This Health Hazard Evaluation (HHE) report and any recommendations made herein are for the specific facility evaluated and may not be universally applicable. Any recommendations made are not to be considered as final statements of NIOSH policy or of any agency or individual involved. Additional HHE reports are available at http://www.cdc.gov/niosh/hhe/reports

HETA 92-0348-2361
OCTOBER 1993
FIRST UNITED METHODIST CHURCH
MANCHESTER, TENNESSEE

NIOSH INVESTIGATOR:
STEVEN W. LENHART, CIH

SUMMARY

The National Institute for Occupational Safety and Health (NIOSH) conducted a health hazard evaluation (HHE) at the First United Methodist Church in Manchester, Tennessee, at the request of the chairperson of the church's Board of Trustees. The request concerned evaluation of the health risks associated with worker exposures to old, deteriorated rock wool insulation and a large accumulation of bat droppings during the removal of these materials from the church's 5000 square foot attic. Rock wool is a mineral wool that is manufactured by melting and fiberizing naturally occurring basaltic rock or siliceous limestone. Exposure to rock wool can cause itching and adverse skin reactions, and eye and upper respiratory tract irritation.

A NIOSH researcher collected samples of bat droppings from the attic which were analyzed for the fungus, *Histoplasma capsulatum*. *H. capsulatum* is the etiologic agent of histoplasmosis, the most common pulmonary mycosis of humans and animals. Acute, severe pulmonary histoplasmosis usually occurs in small epidemics involving exposure to an aerosol containing numerous spores resulting from the disturbance of highly infected material. A primary source of *H. capsulatum* is soil, especially in regions of bird or bat habitats. While wind is probably the most important means of disseminating *H. capsulatum*, the fungus can survive and be transmitted from one location to another on the feet of both birds and bats. Unlike birds, bats can become infected with *H. capsulatum* and consequently may excrete the organism in their feces. Sixteen samples of bat droppings were analyzed by mouse inoculation for *H. capsulatum*, and *H. capsulatum* was isolated from all four mice inoculated with the material from one sample.

A health risk was determined by NIOSH to be associated with exposure to aerosolized rock wool and bat droppings contaminated with *Histoplasma capsulatum* during the removal of these materials from the church's attic. Recommendations were made to reduce exposures to these materials by spraying the rock wool and bat droppings with water prior to and during removal operations. Further, each removal worker was recommended to wear a NIOSH/MSHA-approved full-facepiece powered air-purifying respirator with high-efficiency filters, disposable protective clothing with a hood, disposable latex gloves under cotton work gloves, and disposable shoe coverings. Because the recommended ensemble of disposable personal protective equipment is more insulating than normal work clothing, precautions should be taken during removal activities to reduce the risk of heat stress-related illnesses.

Keywords: SIC 8661 (religious organizations), bats, *Histoplasma capsulatum*, histoplasmosis, respiratory protection, and rock wool.

INTRODUCTION

The National Institute for Occupational Safety and Health (NIOSH) conducted a health hazard evaluation (HHE) at the First United Methodist Church in Manchester, Tennessee, at the request of the chairperson of the church's Board of Trustees. The request concerned evaluation of the health risks associated with worker exposures to old rock wool insulation and a large accumulation of bat droppings during the removal of these materials from the church's attic.

The main sanctuary of the First United Methodist Church was built in 1885. Additions to the original structure were built in 1916 and 1948. During the 1948 construction, rock wool insulation was added to the attics of all three sections, an area of approximately 5000 square feet. In 1992, the church's Board of Trustees initiated a church renovation project which included replacing the old, deteriorated rock wool insulation with new fiberglass insulation. However, upon inspecting the attic, a large accumulation of guano was discovered that had been created over the past several years by a colony of bats residing there. Guano covered the entire area at a depth of 0.5 to 1 inch. Deeper deposits of 10 to 15 inches were found under the three major entry/exit locations for the bats and under roosting locations that appeared to be used most frequently. The droppings covered not only the insulation, but also the top of the rectangular duct work of the church's air-handling system. The first corrective measure taken was to screen the major entry/exit locations. NIOSH was then contacted for guidance concerning a personal protective equipment recommendation to be followed by the employees of the insulation removal contractor.

A NIOSH researcher collected sixteen samples of bat droppings from the attic of the church on September 17 and 18, 1992, which were analyzed for the fungus, *Histoplasma capsulatum*. Bat droppings were collected from eight sampling locations in the attic before and after treatment with a 10% household bleach solution to investigate the ability of sodium hypochlorite to disinfect material potentially contaminated with *H. capsulatum*. This final report presents the results of the analyses of these samples and provides recommendations for protecting workers who might be exposed to aerosols of rock wool dust and bat droppings during renovation activities.

