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Adverse health effects of indoor mold exposure

Allan Lieberman, MD[a], William Rea, MD[b], Luke Curtis, MS, CIH[c]

Article Outline

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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[a] From the Center for Environmental and Occupational Medicine, North Charleston, SC

[b] Environmental Health Center, Dallas, Tex

[c] Norwegian American Hospital, Chicago, Ill

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Position paper on molds is seriously flawed

Vincent A. Marinkovich, MD

Article Outline

To the Editor:

As a longtime member of the Academy, I was shocked and disappointed by the position paper printed in the February issue of the Journal.[1] A number of criticisms come quickly to mind:

1. At least 2 of the authors earn a substantial income testifying against patients in mold-related litigation. The potential conflict of interest is not addressed.

2. This is not a position paper generated from free and open discussion among Academy members. It is a one-sided opinion paper.

3. The authors seem to be ignoring one of the basic tenets of allergy: when symptoms appear after an exposure and abate on its cessation, chances are the patient is reacting to something in that exposure. Before we label her a hypochondriac, let us explore the details. Perhaps we can learn.

4. The authors draw conclusions about the health effects of indoor mold exposure for which they offer no positive support from the literature. The lack of evidence is not evidence against.

5. The authors have selected from the literature articles that, however tenuously, support their opinions and ignore the mountain of evidence that refutes their conclusions.[2], [3]

6. Two peer-reviewed literature references that do not support the authors' conclusions are cited and rejected as “poor quality” without discussion.[4], [5]

7. The authors' review of the literature involving the presence of mold-specific IgG antibodies reflecting the patients' exposure to mold is completely distorted. They seem to suggest that the measurement of mold-specific IgG antibodies cannot be a useful clinical parameter in diagnosing and monitoring the progress of patients with mold-related illness.

8. The conclusion that mycotoxins are not proteins and therefore mycotoxin antibodies are not possible ignores the enormous literature on penicillin reactions (a mycotoxin). One of the articles cited by the authors specifically identifies IgG antibodies against mycotoxins but is given no value in reading their conclusion.[6]

9. No reference is made to the very important work done by the group headed by Dr Sherris, formerly of the Mayo Clinic, now at the University of Buffalo, in which mold-specific IgG antibodies are identified as markers of chronic rhinosinusitis, and no difference between patients and control subjects is seen with IgE antibodies.[7]

I am astounded that the Academy would take such a blatant stand against the best interest of patients and 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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From private practice, Redwood City, Calif

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The role of airborne mold in chronic rhinosinusitis

Jens U. Ponikau, MD, David A. Sherris, MD

Article Outline

To the Editor:

Bush et al[1] 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], [4], [5], [6], [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], [9], [10], [11], [12], [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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From the Department of Clinical Otorhinolaryngology, University at Buffalo, The State University of New York, Buffalo, NY

Disclosure of potential conflict of interest: J. U. Ponikau and D. A. Sherris are employees of the Mayo Foundation, which has a license agreement with Accentia Pharmaceutical, Inc, for methods and materials for treating and preventing inflammation of mucosal tissue, and receive royalties from the Mayo Foundation. J. U. Ponikau testified in a mold litigation case involving a patient's diagnosis; any proceeds from this testimony were donated to charity.

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Respirable trichothecene mycotoxins can be demonstrated in the air of *Stachybotrys chartarum*–contaminated buildings

David C. Straus, PhD, Stephen C. Wilson, PhD

Article Outline

To the Editor:

This correspondence is in response to the American Academy of Allergy, Asthma and Immunology position paper recently published in the Journal and entitled "The medical effects of mold exposure."[1] The authors imply that the most important way that trichothecene mycotoxins could get into the human body is via the inhalation of *Stachybotrys chartarum* (SC) conidia. We have recently shown that the number of SC conidia in the air in a SC-infested building is not a good predictor for the amount of macrocyclic trichothecene mycotoxins (MTMs) in the air. This is because the MTMs can exist in the air on fungal fragments free of conidia, so the number of SC conidia found in the air should play only a small role in determining airborne MTM levels.[2] This becomes very important because it has recently been shown that there are 514 times more SC fungal fragments released by this organism than there are SC conidia released.[3] The authors also imply that the idea that the presence of mycotoxins in a building should give rise to an array of nonspecific complaints is "not consistent with what is known to occur when a toxic dose is achieved." This simply is not the case. Indeed, in a report examining the introduction of this type of mycotoxin (a trichothecene) into human beings, the opposite was observed. We know what kinds of symptoms are observed when a simple trichothecene (a preparation of diacetoxyscirpenol, also known as anguidine) is injected into humans. They are (among others) nausea, vomiting, low blood pressure, drowsiness, ataxia, and mental confusion.[4] These symptoms are consistent with those reported by individuals in SC-infested buildings.[5] The authors also state, "…however, potential levels of mycotoxins in nonagricultural air samples are too low to be measured practically with this technology." That may be true regarding the discussed technology; however, we have measured MTMs in the air of nonagricultural buildings.[6] Finally, the authors stated, "Testing for airborne mycotoxins in nonagricultural environments cannot be used to diagnose mold exposure." This is not the case. We have successfully preformed airborne testing for MTMs in nonagricultural settings.[6] In fact, we have used an ELISA to measure MTMs in the serum of individuals from SC-infested buildings.[7]

In conclusion, we feel that the following statements are true. SC has been shown to grow in buildings where people are having health problems.[5] SC definitely produces MTMs in these situations.[6] These MTMs definitely get into the air in these buildings, where they can be inhaled.[6] They definitely are inhaled by people in these buildings.[7] The following, then, is the final question that remains to be answered: do the MTMs get into human beings in concentrations sufficient to cause the health problems observed in people in SC-contaminated buildings?

We found a number of other issues of contention in the authors' review. However, because of space restrictions, we have limited our response to these points.

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From the Texas Tech University Health Sciences Center, Lubbock, Tex

Disclosure of potential conflict of interest: D. C. Straus and S. C. Wilson state that they have received research support from Assured Indoor Air Quality and served as expert witnesses in mold litigation and as experts for the plaintiff in legal cases associated with mold exposure and the alleged risks or injuries.

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How solid is the Academy position paper on mold exposure?

Maurice H.V. Strickland, MD, FAAAAI

Article Outline

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).

1. Mycotoxin[2], [3], [4] and allergen[5], AppendixSupplementary data have been documented in small fragments released by mold growth indoors. These particles are respirable and for *Stachybotrys* species can exceed spore counts by 500 times and spore deposition by 250 times in the respiratory tract.[E4] These particles are unmeasured and uncharacterized in indoor evaluations[E2,E3,E5,E6] and are a vehicle for mycotoxin and allergen entry into the body.

2. Personal monitoring and the determination of specific IgE sensitization to the individual's own environment through dual immunoassay has not been used routinely in exposure studies.[E3] Personal monitoring should be preferred to area sampling alone.[E7,E8]

3. Germinating spores release more allergen than dormant spores in 8 of 11 molds studied, which is important in environments with active mold growth or when spores germinate or colonize the respiratory tract.[E9,E10]

4. Persons with environmental exposure to *Stachybotrys* species have had measurable stachylysin[E11] and mycotoxin[E12] in their serum.

5. Inhaled mycotoxin is 10-fold more potent than ingested mycotoxin.[E13]

6. Common construction materials permit growth of toxigenic fungi and mycotoxin production.[E14-E19]

7. Mycotoxin can cause local mucosal damage[E20-E22] independent of systemic toxicity. Some mycotoxins persist for many weeks in the body,[E20] possibly permitting chronic toxicity.

8. There are ciliostatic,[E23-E26] cytotoxic,[E25,E27] inflammatory,[E20,E28-E40] and mutagenic,[E41-E46] factors elaborated by fungi, even in the absence of mycotoxin production.[E20,E47-E49]

9. Water-damaged buildings have a distinct fungal ecology than outdoor molds[E50,E51] or non–water-damaged buildings. The human effects of these "water-indicator fungi"[E52] might have overlap with outdoor fungal diseases yet are distinct.[E53]

10. Molds have been shown to cause molecular mimicry and IgE- and T cell–mediated autoimmunity.[E54-E57]

11. Molds cause a variety of immune effects,[E58-E63] including diminishing T_H1 reactivity while not diminishing or even stimulating T_H2 reactivity.[E64-E67]

12. Volatile organic compounds (VOCs) from *Trichoderma virieae* have been shown to trigger histamine release from human pulmonary mast cells.[E68]

13. Preliminary studies (including studies for USC and Mt Sinai) have shown neuropsychologic aberrations in patients exposed to mold. These studies need replication.[E69-E74]

14. Mold has been found to reside in the upper[E59,E75-E81] and lower[E82] airways of many persons with chronic respiratory disease.

15. Fungal intracellular proteins[E83] and protease[E20,E83] and fungal surface protein[E84] have been shown to be allergenic.

16. *Alternaria* species sensitization predicts polysensitization to a variety of fungi, some that are not routinely tested.[E85]

17. Mold growth is accompanied by bacterial growth and potentially inflammatory bacterial products.[E3,E86,E87] Amoebae have been found in these environments.

New knowledge renders virtually every study of indoor mold exposure obsolete. I have mentioned some of the pieces of the puzzle that will have to be used to assemble the entire picture of indoor mold effects. New knowledge and new studies will solve this puzzle.

Appendix. Supplementary data

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From the Department of Internal Medicine, University of Kansas School of Medicine, Wichita, Kan

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Rigor, transparency, and disclosure needed in mold position paper

Ritchie C. Shoemaker, MD[a], Harriett Ammann, PhD[b], Richard Lipsey, PhD[c], Edward Montz, PhD[d]

Article Outline

To the Editor:

Position papers of the American Academy of Allergy, Asthma and Immunology (AAAAI) should meet high standards in 3 areas: rigorous evaluation of the scientific literature, transparency in the process for attaining “AAAAI position paper” status, and full disclosure of authors' potential conflicts of interest. Credibility cannot be attained without high standards in each area. Only credible evaluations of the state of the science and identification of gaps in knowledge can improve clinical care and research. We believe that the AAAAI position paper “The medical effects of mold exposure”[1] promotes continued and unproductive contention among stakeholders rather than credible advancement of the field.

