifungals as a spray failed to demonstrate efficacy,¹⁶ other trials that formulated amphotericin B differently or used squirts for delivery showed a reduction in inflammatory mucosal thickening on computed tomographic scanning, endoscopy, or both, as well as a reduction of intranasal makers of inflammation, when compared with placebo.

None of these recent developments are cited in the "state of the art" review. Instead, it is stated that "evidence supporting a role for fungi in CRS does not exist," citing only Dr Bush's own editorial as evidence. Either the authors were unaware of the emerging evidence for a role of certain molds or choose not to share it with the readers, either of which is unacceptable in a position paper that carries the weight and name of the American Academy of Allergy, Asthma and Immunology and indirectly the Journal.

To withhold crucial scientific information on the role of mold in CRS questions the intentions of the authors. Hopefully, the references cited, when read in reference to one another, will clarify the current "state of the art" and help to understand the role mold-induced inflammation plays in CRS.

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Adverse health effects of indoor mold exposure

To the Editor:

We read with interest your recent position paper on the controversial subject of adverse health effects of mold exposure.¹ When anyone writes a position paper, we question whose ox is being gored. You state in your article that "it is important for the members of the allergy-clinical immunology community who are frequently asked by patients, parents, and other interested parties to render opinions."¹ Who are these other interested parties? Was there a separate agenda for this position paper that also agrees with the American College of Occupational and Environmental Medicine's evidence-based statement on indoor molds?² Dr Andrew Saxon coauthored both these position papers.

In our review paper on the adverse health effects of indoor mold exposure,³ we cited 171 references in contrast to your 44 and the American College of Occupational and Environmental Medicine's 83. You state, "We will review the state of the science of mold-related diseases and provide interpretation as to what is and what is not supported by scientific evidence." What criteria did you use to decide this? Expert scientific testimony is admissible if it is reliable based on methods and procedures of science.

What evidence to the contrary do you have that you summarily dismiss the findings of Gray, Thrasher, Crago, Campbell, and Vojdani? Could you have selectively dismissed the many other references that we cited? What are the "unproved assertions that exposure to indoor molds caused a variety of ill-defined illnesses?" What, for example, is ill defined about neurotoxicity, a disorder ranking in the top 10 causes of occupational injury? Is PCR identification of *Stachybotrys* species and its mycotoxins not "specific" enough for "fungus-fungal products purported to cause the mold-related illness?"

If you condemn the "measurement of clinically useful tests of autoimmunity," as well as "a wide range of nonspecific immunologic parameters," how would you know whether there are adverse reactions to the immune system? The practice of occupational and environmental medicine requires the objective evaluation of biomarkers of exposures and biomarkers of the effect of exposures to establish causation. Your statement that "testing is expensive and does not provide useful information that will benefit in diagnosis, management, or both of disease and is to be discouraged" makes us suspicious of the message of this position paper.

We agree that mold exposure has become a litigious issue. But are we as physicians to choose sides? Or are we to evaluate objectively the alleged effects of toxic mold exposure? We suspect your interpretation of what is and what is not supported by scientific evidence might, at least in part, represent an agenda for the defense.

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How solid is the Academy position paper on mold exposure?

To the Editor:

I disagree with much of the position paper by Bush et al.¹ There is much evidence supporting illness caused by water-damaged, moldy, or damp indoor spaces. Classic allergy accounts for only part of the problem. I am allotted only 500 words, and therefore the numbered statements lead to important references (see this article's additional references in the Online Repository at www.jacionline.org).