BACKGROUND

Histoplasma capsulatum

Histoplasma capsulatum is a dimorphic fungus (i.e., exhibits growth in two different forms in different environments); it has a mycelial form at lower growth temperatures (optimal 25°C) and a yeast form when incubated at 35°C on enriched media.[1] The mycelial form is found in nature and is frequently designated as saprobic (i.e., derives its nutrition from dead or decaying organic matter), whereas the yeast form occurs in a host's tissue and is the pathogenic form. Hyphae, microaleuriospores (microconidia), and macroaleuriospores (macroconidia) are infectious particles of the mycelial form.[2] *H. capsulatum* infections in humans result predominantly from inhalation of these aerosolized spores. The spores of *H. capsulatum* are of a respirable size, with 70% to 95% reported by one author to have diameters less than 4.8 micrometers.[3]

H. capsulatum is the etiologic agent of histoplasmosis, the most common pulmonary mycosis of humans and animals.[4] Forty million people in the United States are estimated to have been infected by *H. capsulatum*, with approximately 500,000 new infections occurring each year.[4] Asymptomatic or mild infections due to *H. capsulatum* are the rule, whereas the serious chronic or disseminated types are fairly uncommon.[2] The extent of acute pulmonary involvement that a person experiences when infected with *H. capsulatum*, whether it be asymptomatic, mild,

moderate, or severe, depends on the inoculum dose and the immunologic status of the host.[2] Acute, severe pulmonary histoplasmosis usually occurs in small epidemics involving exposure to an aerosol containing numerous spores resulting from the disturbance of highly infected material. Symptoms of acute respiratory histoplasmosis, including fever and cough, occur within two weeks of exposure.[5] Approximately 95 per cent of histoplasmosis cases are inapparent, subclinical, or completely benign. These cases are diagnosed only by x-ray findings of residual areas of pulmonary calcification and a positive histoplasmin skin test. Resolution of the benign form confers a certain degree of immunity to reinfection and, in addition, varying grades of hypersensitivity to the antigenic components of the organism. As a consequence, massive reinfection may result in a fatal acute allergic reaction in a person with highly sensitized lungs.[6]

A small percentage of histoplasmosis cases may have a chronic progressive lung disease, a chronic cutaneous or systemic disease, or an acute fulminating, rapidly fatal, systemic infection. The latter form is particularly common in children.[6] In the United States, 1500 to 4000 hospitalizations and 25 to 100 deaths occur annually due to histoplasmosis.[1, 4] These estimates were made before 1980 and do not include the increasing incidence of opportunistic histoplasmosis in patients with acquired immunodeficiency syndrome (AIDS).[1] In addition to AIDS, a rapidly progressive opportunistic infection occurs in some patients with the lymphoma-leukemia-Hodgkin's group of diseases, or those on steroid therapy or other immunosuppressive agents.[6] "*H. capsulatum* is now considered a regularly encountered opportunist in these circumstances and appears to be involved in opportunistic infections more often than the other "true" pathogenic fungi."[6]

For many years, only the severe disseminated form of histoplasmosis was recognized, and the disease was thought to be uniformly fatal. However, in the mid 1940's it was shown that histoplasmin skin reactivity was common in asymptomatic individuals. The skin test antigen, histoplasmin, is a valuable epidemiologic tool.[1] However, a positive histoplasmin test merely indicates that a person has probably lived in an endemic region of the United States at one time, and the test by itself has limited diagnostic value.[1, 6]
In addition, the prevalence of histoplasmin reactivity at any given time underestimates the prevalence of all past and present infections, since the skin test may revert to negative over a period of time with no exposure.[5] The overall incidence of histoplasmin sensitivity in the United States is about 22%.[5] However, the risk of infection is not uniform, but varies from location to location. The region with the highest level of reactivity is the central United States, along the valleys of the Ohio, Mississippi, Missouri, St. Lawrence, and Rio Grande rivers.[1] In a series of studies conducted in the highly endemic area of Kansas City, it was found that, by age 20, between 80 and 90 per cent of the population had a positive histoplasmin skin test. The same is true in the Cincinnati-southern Ohio and southern Indiana region, southern Illinois, central Missouri, and areas of Kentucky, Arkansas, and Tennessee. Interestingly, the first documented human case of histoplasmosis in the United States was reported in 1932, in Tennessee, and epidemiologic surveys have implied that a positive histoplasmin skin test will be found in over 60% of the residents of this state.[7] Focal areas of high endemicity also occur in Michigan, Wisconsin, Minnesota, Georgia, and Louisiana.[6]