The claim by the AAAAI position paper to “review the state of the science of mold-related disease” is questioned because many important studies are not considered, and those considered are often accepted or rejected without evidence-based discussion. Apparent statements of fact are not supported by any references, or are supported only by reference to an author's 1-page editorial,[2] an outdated article,[3] or an article with negative results,[4] while ignoring positive results from the same[5] and other authors. The claim that “studies do not conclusively prove” an association between outdoor or indoor mold exposure and allergic rhinitis is true only in the sense that the scientific method enables rejection, not proof, of hypotheses. The inability of many studies to reject the hypothesis that mold causes allergic rhinitis provided the weight of evidence for the Institute of Medicine's conclusions placed into the Congressional Record by the Centers for Disease Control and Prevention: “airborne fungal allergens were most often associated with … allergic rhinitis/conjunctivitis, allergic asthma, and hypersensitivity pneumonitis” (HP).[6] This testimony and many studies also oppose the authors' unreferenced contention that “exposure to domestic specific indoor fungal spores is an extremely unlikely cause of HP.” In addition to “specific indoor fungal spores,” the AAAAI position paper should consider the complex

mixtures of airborne fungi, mycotoxins, bacteria, endotoxins, antigens, LPSs, and volatile organic compounds observed in water-damaged buildings (WDBs) that show evidence of microbial amplification.[7] The conclusion that “data supporting the role of fungi in CRS [chronic rhinosinusitis] are lacking” cites only an author's editorial[2] while ignoring many supporting studies and a National Institutes of Health press release announcing discovery of a non-IgE–mediated immunologic mechanism for mold-induced CRS.[8] Two articles are cited without critical discussion in concluding that the literature on mold-induced immune system dysregulation “is of particularly poor quality.” Only one 12-year-old article is cited to support the conclusion that “measurement of serum cytokines” and other immunologic parameters “is not appropriate.”[3] Hundreds of studies indicate that many individual components of the complex mixtures in WDBs induce inflammation by stimulating proinflammatory cytokine production. The simultaneous convergence of components such as *Stachybotrys chartarum*, other fungi, their metabolites, and actinomycetes like *Streptomyces californicus* on this common mode of action causes synergistic cytokine production.[9] This evidence indicates that complex-mixture components interact in illness production. It is inappropriate, therefore, to conclude that “mycotoxin-mediated disease” is “highly unlikely at best” because the concentration of any single component is unlikely to reach a threshold level. Yet the position paper reiterates this conclusion originally reached by the American College of Occupational and Environmental Medicine (ACOEM) on the basis of a spore instillation study in rodents and indoor spore concentrations.[10] Neither the AAAAI nor the ACOEM papers discusses evidence indicating that concentrations of airborne mycotoxin-containing fungal fragments are orders of magnitude higher than spore concentrations,[11], [12] and neither paper applies standard risk assessment procedures to the calculations. Both cytokine-mediated and mycotoxin-induced illnesses are consistent with multiple-system symptoms,[5], [13] although the position paper states without reference that “the presence of mycotoxins … is not consistent with … a whole panoply of nonspecific complaints.” Multiple-system symptoms and objective indications of neurologic dysfunction and hormonal imbalances were carefully described in a prospective study of human exposure to WDBs, another study not considered in the position paper.[7] Rigorous, not cursory, reviews of the literature are needed to improve clinical care and design studies that can further describe the mechanistic pathways through which exposure to WDBs affects human health.

The AAAAI Web site states that position papers “contain an extensive bibliography” based on literature review, but the position paper contains only 44 references. The Web site further states that “because of their weight, Position Statements are created only after careful discussion and review” and a “consensus of experts.” The signatories and endorsers (see this article's Online Repository at www.jacionline.org), some of whom are AAAAI members, are unaware of any discussion or peer review. An original author of the paper withdrew his name because his contributions were rewritten to reach unsupported conclusions. The position paper apparently states the opinions of a few, rather than the “consensus of experts.”

Several medical journals recently retracted articles and implemented procedures to disclose authors' conflicts of interest because of postpublication revelations that impugned credibility. The conflicts of interest of position paper authors should be fully revealed, particularly consultant-related and litigation-related activities that invoke position papers, so that informed conclusions can be reached. The signatories of this letter have submitted conflict of interest statements to the AAAAI and thank the Academy for this opportunity to present an opposing viewpoint.

Appendix. Supplementary data

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[a] From the Center for Research on Biotoxin Associated Illnesses, Pocomoke, Md
[b] Olympia, Wash
[c] Lipsey and Associates, Jacksonville, Fla
[d] Indoor Air Solutions, Pottstown, Pa

Disclosure of potential conflict of interest: R. C. Shoemaker owns stock in ChronicNeuroToxins.com, and is an expert witness for plaintiff and defense litigation. H. Ammann has served as an expert witness in mold exposure cases for the state of Washington and has served as an expert witness in mold litigation for which she was compensated. R. Lipsey is employed by Lipsey and Assoc, Inc, and has served as an expert witness for plaintiff and defense litigation. E. Montz is employed by and owns stock in Indoor Air Solutions, and has served as an expert witness in plaintiff and defense litigation. K. Carstens is owner/moderator of a nonprofit support group that assists mold victims, and his wife is pursuing a workmen's compensation claim as a result of illnesses caused by mold exposure in the office environment. J. L. Wright is the director of the Fungal Disease Resource Center, Inc. The rest of the authors have declared that they have no conflict of interest.

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doi:10.1016/j.jaci.2006.07.018

Subj: **Policyholders of America's letter re: Mold position paper**
Date: 2/23/2006 7:36:52 AM Pacific Standard Time
From: MBallardAI
To: bkruger@aaaai.org
BCC: SNK 1955

Attached is a zip file (word documents) for distribution at the March AAAAI board meeting.

Under separate cover I have sent you some supporting material.

Thank you,

Melinda Ballard
President
Policyholders of America

Re: Position Paper by A. Terr, et al

Dear Mr. Kruger: I am one who usually does not write to editors and offer criticism, particularly of original research. However, I have read the position paper authored by Dr. A. Terr and others. I find their position and arguments to be entirely one sided. These authors seldom see patients exposed to molds. When they do, they always find some other mechanism for their illness and health problems. They even express their opinions for the defense (insurance companies) regarding their beliefs.

Their review of the literature is one sided and supports their point of view. A few examples are as follows:

1. They cite the paper of Salvaggio (usually a defense witness) regarding immune and biological markers. This paper was published in 1995. Thus, it does not cover any of the literature between the date of submission (probably 1994) to current). This is eleven or twelve years of thousands of papers dealing with toxic exposure and immune biomarkers.

2. They state that the only reliable testing for fungal (mold) exposure is IgE (skin, Rast, ELISA, etc.). They attempt to allude to the fact that IgG, IgM mold antibodies are meaningless. This again ignores many current and older research papers on the subject. For example, it has been demonstrated that IgG antibodies to molds is diagnostic of farmer's lung disease and also represents mold exposure in damp buildings. They ignore the more recent paper of Vojdani (Archives of Environmental Health) who has clearly demonstrated that if one uses affinity chromatography to purify mold antigens, then little (about 12 %) or no cross reactivity occur in the ELISA assay. Further, I have in my possession the CDC statement to Dr. Martin Belson regarding the CDC publication MMWR, 1/14/06, Vol. 54, No. RR-1, on mold serology. The CDC clearly states "We are not aware of any info that these tests are unreliable; however, we have no information that no individual laboratory's method has been validated either analytically or clinically in the general population. The MMWR says they are not recommended for definition of clinical illness. We do not comment on how someone may choose to use them for evaluating mold exposure. This document is specifically for case definitions, not for routine clinical use. Hope this is helpful." Terr et al also miss the Texas Medical Association, which in 2002 and the AAEM (2005) recommendations for IgG, IgM, IgA and IgE Mold serology. Thus, not all in the medical field endorse the AAAI position paper on this subject. Moreover, the MMWR in a later issue writes on the toxicology of trichothecenes. This writing is ended by the statement: "If you wish to have more information see the papers listed below." Needless to say one of the 4 listed articles is Vojdani, Thrasher et al, 2004 and is not, by the way, cited in the position paper.

3. In their discussion regarding other health effects of fungi, they attempt to gloss over the peer reviewed literature on the subject. For example they cite the papers of Campbell et al and Gray et al by stating they are not creditable, but then cite Salvaggio to support their position. No further explanation is given.

When it comes to neurological assessment and injury they totally miss the peer reviewed papers by Kilburn, Crago et al, and Rae et al, in 2004, Archives of Environmental Health. The paper by Crago et al is very interesting because it shows neurocognitive deficits and changes in the QEEG that have a dose response in mold exposed subjects. Do these papers also lack creditability?

4. They cite one paper by Roponen that shows no symptoms in workers exposed to molds as definitive proof of their position. However, they miss all of the papers by this author and his group as well as other Europeans that show that fungal exposure in damp buildings leads to the production of local (nasal) and systemic (blood) pro-inflammatory cytokines, and in particular TNF-alpha. TNF-alpha is probably the most toxic of the cytokines and can lead to apoptosis and tissue injury. This cytokine is increased in a dose response manner in workers exposed to mycotoxins who are symptomatic. These same workers also have a shift in their LDH

isoenzymes patterns favoring lungs and spleen. Finally, in traumatic injury to the CNS and PNS, TNF-alpha is elevated and believed to lead to apoptosis of neurons and inflammatory changes in neural tissues.

5. They have missed the paper published in Environmental Health Perspectives, 2005 by Cox-Ganser that has followed several hundred individuals in a single building exposed to molds. These authors clearly demonstrate that indoor mold leads to upper and lower respiratory illness that is not IgE mediated. The lower respiratory illness has all of the features of hypersensitivity pneumonitis, which means at least a type III immune response. In addition, there is a very recent paper by Bernstein et al on children and mold exposure. These authors clearly state that the children they observed have health problems beyond IgE mediated illness, but they do not know what the illness is. Dales, et al have also demonstrated chronic health effects in children with immune alterations being present.

