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HISTOPLASMOSIS AND ITS IMPACT ON BLACKBIRD/STARLING ROOST MANAGEMENT

by A. R. Stickley, Jr.[1] and R. J. Weeks[2]

Abstract: Histoplasmosis is a common and sometimes serious fungal disease that primarily affects the lungs, but can also involve other parts of the human body. The disease is characterized by 3 major forms: acute pulmonary, chronic cavitary, and disseminated histoplasmosis. Two fungicides are effective in treatment of the disease, but serious side effects often result. The occurrence of the fungus, *Histoplasma capsulatum*, is associated with large quantities of bird and bat excreta that have come in contact with soil. Roost sites must generally be occupied by birds for at least 3 years before soil conditions are conducive to its growth. Once established, the fungus remains for years, but its development can be prevented by bird dispersal and lethal bird control techniques. Infested sites may, with difficulty, be decontaminated using formalin.

INTRODUCTION

A major aspect of the blackbird (Icterinae)/European starling (*Sturnus vulgaris*) winter roost problem in the Southeast is the threat of histoplasmosis to urban populations. This is a lung disease caused by a fungus (*Histoplasma capsulatum*) that sometimes develops in the soil of roosts. Progress has been made in determining the nature and extent of this disease as well as developing procedures to curb it. Yet, much of this information is not readily available to animal damage control specialists who must deal with the roost problem. To address this need the Denver Wildlife Research Center initiated in 1981 a cooperative agreement with the Centers for Disease Control, U. S. Public Health Service, to produce an interpretative review of the histoplasmosis literature for wildlife management professionals. This paper is a product of that agreement.

DISEASE CHARACTERISTICS

Histoplasmosis is a common and sometimes serious fungal disease of mammals, including man. It is contracted by disturbing the soil where the fungus *H. capsulatum* grows and by inhaling the spores or microconidia. The extent of illness is variable, ranging from a common but minor lung disease to a rapidly developing fatal illness involving the lungs and sometimes other organs. Until the late 1960's and early 1970's, it was commonly misdiagnosed as tuberculosis.

Three major forms of the disease are recognized. They are: acute pulmonary histoplasmosis, chronic cavitary histoplasmosis, and dessiminated histoplasmosis. Acute pulmonary histoplasmosis is characterized by pulmonary lesions detectable by chest x-rays. The onset of the disease is associated with chills, fever, muscle pain, and a non-productive cough. Treatment is not usually required.

Chronic cavitary histoplasmosis is characterized by a cough, sputum containing pus, anorexia, weakness, and fatigue. Unless this disease is treated, it will worsen and can lead ultimately to death.

Disseminated histoplasmosis

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results from spread of the fungus in the lungs throughout the body via the bloodstream. The acute form is characterized by high fever, loss of appetite and weight, and enlargement of the spleen and liver. Although chest x-rays may be clear, adrenal gland function is frequently affected, and death may result in a few weeks unless the patient receives antifungal therapy. In the chronic form, patients often have a low-grade fever, long-term weight loss, and borderline adrenal function. Ulcerative lesions of the mouth are the most common complaint.

Histoplasmosis of the eye, or "presumed ocular histoplasmosis" as it is known medically, is a serious eye disease that can lead to blindness. It is diagnosed by a "recognizable pattern" of localized inflammation in a particular area of the vascular membrane that overlays the retina. The connection between this disease and histoplasmosis is based only on the greater incidence of positive skin test reactions with histoplasmosis of the eye when compared with other types of retinal inflamation.

Two antibiotics, amphotericin B and ketoconazole, have been found to be effective in treating histoplasmosis; however, amphotericin B causes a number of adverse side reactions including damage to kidneys and adrenals. Ketoconazole, introduced only recently, has few side effects except for gastrointestinal distress and possible sterility.

DISEASE TRANSMISSION

H. capsulatum produces 2 types of spores: macronconidia (8-14 millimicrons in diameter); and microconidia (2-4 millimicrons in diameter), but only the microconidia are of concern. They easily become airborne when the soil is disturbed and can be inhaled into the lungs where they are deposited in the alveoli and initiate the infection. Histoplasmosis also can result from contact with items contaminated with the microconidia and from the importation and use of infested soil. In rare cases, infections have occurred in observers who watched from a distance as soil samples were being taken at an infested site and in laboratory technicians who processed soil samples for fungal isolation Infections have even occurred among family members of roost workers who have taken *H. capsulatum* microconidia home on their field clothes. However, human-to-human (note above exception) and animal-to-human transmission has not been reported.