While an *H. capsulatum* infection is most often a pulmonary disease, or a systemically disseminated disorder, a multifocal choroiditis (inflammation of the vascular coat of the eye) termed "presumed ocular histoplasmosis" has been described by many investigators.[7] This disease was called ocular histoplasmosis throughout the early 1960's, even though evidence for ocular histoplasmosis was circumstantial since *H. capsulatum* has not been recovered from eye lesions, cultured, and recovered in an animal model.[8] Although structures suggestive of an organism have been found in such lesions, the identity of the fungus has been difficult to demonstrate.[7] A correlation between exposure to *H. capsulatum* and ocular abnormalities has been suggested from the results of epidemiologic studies, but the characteristic multifocal choroiditis has rarely been

reported in patients who have the typical forms of this disease.[7] While the results of laboratory tests suggest that presumed ocular histoplasmosis is associated with hypersensitivity to *H. capsulatum*,[9] the incident that converts asymptomatic to symptomatic presumed ocular histoplasmosis remains unknown.[7]

A primary source of *H. capsulatum* is soil, especially in regions of bird or bat habitats. While wind is probably the most important means of disseminating *H. capsulatum*, the fungus can survive and be transmitted from one location to another on the feet of both birds and bats.[6] The organism thrives in humid areas where large numbers of birds have roosted over a period of several years. It is found in association with old or unused chicken houses, and under blackbird/starling roosts. Bird excreta provides nutrients that promote the growth of the organism in the soil, although the requirements for growth are not precisely defined. Caves sheltering bats, and soil at the base of buildings fertilized by droppings from bats inhabiting the buildings, also often provide environmental conditions suitable for the existence and propagation of the fungus.[10] Unlike birds, bats can become infected with *H. capsulatum* and consequently may excrete the organism in their feces.[5] *H. capsulatum* has been isolated from bat guano collected from around the world, and by 1970, twenty-five bat species had been reported to harbor this organism. Isolations of *H. capsulatum* from bats captured in the United States have been extensive.[11]

While accumulations of bat droppings alone have been shown to be contaminated with *H. capsulatum*,[11-19] similar results have been reported far less frequently for samples taken from accumulations of bird droppings.[20] In avian habitats, the organism seems to grow preferentially where the guano is rotting and mixed with soil rather than in nests or fresh deposits.[6] Attempts to demonstrate the presence of *H. capsulatum* in the organs and excreta of birds have never proven them to be carriers of the organism.[18] It has been suggested that birds do not harbor *H. capsulatum* because the organism does not survive at elevated avian body temperatures of 41 to 42°C.[5] However, the same temperature has been recorded in certain bats (*Molossus major*) for which *H. capsulatum* was demonstrated from cultures of their internal organs.[18]

Exposure to accumulations of bird droppings alone can not be assumed to be risk-free, however, since disturbance of bird habitats are associated with a risk of infection by *Cryptococcus neoformans* and the development of cryptococcosis.[21] *C. neoformans*, an encapsulated yeast, is ubiquitous in the soil and in avian fecal material, such as pigeon droppings, which apparently provide a reservoir of organisms.[22] *C. neoformans* has the ability to use the creatine found in avian feces as a nitrogen source. There, it gains a competitive advantage over other microorganisms and multiplies exceedingly well in bird droppings.[21] *C. neoformans* has also been recovered from bat droppings and associated dusts during studies for which samples were also found to contain *H. capsulatum*.[14, 15, 19] Unlike outbreaks of other mycoses, outbreaks of cryptococcosis traced to environmental sources have not been described, and it is presumed that most people can mount adequate host defenses when exposed to the organism.[23] However, as with histoplasmosis, the prevalence of cryptococcosis is markedly increased among immunocompromised patients.[23] More detailed information on *C. neoformans* and cryptococcosis is available elsewhere.[22-24]

Rock Wool

Mineral wool fiber is one member of a family of products known collectively as man-made mineral fibers. Due to their synthetic amorphous nature, they have also been called man-made vitreous (glassy) fibers. These products share a common manufacturing origin in that they are all created from molten raw materials under highly controlled conditions. Mineral wool fiber was originally developed in the early 1900's for use in industrial and home insulation.[25]