6. Finally, they take the position that the ACOEM position paper that attempts to demonstrated that mold spore counts and mycotoxins concentrations inside of water-damaged buildings are not sufficiently concentrated to cause ill health effects. They fail to point out the following aspects of this position paper, confirmed in a deposition of Dr. Kelman (one of the authors) as follows:

a. Rao's paper that is relied upon was done on rats and used an unknown strain of Stachybotrys isolated from a building. Thus, it is not known what the concentration or what mycotoxins these spores had because the strain of Stachybotrys is not known. Further, Rao used rats that are the least sensitive animal to mold/mycotoxins exposure. Other authors, e.g. Nikulin, Rand, etc. have clearly demonstrated that mice are more sensitive. Furthermore, the EPA recommends the use of the most sensitive animal model in risk assessment and then uses the denominator of 10 to apply to adult humans and another 10 to the very young. None of this was done in this position paper.

b. The ACOEM position paper does not include the fact that fine particulate matter (less than 2 microns) is shed by molds in the indoor air. The fine particulate matter is some 300 times more concentrated than are the spores (Gorny) and contains mycotoxins (Brasel et al, 2005). Thus, the calculations in the position paper are absurd at best. Furthermore, they use numbers from the FDA regarding mycotoxins concentrations and exposure limits in foods. What does this have to do with inhalation? Last of all, they do not cite the paper by Brasel et al, 2005 which demonstrates the presence of trichothecenes in the blood of symptomatic individuals following mold exposure. Nor do the reference the paper by von Emon et al who demonstrated the presence of Stachylysin in the blood of symptomatic adults. The only way this could occur is via inhalation of particulate matter, especially the fine particulate fraction.

c. Dr. Kelman was deposed by an attorney, Richard Langerman regarding the ACOEM position paper. It is noted in the deposition that Dr. Kelman's business received $40,000 for his participation in the layman's re-write of this paper and that there are serious questions regarding the appropriateness of the peer reviewed process used in reviewing this paper. Apparently, the authors may have done their own reviewing and the position paper apparently was not sent out to members of the ACOEM who have had research and clinical experience in mold exposure.

7. Finally, the authors take the position of the IOM monograph. It must be pointed out that this monograph did not review the literature published after their closing date in late 2002. Thus, the IOM publication does not include the medical/scientific literature published since the date of closing. The authors of the IOM monograph are careful to point this out and the contributors clearly state that at the time of their review there was insufficient information to draw definitive conclusions on the subject of mold exposure and systemic health effects.

8. I am also very familiar with the all of the papers published on fungal rhinosinusitis. Why does Bush et al only cite Bush's one page editorial position on this subject? Has he not read the host of information available on this subject and probable colonization of sinuses by molds? Further,

has he not read the papers on Aspergillus colonization/infection and the presence of gliotoxin in the blood of affected individuals? Furthermore, the work of Ponkinau using special stains for chitin, demonstrate clearly new fungal growth in sinuses, which supports their observations on fungal rhinosinusitis.

It appears to me that these authors are attempting to put out their position on the mold issue and make it look like their position is endorsed by the AAAAI so that their position on the witness stand will have some form of validity. Already, their position with respect to the ACOEM position paper has been undermined by Kelman's own testimony under oath. I truly pray that the AAAAI will not allow itself to become part of this political deception that has been ongoing and truly involves major insurance companies in the U.S and probably world wide.

Jack D. Thrasher, Ph.D.
Toxicologist/Immunotoxicologist
Director of Research
Medical Center for Immune and Toxic Disorders
Spring, Texas 77386

Subj: **revised and resent**
Date: 3/1/2006 7:15:15 AM Pacific Standard Time
From: SBRINCHMAN
To: SNK 1955

Dear Mr. Kruger,
I have found one error (sound has been replaced with good in paragraph 2) and **wish this letter of concern to replace the last**. I wrote it late at night, I apologize.
S. Brinchman

H. Bruce Kruger
Managing Director
Practice and Policy
AAAAI
February 28, 2006

Dear Mr. Kruger,

As the Executive Director and Founder of The Center for School Mold Help, I would like to say, after reviewing the position paper, The medical effects of mold exposure, by Bush, Portnoy, Saxon, Terr, and Wood, that the paper may ultimately discredit the AAAAI, as well as all of its authors, because it appears to be so politically biased.

The paper appears to reflect what has been called the **"Orwellian concept of 'sound science,'** which is clearly understood by the scientific community to mean **the misrepresentation of scientific data to reflect ... political and social agendas." (Schubert, UT '04)** I would also add, private industry and financial agendas. It is a fact that many industries and lobbyists have suppressed the science that does exist on mold - and this paper, which is not based on good science, has no basis in reality. I am reminded of the fight the tobacco industry has put up to deny harm from its products.

The immense suffering of the American people in our dilapidated and often very damp schools (20% of Americans occupy schools each weekday, and 50% have indoor air problems, with more than 50% having moisture problems) is magnified by their inability to obtain proper medical assistance for resulting illnesses. Physicians may be misled by papers such as this one, that purport to reflect an adequate summation of the state of scientific knowledge on a health topic, and may then misdiagnose or ignore serious mold or damp-building related illnesses. This is often the case due to one other similar paper, and to add this one to your association's archives is to further confound the truth.

This position paper is no "impartial search for understanding", as Schubert describes it in his article about the turning of a deaf ear to reality. In fact, the very name "position paper" indicates bias exists.

The reality is that mold, indeed, makes people ill - many of the types that grow in buildings produce potent toxins - and this is well documented. School buildings are the most neglected of all the gov't buildings - and the gov't buildings are the most neglected of the commercial buildings. Estimates for damp schools range from 10-50% and above. Schools allow leaks and flooding to occur for decades, without intervention. Stachybotrys is common in these leaky schools. The attack of molds, bacteria and other agents in damp buildings profoundly and visibly impacts the occupants, much as AIDS impacts its unfortunate victims. Just today, a press release from Michigan State University described a study that showed how a toxin produced by stachybotrys kills nerve cells in the nasal passages and brains of mice. Please visit our Research page to read more studies like these.

Our children and school staff deserve to have impartial and fair, respectful treatment. These comprise our next generation and their teachers. If we turn our backs on them and their illnesses from the plethora of damp government school buildings, what will we have? What future can America offer when its population increasingly becomes sickened and our youth are not only exposed to increasingly more dangerous building environments in our schools, but cannot receive a diagnosis or treatment due to lack of recognition of the problem? The most important thing one can do is get out of the exposure. How can this occur when physicians wrongly are led to believe mold is only harmful to the immuno-compromised?

Consider the statement from the The California Air Resource Board (CARB): *Indoor Mold: A General Guide to Health Effects, Prevention, and Remediation. Jan. 2006:*

*"*What seems inarguable is that at least some mold-produced toxins can be very dangerous, as this passage from the Textbook of Military Medicine suggests:

[T]richothecene mycotoxins are proven lethal agents in warfare. Symptoms include vomiting, pain, weakness, dizziness, ataxia, anorexia, diarrhea, bleeding, skin redness, blistering, and gangrene, as well as shock and rapid death.34

It appears reasonable to conclude that there is a potential risk to humans from toxic effects of inhalation of mold spores and other mold by-products, including fragments and dust that may have adsorbed mycotoxins (taken them on the surface). The level of risk would depend on the amount of the exposure and on individuals' susceptibility. Highly contaminated environments and long exposures increase risk. Lesser exposures might have minor or transient effects or effects too small to draw notice. Individual genetic factors, prior or concurrent illnesses, age, weight, and other risk factors affect risks presented by an environment containing mycotoxins." (CA EPA, Indoor Mold, Jan.'06, p.18)

Considering school-aged children and their school staff, including teachers, principals, custodians, secretaries, clerks, aides, and volunteers are becoming sickened in shocking manner, very much like the above description, in our damp schools in great numbers, it would be a great disservice to America's youth, their families and loved ones, and our society as a whole, to deny them help. It is a profound shame that our government has not admitted this problem more acutely. Our medical associations must support health, not deny or block it.

As a victim of mold in schools, and as a representative of those crying out for help and medical understanding in our nation's schools, I ask you and your colleagues not to publish this paper, which does not reflect scientific or social reality.

Sincerely,

Susan Brinchman
Founder and Executive Director,
The Center for School Mold Help
P.O. Box 3422
La Mesa, CA 91944-3422
www.schoolmoldhelp.org

THE JOURNAL OF Allergy AND Clinical Immunology

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Adverse reactions to fungal metabolic products in mold-contaminated areas

Gerald B. Goldstein, MD

Article Outline

To the Editor:

I wish to commend the authors on their well-written position paper entitled, “The medical effects of mold exposure,”[1] which appeared in the February 2006 issue of the Journal. Specifically I wish to address the portion dealing with toxic effects of mold exposure. Mycotoxins, cell wall glucans, and mold volatile organic compounds are discussed, and rightfully the authors express the difficulty in measuring the concentrations of these mold products. In addition, they imply the difficulties in defining the intricacies of the aerobiologic pathway leading from the mold source, to aerosolization and exposure, and finally to tissue deposition and potential tissue damage. My only reservation concerning this portion of the paper is that potential toxic effects of mold exposure are considerably downplayed by phrases such as “transient symptoms-signs,” “its occurrence is improbable,” and so forth.

A few years ago I had the opportunity to take histories from 93 former residents of an apartment complex with chronic visible mold contamination from recurrent water leaks. In most cases, the indoor mold spore counts were considerably higher than the comparable outside measurements. The residents' complaints were multiple and varied in severity, but most commonly elicited were cough (49%), rhinitis (44%), wheeze (31%), and headache (41%). With the exception of the latter, these symptoms were determined in the 2004 Institute of Medicine report[2] to have an association with living and working in mold-contaminated environments.

There are several carefully performed clinical studies in the environmental medicine literature documenting significant respiratory disease in subjects exposed to fungal contamination in schools,[3] office buildings,[4] a courthouse,[5] and homes.[6], [7] In many instances, *Stachybotrys chartarum* was isolated, but in other studies, *Alternaria*, *Aspergillus*, *Penicillium*, *Cladosporium*, and *Zygomycetes* were implicated.

