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MOLD

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Health Effects of

Toxin-Producing Indoor Molds in California

CA Department of Health Services

Environmental Health Investigations Branch

Due to excessive rainfall this winter many Californians are experiencing increased exposure to indoor microorganisms. Several fungal species capable of producing toxic substances have been found in water-damaged California homes and offices. This article provides information about potential health effects from exposure to *Stachybotrys chartarum* (a.k.a. *S. atra*), a toxigenic mold that has received increasing attention recently among indoor air reseachers and the public. Within the last 12-18 months several scientific reports (and media attention) have focused on *Stachybotrys*, a ubiquitous saprophytic fungus that grows on nitrogen-poor, cellulose rich materials such as hay, straw and building materials (ceiling tiles, wall paper, paper covering on gypsum wallboard). The statewide prevalence of this fungus in homes or work places is unknown, although one report found Stachybotrys in 2-3% of a small survey of southern California homes (Kozak, 1979).

Mechanism of Action

Some strains of *Stachybotrys chartarum* can produce mycotoxins of the trichothecene and spirolactone families. The trichothecene mycotoxins satratoxins G and H are potent protein synthesis inhibitors and cause immunosuppression in laboratory animals. In experimental animal studies, the trichothecenes affect rapidly proliferating tissues such as skin and mucosa, as well as lymphatic and hematopoietic tissues (Ueno, 1983). In laboratory animals, acute exposure to large amounts of trichothecene toxins results in a rapid release of sequestered white blood cells into circulation, while repeated or chronic exposure destroys granulocytic precursor cells in

bone marrow leading to white cell depletion. Among the reported cellular effects are: mitogen B/T lymphocyte blastogenesis suppression; decrease of IgM, IgG, IgA; impaired macrophage activity and migration-chemotaxis; broad immunosuppressive effects on the cellular and humoral-mediated immune response leading to secondary infections; and, paradoxically, increased spontaneous antibody producing cells in the spleen (Corrier, 1991).

Toxigenic strains of SC may also produce spirolactones (stachybotrylactone) and spirolactams (stachybotrylactam), toxins which produce anticomplement effects (Jarvis, 1995). Possible synergistic effects between the trichothecenes and these mycotoxins have not yet been evaluated. Although laboratories can test a sample of *Stachybotrys chartarum* for its ability to produce mycotoxins, *in vitro* results do not necessarily equate with the *in vivo* situation. Therefore, a fungus that produces toxins in the lab may not do so in the field, or vice versa. It has been suggested that to assure the safety of any exposed individual, whenever *Stachybotrys chartarum* is identified, it should be considered as a potential mycotoxin-producing organism (Jarvis, 1994).

Positive skin reactions to the fungus have been found in some asthmatics living or working in *Stachybotrys*-contaminated rooms, suggesting a hypersensitivity component in addition to the potential for mycotoxicosis. Thus the fungal spores themselves or chemicals carried on the spores may produce either allergenic or toxigenic effects (Flannigan, 1991).

Routes of Exposure

Due to its wet, slimy growth characteristics, it is unusual for spores from active *Stachybotrys* colonies to become aerosolized. However, when colonies of this fungus die and become dehydrated, there is increased risk for air dispersion. Portals of possible entry into the body include inhalation and dermal absorption when the fungus is found on walls or in carpets.

Case Reports

Historically, toxicologic effects from this fungus were reported in Europe, where horses, sheep and cattle suffered fatal hemorrhagic disorders following ingestion exposures (Forgacs,1972)). Human occupational exposures to contaminated straw or hay resulted in nasal and tracheal bleeding, skin irritation and alterations in white blood cell counts (Hintikka, 1987).

The first U.S. case of Stachybotrys-associated health effects from inhalation exposure was reported in a suburban Chicago family (Croft, 1986). The fungus had contaminated the ventilation system

and ceilings of the house. Health effects reported by the family included chronic recurring cold and flu-like symptoms, sore throat, diarrhea, headache, fatigue, dermatitis, intermittent focal alopecia and generalized malaise. Workers who cleaned and removed contaminated material from this house also experienced skin irritation and respiratory symptoms. After *Stachybotrys* contamination was removed the house was reoccupied and residents reported no recurrence of clinical symptoms.

Stachybotrys and satratoxin H (one of the trichothecene mycotoxins) were subsequently identified in a water-damaged office building in New York City. A small case-control study showed workers exposed to the fungus were at statistically significant higher risk for nonspecified disorders of the lower airways, eyes and skin; fevers and flu-like symptoms, and chronic fatigue (Johanning, 1993, 1996). No significant differences in specific *S. chartarum* IgE and IgG levels were noted between cases and controls. Although *Stachybotrys chartarum* specific IgE (RAST) and IgG (ELISA) tests are available, their sensitivity and specificity have not yet been determined.

A recent report describes identification of 10 likely or possible cases of building-related asthma in a courthouse contaminated with *Stachybotrys* and *Aspergillus* species (Hodgson, 1998). Self-reported symptoms among co-workers included fever, headache, rhinitis, coughing, dyspnea and chest tightness. Chest radiographs were negative and *Stachybotrys*-specific serology was uninformative.

Stachybotrys chartarum, along with other fungi and environmental tobacco smoke, was recently postulated to have an association with pulmonary hemosiderosis in a cluster of Cleveland, Ohio infants (Montana, 1997; MMWR, 1997)). While *SC* was found more frequently in the homes of case infants compared to controls, exposure of case infants to mycotoxins in the home could not be determined. Because there is no field test for airborne mycotoxins, it is not currently possible to determine if toxins were actually present in the living space of case infants, and if so, at what levels. However, since *Stachybotrys chartarum* spores containing mycotoxins have been shown to produce pulmonary alveolar and intra-bronchiolar inflammation and hemorrhage in mice (Nikulin, 1996, 1997), more research into the inhalation effects of these toxins, especially on immature alveoli and pulmonary vascular walls, is critically needed.

Pulmonary hemosiderosis is a condition characterized by recurrent alveolar hemorrhage resulting in clinical signs of cough, wheeze, hemoptysis, tachypnea, low grade fever, and microcytic hypochromic anemia. Chest radiographs typically show patchy infiltrates and sputum specimens, laryngeal swabs or gastric aspirates reveal hemosiderin-laden macrophages. The association of some cases with

allergy to cow's milk (Heiner syndrome) and its association with glomerulonephritis in Goodpasture's syndrome suggests an immunologic etiology but immunologic findings in idopathic cases have been inconsistent. Some familial case reports also suggest a genetic component.

California Department of Health Services staff reviewed statewide hospital discharge data for 1989-1995 (last year for which data is available) and identified a total of eight hospitalizations and no deaths during these years for hemosiderosis in infants less than one year of age. There were no more than 3 cases in any year and no geographic clustering.

American Academy of Pediatrics

On April 6, 1998, the American Academy of Pediatrics (AAP) Committee on Environmental Health released a statement concerning toxic effects of indoor molds and acute idiopathic pulmonary hemorrhage in infants. They recommend that until more information is available on the etiology of this condition, pediatricians should try to ensure that infants under 1 year of age are not exposed to chronically moldy, water-damaged environments (AAP, 1998).

Sources of Additional Information/Assistance:

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