Rock wool is a mineral wool that is manufactured by melting and fiberizing naturally occurring basaltic rock or siliceous limestone.[26, 27] The fibers produced by this process are collected as a wool; approximately 10% of the mass forms small mineral pearls instead of fibers.[28] The main components of rock wool are oxides of silicon, calcium, magnesium, aluminum, and iron.[26] Health risks are associated with rock wool from skin, eye, and respiratory exposures. Skin exposure to rock wool is known to cause severe itching and skin reactions on direct contact, and these reactions are best described as a primary irritant contact dermatitis induced mechanically by the fibers.[28] An intense itching can occur, affecting the hands, face, and neck that likely is the result of the release of histamine or kinins when fibers pierce the epidermis. This phenomenon, which is worse in hot, humid weather, usually resolves in a few weeks with continuous exposure through a poorly understood "hardening " process.[27] The eye irritation that may occur during exposure to rock wool dust is generally not an important problem, except in very dusty conditions, but can be prevented by wearing eye protection.[27]

Because of the various adverse health effects associated with respiratory exposure to asbestos fibers, there is concern for the potential health implications of inhalation exposures to man-made mineral fibers beyond the effects of upper respiratory tract irritation, such as pharyngitis and rhinitis that can be caused by exposure to unusually dusty conditions. Currently available data indicate that man-made mineral fibers represent a substantially lower order of health risk to humans in comparison to what has been associated with asbestos exposure.[27] "On the basis of epidemiological studies and experimental evidence, The U.S. Environmental Protection Agency[29] and the International Agency for Research on Cancer[30] have classified mineral wool, glass wool, and special purpose glass fibers as either possible or probable human carcinogens. These agencies have concluded that the scientific evidence is currently inadequate to support a determination that they are confirmed human carcinogens. Epidemiological studies have shown some evidence that respiratory cancer mortality rates may be significantly elevated in production workers. However, clear and consistent dose-response relationships are lacking; these are critical to supporting a causal relationship between exposure and production of cancer."[31]

The NIOSH Recommended Exposure Limits for mineral wool are 3 fibers (f)/cubic centimeter (cm^3) (fibers $\leq$ 3.5 micrometers (µm) in diameter and $\geq$ 10 µm long) and a Time-Weighted Average (TWA) of 5 milligrams/m^3 (total mineral wool dust).[32] Some manufacturers of man-made mineral fibers have established self-imposed guidelines ranging from 1 to 2 f/cm^3.[27] The Occupational Safety and Health Administration (OSHA) is reviewing its regulatory position regarding worker exposure to man-made mineral fibers.[33] One union organization has recommended that OSHA establish a Permissible Exposure Limit for respirable fibers of 1.0 f/cm^3 as a TWA for all man-made mineral fibers other than refractory ceramic fibers.[31] The basis for the recommendation is that this exposure concentration "is consistent with the current state of our understanding of health risks, including the suggestive but not conclusive indication that these fibers may possess the ability to produce respiratory system cancer in exposed workers."[31]

METHODS

Sixteen samples of bat droppings were collected from eight sampling locations in the attic of the First United Methodist Church in Manchester, Tennessee, and were analyzed for *H. capsulatum*. Only one bat was observed in the attic during the collection of bat dropping samples. Samples were collected from four locations of the attic above the main sanctuary, from two locations of the 1916 addition, and from two locations of the 1948 addition. Each sample was collected in a sterile, nonpyrogenic plastic 50-milliliter (ml) centrifuge tube. The volume of droppings collected at each sampling location ranged from 20 to 50 ml (approximately 7 to 17 grams). While collecting

samples, the NIOSH investigator wore a NIOSH/Mine Safety and Health Administration (MSHA)-approved full-facepiece powered air-purifying respirator with high efficiency filters, disposable protective clothing with a hood, disposable latex gloves, and disposable shoe coverings.

After collection of a sample from each of the eight sampling locations was completed, the remaining bat droppings at each sampling location were soaked with a bleach and water solution prepared by adding 7 ounces of Chlorox® bleach to 2 quarts of tap water. The resulting 10% bleach solution, containing approximately 5,000 parts per million of sodium hypochlorite, was sprayed at each sampling location. Eight additional samples of bat droppings were collected from the same sampling locations the next day to permit sufficient contact-time between the sodium hypochlorite solution and any microbial contamination.

The bat dropping samples collected at the First United Methodist Church were analyzed for *H. capsulatum* at the University of Cincinnati Medical Center in Cincinnati, Ohio. One-half gram of the material from each sample was diluted 1:20 (weight/volume) and 1 ml was injected intraperitoneally into mice. Four mice were tested for each sample. The mice were observed for four weeks and then sacrificed. The spleens were removed from the mice, homogenized, and the homogenate was streaked onto brain heart infusion agar plates supplemented with sheep erythrocytes, glucose and L-cysteine.