As noted in the position paper, fungal metabolic products and cell wall components are indeed difficult to measure. However, *Stachybotrys chartarum* macrocyclic trichothecene mycotoxins have been measured by ELISA in contaminated areas in amounts greater than 1300 pg/m^3.[8] The same toxins have been detected in blood[9] and urine[10] of exposed persons.

These citations lead me to believe that there is sufficient evidence that the concept of mold toxicity is real and that it should not be downplayed as a potential public health problem. We as allergists can help by collaborating with environmental health specialists and medical toxicologists in further elucidating this subject.

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From Allergy, Asthma Associates, P.C., Tucson, Ariz

Disclosure of potential conflict of interest: G. B. Goldstein was an expert witness for a plaintiff in a mold exposure case.

PII: S0091-6749(06)01383-2

doi:10.1016/j.jaci.2006.06.030

Subj: **Re: Position paper**
Date: 2/27/2006 7:02:50 AM Pacific Standard Time
From: moneynp@muohio.edu
To: SNK1955@aol.com

Sharon, I have read the position paper and offer the attached brief commentary in the form of a memo. Best wishes, Nik.

Nicholas P. Money, Ph.D.
Professor
Department of Botany
Miami University
Oxford, OH 45056, USA

Phone: (513) 529-2140
Fax: (513) 529-4243

Subj: **Mold Induced Disease Position Paper**
Date: 2/21/2006 2:07:40 PM Pacific Standard Time
From: DSherris@KaleidaHealth.Org
To: bkruger@aaaai.org
CC: SNK1955@aol.com, jponikau@buffalo.edu

Dear Mr. Kruger,

I was sent a copy of the position paper on mold induced disease that is about to be published in JACI from Sharon Kramer and asked to comment from my perspective, as an expert on mold induced chronic rhinosinusitis. My expertise arises from the fact that I am one of the principle investigators on the initial research from the Mayo Clinic, and on subsequent research from University at Buffalo, where I am Professor and Chairman of Otolaryngology, and continue the research with my Co-investigator, Dr. Jens Ponikau, who joined me here from the Mayo Clinic. I gave testimony on the subject at a Senate Staff Briefing to the Health, Education, Labor and Pensions Committee at the request of Senator Kennedy's office on January 12th. At the meeting, we were working to clear up the exact type of confusion that the Bush article promises to propagate.

The fact that Dr. Bush has only sighted his one page editorial on chronic sinusitis and its link to fungi in this review article and not our countless research papers on the subject indicates his bias on the subject, or lack of understanding, or both. I am including just some of the various articles we have published, starting in 1999 (many of which were published in JACI, and 2 of which received editor's choice citations). The NIH put out a press release praising the fact that it appeared we had discovered a new immunologic mechanism implicated in chronic rhinosinusitis induced by mold, and alternaria in particular.

Dr. Bush also has confused the diagnostic criteria for an entity originally called "Allergic Fungal Sinusitis (AFS)". Some authors have required IgE mediated disease to "anything" as a diagnostic criteria, while other authors have delineated IgE mediated disease to the specific fungi cultured as a diagnostic criteria, while finally other authors do not require IgE mediated disease presence to make the diagnosis. This last group is where my coauthors and I sit for a variety of reasons. First, our initial paper in Mayo Clinic Proceedings demonstrated that all of the other criteria for disease that Dr. Bush cited were present in 96% of consecutive patients who were diagnosed with the general disease chronic rhinosinusitis (CRS). The fact that fungus was present in both patients and normal controls was true. But, the IMPORTANT differentiating factor that Dr. Bush failed to recognize was that the eosinophilic mucin, which is a diagnostic criteria for AFS, was present in 96% of all chronic rhinosinusitis patients, but in no healthy controls. The presence of the eosinophils and breakdown products of the eosinophils is the differentiating factor between those with disease and those without disease.

Our subsequent papers demonstrated the T-cell response that fungi, and alternaria in particular, induce in patients with CRS, but not in healthy controls. This T-cell response activates the eosinophils that clump on and attack the fungi in the mucus of diseased patients, but not in normal people. There, the eosinophils dump their toxic proteins, and destroy the fungi, but also damage the intranasal membranes, leading to the epithilial erosion seen in CRS. We have recently had FDA approval for a diagnostic test for chronic rhinosinusitis approved and had it launched (IMMCO Diagnostics, Williamsville, NY) based on our data. It is a test based on the presence or absence of major basic protein (MBP) in the nasal mucus. We have demonstrated its presence in people with CRS, and its absence in healthy controls. It is the first specific test for CRS available to the public. Dr. Bush never mentioned the test in the section entitled "Laboratory Assessment".

We have also demonstrated in both an open and double blinded clinical trial that Amphotericin-B applied intranasally improves the inflammation of the sinuses in CRS sufferers (both of which were published in the JACI). Other authors have demonstrated the same. We are presently working on multi-centered clinical trials through the FDA to provide the first drug that would be FDA approved to treat this common entity.

Finally, to get back to that IgE mediated issue--- in every paper we published we looked separately at whether the responses immunologically, or the inflammation histologically, or the response to therapy was different in those with IgE mediated allergy or in those without IgE mediated allergy. Our answer was always NO. There is no evidence that IgE plays any role in chronic rhinosinusitis. If it did, one would expect that antihistamines, or other allergy treatments would improve chronic rhinosisusitis, and there would be an FDA approved product for the 32 million adult sufferers in the US (per the CDC). Unfortunately, there is not.

Dr. Bush's paper will continue to confuse the physicians and patients out there. His statement that "the data supporting the role of fungi in CRS are lacking at this time" is patently incorrect. Maybe he did not read all of the

papers, so it is lacking in his mind, or maybe he is forwarding a personal agenda or bias that I am unclear of. Either way, I hoped that the JACI, and the AAAAI in general propagated information based on facts and scientific information, not on personal biases and unsupported statements. I do not know if the paper's publication can be delayed or stopped, but I do believe if it is published in its present form it will contain unsupported claims and conclusions and be called a "Position Paper", thereby carrying the weight and name of the AAAAI with it. I do believe with appropriate editing, and with input from experts in the field a decent paper could be generated.

By the way, Dr. Ponikau, my coauthor, will be presenting a workshop at the AAAAI meeting in Miami explaining the connection of CRS and Fungi, and I will be presenting a workshop on Endoscopic Sinus Surgery, both at the AAAAI request. Maybe Dr. Bush, or you would like to come to get educated on the subject, like the participants will.

If I can provide any further information, or the opportunity to write an editorial on the paper if its publication in its present form is inevitable, please feel free to contact me.

Yours,

David A. Sherris, MD
Professor and Chair
Department of Otolaryngology
University at Buffalo

dsherris@buffalo.edu
(716) 887-5101

Subj: **RE: Mold Induced Disease Position Paper**
Date: 2/23/2006 3:30:16 PM Pacific Standard Time
From: jponikau@buffalo.edu
CC: SNK1955@aol.com, sawhneyra@od.nih.gov

Dear Bruce,

I can only second what David Sherris has eluded to.
First, please allow me to state that there is obviously a certain bias in myself since I am directly involved in the research on the role of fungi in CRS.
As an ENT, I have always seen the AAAAI as an organization which has promoted the science of allergy (both IgE and non-IgE mediated disorders), hypersensitivities and immunological disorders, not being married to only one mechanism. In addition, I believe the AAAAI as well as the JACI are where they are today because they welcome novel approaches to unsolved problems and present a balanced and objective view their members/readers. For that reason, the JACI has become my top choice to publish original articles in, and the AAAAI meeting has been my "go to" meeting as invited faculty for the last 5 years. I was on the rhinosinusitis task force by the ENT-Academy, although I withdrew my name from their position paper in 2003 because I felt that their review was bias towards an infectious cause and a surgical approach for chronic rhinosinusitis (CRS). I participated in the rhinosinusitis definition effort in March 2005 by the AAAAI which was initiated by Dan Hamilos and Eli Meltzer. I have been actively involved in explaining specifically ENT physicians the non-infectious nature of CRS and I hope to have helped shifting the CRS paradigm from "bacterial infectious" towards a immunological (hypersensitive) background to better recognize the underlying eosinophilic inflammation.

Having said all this, I was deeply disappointed by the "state of the art" review which is written up as a position paper on the effects of molds. It is one thing to have a biased review by certain individuals published, but as a position paper representing the AAAAI it does become a political issue. Not only recognizes the review only IgE mediated inflammation as contributing factors and thus and thus disfavors the increasing knowledge of non-IGE mediated hypersensitivities, but also withholds important and exciting information which support a role for fungi in CRS. They are as follows:

. CRS is strongly associated with an eosinophilic inflammation.
. Patients as well as healthy controls nasal and sinus secretions are colonized with fungi.
. The immune system (lymphocytes) in patients, but not healthy controls, react to certain fungi with the production of cytokines, which elicit the eosinophilic inflammation.
. This abnormal immune response occurred regardless from the allergy status of the patient.
. The same fungi induced the degranulation of eosinophils.
. The degranulation activity is induced by a 60 kDa antigen from Alternaria alternata, is highly heat labile, and works protease dependant through a G protein-coupled receptor
. Eosinophils in CRS patients migrate into the mucus and target fungi in vivo.
. During that attack, eosinophils release toxic proteins onto the fungi, which also erodes the epithelium and explains the secondary bacterial infections.
. This attack is reproducible in vitro, but dependant on a signal from CRS patients' PBMCs. Healthy control PBMCs lack this signal.
. Intranasal antifungal medication reduce the patients symptoms, the

inflammatory thickening of the mucosa and the eosinophilic inflammation.

The following references support the above, none of which have been mentioned in the article:

Ponikau JU, Sherris DA, Kephart GM, Kern EB, Gaffey TA, Tarara JE, Kita H. Features of Airway Remodeling and Eosinophilic Inflammation in Chronic Rhinosinusitis: Is it the Histopathology Similar to Asthma? J Allergy Clin Immunol. 2003; 112(6):877-882

Ponikau JU, Sherris DA, Kephart GM, Kern EB, Congdon DJ, Adolphson CR, Springett MJ, Gleich GJ, Kita H.Striking deposition of toxic eosinophil major basic protein in mucus: implications for chronic rhinosinusitis. J Allergy Clin Immunol. 2005 Aug;116(2):362-9.

