**For full article see: Epidemics of mold poisoning past and present**

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**A Summary Overview of Hurricanes and Mold Epidemics**

Extensive flooding from Hurricane Floyd that hit coastal North Carolina in 1999, Hurricane Rita that hit the gulf coast in 2004, and Hurricane Katrina that hit the Louisiana-Mississippi coast in 2004 led to wide- spread anecdotal reports of illness related to mold exposures in flooded buildings, but there is a paucity of documentation of the health effects associated with these exposures. An inspection of 112 buildings dam- aged by flood waters after Hurricanes Rita and Katrina found that mold growth was present in 45% of 112 inspected homes, with heavy mold growth in 17% of homes. Homes with flood levels greater than 6 feet were more likely to be affected. The most common species found were Aspergillus and Penicillium. Proper respiratory protection was lacking in those exposed, but health effects were not adequately studied (Centers for Disease Control and Prevention [CDC], 2006b).

A study of indoor air of homes flooded in Louisi- ana by Hurricane Katrina in 2005 found mold spore counts to be double those of non-flooded homes, with Cladosporium and Aspergillus being the most com- mon species found. Stachybotrys species were found in some homes. Endotoxin concentrations did not dif- fer between flooded homes, non-flooded homes, and outdoor air (Solomon et al., 2006).

When Hurricane Floyd hit coastal North Carolina in 1999, there was an increase in hospital admission for asthma. A Centers for Disease Control study of New Orleans firefighters and police officers motivated by reports of an increased number of illnesses after Hurricane Katrina found that upper respiratory symptoms of head and sinus congestion and throat and nasal irritation were very common. Skin rash was reported by 54% of police officers and 49% of firefighters. Neither the etiology nor extent to which mold contributed to these symptoms was determined (CDC, 2006a). After Hurricane Katrina, there was an outbreak of acute respiratory illnesses that increased with time over a 3-week period (Williams et al., 2006), though there is no data on specific etiol- ogies. It can be concluded that flooding after hurri- canes leads to visible mold growth in flooded buildings and increases in respiratory and dermal illnesses, but the extent to which molds contribute to these illnesses has not been adequately assessed.

**Stachybotrys chartarum**

S. chartarum is a mold that is found in water-damaged homes. It grows on high cellulose and low nitrogen content gypsum board, fiberboard, and other sub- strates, including compostable pots (Dill et al., 2004). Construction techniques now in use, with large amounts of fiberboard and gypsum board and moisture arising from leaky roofs, water pipes, and condensation, have produced an epidemic of Stachybotrys contamination of buildings. While this contamination is not controversial, the extent of the health consequences has the usual naysayers about emerging environmental-related illness. They produced position statements that there is insufficient evidence at this time for an association to be made between mycotoxins and building-related illnesses (Bush et al., 2006; Hardin et al., 2003). A contrary statement documenting the evidence has been produced (Curtis et al., 2004). Anecdotally, chemically sensitive individuals report illnesses related to the odors associated with moldy basements and similar damp environ- ments that are due to volatile mycotoxins, suggesting individual susceptibility may play a role in adverse reactions to airborne mycotoxins.

Hodgson and Dearborn (2002) have pointed out that though human evidence of an association between mycotoxins and human health is anecdotal and based on case reports and series, the standards generally regarded as evidence in environmental medicine are met, including controlled studies in animals, the development of animal models with diseases that parallel the human experience, and studies of the mycotoxins at subcellular levels (Hodgson and Dear- born, 2002 and references therein).

S. chartarum produces the T2 or tricothecene mycotoxins (Holstege et al., 2007) that, when concen- trated, produce a deadly cytotoxic syndrome by shutting down protein synthesis. Severe acute poisoning is characterized by multiple organ system failure and necrosis of bowel and airway, bone marrow dysfunction with pancytopenia, and cardiovascular collapse. Severe irritation of the skin, mucous membranes of the oral pharynx and respiratory tract, and conjunctiva occurs, which is somewhat unique among biological toxins relative to chemical toxins. While this devastating toxicity is not seen among inhabitants of moisture-damaged buildings, the presence of

S. chartarum in buildings, sometimes in association with Aspergillus species or other molds, has been associated with asthma, hypersensitivity pneu monitis, and the constellation of respiratory and neurological symptoms associated with poorly ventilated buildings (Cooley et al., 1998; Hodgson and Dearborn, 2002; Jarvis et al., 1998; Johanning et al., 1996; Straus and Wilson, 2008, 2009). Tri- chothecene mycotoxins have been detected in the sera of individuals from contaminated buildings by ELISA assays (Brasel et al., 2004).

A cluster of cases of pulmonary hemorrhagic and hemosiderosis occurred in a hospital nursery in Cle- veland during the period November 1994 to January 1995 and was investigated by the Centers for Disease Control. It was concluded that heavy mold growth in the building was responsible for the outbreak (CDC, 1997). This finding was retracted after internal and external reviews of the investigation, which cited the quality of evidence insufficient to conclude an association (CDC, 2000). A growing body of evidence sup- ports that S. Charatrum may be the cause of epidemics and isolated cases of pulmonary hemorrhage and hemosiderosis in infant lungs. An updated report did find an association, and infants who relapsed after returning home were more likely to have water damage in their homes (CDC, 2000 update). A case of pulmonary hemorrhage in an infant exposed to Stachybotrys has been reported in Delaware (Weiss and Chidekel, 2002). A strain of S. chartarum was isolated from the lung of a pulmonary hemorrhage and hemosiderosis patient in Texas, which produced toxins with biological plausibility for producing pulmonary hemorrhage and hemosiderosis (Vesper et al., 2000a). S. Charatrum was found in the home of a 1-month-old infant in Kansas City, who developed pulmonary hemorrhage and hemosiderosis (Flappan). The mycotoxin stachylysin is more likely to be found in strains of S. Charatrum isolated from homes of children with pulmonary hemorrhage and hemosiderosis than strains from control homes (Vesper et al., 2000b), suggesting a role of this mycotoxin that produces vascular leak in an animal model.

**Conclusion**

Throughout history, mold infestations have been recognized as a cause of disease epidemics, and in many cases, the causative mycotoxins have been iso lated and studied. Though controversy exists about the role mycotoxins play in current disease epidemics

associated with poorly ventilated buildings, sensitivity to indoor air, and airway inflammation and hemorrhage, there is a growing body of evidence that mandates open inquiry into these agents and their role in diseases.

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576

Toxicology and Industrial Health 25(9-10)

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