In addition, six samples (three samples collected before and three samples collected after treatment with the bleach solution) were subjected to direct plating by adding 100 microliters of the diluted material to culture plates that contained the same agar as described above. All plates were observed for growth for four weeks.

Personal breathing zone and area air sampling was planned to estimate worker exposures to aerosolized rock wool during the removal operations in the attic of the church. However, no air sampling was conducted because of scheduling conflicts.

RESULTS

H. capsulatum was isolated from all four mice inoculated with material from one of the two bat dropping samples collected at the base of the back wall of the original 1885 building. *H. capsulatum* was not isolated from any of the other inoculated mice, and no mold growth was found on any of the agar plates that were direct-plated. Although the sample containing *H. capsulatum* was collected after treatment with a 10% bleach solution, a conclusion on the effectiveness of such treatment cannot be reached based on the sampling results of this study.

DISCUSSION

Mice are extremely susceptible to infection with *H. capsulatum* spores, and infection of mice inoculated with single spores has been demonstrated experimentally.[34] However, while mouse inoculation is the most reliable method for detecting *H. capsulatum* in environmental samples such as the bat droppings collected during this study, the method has a disadvantage of requiring several weeks before results are available. The method also has the limitation of using only a very small portion of a sample. This limitation might explain why *H. capsulatum* was isolated from a sample collected at the same location from which a negative sample was collected on the previous day.

Nevertheless, the laborious and time-consuming procedure required for the isolation of this fungus from its natural sources remains the important factor that restricts more extensive investigation into ecological relationships. The expense, space, and personnel required for large-scale studies are also important limiting factors.[10] Direct isolation of *H. capsulatum* in culture from soil samples has been accomplished,[35] but the sensitivity of the method is inferior to mouse inoculation.[18]

To overcome the disadvantages associated with mouse inoculation, development of a simple and reliable technique is necessary for the detection of *H. capsulatum* in samples collected from its natural environment.[10] Researchers are currently experiencing success identifying pathogenic fungi in clinical samples using polymerase chain reaction (PCR) probe detection systems and chemiluminescent DNA probe assays.[36-39] PCR probe systems have an advantage over DNA probe assays in that identification of pathogenic fungi in samples can be accomplished directly, without the need to wait for the growth of isolates from culture. A PCR probe system would also be capable of analyzing a larger portion of a sample of material at one time than the very small portion used with the mouse inoculation method. More importantly, development of a PCR probe system for the analysis of *H. capsulatum* in environmental samples would reduce the time presently necessary for analysis using mouse inoculation from weeks to only a few days.

Disinfection of soils contaminated with *H. capsulatum* has been tried with various chemicals. Formaldehyde has fungicidal properties, and it has been shown to be the most effective of the chemical agents tried based on the performance of pre- and post-treatment sampling for *H. capsulatum*.[2] A 37% to 40% solution by weight (formalin) stabilized with 10% to 15% methanol has been the basic formulation used. For decontamination procedures outdoors, a 3% formalin solution has been found to be effective.[21, 40, 41] However, exposures to formaldehyde during soil disinfection operations have been reported to cause adverse health effects among applicators. Workers at one site reported burning eyes and mucous membrane irritation,[40] while workers at another site reported nausea with vomiting.[41]

In addition to soil disinfection, formaldehyde has also been reported to be effective for disinfecting *H. capsulatum*-infected accumulations of bat droppings in the attics of buildings, using formalin concentrations of 3%[15] and 4%.[19] Formaldehyde solutions should be used with caution since this chemical may cause adverse health effects following exposure via inhalation, ingestion, or dermal or eye contact.[42] Mild to unpleasant eye irritation occurs in acclimated workers at 2 to 10 ppm, and intolerable irritation (tissue damage possible) occurs at levels above 25 ppm.[42] Workers exposed to 0.3 ppm of formaldehyde have reported symptoms of upper respiratory and acute bronchial irritation during a work shift.[43] There have also been reports of primary skin irritation and allergic dermatitis as a result of skin contact with water solutions of formaldehyde. Although a threshold for the development of these skin conditions has not been clearly defined, it is estimated to be a water solution containing less than 5 percent formaldehyde.[44] Based upon the results of laboratory tests which have demonstrated carcinogenic and mutagenic activity of formaldehyde in animals, NIOSH and OSHA recommend that formaldehyde be handled in the workplace as a potential occupational carcinogen.[45, 46] NIOSH recommends that occupational exposures to formaldehyde be controlled to the lowest feasible limit.[45]

CONCLUSIONS AND RECOMMENDATIONS

H. capsulatum was isolated from one of sixteen samples of bat droppings collected in the church's attic. Therefore, precautions should be taken to protect workers from inhalation exposure to dust disturbed during the removal of the rock wool and bat droppings. Samples of bat droppings were collected and analyzed primarily to investigate the effectiveness of a bleach solution to disinfect the bat droppings. Because of the large accumulation of bat droppings in the church's attic and

because the church is located in an endemic region for *H. capsulatum*, it would have been prudent to assume that a health risk existed if none of the samples was positive, or even if no samples had been collected and analyzed.