Kita, H., Adolphson, C.R., Gleich, G.J., Chapter 19, Biology of Eosinophils, in Middleton's Allergy Principles & Practice, Sixth Edition, ed. N.F. Adkinson, Jr, B.S. Bochner, J.W. Yunginger, S. T. Holgate, W. W. Busse and F. E. R. Simons, Mosby, Philadelphia PA, 2003, pp 305-332

Sasama J, Sherris DA, Shin SH, Kephart GM, Kern EB, Ponikau JU. New paradigm for the roles of fungi and eosinophils in chronic rhinosinusitis. Curr Opin Otolaryngol Head Neck Surg. 2005 Feb;13(1):2-8.

Ponikau JU, Sherris DA, Kern EB, Homberger HA, Frigas E, Gaffey TA, et al. The diagnosis and incidence of allergic fungal sinusitis. Mayo Clin Proc. 1999; 74(9):877-84.

Braun H, Buzina W, Freudenschuss K, Beham A, Stammberger H. 'Eosinophilic fungal rhinosinusitis': a common disorder in Europe? Laryngoscope 2003; 113(2):264-9.

Lackner A, Freudenschuss K, Buzina W, Stammberger H, Panzitt T, Schosteritsch S, Braun H. Fungi: a normal content of human nasal mucus. Am J Rhinol. 2005 Mar-Apr;19(2):125-9.

Taylor MJ, Ponikau JU, Sherris DA, Kern EB, Gaffey TA, Kephart G, Kita H. Detection of fungal organisms in eosinophilic mucin using a fluorescein-labeled chitin-specific binding protein. Otolaryngol Head Neck Surg. 2002 Nov; 127(5):377-83.

Gosepath J, Brieger J, Vlachtsis K, Mann WJ. Fungal DNA is present in tissue specimens of patients with chronic rhinosinusitis. Am J Rhinol. 2004; 18(1):9-13.

* This study demonstrates the existence of fungal DNA in polypoid nasal tissues of 100% of patients with CRS. Furthermore, PCR screening specifically for Alternaria tested positive in 100% of the CRS patients, whereas none of the healthy control samples tested positive for Alternaria DNA.

Shin S-H, Ponikau JU, Sherris DA, Congdon D, Frigas E, Homburger HA, Swanson MC, Gleich GJ, Kita H. Rhinosinusitis: An enhanced immune response to ubiquitous airborne fungi. J Allergy Clin Immunol, 2004;114:1369-75.

** This study demonstrates that CRS patients have exaggerated humoral and cellular responses, both TH1 and TH2 types, to common airborne fungi, particularly Alternaria, linking them to the eosinophilic inflammation

Ponikau JU, Sherris DA, Weaver A, Kita H. Treatment of chronic

rhinosinusitis with intranasal amphotericin B: A randomized, Placebo-controlled, double-blinded pilot trial. J Allergy Clin Immunol. 2005 Jan; 115(1):125-31

Inoue Y, Matsuwaki Y, Shin S-H, Ponikau JU, Kita H
Non-pathogenic, environmental fungi induce activation and degranulation of human eosinophils
J Immunol 2005 Oct; 175: 5439-5447

Krouse JH, Shah AG, Kerswill K.
Skin testing in predicting response to nasal provocation with alternaria. Laryngoscope. 2004 Aug;114(8):1389-93.

Ponikau JU, Sherris DA, Kita H, Kern EB. Intranasal antifungal treatment in 51 patients with chronic rhinosinusitis. J Allergy Clin Immunol. 2002; 110(6):862-6.

Ricchetti A, Landis BN, Maffioli A, et al. Effect of anti-fungal nasal lavage with amphotericin B on nasal polyposis. J Laryngol Otol. 2002; 116(4):261-3.

The authors conclude that "...data to support a role for fungi in CRS is lacking at this time.". The only reference they use as an argument against it is Dr. Bush's own editorial on the subject. This brings up the thought in me that either the authors are not updated on the current literature, or have chosen not to share it with the readers, both of which is unacceptable in a position paper. I have enclosed a current review presenting the current knowledge about the role of fungi in CRS. To withhold crucial and exciting new peer reviewed and published information about a non-IgE mediated role of fungi in CRS to the audience to provide a more balanced view threatens the integrity of the JACI and the AAAAI as a science driven organization. I am deeply saddened to see this happen.

Sincerely yours

Jens Ponikau, M.D.
Assistant Professor of Clinical Otorhinolaryngology
University at Buffalo
The State University of New York
3 Gates Circle
3c41 Millard Fillmore Hospital
Buffalo, NY 14209
Phone: 716 (887-5101)
Fax: 716 (887-5073)
e-mail: jponikau@buffalo.edu

Subj: **Fwd: AAAAI Position Paper on Mold**
Date: 2/22/2006 11:44:07 AM Pacific Standard Time
From: RHaynes668
To: SNK 1955

Forwarded Message:

Subj: **Re: AAAAI Position Paper on Mold**
Date: 2/22/2006 7:14:50 AM Pacific Standard Time
From: bkruger@aaaai.org
To: RHaynes668@aol.com
Sent from the Internet (Details)

Thank you for forwarding your comments. I will pass them on to our Board.
Bruce

>>> <RHaynes668@aol.com> 02/22/06 08:31AM >>>
Mr. Kruger,

Sharon Kramer passed the position paper on to me this week and, as a mold victim and victim's advocate, I'd like to take a minute to speak to you from that perspective.

My family built a new house several years ago when my boys were ages two and four. They had developed normally to that point and when we moved into the house, their development came to a screeching halt and even regressed in many ways right before my eyes. Both were in special education classrooms last year. My youngest will remain in one for some time. His symptoms look like autism. They've suffered more than I can convey. If I hadn't seen it myself, I would never have believed what happened to them.

When we won our lawsuit, our story was covered by local television and newspapers and is can be found on the web. I was BARRAGED with phonecalls. They were so sad and so frequent at first that I couldn't answer my phone some days. I continue to receive calls one year later. I have lost COUNT of the number of people who have cried over the phone to me as they tell me their stories.

One woman in Texas gave birth to twins while in her moldy house and one baby died of respiratory problems. Another woman in Washington state living in a camper behind her moldy house finally took her own life she was so depressed by her ruined health and hopeless financial situation. A woman in California had five different abdominal surgeries before being properly diagnosed and treated by one of the handful of doctors who understand this. MANY of the people who call me have children near the ages of mine displaying the exact same neurological symptoms. Frequently I've crossed paths with people in other settings, our furnace repairman for example, who is knowingly living in a moldy environment and had no idea it was connected to his son's problems at school.

These people are physically and financially devastated and the vast majority have no recourse. They can't live in their homes, they are sick and many can't work, they are unable to afford an attorney. Their children are sick and their physicians are doing the worst possible things to treat them because the don't have accurate information about how mold effects children or how to treat it. PEOPLE DON'T KNOW because the truth is suppressed by very powerful people with a lot to lose. It's wrong that I, a layman, have more useful medical information for them than their own pediatrician.

I spent many nights, as my children slept, researching their health problems

and the very limited resources for treatment. I'm happy to report that my oldest, being treated by people who understand mold, has gone from being unable to hold a pencil or identify his letters and numbers at age six to the best reader in his 24-person classroom at age seven. THERE IS HELP, if people just receive the right information.

I implore you, don't make it so hard for people to find. If what I've witnessed firsthand is even a fraction of the suffering in this country, it is epidemic. I firmly believe the cost of mold UNTREATED is much higher than the cost would be if it were acknowledged and treated. But there is no time to waste.

Last week the Oregonian ran an article about autism rates in our state rising exponentially. They suspect environmental causes. I know in our case, our self-composting house was the cause. I watched it happen. Oregon is obligated to educate all children to the age of 21. For an autistic child, the pricetag is $2 million. We're already paying in dollars. The human cost is beyond measure.

Sincerely,

Renee Haynes

Renee Haynes, Oregon Representative, 503-668-0889
Homeowners Against Deficient Dwellings INC. (www.HADD.com)
A National Not for Profit Organization

"Because Sick Buildings Make Sick Children"
Whether you've been a victim (yet) or not, please sign this petition requesting
a Congressional hearing concerning accountability of the home building industry
http://www.thepetitionsite.com/takeaction/322833272?ltl=1110496374

Subj: **Comments on AAAAI Position Paper (fwd)**
Date: 3/1/2006 4:48:57 AM Pacific Standard Time
From: jeff@mayindoorair.com
To: SNK1955@aol.com

Sharon,

I sent this to Mr. Kruger and forwarded your info to three more docs: Charlie Reed, Jim Sublett and Richard Irwin.

Jeff
May Indoor Air Investigations LLC
1522 Cambridge Street
Cambridge, MA 02139
617-354-1055
www.mayindoorair.com
www.myhouseiskillingme.com

----------Forwarded message ----------
References: <1ee.4c6985f3.3133475d@aol.com>
In-Reply-To: <1ee.4c6985f3.3133475d@aol.com>
From: "Jeff May" <jeff@mayindoorair.com>
To: "Mr. Kruger" <bkruger@aaaai.org>
Subject: Comments on AAAAI Position Paper
Date: Wed, 01 Mar 2006 07:34:22 -0500
Mime-Version: 1.0
Content-Type: text/plain; format=flowed; charset="iso-8859-1"
Content-Transfer-Encoding: 8bit

Mr. Kruger,

The AAAI position paper “The Medical Effects of Mold Exposure” by Bush et al. claims to “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.” With respect to hypersensitivity pneumonitis (HP), the position paper states that “exposure to domestic specific fungal spores is an extremely unlikely cause of HP, except in highly unusual circumstances, such as workplace exposure” and concludes that “HP is an uncommon but important disease that can occur as a result of mold exposure, particularly in occupational settings with high levels of exposure.” These statements are incorrect.