ENVIRONMENTAL FACTORS

The occurrence of *H. capsulatum* is associated with large quantities of bird and bat excreta that have come in contact with soil. Thus chicken houses with accumulated manure are notorious sources of this fungus. In natural situations, *H. capsulatum* is often found where gregarious birds congregate, such as in large blackbird/starling roosts, ring-billed gull (*Larus delawarensis*) nesting colonies (Waldman et al. 1983), oil bird (*Steatornis caripensis*) colonies (Ajello et al. 1962), and rock dove (*Colomba livia*) roosts (Grayston and Furcolow 1953). The fungus can also be found in caves, hollow trees, and attics where bats roost in numbers.

H. capsulatum-infested soils have a higher nitrogen, phosphorus, and organic matter content, and a greater water-holding capacity than do uncontaminated soil (Vining and Weeks 1974). The fungus generally grows in the upper 2 to 12 cm of the soil, but can be found as deep as 37 cm (Smith et al. 1966). It survives within a pH range of 5 to 10. The fungus tolerates temperatures below $0^{o}C$ and can withstand $40^{o}C$ or higher for extended periods (Goodman and Larsh 1967). While soil moisture and high relative humidity encourage growth of the fungus (Menges et al. 1952), lack of moisture is not lethal. The conidia

are able to survive long periods in dry soil (Goodman and Larsh 1967). Although wet weather will reduce normal spore dissemination, no seasonal fluctuations in infection patterns for H. capsulatum are known. Epidemics of histoplasmosis, as well as sporadic cases, have occurred every month of the year.

Studies of the chemical and physical properties of soil and the distribution of fungi in bird roosts indicate that antagonistic soil factors together with the competitive activity of other soil organisms prevent the establishment, survival, and multiplication of H. capsulatum in soils not contaminated with bird or bat droppings (McDonough 1963). But once established in contaminated soil, it competes successfully with other organisms (Brandsburg et al. 1969), and is likely to be present for years (Smith et al. 1964).

Apparently roost sites must be occupied by birds for at least 3 years (or winters) before the soil becomes conducive to the growth of H. capsulatum (Chin et al. 1970). We investigated this phenomenon further in a complex of 11 roosts near Milan, Tennessee where the blackbird/starling roosting chronology had been recorded continuously from the late 1960's. The number of years the birds had used the roosts ranged from 1 to 5 years. H. capsulatum was isolated only from the 1 roost occupied for 5 years.

In Kentucky, Pass (unpubl. rpt.) cultured 83 of the 125 known bird roost sites in the State as of 1981. He found that 24 (29%) contained H. capsulatum. From these data, he drew the following profile of a histoplasmosis-positive roost: the average size was 4 ha; the average number of years the roost had been occupied by birds was 6; the average number of years between the time birds had last occupied the roost and the time that the roost was sampled for the fungus was 1.7 years; 44% of the roosts that had had 3 or more years of bird activity contained H. capsulatum; and 41% of the roosts that had contained a million birds or more were positive for the fungus.

How the fungus is spread from infested sites to susceptible ones has not been satisfactorily shown, but circumstantial evidence suggests that wind dissemination of spores is a major contributor. Tosh et al. (1966) not only found H. capsulatum in samples of accumulated dirt and dust on the roofs of buildings in Mason City, Iowa during a histoplasmosis epidemic, but also found a strong correlation between the incidence of histoplasmosis in the local population and the prevailing wind conditions from the infested roost site. Several studies (Furcolow 1961, Tosh et al. 1966, Chin et al. 1970, and Chick et al. 1981) have shown strong correlations between the incidence of histoplasmosis among human populations and distance from H. capsulatum-infested sites. Even though airborne spores have been found 1.5 m above infested chicken house floors (Ibach et al. 1954), their presence in free air samples away from infested sites has not been specifically reported.

Birds have been precluded as instruments of dissemination because their high body temperature (41-42°C) prevents growth of H. capsulatum (Menges and Habermann 1955). Although the asexual form of the fungus may be able to survive for a period of time at 41°C, it does not multiply. In a study of over 1000 blackbirds and starlings collected in a bird roost positive for H. capsulatum, the fungus was not isolated from feathers, feet, internal organs, or intestinal contents (Weeks, unpubl. data).

In contrast, bats may play a role in the dissemination of H. capsulatum (Zamora, 1977). Various bat species roost in attics, chicken houses, bird roosts, hollow trees, and caves--all areas where

the fungus is found. Theoretically bats could become infected from 1 of these H. capsulatum-positive sites, and could infect a new site favorable for growth of the fungus by depositing infected guano at the new site. Experimentally, natural soils made favorable for the growth of the fungus have been colonized by H. capsulatum derived from infected animal carcasses that were buried in the soil (Weeks, unpubl. data). After colonization, the fungus persisted for a number of years.