Prior to the start of removal activities, the health risks associated with exposure to *H. capsulatum* should be communicated to each worker who might be exposed to bat droppings during the course of the project. Individuals with compromised cell-mediated immunity are at greater risk of clinical histoplasmosis should infection occur, so such workers should avoid exposure to all materials potentially contaminated with *H. capsulatum*. Also before removal activities begin, all entry/exit locations for bats should be eliminated.

To reduce the potential for aerosolization of both rock wool dust and bat dropping dust, these materials should be sprayed with water. Then, the dampened materials should be collected in heavy-duty trash bags, and immediately disposed of at a landfill. Because the water will evaporate over the course of the removal operation, additional water will need to be sprayed as needed. The addition of a surfactant (wetting agent), such as a small amount of detergent, to the water may improve the dust suppression ability of the water alone. Dust remaining in the attic after removal of the bulky material, should be removed with an industrial vacuum cleaner equipped with a high-efficiency particulate air (HEPA) filter.

Workers should wear personal protective equipment while spraying water on the rock wool and bat droppings, and while collecting these materials in plastic bags. A NIOSH/MSHA-approved full-facepiece powered air-purifying respirator with high efficiency filters, disposable protective clothing with a hood, disposable latex gloves under cotton work gloves, and disposable shoe coverings should provide adequate protection. Respirators should be used in accordance with the regulations of OSHA[47] and the recommendations of NIOSH.[48] Since the recommended ensemble of disposable personal protective equipment is more insulating than normal work clothing, sweat evaporation will be impeded during removal activities. Therefore, precautions should be taken during these activities to reduce the risk of heat stress-related illnesses. If possible, removal activities should be scheduled when temperatures in the attic can be expected to be relatively cool.

Health risks are associated with exposures to even low air concentrations of formaldehyde.[44] Therefore, alternative chemicals should be used to disinfect those materials for which removal is impractical, such as a large volume of contaminated soil. Household bleach is one possible alternative since it contains sodium hypochlorite, which has bactericidal and sporicidal properties. Household bleach also has the practical advantages of being readily available and less expensive than most other chemical bactericidal and sporicidal agents. However, a disadvantage of hypochlorites is that their activity is greatly reduced in the presence of organic matter.[49] Because of the limited number of positive samples collected during this study, the effectiveness of bleach solutions to disinfect bat droppings containing *H. capsulatum* could not evaluated. The effectiveness of bleach solutions or other disinfectants should be documented before their use is recommended for decontaminating environmental materials containing *H. capsulatum*.

REFERENCES

1. Mitchell TG [1992]. Systemic mycoses. In: Joklik WK, Willett HP, Amos DB, Wifert CM, eds. Zinsser microbiology. 20th ed. Norwalk, CT: Appleton and Lange, pp. 1091-1112.

2. Larsh HW [1983]. Histoplasmosis. In: DiSalvo AF, ed. Occupational mycoses. Philadelphia, PA: Lea and Febiger, pp. 29-41.

3. Furcolow ML [1961]. Airborne histoplasmosis. Bacteriological Reviews *25*:301-309.

4. Walker EM Jr., Gale GR [1991]. Fungistatic and fungicidal compounds for human pathogens. In: Block SS, ed. Disinfection, sterilization, and preservation. 4th ed. Philadelphia, PA: Lea and Febiger, p. 389.

5. George RB, Penn RL [1986]. Histoplasmosis. In: Sarosi GA, Davies SF, eds. Fungal diseases of the lung. Orlando, FL: Harcourt Brace Jovanovich, pp. 69-85.

6. Rippon JW [1988]. Medical mycology, the pathogenic fungi and the pathogenic actinomycetes. 3rd ed. Philadelphia, PA: W.B. Saunders Company, pp. 381-423.