I have investigated several cases in which physician-diagnosed HP was the result of home exposure to mold:

#1: A 27 year-old female living in a 2 ½ year-old house with a recently finished and carpeted basement (where the laundry was located), was hospitalized twice with shortness of breath and was referred by her pulmonologist who diagnosed HP. She had and weakly positive serum reactivity to Aspergillus, Candida, Cladosporium, Mucor, Rhodotorula and pigeon but not to her dog or cat. The concentration of spores (NV air sample) in the basement, where she exercised, was about 50,000 /m3 (majority Pen/Asp), but the result of culturable sampling was only about 154 total CFU/ m3 (less than 100 Penicillium CFU/ m3, but still about twice the culturable exterior concentration). Dust in basement tape samples from the carpet, and mildew from the baseboard contained Penicillium, Cladosporium, Aureobasidium pullulans and Aspergillus nidulans. Microscopy of dust and non-viable (NV) air samples were indicative of active mold growth (hyphae and Aspergillus conidiophores) in the basement carpet. During NV air

sampling in the living room, a child jumped on the couch and an airborne dust mite was trapped in the sample. The woman avoided entering her basement and her HP symptoms abated within months.

#2: A 60+ year-old partially disabled female, forced to spend a significant portion of each day in bed (due to an unrelated illness), was referred by her pulmonologist who had diagnosed HP. The home had forced hot air heat (with a dirty blower and mold growing in the dust on the return grilles) and a finished basement (no carpeting) that had flooded several times. A furnace humidifier contained no mold growth, though bacteria and unidentified, flagellate unicellular organisms were present. The concentration of Pen/Asp spores was over 1000 / m3 at a heat register (over 100 times the exterior concentration and almost 8 times greater than the indoor ambient level before operation of the blower, all NV samples) but the culturable sample from the vent (reported by J. Fink) grew only “a few Penicillium” colonies (less than 50 CFU/ m3). Tape samples from the blower contained numerous Penicillium and yeast, as determined by microscopy and culturing. The woman’s blood serum did not react to a commercial Penicillium antigen and reacted only “weakly positive” to Aspergillus in the HP panel of antigens, but was, from a culture of the tape sample, “highly positive” (IgG) to the Aspergillus and Penicillium from the blower (yet negative in IgE reactivity to both). In addition to significant exposures from the heating system, the woman also had bioaerosol exposures while in bed due to the mites and mold colonizing the feather bedding, most probably due to the body moisture she supplied while bedridden. The heating system was professionally cleaned and a media filter installed and the woman’s symptoms diminished, but were exacerbated about two years later. Despite previous recommendations, the woman did not eliminate her feather pillow or encase the mattress in allergen control covers. Upon subsequent testing, a dust sample from the bedding contained entire Aspergillus and Penicillium conidiophores, suggesting active growth, and her serum IgG reactivity to Penicillium was “strongly positive.”

#3: A 70+ year old female, referred by her pulmonologist and diagnosed with HP, had suffered from chronic cough, and for three years had not slept through the night without experiencing disruptive coughing fits (one of which resulted in a hernia). She and her retired husband had lived in the house, which had a dirt basement floor and a steam boiler, for over 50 years, but a few years previously had moved their bedroom into a converted porch above a dirt crawl space. The couple had a dog and had used both an evaporative and a cool mist humidifier, and burned soot-producing jar candles. Dust from the living room furniture contained numerous dog dander particulates as well as many dust mite fecal pellets. There was visible mildew on the walls of the carpeted and cluttered bedroom. Air (NV) samples in all the rooms contained dog dander particulates, elevated numbers of Pen/Asp spores and skin scale fragments (possibly due to bacterial degradation caused by annual carpet washing) in a range of 2-12 microns. Snow covered the ground at the exterior, and the indoor air yielded (culturable samples) 92, 58 and 23 CFU/ m3 (most of which consisted of Aspergillus and Penicillium spp.) in the master bedroom, dining room and basement, respectively. The woman stopped coughing as soon as she put on a NIOSH N95 disposable mask, and did not cough for three hours, but resumed as soon as she took the mask off. She spent the night in the guest room, where there was hardwood flooring and slept soundly to morning.

The authors have ignored a number of papers:

1-Lee YM, Kim YK, Kim SO, Kim SJ, Park HS J Korean Med Sci. 2005 Dec;20(6):1073-5.

“A case of hypersensitivity pneumonitis caused by Penicillium species in a home environment”

We report a case of hypersensitivity pneumonitis in a 30-yr-old female housewife caused by Penicillium species found in her home environment. The patient was diagnosed according to history, chest radiograph, spirometry, high-resolution chest CT, and transbronchial lung biopsy. To identify the causative agent, cultured aeromolds were collected by the open-plate method. From the main fungi cultured, fungal antigens were prepared, and immunoblot analysis with the patient's serum and each fungal antigen was performed. A fungal colonies were isolated from the patient's home. Immunoblotting analysis with the patient's sera demonstrated a IgG-binding fractions to Penicillium species extract, while binding was not noted with control subject. This study indicates that the patient had hypersensitivity pneumonitis on exposure to Penicillium species in her home environment.

2. Ikeda T, Kuroda M, Ueshima K., Nihon Kokyuki Gakkai Zasshi. 2002 May;40(5):387-91.
“A case of hypersensitivity pneumonitis caused by Gyrodontium versicolor”

A 36-year-old woman was admitted to our hospital because of fever, dry cough, dyspnea on exertion and body weight loss in August 2000. Chest radiography and CT scanning showed diffuse ground glass opacity and small centrilobular nodules in the middle and lower lung fields of both lungs. Serum antibody against Trichosporon cutaneum was positive; and summer-type hypersensitivity pneumonitis was therefore initially diagnosed. Treatment with methylprednisolone and prednisolone decreased the symptoms, but the dyspnea reappeared when the patient was at home. Inspection of her house revealed the presence of fungi under the floor. After these were removed, her symptoms disappeared completely. The lymphocytic stimulation test of the peripheral blood was positive for the fungi, and it was therefore suggested that they were the cause of her hypersensitivity pneumonitis. The fungi were identified as Gyrodontium versicolor. This is the first report of hypersensitivity pneumonitis caused by Gyrodontium versicolor.

3. Lee SK, Kim SS, Nahm DH, Park HS, Oh YJ, Park KJ, Kim SO, Kim SJ. Allergy. 2000 Dec;55(12):1190-3.
“Hypersensitivity pneumonitis caused by Fusarium napiforme in a home environment”

BACKGROUND: We report a case of hypersensitivity pneumonitis (HP) in a 17-year-old male student caused by Fusarium napiforme found in his home environment. METHODS: The patient was diagnosed according to history, chest radiograph, spirometry, high-resolution chest CT, and transbronchial lung biopsy. To identify the causative agent, cultured aeromolds were collected by the open-plate method. From the main fungi cultured, fungal antigens were prepared, and immunoblot analysis with the patient's serum and each fungal antigen was performed. RESULTS: Five fungal species were isolated from the patient's home. Immunoblotting analysis with the patient's serum demonstrated more than 10 IgG-binding fractions to F. napiforme extract only, while little binding was noted with the other fungal antigens. CONCLUSIONS: We should be aware that HP may be caused by F. napiforme in the home environment.

4. Wright RS, Dyer Z, Liebhaber MI, Kell DL, Harber P., Am J Respir Crit Care Med. 1999 Nov;160(5 Pt 1):1758-61.

“Hypersensitivity pneumonitis from Pezizia domiciliana. A case of El Nino lung”

A previously healthy woman developed severe dyspnea and was found to have

restrictive lung disease and evidence of alveolitis. Open lung biopsy revealed extrinsic allergic alveolitis (hypersensitivity pneumonitis). The etiology was not initially apparent, but a home inspection showed an unusual mushroom growing in the patient's basement. Air sampling and serum precipitins against the fungal antigens confirmed that Pezizia domiciliana was the cause of the patient's hypersensitivity pneumonitis. This is the first described case of hypersensitivity pneumonitis cause by P. domiciliana. We speculate that unprecedented rainfall and flooding of the patient's basement as a result of El Nino rains produced ideal factors for the growth of this fungus.

5. Jacobs RL, Andrews CP, Coalson JJ. Ann Allergy Asthma Immunol. 2005 Aug;95(2):115-28. Ann Allergy Asthma Immunol. 2005 Aug;95(2):99.

“Hypersensitivity pneumonitis: beyond classic occupational disease-changing concepts of diagnosis and management”

OBJECTIVE: To review inhaled antigens in home environments that cause hypersensitivity pneumonitis (HP) of varied clinical expressions and histopathologic patterns. DATA SOURCES: Computer-assisted MEDLINE and manual searches for articles concerning HP, interstitial lung disease (ILD), epidemiology of HP and ILD, challenge procedures of HP, and indoor fungi. STUDY SELECTION: Published articles concerning inhaled antigens in home environments and HP were selected. RESULTS: Current criteria for the diagnosis of HP are too restrictive, because most apply only to the classic acute presentation and are of limited value in the subacute and insidious forms. Clinical expressions vary across the gamut of respiratory tract signs and symptoms. Patterns on lung biopsy may include all histopathologic descriptions of idiopathic ILD. The home is the likely causative environment rather than the workplace. Exposures may be occult and require in-depth environmental histories and on-site investigations to detect antigens and sources. CONCLUSIONS: Natural or environmental challenges have become an important tool for diagnosing HP and determining effectiveness of remediation. Early diagnosis and effective remediation of the cause lead to a high survival rate, whereas diagnosis in advanced stages leads to disability and/or premature death.

6. Venkatesh P, Wild L.,Paediatr Drugs. 2005;7(4):235-44.

Hypersensitivity pneumonitis in children: clinical features, diagnosis, and treatment.