The Extent and Severity of Histoplasmosis

Histoplasmosis is found worldwide, and the highest skin reactor rates are generally found in tropical and subtropical climates. The United States is an exception because the highest reactor rates occur in the temperate east-central portions of the country. An estimated 500,000 people are infected by H. capsulatum in the United States yearly (CDC 1969). Hammerman et al. (1974) estimated that 2700 hospital cases of histoplasmosis occur yearly and result in 50 deaths in the United States.

The disease has been reported frequently in 31 of the 48 contiguous States. Edwards et al. (1969) found that the overall frequency of positive skin test reactions (a measure of past or present infections) in the United States was about 16% in 18 to 20 year old males. The average sensitivity rates of States ranged from 1% in Rhode Island to 69% in Kentucky. Ajello (1971) reported that positive skin test reactions were most prevalent in Arkansas, Kentucky, Missouri, and Tennessee where positive rates in excess of 50% were found in young men in both rural and urban areas. Rural rates are only somewhat less in Illinois, Indiana, Ohio, and Oklahoma where they were still above 50%. In addition to these high-prevalence States, others with one or more counties whose lifetime residents had prevalence rates of 50% or more were Alabama, Kansas, Louisiana, Maryland, Mississippi, Texas, and West Virgina.

The economic impact of histoplasmosis is considerable. In 1976 the number of patients hospitalized with a primary diagnosis of histoplasmosis was estimated to be 4600 (Fraser et al. 1979). The same study estimated that the hospital costs for these patients amounted to $3,780,000. The financial impact must also include the cost of time lost from employment during the hospitalization period, convalescence until complete recovery, and worker compensation payments.

Histoplasmosis is not a disease that by law must be reported; therefore, accurate determination of its effect is impossible. Its prevalence is probably underestimated since it mimics tuberculosis in those patients having pulmonary lesions. Although 28 States reported 551 acute cases of histoplasmosis to the CDC in 1979, 363 of these were in Missouri where knowledge of and interest in histoplasmosis was great.

Both wild and domestic mammals are susceptible to infections caused by H. capsulatum as demonstrated both by the use of serologic tests and by the isolation of the fungus from body organs. Infections have been found in cats, cattle, dogs, horses, and pigs, as well as in brown bears (Ursus arctos), grey (Urocyon cinereoargenteus) and red foxes (Vulpes vulpes), house (Mus musculus) and white-footed mice (Peromyscus leucopus), common opossums (Didelphis virginiana), raccoons (Procyon lotor), Norway (Rattus norvegicus), black (Rattus rattus) and spiny rats (Proechimyus semispinosas), least shrew (Cryptotis parva), spotted (Spilogale putorius) and striped skunks (Mephitis mephitis), 13-lined ground squirrels (Spermophilos tridecemlineatus),

woodchucks (Marmota monax) and several genera of bats (Menges 1971). A greater variety of mammals is probably susceptible and would be found to be infected if more thorough studies were conducted at infested environmental sites.

EPIDEMIOLOGY OF HISTOPLASMOSIS

Immunity is acquired after inhalation of a sublethal quantity of microspores, which result in extremely slight to very serious illnesses. Outbreaks of histoplasmosis occur most frequently among individuals who lack this immunity (demonstrate negative skin tests). In endemic areas, outbreaks frequently occur among families, particularly children, who "return to the farm" from urban areas where they have had no exposure to histoplasmosis (Lehan and Furcolow 1957). When they clean up and utilize old farm buildings that have housed chickens in the past, they often contract the disease.

Most serious outbreaks of histoplasmosis tend to occur in nonendemic or marginally endemic areas. These include cities in endemic regions where many residents do not have immunity (as indicated by the lack of positive skin tests). At least 15 major outbreaks of histoplasmosis were reported between 1960 and 1982 in urban areas where bird roosts had been disrupted by clearing. This activity resulted in infections contracted by roost workers as well as dissemination of the fungus throughout the communities. A small epidemic (4 cases) occurred in Mexico, Missouri when several troops of Boy Scouts cleaned up a city park that had been a bird roost about 4 years before (Furcolow et al. 1969). Similarly, as a result of a large roost in the middle of Hopkinsville, Kentucky being cleaned up and bulldozed, 15 residents became ill from histoplasmosis over a 22-month period (Latham et al. 1980).

