

Louisiana



REPORTED MORBIDITY  
MARCH, 1978

RECEIVED  
MONTHLY MORBIDITY REPORT

TULANE UNIV.  
Provisional Statistics  
RUDOLPH HARTAS MEDICAL LIBRARY

DEPARTMENT OF HEALTH  
AND HUMAN RESOURCES  
OFFICE OF HEALTH SERVICES  
AND ENVIRONMENTAL QUALITY

from the

OFFICE OF PUBLIC HEALTH STATISTICS

*Louisiana editor's note:*

*Histoplasmosis and blastomycosis are important fungal diseases endemic in Louisiana. This month's Morbidity Report is concerned with histoplasmosis. Next month's issue will carry an article on blastomycosis. References for both will accompany next month's article.*

## HISTOPLASMOSIS - NORTHERN LOUISIANA\*

An outbreak of acute pulmonary disease, presumptively identified as histoplasmosis, occurred in early September 1977 in northwest Louisiana. The outbreak involved all 6 men who cleared a field of bamboo cane on August 25 and 26. The field was known to be a blackbird roosting site and was located in an area considered to be heavily endemic for histoplasmosis (1).

Although no birds were present when the work was done, the grounds and a doghouse in the middle of the field were covered with several inches of bird droppings. One man, in addition to clearing the cane, cleaned off the roof of the doghouse while the others stood nearby. After the cane was bulldozed, it was burned and buried, and a fresh topping of soil was applied to the field.

The first person to become ill was the oldest participant, a 38-year-old man, whose illness began with low-grade fever and generalized aching 6 days after exposure. Over the next few days he had abdominal cramps and diarrhea followed by increasing fever, cough, chest pain, and dyspnea. His chest X ray showed widespread miliary infiltrates. The patient was hospitalized and improved over the next 2 weeks. The other 5 workers, all college students in their early 20s, developed acute illness with symptoms of fever, body aches, cough, and dyspnea 11-13 days after exposure. Chest X rays on all showed widespread miliary infiltrates. Bacterial and fungal cultures of sputum and blood were negative.

One patient developed respiratory failure, was treated

with amphotericin B, and recovered. The others all recovered without specific anti-fungal therapy. Five had 4-fold or greater rises in titer to *Histoplasma capsulatum* yeast-phase complement fixation (CF) antigen, reaching a level of  $\geq 1:64$  in 4 patients and 1:32 in the fifth. The other man had a single titer of 1:16. Histoplasmin skin tests on 4 people, placed at the same time that the acute-phase serum specimens were drawn, were all negative. An aqueous suspension of the bird droppings from the doghouse showed structures identified as the tuberculate macroconidia of *H. capsulatum*. Cultures of the soil and the bird droppings from the doghouse roof

(Continued on page 2)

### HISTOPLASMOSIS

JOHN G. BURFORD, M.D.

Department of Medicine

Pulmonary Disease Section

L.S.U. School of Medicine in Shreveport

**DEFINITION:** Histoplasmosis is a common granulomatous disease of world-wide distribution caused by the pathogenic fungus *Histoplasma capsulatum*. Its manifestations range from mild, localized lesions to acute or chronic, generalized systemic disease. Approximately 80% of infections are asymptomatic and 15 to 20% present as mild to moderate influenza-like illnesses. Less than 1% will develop into progressive pulmonary histoplasmosis and less than .01% progressive disseminated histoplasmosis.

**INFECTIOUS AGENT:** *H. capsulatum* is a diphasic fungus. In moist soil enriched by the droppings of birds or bats, it exists as a saprophyte in mycelial phase. When this soil growth is disturbed, the infectious spores become airborne and gain entry into humans by inhalation. The organism then assumes its yeast form and resides in cellular cytoplasm, resulting in granulomatous inflammation. Spores are present in enormous numbers at "point sources of infection" (chicken coops, starling or black bird roosts, caves, etc.) but may be airborne in small numbers almost anywhere throughout an endemic area.

**EPIDEMIOLOGY:** On the basis of large scale epidemiological surveys using the histoplasmin skin test, it is estimated that approximately 40 million people in the United States have had *Histoplasma* infections, with a new case rate of about 500,000 infections per year. The overall histoplasmin sensitivity rate in the United States is about 20%. The

(Continued on page 3)

\* Reprinted from Morbidity and Mortality Weekly Report, Vol. 26, No. 45, Center for Disease Control, D.H.E.W., November 11, 1977, p. 375.