Hypersensitivity pneumonitis (HP), or extrinsic allergic alveolitis, is a form of immune-mediated inflammatory lung disease involving the distal portions of the lungs associated with intense or repeated exposure to a variety of finely dispersed environmental antigens. Although once believed to be a disease of adults because of its frequent association with the occupational setting, HP exists in the pediatric population and often goes unrecognized. Childhood HP is often associated with exposure to antigens in the home environment as well as with certain hobbies. Patients present in any one of the three disease stages: acute, subacute, and chronic, all with unique clinical presentations. Histopathologic findings depend on the disease stage at the time of evaluation. The immuno-pathogenesis is complex, but immune-complex (type III hypersensitivity) and cell-mediated (type IV hypersensitivity) immune responses appear to be the primary immune mechanisms involved in the pathogenesis of HP. Diagnosis can be very challenging. Although no single diagnostic or clinical laboratory test is available to diagnose HP, the most significant diagnostic tool is a detailed environmental exposure history. Avoidance of the inciting antigen is the most important form of treatment. Acute HP is responsive to antigen removal alone. However, a short course of prednisone for 2-3 weeks can be useful in

patients with severe attacks. Subacute and chronic HP may require higher doses of corticosteroids for a longer duration (i.e. months); however, the long-term efficacy of using corticosteroids is still not well defined. As with most hypersensitivity diseases, early diagnosis provides the best prognosis.

7. Moran JV, Greenberger PA, Patterson R., Allergy Asthma Proc. 2002 Jul-Aug;23(4):265-70.

“Long-term evaluation of hypersensitivity pneumonitis: a case study follow-up and literature review”

This study reports a 3-year follow-up of a classic presentation of hypersensitivity pneumonitis (HP), originally reported elsewhere, after removal of the causative antigens. The literature is reviewed and this case is compared with outcomes of series previously reported. The patient was reevaluated by clinical, serologic, radiographic, and pulmonary function testing 3 years after removal of her home's contaminated humidifier, cleaning of the home, and administration of a course of prednisone. Repeat serologic measurements revealed positive serum precipitins only for Aspergillus flavus and Phoma herbarum, significantly fewer than her original panel, which revealed precipitating antibodies to her humidifier water and 10 other specific antigens. Pulmonary function tests remained stable. Physical exam revealed bibasilar rales. Computed tomography scan revealed pulmonary fibrosis, bronchiectasis, and honeycombing that was compared with 3 years earlier. Although most of the data obtained on reevaluation suggest remission, radiographic findings have not remitted. Long-term follow-up of parameters of HP disease activity do not always reveal consistent findings. This patient appears to be in a category of HP between the classic subacute and chronic stages.

8. Yoshizawa Y, Ohtani Y, Hayakawa H, Sato A, Suga M, Ando M.,J Allergy Clin Immunol. 1999 Feb;103(2 Pt 1):315-20.

“Chronic hypersensitivity pneumonitis in Japan: a nationwide epidemiologic Survey”

BACKGROUND: Pulmonary fibrosis inevitably develops in patients with chronic hypersensitivity pneumonitis (HP). OBJECTIVE: We conducted a nationwide epidemiologic study in Japan to evaluate the frequency and clinical characteristics of chronic HP. METHODS: This report is on 36 cases of chronic HP, including 10 patients with summer-type HP, 5 patients with home-related HP, 7 patients with bird fancier's lung, 5 patients with isocyanate-induced HP, 4 patients with farmer's lung, and 5 patients with other types of chronic HP. Chronic HP was further subgrouped into 2 types: one type of patients were first seen with chronic disease (9 patients), and the other type became chronic with fibrosis after repeated acute episodes (27 patients). RESULTS: The upper lung field was frequently involved in chronic HP (17%). Ground-glass opacities were observed in 57% and air space consolidation in 30% of the patients. Honeycombing was apparent in 37%. Twenty-six of 28 patients had antibodies to the presumptive antigens. Five of 8 patients with chronic HP were positive for antigen-induced lymphocyte proliferation. In 2 cases patients did not have detectable antibodies to causative antigens, although antigen-induced lymphocyte proliferation was detectable. The ratio of CD4 to CD8 in BAL lymphocytes was lowest in isocyanate-induced HP (mean 0.22) and tended to be high in farmer's lung and bird fancier's lung. Granulomas were observed in 39% and Masson bodies in 42% of specimens on histologic examination. Administration of prednisolone was effective in 58% of patients. CONCLUSIONS: The insidious form of chronic HP has probably been misdiagnosed as idiopathic pulmonary fibrosis when a good history was not taken and immunologic (especially antigen-induced

lymphocyte proliferation) and BAL testing were not counted.

Also:

Apostolakos, M.J., Rossmoore, H., Beckett, W.S. 2001, “Hypersensitivity pneumonitis from ordinary residential exposures,” Environ. Health Perspect., vol. 109, no.9, pp. 979-81.

Hirakata, Y., Katoh, T., Ishii, Y., Kitamura, S., Sugiyama, Y.,2002, “Trichosporon asahii-induced asthma in a family with Japanese summer-type hypersensitivity pneumonitis,” Ann. Allergy Asthma Immunol., vol.88, no.3, pp. 335-8.

Park, H.S., Jung, K.S., Kim, S.O., Kim, S.J, 1994, “Hypersensitivity pneumonitis induced by Penicillium expansum in a home environment,” Clin. Exp. Allerg., vol. 24, no.4, pp. 383-5.

Patel, A.M., Ryu, J.H., Reed, C.E., 2001, “Hypersensitivity pneumonitis: current concepts and future questions,” J. Allergy Clin. Immunol., vol.108, no.5, pp. 661-70.

Suda, T., Sato, A., Ida, M., et. al., 1995, “Hypersensitivity pneumonitis associated with home ultrasonic humidifiers,” Chest, vol.107, no.3, pp. 711-7.

Yoshida, K., Ando, M., Sakata, T., Araki, S., 1989, “Prevention of summer-type hypersensitivity pneumonitis: effect of elimination of Trichosporon cutaneum from the patients' homes,” Arch. Environ. Health, vol.44, no.5, pp. 317-22.

HP as a result of home exposure and home exposure to mold is probably under-diagnosed in the U.S. The illness is a serious public health concern (no doubt with more cases than illness due to exposure to radon, for example, for which there has been great expenditures).

The AAAI position paper should modified to reflect importance of home exposures to antigens.

Jeffrey C. May, M.A., Author
May Indoor Air Investigations LLC
1522 Cambridge Street
Cambridge, MA
617-354-1055
www.mayindoorair.com
www.myhouseiskillingme.com

Subj: **Re: My Comments on the AAAAI article**
Date: 2/24/2006 4:32:25 AM Pacific Standard Time
From: RIlipsey87
To: bkruger@aaaai.org.
BCC: SNK 1955

In a message dated 2/22/2006 11:16:13 A.M. Eastern Standard Time, RIlipsey87 writes:

> **Dr. Kruger:**
>
> **Thank you for the opportunity to comment on the recent article entitled, "The Medical Effects of Mold Exposure", by Bush, et al, , AAAAI, in the JACI, Feb. 2006.**
>
> **My PhD is in fungicides and I testify about 90 times a year as a forensic toxicologist. I try to stay 50:50 plaintiff / defense in the cases I take nationwide. I also peer review articles for several scientific societies.**
>
> **A fair and balanced review article is very helpful to everyone. A "review" article by one side or the other in a new area of science serves no useful purpose. After Silent Spring by Rachael Carson, came out, we saw the same problem regarding the pesticide controversy. "Experts" paid by the pesticide industry wrote review articles slanted toward the industry, ie Dursban (chlorpyrifos)" is 50 times safer that aspirin and 11 times safer than table salt " (comparing only the LD50 in rat studies). We now know about axonal blocking, damage to the myelin sheath, etc. leading to paralysis in children and now Dursban is being taken off the market.**
>
> **The article by Bush, et al, is a prime example of the authors listing an impressive number of articles in their review, but rejecting key articles that disagree with their basic premise as flawed or lacking credibility or problematic, etc. When I see the names of professional defense experts on a list of authors, I know not to include that article in any serious discussion on the subject. I know they will probably quote each other and ignore as flawed or problematic key articles that express an opposing view.**
>
> **Therefore, for Bush, et al, to imply that mycotoxins do not get into the air is wrong. They travel on spores, hyphae or particles and dust. Mycotoxins do not have to be volatile to get into the air in a home with the air handler running. They state that bulk sampling for mycotoxins does not provide evidence that mycotoxins get into the air because mycotoxins, spores and hyphae, or particles, are nonvolatile. This is wrong. This statement totally ignores the fact that analyzing the dust in air handler filters for mycotoxins, which is done on a regular basis now, is a valid way to determine the gross levels of mold spores and mycotoxins in the air inside homes.**
>
> **They attacked Dr. Johanning's work on mycotoxin "crude" cytotoxicity as "lacking sensitivity" yet quote the ACOEM article misusing the Rao's study on visible hemorrhaging in the lungs of rats as sensitive in order to make their argument that mycotoxins cannot get into the air in sufficient quantities to cause injury. This is wrong.**
>
> **Attached is my review of the misuse of the Rao study by GlobalTox employees, which became the very controversial ACOEM position document on mycotoxins and human health based largely on the misuse of the Rao study involving rats and a single dose of an unknown quantity and an unknown strain and unknown purity of mycotoxins. They implied that children living in moldy homes and breathing contaminated air for weeks and months could not possibly be harmed. This is wrong.**
>
> **(I am not ready to mention my Katrina study, yet)**

Dr. Richard L. Lipsey (904)398-2168

550 Water St, #1230, Jacksonville, FL 32202
Forensic Toxicologist and former Adjunct Professor,
Univ. N. Florida, Div. Continuing Educ., HazMat/OSHA
Fla. Comm. College Jax, Institute of Occ. Safety & Health,
Clinical Toxicology Advisory Comm., Florida Poison Info Center, Jax.
www.richardlipsey.com

To: The Editors, JACI, and the Board of Directors, American Academy of Allergy, Asthma and Immunology.

As a long time member of the Academy, I was shocked and disappointed by the "Position Paper" printed in the February issue of the JACI (Bush RK, et al. The medical aspects of mold exposure). A number of criticisms come quickly to mind:

1. At least two of the authors earn a substantial income testifying against patients in mold related litigation. The potential conflict of interest is not addressed.
2. This is not a position paper generated from free and open discussion among Academy members. It is a one-sided opinion paper.
3. The authors seem to be ignoring one of the basic tenets of allergy: when symptoms appear following an exposure and abate on its cessation, chances are the patient is reacting to something in that exposure. Before we label her a hypochondriac, let's explore the details. Perhaps we can learn.
4. The authors draw conclusions about the health effects of indoor mold exposure for which they offer no positive support from the literature. The lack of evidence is not evidence against.
5. The authors have selected from the literature articles that, however tenuously, support their opinions and ignore the mountain of evidence which refutes their conclusions. c.f. Straus D, (ed). Sick Building Syndrome: Advances in applied microbiology. 55, 2004, and Johanning E. Bioaerosols, fungi, bacteria,

mycotoxins and human health. Fungal Research Group Foundation, Albany, 2005.

