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Committee on Environmental Health

Toxic Effects of Indoor Molds

ABSTRACT. This statement describes molds, their toxic properties, and their potential for causing toxic respiratory problems in infants. Guidelines for pediatricians are given to help reduce exposures to mold in homes of infants. This is a rapidly evolving area and more research is ongoing.

ABBREVIATIONS. SIDS, sudden infant death syndrome; CDC, Centers for Disease Control and Prevention.

The growth of molds is pervasive throughout the outdoor environment. Given the proper conditions, molds may also proliferate in the indoor setting. Because Americans spend 75% to 90% of their time indoors,¹ they are exposed to molds that are growing indoors.

Molds readily enter indoor environments by circulating through doorways, windows, heating, ventilation systems, and air conditioning systems. Spores in the air also deposit on people and animals, making clothing, shoes, bags, and pets common carriers of mold into indoor environments. The most common indoor molds are *Cladosporium*, *Penicillium*, *Aspergillus*, and *Alternaria*.^{2,3}

Molds proliferate in environments that contain excessive moisture, such as from leaks in roofs, walls, plant pots, or pet urine.⁴⁻⁶ Many building materials are suitable nutrient sources for fungal growth. Cellulose substrates, including paper and paper products, cardboard, ceiling tiles, wood, and wood products, are particularly favorable for the growth of some molds. Other substrates such as dust, paints, wallpaper, insulation materials, drywall, carpet, fabric, and upholstery commonly support mold growth.³ Molds also may colonize near standing water.⁷⁻⁹

Some indoor molds have the potential to produce extremely potent toxins called mycotoxins.¹⁰⁻¹² Mycotoxins are lipid-soluble and are readily absorbed by the intestinal lining, airways, and skin.¹³ Species of mycotoxin-producing molds include *Fusarium*, *Trichoderma*, and *Stachybotrys*. In general, the presence of these molds indicates a long-standing water problem.

DIRECT TOXIC EFFECTS FROM MOLD EXPOSURE

The toxic effects from mold exposure are thought to be associated with exposure to toxins on the sur-

face of the mold spores, not with the growth of the mold in the body. Until recently, there was only one published report in the United States linking airborne exposure to mycotoxins with health problems in humans.¹⁴ This report described upper respiratory tract irritation and rash in a family living in a Chicago home with a heavy growth of *Stachybotrys atra* (also known as *Stachybotrys chartarum*). The investigators documented that this mold was producing trichothecene mycotoxins. The symptoms disappeared when the amount of mold was substantially reduced.

More recently, molds that produce potent toxins have been associated with acute pulmonary hemorrhage among infants in Cleveland, Ohio.¹⁵ In November 1994, physicians and public health officials in Cleveland reported a cluster of eight cases of acute pulmonary hemorrhage and hemosiderosis that had occurred during January 1993 through November 1994 among infants in neighborhoods of eastern metropolitan Cleveland.¹⁶ Two additional cases were identified in December 1994. Pulmonary hemorrhage recurred in five of the discharged infants after they returned to their homes; of these infants, one died from pulmonary hemorrhage.

A case-control study comparing those 10 infants who had acute pulmonary hemorrhage and hemosiderosis with 30 age-matched control infants from the same area in Cleveland¹⁷ revealed that the infants with pulmonary hemorrhage were more likely to have resided in homes with major water damage from chronic plumbing leaks or flooding (95% confidence interval = 2.6 to infinity). The quantity of molds, including the toxigenic fungus *Stachybotrys atra*, was higher in the homes of infants with pulmonary hemorrhage than in those of controls. Simultaneous exposure to environmental tobacco smoke appeared to increase the risk of acute pulmonary hemorrhage among these infants.

Stachybotrys atra requires water-saturated cellulose-based materials for growth in buildings. In studies conducted in North America, it has been found in 2% to 3% of home environments sampled.^{8,18} Although *Stachybotrys atra* has been associated with gastrointestinal hemorrhaging in animals that had consumed moldy grain,¹⁹ the fungus previously had not been associated with disease in infants. Infants may be particularly susceptible to the effects of these inhaled mycotoxins because their lungs are growing very rapidly. In an animal model, intranasal administration of toxic spores of *Stachybotrys atra* to mice resulted in severe interstitial inflammation with hemorrhagic exudates in the alveoli.²⁰

The county coroner re-examined all infant deaths

The recommendations in this statement do not indicate an exclusive course of treatment or serve as a standard of medical care. Variations, taking into account individual circumstances, may be appropriate.