## HISTOPLASMOSIS - NORTHERN LOUISIANA

(Continued from page 1)

are pending.

Reported by ES Butler, MD, Haynesville, Louisiana; RB George, MD, E Kotcher, ScD, AD Oberle, MS, LSU Medical School, Shreveport; CT Caraway, DVM, MPH, State Epidemiologist, Louisiana State Health and Human Resources Administration; Field Services Div, Special Pathogens Br, Bacterial Diseases Div, Bur of Epidemiology, CDC.

**Editorial Note:** As is often the case with this disease, the organism was not cultured from any of the patients in this outbreak. The diagnosis is supported, however, by the clinical illness, X rays, and serologic and epidemiologic findings. Numerous reports have indicated that a single histoplasmin skin test may stimulate humoral antibodies to *H. capsulatum* antigens in histoplasmin-hypersensitive individuals. In one study of 139 individuals who were skin-tested and later bled, none of the 25 who had negative skin tests had measurable antibodies, while 12 of the 114 who were skin-test positive had CF titers  $\geq 1:8$ . Those 12 included 10 who responded to a level of  $\geq 1:8$  to the mycelial phase CF antigen and 2 who responded to a 1:8 level to the yeast-phase CF antigen. The highest titers observed were in 2 patients who had levels of

1:32 to the mycelial phase antigen (2).

In this outbreak the patients skin-tested were all skin-test negative and showed high titer serologic responses to the yeast-phase antigen. Thus, it is unlikely that the serologic responses are the result of skin-testing. As a general rule, however, skin-testing plays no role in the diagnosis of acute histoplasmosis since it usually does not provide helpful diagnostic information and potentially confuses the interpretation of serologic tests.

The danger of working with soil containing *H. capsulatum* organisms can be minimized if only workers with positive histoplasmin skin tests are involved and if the soil is decontaminated beforehand with 3% formalin.

### References

1. Edwards LD, Acquaviva FA, Livesay VT: An atlas of sensitivity to tuberculin, PPD-B, and histoplasmin in the United States. *Am Rev Respir Dis* 99: 1-132, 1969
2. Kaufman L, Terry RT, Schubert JH, and McLaughlin D: Effects of a single histoplasmin skin test on the serological diagnosis of histoplasmosis. *J Bacteriol* 94: 798-803, 1967

### Louisiana Editor's Note: Follow-up.

Bird droppings from the dog house roof and soil specimens collected from the cane field where the outbreak described here occurred have yielded *H. capsulatum*. All 6 patients involved have shown 4 fold rises in antibody titer to the yeast or mycelial antigen of *H. capsulatum*, as measured

by complement fixation. In addition, all 6 have shown antibodies to *H. capsulatum* on the immunodiffusion test. This result is specific for histoplasmosis, because unlike the complement fixation test the immunodiffusion test is not affected by cross reactions with other fungi.

## RUBELLA IMMUNIZATION OF POST-PUBERTAL FEMALES

Up to twenty percent of females of childbearing age may be susceptible to rubella. Public health officials recommend that females of childbearing age who have no record of rubella immunization be serologically tested to determine their susceptibility to rubella. Those found susceptible who agree to use a medically acceptable method to prevent pregnancy for 60 days should be vaccinated.

Health units throughout the state at present do not administer rubella vaccine to post pubertal females because of their inability to adequately screen for pregnancy or to provide contraceptive services under full time medical supervision. Health units have been advised, however, to test previously unvaccinated post-pubertal girls for rubella and to refer those without immunity to their personal physicians for vaccination.

Public health nurses have been authorized to administer the vaccine only by direct order of a physician. To avoid any misunderstanding between private physician and the health unit, it is strongly suggested that a physician who refers a patient to the health unit for vaccination write the orders and state that the patient is not pregnant and has agreed to a medically acceptable method to prevent pregnancy for 90 days.

The rubella hemagglutination inhibition test (HI) is performed at the Central Laboratory of the Office of Health Services and Environmental Quality (OHSEQ) in New Orleans. Blood or serum may be submitted directly or through one of the regional laboratories of OHSEQ. A titer of 1:10 or greater is considered adequate for protection and such individuals need not be vaccinated.