7. Feman SS, Tilford RH [1985]. Ocular findings in patients with histoplasmosis. J Am Med Assoc *253*:2534-2537.

8. Ganley JP [1984]. Epidemiology of presumed ocular histoplasmosis. Arch Ophthalmol *102*:1754-1756.

9. Newell FW [1992]. Ophthalmology principles and concepts. 7th ed. St. Louis, MO: Mosby Year Book, p. 439.

10. Bernstein IL, Calpouzos L, Edmonds RL, Hasenclever HF, Leedom JM, Loosli CG, et al. [1979]. Impact of airborne materials on living systems. In: Edmonds RL, ed. Aerobiology: the ecological systems approach. Stroudsburg, PA: Dowden, Hutchinson and Ross, Inc., pp.199-274.

11. DiSalvo AF [1971]. The role of bats in the ecology of *Histoplasma capsulatum*. In: Ajello L, Chick EW, Furcolow ML, eds. Histoplasmosis proceedings of the second national conference. Springfield, IL: Charles C Thomas, pp. 149-161.

12. Emmons CW [1958]. Association of bats with histoplasmosis. Public Health Rep *73*:590-595.

13. Furcolow ML [1965]. Environmental aspects of histoplasmosis. Arch Environ Health *10*:4-10.

14. Gordon MA, Ziment I [1967]. Epidemic of acute histoplasmosis in western New York state. NY State J Med *67*:235-243.

15. Ajello L, Hosty TS, Palmer J [1967]. Bat histoplasmosis in Alabama. Am J Trop Med Hyg *16*:329-331.

16. Chick EW, Bauman DS, Lapp NL, Morgan WKC [1972]. A combined field and laboratory epidemic of histoplasmosis. Am Rev Respir Dis *105*:968-971.

17. Sorley DL, Levin ML, Warren JW, Flynn JPG, Gerstenblith J [1979]. Bat-associated histoplasmosis in Maryland bridge workers. Am J Med *67*:623-626.

18. Schwarz J [1981]. Histoplasmosis. New York, NY: Praeger Publishers, pp. 179-186.

19. Bartlett PC, Vonbehren LA, Tewari RP, Martin RJ, Eagleton L, Isaac MJ, Kulkarni PS [1982]. Bats in the belfry: an outbreak of histoplasmosis. Am J Public Health *72*:1369-1372.

20. Dean AG, Bates JH, Sorrels C, Sorrels T, Germany W, Ajello L, et al. [1978]. An outbreak of histoplasmosis at an Arkansas courthouse, with five cases of probable reinfection. Am J Epidemiol *108*:36-46.

21. Ajello L, Weeks RJ [1983]. Soil decontamination and other control measures. In: DiSalvo AF, ed. Occupational mycoses. Philadelphia, PA: Lea and Febiger, pp. 229-238.

22. Mitchell TG [1992]. Opportunistic mycoses. In: Joklik WK, Willett HP, Amos DB, Wifert CM, eds. Zinsser microbiology. 20th ed. Norwalk, CT: Appleton and Lange, pp. 1135-1157.

23. Levitz SM [1991]. The ecology of *Cryptococcus neoformans* and the epidemiology of cryptococcosis. Rev Infect Dis *13*:1163-1169.

24. Bodet CA, Graybill JR [1986]. Cryptococcal pulmonary disease. In: Sarosi GA, Davies SF, eds. Fungal diseases of the lung. Orlando, FL: Harcourt Brace Jovanovich, pp. 131-152.

25. TIMA [1988]. Health and safety aspects of mineral wool fiber. Stamford, CT: Thermal Insulation Manufacturers Association.

26. Singh J, Coffman MA [1991]. Man-made mineral fibers. In: Clayton GD, Clayton FE, eds. Patty's industrial hygiene and toxicology. 4th rev. ed. Vol. IB. New York, NY:Wiley-Interscience Publishers, pp. 289-327.

27. Lockey JE, Weise NK [1992]. Health effects of synthetic vitreous fibers. Clin Chest Med *13*:329-339.

28. Björnberg A, Löwhagen G-B [1977]. Patch testing with mineral wool (rockwool). Acta Dermatovener (Stockholm) *57*:257-260.

29. Vu VT [1988]. Health hazard assessment of nonasbestos fibers. Office of Toxic Substances, U.S. Environmental Protection Agency, Washington, DC.

30. IARC [1988]. IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans, man-made mineral fibres and radon. Vol. 43. Lyon, France: World Health Organization, International Agency for Research on Cancer.

31. Kojola WH, Moran JB [1992]. Exposure limits for man-made mineral fibers. position of the building and construction trades department, AFL-CIO. Appl Occup Environ Hyg *7*:724-733.

32. NIOSH [1992]. NIOSH recommendations for occupational safety and health compendium of policy documents and statements. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 92-100.