These cases could have represented first-time infections among urban residents who, although living in highly endemic areas, had somehow not come into contact with the fungal spores of H. capsulatum. But some also could have resulted from massive exposure to microconidia, which have been known to trigger reinfections in previously exposed individuals, especially those who have not been exposed to the fungus for a period of time (Goodwin and Des Prez 1978). Positive skin tests will eventually revert to a negative condition unless the immunity is reinforced from time to time by contact with the conidia of H. capsulatum.

In endemic areas, histoplasmosis occurs primarily among residents living close to bird roost sites infested with the fungus. Several studies have compared histoplasmin skin test sensitivity rates (1) in school children in 2 towns, similar in size and environment, a few miles from each other (Chin et al. 1970), (2) in children who lived close to a H. capsulatum contaminated blackbird roost (Tosh et al. 1970), and (3) in a statewide population (Chick et al. 1981). These studies revealed that the presence of and the distance from a contaminated blackbird roost influenced the rates of histoplasmin positivity. They also showed that if an H. capsulatum contaminated roost was disturbed, positive skin test sensitivity rates in the surrounding poplations were greatly increased.

MANAGEMENT IMPLICATIONS

Some southeastern States tend to ignore or mimimize the problem of histoplasmosis and bird roosts. Others, however, such as Alabama, Arkansas, Kentucky, and Tennessee, with or without help from the Division of Wildlife Assistance, U. S. Fish and Wildlife Service, do have active bird dispersal and/or lethal roost control programs. When histoplasmosis epidemics strike, most States rely on the Division of Mycotic Diseases, Centers for Disease Control, U. S. Public Health

Service, to determine the presence or absence of H. capsulatum in roosts. However, Alabama, Missouri, and South Carolina have their own programs for determining the presence of the fungus in bird roosts. In lieu of collecting and culturing soil samples, Tennessee has developed a point system for determining the histoplasmosis hazard potential of roosts. Point values are assigned to site characteristics such as age of roost, visible droppings on the ground, and the distance from human population centers. A high point total means the site should be decontaminated before development.

Since only those roosts occupied by birds for 3 or more years have been shown to be infested with H. capsulatum, birds roosting at a site the first winter might be allowed to remain, unless nuisance complaints dictate otherwise. If the birds return the following fall or winter, they should be dispersed or removed unless it is known positively that birds have not roosted there before.

Whether roosting birds are dispersed or killed could depend on the existence of alternate roost sites. If nearby sites are available that birds have not occupied in the past, then dispersal might be the better option. If few alternate sites are available to birds and those that are have already been occupied for one or 2 years, lethal control should be considered.

There is little need to test a bird roost site for the presence of H. capsulatum unless it is being used frequently by the public or is about to be developed. If such a site is found to be positive, decontamination should be undertaken with care not to disturb the soil or alter the habitat any more than necessary. Lethal control should probably not be carried out until after the site has been decontaminated.

Various attempts have been made to remove the fungus from sites by causing biological and physical changes in the soil, and by using fungicidical chemicals (Weeks and Tosh 1971). The only techniques that have proven successful have involved the application of fungicides including cresol compounds, formalin solution, and pentachlorophenol in fuel oil.

Formalin has been found to be the most desirable and practical choice (Weeks and Tosh 1971). It is prepared commercially by diluting formaldehyde, which contains 37% formaldehyde gas by weight, with water. When air and soil temperatures are between 16 and 32°C, this aqueous solution is applied to the soil at the rate of 13.5 liters/m^2 of the surface area for each of 3 applications. Each application should be made on a separate day to allow the solution to penetrate the soil. Formalin, however, is a suspected carcinogen (Yodaiken, 1981), and proper application is difficult, potentially hazardous, and expensive (Bartlett et al. 1982). (Further application details are given in "Histoplasmosis Control", U. S. Department of Health, Education, and Welfare Brochure #00-3021.) There are several pest control companies in the Kentucky-Tennessee area that do this work commercially.

Decontamination of all suspected infested sites in endemic areas is neither realistic nor practical. Therefore, other suggestions to reduce the incidence and prevalence of infections in the general population are: 1. Encourage occupational or avocational groups who may have need to come into close contact with suspected positive roost soils to restrict such contact to those of their group with histoplasmin-positive skin tests. 2. Inform the public about the existence of roost sites that are potential sources of H. capsulatum and the means available to test and decontaminate, if necessary, the sites before they are disturbed. 3. Update the medical community regarding the prevalence of histoplasmosis, its

diagnostic characteristics, and the epidemiology of the disease. 4. Encourage the passage of laws for the mandatory reporting to State health departments of all cases of histoplasmosis in an effort to determine the public health importance of this disease.

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