## HISTOPLASMOSIS

(Continued from page 1)

risk of being a histoplasmin reactor increased with increased contact with soil as evidenced by reactor rates of 11%, 20%, and 29% recorded for metropolitan, non-metropolitan non-farm, and farm areas, respectively.

Although histoplasmosis is considered to be endemic in 31 of the 48 contiguous states, Louisiana is one of 15 states in the central United States making up the major histoplasmosis region. The overall histoplasmin sensitivity rate in Louisiana is 27% with average rates of 18%, 34%, and 38% noted for metropolitan, non-metropolitan non-farm, and farm areas, respectively. Rates of 80 to 90% occur in some of the northern and central parishes. Infections are unevenly distributed, even in highly endemic areas, probably reflecting "point sources" of heavily contaminated soil.

Persons of all ages may be affected. The disease may be particularly virulent in infants, the elderly, and patients with lymphoma or on immunosuppressive therapy. Although symptomatic histoplasmosis is common in adult white males over 45 and uncommon in blacks, skin test surveys indicate that the overall incidence of infection is probably similar in blacks and females. Cases occur both sporadically and in outbreaks. The latter have usually occurred after blackbird roosts or old chicken coops have been disturbed (see accompanying article).

**CLINICAL MANIFESTATIONS:** Pulmonary histoplasmosis may be considered as acute or chronic. Virulent disseminated disease can occur with either form.

The symptoms and roentgenographic manifestations of acute pulmonary histoplasmosis probably are related both to the degree of recent exposure and prior history of exposure. Most cases of acute histoplasmosis have no specific symptomatology and probably reflect light spore exposure. Occurrence is documented by histoplasmin skin test conversion. The clinical picture with larger exposures is a mild to severe influenza-like illness beginning 7 to 21 days after exposure and lasting from a week to a month or more. Symptoms may include cough (which may or may not be productive), chills, fever, chest pain (which may or may not be pleuritic), headaches, malaise, and generalized aches and pains. A feeling of easy fatigability often persists for several weeks after other symptoms have cleared. The disease is characterized roentgenographically by small, patchy, soft areas of pneumonitis which vary from a simple focal area of infiltration to multiple infiltrates scattered throughout both lung fields. With very heavy exposure the patchy infiltrates may become confluent to produce widespread consolidation with "white lungs" on x-ray and severe hypoxemia. Prominent hilar adenopathy is usually also present and pleural effusions are rare. Healing of the foci of infection occurs by necrosis, caseation, fibrous encapsulation, and eventual calcification.

Symptomatic disease probably does not occur after a light spore reexposure in a previous sensitized host, but an acute illness can occur after heavy spore reexposure. The illness is similar to that of previously uninfected persons, but is usually milder with a shorter incubation period. The chest x-ray usually reveals fine nodulations without hilar adenopathy. Whether this illness represents a true reinfection or is a manifestation of cellular hypersensitivity in the previously sensitized host is unclear.

Chronic pulmonary histoplasmosis is thought to result from reinfection, either exogenous or endogenous. Reports suggest that both possibilities exist. Established underlying emphysematous chronic lung disease is a predisposing factor. Symptoms include mild fever, weight loss, malaise, anorexia, cough, and chest pain. The basic, or early, lesion appears to be an intense interstitial pneumonitis usually involving segmental areas of the apical-posterior regions of the lung. Radiographically the infiltrates often outline emphysematous spaces which appear as radiolucencies. New lesions may appear at the same time older lesions are healing. Although there is a general tendency for these lesions to heal, prolonged resolution which may occur over several months by progressive contraction and organization may result in the loss of considerable lung volume. Approximately 20% of the early lesions develop persistent cavitation. This form of the disease is slowly progressive, is associated with pulmonary fibrosis, and if untreated usually leads to fatal pulmonary insufficiency.

Although the majority of hematogenous disseminations of *H. capsulatum* are benign self-limited processes occurring in the prehypersensitivity period of acute pulmonary histoplasmosis, on rare occasions virulent disseminated disease does occur in both the acute and chronic pulmonary diseases. Infants, the elderly, and patients with lymphoma or on immunosuppressive therapy are most commonly affected. The clinical spectrum ranges from an acute fulminant febrile form, usually seen in infants, characterized by wasting, hepatosplenomegaly, anemia, and leukopenia to a more slowly progressive form, usually seen in adults, characterized by specific organ inflammation and tissue destruction. In the latter category are found gastrointestinal ulcerations, endocarditis, adrenal gland destruction with Addison's disease, diffuse meningitis or central nervous system focal lesions, and nodular ulcerative lesions of skin and mucous membranes. The virulent disseminated disease is usually fatal when untreated.