6. Two peer-reviewed literature references that do not support the authors' conclusions are cited and rejected as "poor quality" without discussion.[1,2]
7. The authors' review of the literature involving the presence of mold specific IgG antibodies reflecting the patients' exposure to mold is completely distorted. They seem to suggest that the measurement of mold specific IgG antibodies cannot be a useful clinical parameter in diagnosing and monitoring the progress of patients with mold related illness.
8. The conclusion that mycotoxins are not proteins and therefore mycotoxin antibodies are not possible ignores the enormous literature on penicillin reactions (a mycotoxin). One of the papers cited by the authors specifically identifies IgG antibodies against mycotoxins but is given no value in reading their conclusion.[3]
9. No reference is made to the very important work done by Dr. Sherris' group, formerly of the Mayo Clinic, now at the University of Buffalo, in which mold specific IgG antibodies are identified as markers of chronic rhinosinusitis, and no difference between patients and controls is seen with IgE antibodies.[4]

I am astounded that the Academy would take such a blatant stand against the best interest of patients and disburse biased opinions as facts to its membership. I believe this paper does not meet the minimal standard for a position paper by the Academy. It should be

withdrawn. The Academy would de well to sponsor an open forum in which to debate the issues of health effects from mold exposure in the Journal.

Sincerely Yours,

Vincent A. Marinkovich, M.D.

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Subj: **FW: Commentary - AAAAI P0sition Paper, Medical Effects of Mold Exposure**
Date: 2/21/2006 2:12:35 PM Pacific Standard Time
From: mmaizel@oneimage.com
To: SNK1955@aol.com, mch@mchudson.com
CC: ritchieshoemaker@msn.com, jrobbin@ricercar.com

Please see attached.
Regards to all,

Margaret

From: Margaret Maizel [mailto:mmaizel@oneimage.com]
Sent: Tuesday, February 21, 2006 3:06 PM
To: 'bkruger@aaaai.org'
Subject: Commentary - AAAAI P0sition Paper, Medical Effects of Mold Exposure

Dear Mr. Kruger,
Please see the attached letter to you dated to-day regarding the AAAAAI Position Paper: "The Medical Effects of Mold Exposure". Thank you for your interest in hearing from individuals with an ongoing interest in encouraging Defensible Science in the Public Interest.

Margaret Maizel
ONEIMAGE LLC
1810 Linden Lake Road
Fort Collins, Colorado 80524
Phone:970-407-0506
Fax: 970-407-0512
Cell: 970-227-4703

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Nondisclosure of conflicts of interest is perilous to the advancement of science

Kaye H. Kilburn, MD[a], Michael Gray, MD, MPH, CIME[b], Sharon Kramer, BBA[c]

Article Outline

To the Editor:

We are requesting that the American Academy of Allergy, Asthma and Immunology retract, as an official position statement representative of 7000 physicians, “The medical effects of mold exposure” by Bush et al.[1] We are dismayed by the article's interpretation of “state-of-the-art” understanding of illnesses caused by molds and mycotoxins. The document appears to be based on many statements that do not reflect state-of-the-art science but are anecdotal in origin.[1], [2]

A significant finding of the position statement relies on a review piece of another medical association, the American College of Occupational and Environmental Medicine (ACOEM).[3] The ACOEM mold statement is also widely promoted as a state-of-the-art scientific review by an influential medical association. The authors are 2 PhD principals of a defense litigation support corporation and a physician who is an author of both the ACOEM and the Academy's mold statement. This physician also provides expert testimony for the defense in mold litigation.

The ACOEM position states, “Levels of exposure in the indoor environment, dose response data in animals, and dose-rate considerations suggest that delivery by the inhalation route of a toxic dose of mycotoxins in the indoor environment is highly unlikely at best, even for the hypothetically most vulnerable subpopulations.” Of the 83 references “reviewed” by the ACOEM, only one comes to the conclusion that human illness is “highly unlikely at best.” It was written by an ACOEM author and fellow principals in the litigation defense support corporation.[4] The finding of “highly unlikely at best” is based solely on the mathematic extrapolation from a single rat study and calculated by the litigation defense corporation principals. The extrapolations have been questioned by credentialed scientists active in the field of mold and mycotoxin research.[5]

The new Academy position paper states that “the occurrence of mold-related toxicity (mycotoxicosis) from exposure in nonoccupational settings is not supported by the current data, and its occurrence is improbable.” There are 44 references listed for the Academy position statement. Other than the ACOEM statement of “highly unlikely at best,” the Academy mold position paper cites, as reference 29, another article based on rodent studies and extrapolated math and written by the same expert defense witness authors in support of this statement.[6] This article was recently found by the courts not to be based on sound scientific protocol to deduce absence of human illness. Not one of the other 44 references supports the statement that “its occurrence is improbable.”

Are the members of the Academy of the opinion that it is accepted scientific protocol for 2 influential medical associations to deduce that all human illness is “highly unlikely at best” and “its occurrence is improbable” based solely on questioned math from a rodent study? We ask the authors of the Academy position paper to cite any epidemiologic or mechanistic research that supports the statements of “highly unlikely at best” or “its occurrence is improbable.” We are not aware of the existence of any such studies, other than the articles by the defense litigation support corporation mentioned above.

We are concerned by the fact that the Academy's authors are nationally known expert witnesses for the defense in mold litigation. Yet no conflict of interest disclosures were attached to the document for the Academy members' perusal when researching appropriate diagnoses and treatment protocols for their patients. We are concerned the Academy position will cause those with serious non-IgE–mediated illnesses from exposure to molds and mycotoxins to continue to be misdiagnosed and untreated.[7] The Academy position does not accurately reflect illnesses being reported by thousands from across the United States. It does not reflect state-of-the-art research.[8] It does, however, reflect a medical position that is beneficial to industry, insurers, and the medical experts that support them in mold litigation.[9] Because it is the goal of physicians and researchers to advance science to help the sick and because the current understanding of mold-induced illnesses is highly debated, complex, and contentious within the medical community and the courts, the utmost diligence is required to ensure that journals and medical associations are not misused to strengthen a litigation position.

To advance an appropriate review on this issue and potentially others, we are asking the Academy to consider adopting a transparent conflict of interest policy that will guide the publication of all future articles and position statements. We are requesting this article be retracted as an official position of the Academy until such appropriate review of the matter can be provided.

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We thank the Editor of the Journal for the opportunity to present a differing perspective to the members of the Academy in regard to a serious issue that affects the health and safety of countless citizens.

References

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[a] From private practice, Pasadena, Calif

[b] private practice, Benson, Ariz

[c] Escondidio, Calif

Disclosure of potential conflict of interest: K. H. Kilburn has served as an expert witness in mold litigation primarily retained by the plaintiff bar and is president of Neuro-Test, Inc. M. Gray has served as an expert witness in mold litigation primarily retained by the plaintiff bar, is sole proprietor of ImmunoTox, and has a patent pending with Realtime Laboratories. S. Kramer has been a party in mold litigation represented by the plaintiff bar, volunteers to assist those made ill from mold, and is a real estate agent by profession.

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Signators for the Letter Entitled “Non-Disclosure of Interests Are Perilous For The Advancement of Science” aka Kramer Letter

Ms. Lorri Cavaliere
Ms. Andrea Oswald
Mr. Erik Johnson
Dr and Mrs. Mark O’Hara
Dr. Linda J. Leffel
Mr. Mark Hudson
Ms. Donna Cole, (Mold Victim & Air Traffic Controller)
Ms. Debra Rubin
Ms. Harriet Unis
Ms. Jessie Delano
Ms. Diana Leicht
Ms. Karen Scinto
Mr. Scott B. Whitenack, Esq.
Mr. and Mrs. Gary Arthur
Ms. Jessica Johnson
Ms. John Taylor
Ms. Cindy Schnackel
Ms. Evon Kochey
Mr. Gene Brooks
Ms. Coralee Reiss
Ms. Kim Eberhart
Mr. David L. Parker
Mr. Jonathan Lee Wright
Ms. Charlotte Leslie
Mr. Kelly Vance, Esq.
Ms. Andrea Johanna Olafsdottir
Connecticut Foundation for Environmentally Safe Schools 501 c 3
Mr. Larry Neil McQuarie, Sr.
Ms. Diane Ethier, IAQ In Schools Mentor and Trainer
Mrs. Judy O'Reilly
Mr and Mrs. Richard and Joellen Lawson
Mrs. Nancy Davis
Ms. Lisa Wheatley
Ms. Angela Page
Ms. Marilyn Hoffman
Mr. Will Schachter
Mrs. Jayne Abernathy
Ms. Ellie Goldberg, M.Ed.
Mr. Kevin Carstens
Ms. Joyce Mermet
Ms. Phylis Schachter
Ms. Lorrie Nadel
Mrs. Helen E. Noonan

Mr. John Wallace
Ms. Tracy Brandaw
Mr. Harold Hyams, Esq.
Mrs. Renee Haynes
Ms. Elvira Williams
Ms. Susan Maxey
Ms. Amy Belisle.
Ms. Valerie Madeska
Ms. Letitia Peters
Ms. Perri Larson
Mr. Chris Beaumont
Mr. Terry Vinocur
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Mr. Shawn Mayfield
Mr. John Stuart
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Mrs. Roxane Riggio Catalanotto
Mr. Jamie Garland
Mrs. Linda Yarbrough
Ms. Katy O'Reilly
Dr. David T. Denmead,
Dr. Vincent Marinkovich
Prof. Matthew Hudson
Ms. Margaret Maizzel
Mr. and Mrs. Michael A. Kramer.