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in Cleveland during January 1993 through December 1995 to determine whether pulmonary hemosiderin-laden macrophages were present in the lung tissue. Postmortem examinations were reviewed for all 172 infants who died during that period, including 117 deaths attributed to sudden infant death syndrome (SIDS). Pathologic lung specimens were sectioned, stained with Prussian blue, and screened for the presence of hemosiderin. The presence of hemosiderin-laden macrophages in alveoli indicates alveolar bleeding at least 2 days before death.²¹

Hemosiderin-laden macrophages were abundantly present in the lung tissue of nine (5%) infants. Of these nine deaths, two resulted from homicide, and one had a recent history of child abuse. The other six deaths that were accompanied by hemosiderin-laden macrophages in the lung thus may have been misclassified as deaths from SIDS. All six infants had lived in the same limited geographic area as the previously described cases of pulmonary hemosiderosis.

The extent of this problem in other areas of the United States is still unknown. Further investigation is needed to establish causation and prevent further health effects if the findings in Cleveland are confirmed in other areas.

CONCLUSION

Very little is currently known about acute idiopathic pulmonary hemorrhage among infants. This is a newly recognized problem and knowledge is expected to be evolving rapidly. In view of the severity of the problem, environmental controls to eliminate water problems and to reduce the growth of indoor molds are wise. Until more is known about the etiology of idiopathic pulmonary hemorrhage, prudence dictates that pediatricians try to ensure that infants under 1 year of age are not exposed to chronically moldy, water-damaged environments.

Coroners and medical examiners should consider using the recently published *Guidelines for Death Scene Investigation of Sudden, Unexplained Infant Deaths*, which includes a question about dampness, visible standing water, or mold growth.

Little is known about the prevalence of toxigenic molds in homes, nor is it clear how extensive measures must be to achieve environments sufficiently free of molds to avoid disease. Bulk mold must be removed, followed by a thorough cleaning with soap and water. Caution must be used, because it is possible that homeowners could actually increase the levels of mold spores in the air by attempting extensive clean-up efforts without guidance from a professional (a certified industrial hygienist or ventilation engineer). These specialists can be found in the yellow pages in the telephone directory under the listing for Industrial Hygiene Consultants. Additional research is needed before the most appropriate recommendations for home clean-up can be determined. Until then, interim guidelines have been formulated.

RECOMMENDATIONS

1. In areas where flooding has occurred, prompt cleaning of walls and other flood-damaged items with water mixed with chlorine bleach, diluted four parts water to one part bleach, is necessary to prevent mold growth. Never mix bleach with ammonia. Moldy items should be discarded.
2. Pediatricians should ask about mold and water damage in the home when they treat infants with idiopathic pulmonary hemorrhage. If mold is in the home, pediatricians should encourage parents to try to find and eliminate sources of moisture. Testing the environment for specific molds is usually not necessary. It appears to be important to clean up moldy conditions before the infant is discharged from the hospital to prevent recurrent pulmonary hemorrhage, although this needs further study. Interim clean-up guidelines are available through the Centers for Disease Control and Prevention (CDC), 1600 Clifton Rd, Atlanta, GA 30333.
3. Infants with idiopathic pulmonary hemorrhage must not be exposed to environments in which smoking occurs.
4. Pediatricians should report cases of idiopathic pulmonary hemorrhage and hemosiderosis to state health departments. A reporting form is available through the CDC.
5. Pediatricians should be aware that there is currently no method to test humans for toxigenic molds such as *Stachybotrys* or mycotoxins.
6. Infants who die suddenly without known cause should have an autopsy done including a Prussian blue stain of lung tissue to look for the presence of hemosiderin.