**DIAGNOSIS:** Since definitive diagnosis can only be obtained by culture of *H. capsulatum* or its morphological demonstration in clinical specimens, proper collection and prompt processing of specimens is mandatory. After vigorous mouth rinsing, sputum should be freshly coughed into a sterile container and promptly delivered (*H. capsulatum* cells die quickly in clinical materials kept at room temperature) to the microbiology laboratory for processing. As the standard potassium hydroxide (KOH) preparation seldom demonstrates the fungal elements in histoplasmosis, a sputum smear should also be stained by the Giemsa or Wright method. The sputum should be plated on at least two culture media, preferably Sabouraud's agar and blood agar. At least 6 sputum specimens on successive mornings should be obtained in suspect cases. When sputum cannot be produced by coughing, induction by aerosol inhalation should be attempted. If induction is also unsuccessful, bronchoscopy to obtain bronchial aspirates and brushings and transbronchial lung biopsy should be considered. Other clinical specimens often useful for examination and culture in selected cases include the buffy coat of venous blood, bone marrow, spinal fluid, urine, and lymph nodes. Stains of tissue specimens should include the



## HISTOPLASMOSIS

(Continued from page 1)

risk of being a histoplasmin reactor increased with increased contact with soil as evidenced by reactor rates of 11%, 20%, and 29% recorded for metropolitan, non-metropolitan non-farm, and farm areas, respectively.

Although histoplasmosis is considered to be endemic in 31 of the 48 contiguous states, Louisiana is one of 15 states in the central United States making up the major histoplasmosis region. The overall histoplasmin sensitivity rate in Louisiana is 27% with average rates of 18%, 34%, and 38% noted for metropolitan, non-metropolitan non-farm, and farm areas, respectively. Rates of 80 to 90% occur in some of the northern and central parishes. Infections are unevenly distributed, even in highly endemic areas, probably reflecting "point sources" of heavily contaminated soil.

Persons of all ages may be affected. The disease may be particularly virulent in infants, the elderly, and patients with lymphoma or on immunosuppressive therapy. Although symptomatic histoplasmosis is common in adult white males over 45 and uncommon in blacks, skin test surveys indicate that the overall incidence of infection is probably similar in blacks and females. Cases occur both sporadically and in outbreaks. The latter have usually occurred after blackbird roosts or old chicken coops have been disturbed (see accompanying article).

**CLINICAL MANIFESTATIONS:** Pulmonary histoplasmosis may be considered as acute or chronic. Virulent disseminated disease can occur with either form.

The symptoms and roentgenographic manifestations of acute pulmonary histoplasmosis probably are related both to the degree of recent exposure and prior history of exposure. Most cases of acute histoplasmosis have no specific symptomatology and probably reflect light spore exposure. Occurrence is documented by histoplasmin skin test conversion. The clinical picture with larger exposures is a mild to severe influenza-like illness beginning 7 to 21 days after exposure and lasting from a week to a month or more. Symptoms may include cough (which may or may not be productive), chills, fever, chest pain (which may or may not be pleuritic), headaches, malaise, and generalized aches and pains. A feeling of easy fatigability often persists for several weeks after other symptoms have cleared. The disease is characterized roentgenographically by small, patchy, soft areas of pneumonitis which vary from a simple focal area of infiltration to multiple infiltrates scattered throughout both lung fields. With very heavy exposure the patchy infiltrates may become confluent to produce widespread consolidation with "white lungs" on x-ray and severe hypoxemia. Prominent hilar adenopathy is usually also present and pleural effusions are rare. Healing of the foci of infection occurs by necrosis, caseation, fibrous encapsulation, and eventual calcification.

Symptomatic disease probably does not occur after a light spore reexposure in a previous sensitized host, but an acute illness can occur after heavy spore reexposure. The illness is similar to that of previously uninfected persons, but is usually milder with a shorter incubation period. The chest x-ray usually reveals fine nodulations without hilar adenopathy. Whether this illness represents a true reinfection or is a manifestation of cellular hypersensitivity in the previously sensitized host is unclear.

