Correspondence

Reply

To the Editor:

Hypersensitivity pneumonitis (HP) is diagnosed when a sufficiently intense exposure to a known or strongly suspected specific airborne allergen occurs in a patient with an appropriate immune response and relevant clinical expression. Given that residence-related HP cases are rarely reported and that exposure to molds in residential settings is universal, the prevalence of such events is likely to be extraordinarily rare, because the denominator is the entire population. Therefore, in the American Academy of Allergy, Asthma & Immunology (AAAAI) position paper,[1] we deliberately ended the section on HP with the statement, "Exposure to domestic specific indoor fungal spores is an extremely unlikely cause of HP, except in highly unusual circumstances, such as workplace exposure." In this respect, molds are unique among known domestic causes of HP, which are typically based on special environmental circumstances such as pigeon droppings, thermophilic actinomycetes, and so forth. We did not attempt to give proportional estimates of mold as a cause of HP or of mold-caused HP cases that are domestic.

Jacobs[2] strongly objects to our conclusion on the frequency of indoor molds causing HP, citing 4 references. Fink et al[3] report a case of HP from a *Micropolyspora* species, which is a filamentous bacterium, not a mold. Patterson et al[4] cite two cases of HP in which *Cephalosporium* was the allergen, and a single case from multiple fungi contaminating a humidifier.[5] Jacobs et al[6] concluded that environmental factors could be responsible for many idiopathic inflammatory and fibrotic lung diseases, suggesting that a diagnosis of specific HP would be more appropriate. Thus, none of these publications address the issue of prevalence of indoor mold exposure causing HP, which we consider to be extremely unlikely because exposure must be sufficiently intense to produce disease in a patient with a specific immune response to that particular mold antigen. The 2 studies published since our report cited by Jacobs include 2 siblings with HP caused by a mold in 1 household[7] and a case series confirming our statement that the majority of HP cases are occupational.[8]

Therefore, we feel that we did accurately describe the likelihood of indoor mold exposure causing HP and that our statement should in no way be "annoying" to authors, journal editors, researchers, clinicians, or anyone else. On the contrary, we had every intention of remaining evidence-based, thus satisfying the requirements of AAAAI Position Statements.[9-16] As for the "extensive criticism" Dr Jacobs[2] mentions, the majority of those whose opinions were published[17] were non-Academy members who in fact had served as experts in mold litigation (not in every case declared).

Finally, the correspondence by Jacobs[2] brings up the topic of disclosure of interest in publications. This is important for all AAAAI activities, especially where the Academy takes a published position. Each author of "The medical effects of mold exposure" submitted a disclosure statement before publication as a required condition of authorship. The Journal inadvertently failed to include it in the final printing but corrected the error in a subsequent issue.[18]

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