33. Mahoney DP [1992]. Controlling exposures to refractory ceramic fibers. Professional Safety *37*:25-38.

34. Ajello L, Runyon LC [1953]. Infection of mice with single spores of *Histoplasma capsulatum*. J Bacteriol *66*:34-40.

35. Smith CD, Furcolow ML, Tosh FE [1964]. Attempts to eliminate *Histoplasma capsulatum* from soil. Am J Hygiene *79*:170-180.

36. Bowman BH [1992]. Designing a PCR/probe detection system for pathogenic fungi. Clin Immunol Newsletter *12*:65-69.

37. Huffnagle KE, Gander RM [1993]. Evaluation of Gen-Probe's *Histoplasma capsulatum* and *Cryptococcus neoformans* AccuProbes. J Clin Microbiol *31*:419-421.

38. Woods JP, Kersulyte D, Goldman WE, Berg DE [1993]. Fast DNA isolation from *Histoplasma capsulatum*: methodology for arbitrary primer polymerase chain reaction-based epidemiology and clinical studies. J Clin Microbiol *31*:463-464.

39. Stockman L, Clark KA, Hunt JM, Roberts GD [1993]. Evaluation of commercially available acridinium ester-labeled chemiluminescent DNA probes for culture identification of *Blastomyces dermatitidis*, *Coccidioides immitis*, *Cryptococcus neoformans*, and *Histoplasma capsulatum*. J Clin Microbiol *31*:845-850.

40. Tosh FE, Weeks RJ, Pfeiffer FR, Hendricks SL, Greer DL, Chin TDY [1967]. The use of formalin to kill *Histoplasma capsulatum* at an epidemic site. Am J Epidemiol *85*:259-265.

41. Bartlett PC, Weeks RJ, Ajello L [1982]. Decontamination of *Histoplasma capsulatum*-infested bird roost in Illinois. Arch Environ Health *37*:221-223.

42. NIOSH [1988]. Occupational safety and health guidelines for chemical hazards. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 89-104, Supplement II-OHG.

43. Alexandersson R, Kolmodin-Hedman B, Hedenstierna G [1982]. Exposure to formaldehyde: effects on pulmonary function. Arch Environ Health *37*:274-283.

44. ACGIH [1992]. 1989 Supplementation documentation-formaldehyde. American Conference of Governmental Industrial Hygienists (ACGIH), Appl Occup Environ Hyg *7*:852-874.

45. NIOSH/OSHA [1980]. Current intelligence bulletin 34: Formaldehyde: evidence of carcinogenicity. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 81-111.

46. 57 Fed. Reg. 22290 [1992]. Occupational Safety and Health Administration: occupational exposure to formaldehyde; final rule. (codified at 29 CFR 1910.1048.)

47. Code of Federal Regulations [1992]. 29 CFR 1910.134. Washington, D.C.: U.S. Government Printing Office, Federal Register.

48. NIOSH [1987]. NIOSH guide to industrial respiratory protection. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 87-116.

49. Russell AD [1991]. Chemical sporicidal and sporostatic agents. In: Block SS, ed. Disinfection, sterilization, and preservation. 4th ed. Philadelphia, PA: Lea and Febiger, p. 389.

AUTHORSHIP AND ACKNOWLEDGEMENTS

Principal investigator:	Steven W. Lenhart, CIH Industrial Hygienist Industrial Hygiene Section
Sample analyses conducted by:	George S. Deepe, Jr., M.D. Associate Professor of Medicine College of Medicine University of Cincinnati Medical Center Cincinnati, Ohio 45267
Editorial and technical guidance provided by:	Millie P. Schafer, Ph.D. Research Chemist Methods Research Branch Division of Physical Science and Engineering
Report formatted by:	Donna M. Pfirman Office Automation Assistant Industrial Hygiene Section
Originating office:	Hazard Evaluations and Technical Assistance Branch Division of Surveillance, Hazard Evaluations and Field Studies

DISTRIBUTION AND AVAILABILITY OF REPORT

Copies of this report may be freely reproduced and are not copyrighted. Single copies of this report will be available for a period of 90 days from the date of this report from the NIOSH Publications Office, 4676 Columbia Parkway, Cincinnati, Ohio 45226 (513) 533-8287. To expedite your request, please include a self-addressed mailing label with your written request. After 90 days, copies of this report can be purchased from the National Technical Information Service (NTIS), 5285 Port Royal Road, Springfield, Virginia 22161 (703) 487-4650. Information regarding the NTIS stock number of this report may be obtained from the NIOSH Publications Office.

Copies of this report were sent to:

1. Chairperson of the Board of Trustees, First United Methodist Church (Manchester, Tennessee)

2. OSHA Region IV (Atlanta, Georgia)