Health Effects Associated With Exposure To Airborne Toxigenic Fungi

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Introduction

The health effects of mold exposure have been described in a number of case reports, but the specific mechanism of disease remains incompletely understood and inadequately studied. Extensive attention has long been given to airborne molds as potential allergens or infectious agents. However, the few published studies of occupants of mold-contaminated buildings have not convincingly demonstrated the clinical syndrome is consistent with an immediate (e.g., allergic rhinitis, asthma) or delayed (e.g., hypersensitivity pneumonitis) hypersensitivity reaction, with no immunological markers (antibodies or other immune measures) correlating with clinical illness, nor with conditions involving immunosuppression, such as viral or bacterial respiratory tract infections. Most clinical studies of this problem have been based on the assumption that mold causes these specific respiratory diseases, and thus have failed to address the significant, non-respiratory symptoms (e.g., fatigue, neurocognitive impairment, rash) of this illness, leading to potential disease misclassification. Indeed, the consistent finding that most occupants of mold-contaminated buildings experience a rapid onset of symptoms upon entering the building and rapid resolution each time they leave strongly suggests a toxicological, rather than allergic/immunological or infectious mechanism.

Sick Building Syndrome and Building-Related Illness

The term "sick building syndrome" (SBS) was first applied to unexplained symptoms of occupants primarily in office buildings in the early 1970's. Numerous studies of "sick buildings" and their occupants focused on a presumed chemical or physical etiology, such as off-gassing of formaldehyde from new carpets or inadequate mechanical ventilation. These studies consistently failed to investigate for any building history of water damage or measure for toxigenic fungal spore contamination. The same clinical syndrome of SBS has more recently been described as a "building-related illness arising from microbial contamination of building materials caused by condensation and leaks" (AIHA 1996) and more vaguely as "building related symptoms" (ACGIH 1999). Only recently has SBS been directly associated with indoor exposure to toxigenic mold through extensive clinical and epidemiological investigation, with the introduction of a new building-related illness, termed Non-Infectious Fungal Indoor Exposure Syndrome (NIFIES) to describe the illness typically first called SBS (Craner 1999). Symptoms of NIFIES include eye, nose and throat irritation/inflammation, respiratory symptoms such as cough and chest tightness, fatigue, papular rash, and neurocognitive symptoms such as inattentiveness and short-term memory impairment.

Theories of Disease Mechanism

The leading theory of the mechanism of illness arising from exposure to toxigenic fungi is that mycotoxins attached to and/or liberated from the spore wall are the causative agent of disease. Inhalation exposure to mycotoxins was first recognized in agricultural workers exposed to extremely high levels of airborne molds. A variety of fungal and bacterial toxins were potentially involved in the acute toxicological disease, termed organic dust toxic syndrome (ODTS). However, the predominantly constitutional manifestations of ODTS (delayed onset of fevers, chills, muscle aches, and sometimes cough) are not clinically

consistent with SBS/NIFIES. Ongoing attempts to demonstrate an allergic mechanism have largely failed, including currently available antibody tests for *Stachybotrys* that have demonstrated poor predictive value as a marker of disease or exposure. Currently, no reliable biological marker (e.g., blood test) has been developed to demonstrate the presence of fungal mycotoxin or its metabolites in the body.

Route of Exposure

Spores and mycotoxins affect occupants in buildings primarily through inhalation. Many microscopic mold spores (2-10 μm) are respirable into the alveoli, the terminal portion of the lungs where oxygen exchange between the lungs and blood occurs, and in which soluble toxins contained in the spores enter the blood stream. However, the toxicology and pathophysiological mechanism by which mycotoxins enter and distribute throughout the body and selectively produce symptoms remains poorly understood. Skin contact and ingestion do not appear to be significant exposure sources in non-industrial/nonagricultural settings, even though dermatological and gastrointestinal manifestations can occur. Generally, individuals have complete clinical improvement shortly after removal of the mycotoxin source, either through relocation or remediation of the mold contaminated environment. In contrast, medications such as antibiotics, anti-fungal agents, antihistamines, and asthma drugs are usually ineffective because the disorder is non-infectious, non-communicable, and does not elicit a measurable immune response.

References

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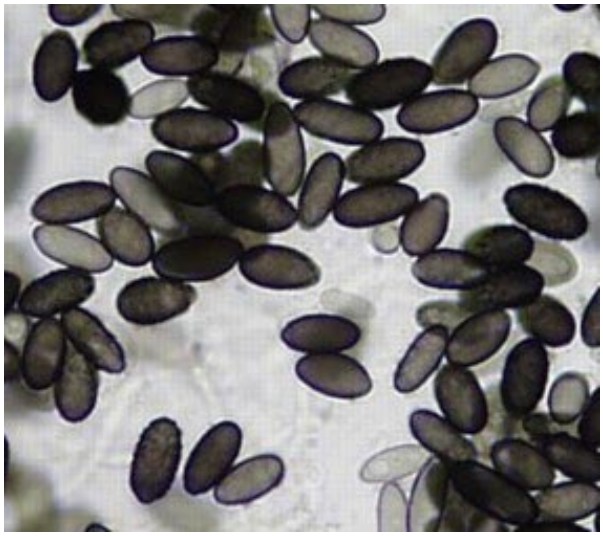

FIGURE 1: Spores of *Stachybotrys chartarum*