Reviewer's report

Title: Fungal exposure in homes of patients with sarcoidosis

Version: 2 Date: 6 October 2010

Reviewer: Päivi Salo

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Although the authors have revised their manuscript based on the reviewers' comments, some major issues remain to be addressed.

Major comments

1) This study may not justify any recommendations regarding fungal remediation; no remedial actions were performed in the study. To investigate whether remediation affects exposure levels and health outcomes (sarcoidosis/other outcomes), information on exposure(s) and outcome(s) is needed before and after performing remedial actions. Reword your conclusions (Abstract, p. 2).

2) N-Acetylhexosaminidases have been shown to be universally distributed among most types of living organisms, both prokaryotic and eukaryotic (Slámová et al., #N-Acetylhexosaminidase: What's in a name…? Biotechnology Advances 28 (2010) 682–693). There is some evidence that the effects of fungal exposure on respiratory health may be modified by bacterial exposure (Park et al., Fungal and endotoxin measurements in dust associated with respiratory symptoms in a water-damaged office building. Indoor Air 2006;16:192-203). Did the authors assess airborne bacterial exposures? Damp indoor environments are known not only to promote fungal growth, but also to influence bacterial growth (IOM:Damp Indoor Spaces and Health, The National Academies Press, Washington, 2004 ). As mentioned before, fungal exposure may contribute to the development of sarcoidosis, but other agents (e.g., bacteria) have also been considered important in etiology.

3) Studies have demonstrated that tobacco smoke contains bacterial (e.g., endotoxin, muramic acid) and fungal components (Larsson et al., Identification of bacterial and fungal components in tobacco and tobacco smoke. Tobacco Induced Diseases 2008;4:4; Sebastian et al., Elevated concentrations of endotoxin in indoor air due to cigarette smoking. J Environ Monit 2006;8:519-22; Hasdey et al., Bacterial Endotoxin Is an Active Component of Cigarette Smoke. Chest 1999; 115:829–835). Can the authors ascertain that the source(s) of airborne NAHAs was other than tobacco smoke? What proportion of the cases were smokers? Do NAHA concentrations correlate with smoking status or other smoking related variables? Studies have shown that a smoking habit affects the morphologic and functional correlations in pulmonary sarcoidosis (Terasaki et al., Pulmonary Sarcoidosis: Comparison of Findings of Inspiratory and Expiratory High-Resolution CT and Pulmonary Function Tests Between Smokers and Nonsmokers.AJR 2005;185:333-38). The authors need to provide additional
information on smoking.

4) Because all controls were non-smokers, the control group may not be a representative sample of the source population.

Minor comments

1) Check the manuscript for typographical errors. For example, replace “ocular inspection” with either “visual inspection” or “ocular inspection” (p. 8).

**Level of interest:** An article whose findings are important to those with closely related research interests

**Quality of written English:** Needs some language corrections before being published

**Statistical review:** No, the manuscript does not need to be seen by a statistician.

**Declaration of competing interests:**

I declare that I have no competing interests.