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National Institute of Environmental Health Sciences

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CONSULTANT

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REFERENCES

1. Lebowitz MD. Health effects of indoor pollutants. *Annu Rev Public Health*. 1983;4:203-221
2. Miller JD. Fungi as contaminants in indoor air. *Atmospheric Environ*. 1992;26:2163-2172
3. Gravesen S, Frisvad JC, Samson RA. *Microfungi*. Copenhagen, Denmark: Munksgaard Publishing; 1994

4. Solomon WR. Fungus aerosols arising from cold-mist vaporizers. *J Allergy*. 1974;54:222–228
5. Kozak PP, Gallup J, Cummins LH, et al. Currently available methods for home mould surveys: II. examples of problem homes studied. *Ann Allergy*. 1980;45:167–176
6. Fergusson RJ, Milne LJ, Crompton GK. Penicillium allergic alveolitis: faulty installation of central heating. *Thorax*. 1984;39:294–298
7. Kapyla M. Frame fungi on insulated windows. *J Allergy*. 1985;40:558–564
8. Miller JD, Laflamme AM, Sobol Y, et al. Fungi and fungal products in some Canadian houses. *Int Biodeterioration*. 1988;24:103–120
9. Pasanen P, Pasanen AL, Janunen M, et al. Water condensation promotes fungal growth in ventilation ducts. *Indoor Air*. 1993;3:106–112
10. Burge HA. Toxigenic potential of indoor microbial aerosols. In: Sandu SS, DeMarini DM, et al, eds. *Short-term Bioassays in the Analysis of Complex Environmental Mixtures*. New York, NY: Plenum Press; 1987
11. Jarvis BB. Mycotoxins and indoor air quality. In: Morey PM, Feeley JC, Otten JA, eds. *Biological Contaminants in Indoor Environments*. Philadelphia, PA: American Society for Testing and Materials; 1990
12. Hendry KM, Cole EC. A review of mycotoxins in indoor air. *J Toxicol Environ Health*. 1993;38:183–198
13. Kemppainen BW, Riley RT, Pace JG. Skin absorption as a route of exposure for aflatoxin and trichothecenes. *J Toxicol/Toxin Rev*. 1988;7:95–120
14. Croft WA, Jarvis BB, Yatawara CS. Airborne outbreak of trichothecene toxicosis. *Atmos Environ*. 1986;20:549–552
15. Centers for Disease Control and Prevention. Update: pulmonary hemorrhage/hemosiderosis among infants—Cleveland, Ohio, 1993–1996. *MMWR Morb Mortal Wkly Rep*. 1997;46:33–35
16. Centers for Disease Control and Prevention. Acute pulmonary hemorrhage/hemosiderosis among infants—Cleveland, January 1993–November 1994. *MMWR Morb Mortal Wkly Rep*. 1994;43:881–883
17. Montaña E, Etzel RA, Allan T, Horgan TE, Dearborn DG. Environmental risk factors associated with pediatric idiopathic pulmonary hemorrhage and hemosiderosis in a Cleveland community. *Pediatrics*. 1997;99(1). URL: <http://www.pediatrics.org/cgi/content/full/99/1/e5>
18. Kozak PP Jr, Gallup J. Endogenous mold exposure: environmental risk to atopic and non-atopic patients. In: Gammage RV, Kay SV, eds. *Indoor Air and Human Health*. Chelsea, MI: Lewis Publishers; 1985:149–167
19. Hintikka E-L. Stachybotryotoxicosis as a veterinary problem. In: Rodricks JV, Hesseltine CW, Mehlman MA, eds. *Mycotoxins in Human and Animal Health*. Park Forest, IL: Pathotox Publishers; 1977:277–284
20. Nikulin M, Reijula K, Jarvis BB, Veijalainen P, Hintikka E-L. Effects of intranasal exposure to spores of *Stachybotrys atra* in mice. *Fund Appl Toxicol*. 1997;35:182–188
21. Stewart S, Fawcett J, Jacobson W. Interstitial haemosiderin in the lungs of sudden infant death syndrome: a histological hallmark of 'near-miss' episodes? *J Pathol*. 1985;145:53–58

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