Chronic pulmonary histoplasmosis is thought to result from reinfection, either exogenous or endogenous. Reports suggest that both possibilities exist. Established underlying emphysematous chronic lung disease is a predisposing factor. Symptoms include mild fever, weight loss, malaise, anorexia, cough, and chest pain. The basic, or early, lesion appears to be an intense interstitial pneumonitis usually involving segmental areas of the apical-posterior regions of the lung. Radiographically the infiltrates often outline emphysematous spaces which appear as radiolucencies. New lesions may appear at the same time older lesions are healing. Although there is a general tendency for these lesions to heal, prolonged resolution which may occur over several months by progressive contraction and organization may result in the loss of considerable lung volume. Approximately 20% of the early lesions develop persistent cavitation. This form of the disease is slowly progressive, is associated with pulmonary fibrosis, and if untreated usually leads to fatal pulmonary insufficiency.

Although the majority of hematogenous disseminations of *H. capsulatum* are benign self-limited processes occurring in the prehypersensitivity period of acute pulmonary histoplasmosis, on rare occasions virulent disseminated disease does occur in both the acute and chronic pulmonary diseases. Infants, the elderly, and patients with lymphoma or on immunosuppressive therapy are most commonly affected. The clinical spectrum ranges from an acute fulminant febrile form, usually seen in infants, characterized by wasting, hepatosplenomegaly, anemia, and leukopenia to a more slowly progressive form, usually seen in adults, characterized by specific organ inflammation and tissue destruction. In the latter category are found gastrointestinal ulcerations, endocarditis, adrenal gland destruction with Addison's disease, diffuse meningitis or central nervous system focal lesions, and nodular ulcerative lesions of skin and mucous membranes. The virulent disseminated disease is usually fatal when untreated.

**DIAGNOSIS:** Since definitive diagnosis can only be obtained by culture of *H. capsulatum* or its morphological demonstration in clinical specimens, proper collection and prompt processing of specimens is mandatory. After vigorous mouth rinsing, sputum should be freshly coughed into a sterile container and promptly delivered (*H. capsulatum* cells die quickly in clinical materials kept at room temperature) to the microbiology laboratory for processing. As the standard potassium hydroxide (KOH) preparation seldom demonstrates the fungal elements in histoplasmosis, a sputum smear should also be stained by the Giemsa or Wright method. The sputum should be plated on at least two culture media, preferably Sabouraud's agar and blood agar. At least 6 sputum specimens on successive mornings should be obtained in suspect cases. When sputum cannot be produced by coughing, induction by aerosol inhalation should be attempted. If induction is also unsuccessful, bronchoscopy to obtain bronchial aspirates and brushings and transbronchial lung biopsy should be considered. Other clinical specimens often useful for examination and culture in selected cases include the buffy coat of venous blood, bone marrow, spinal fluid, urine, and lymph nodes. Stains of tissue specimens should include the

# SELECTED REPORTABLE DISEASES

(By Place of Residence)

STATE AND PARISH TOTALS Reported Morbidity March, 1978	ASEPTIC MENINGITIS	DIPHTHERIA	ENCEPHALITIS	ENCEPHALITIS, POST INFECTION	HEPATITIS A AND UNSPECIFIED	HEPATITIS B	TUBERCULOSIS, PULMONARY	MENINGOCOCCAL INFECTIONS	PERTUSSIS	RABIES IN ANIMALS	RUBELLA*	SEVERE UNDERNUTRITION	SHIGELLOSIS	TYPHOID FEVER	OTHER SALMONELLOSIS	TETANUS	MEASLES	GONORRHEA	SYPHILIS, PRIMARY AND SECONDARY
TOTAL TO DATE 1977	0	0	2	0	161	33	142	42	0	1	8	2	11	0	14	1	55	4129	144
TOTAL TO DATE 1978	1	0	0	0	148	41	138	27	1	3	175	3	20	0	12	1	249	5278	163
TOTAL THIS MONTH	1	0	0	0	81	19	53	9	1	1	144	2	4	0	4	1	134	1893	58
ACADIA							1	1									2	12	
ALLEN																		5	
ASCENSION							1	1				2						5	
ASSUMPTION																		0	
AVOUELLES																		8	
BEAUREGARD																		9	
BIENVILLE																		1	1
BOSSIER						2	1											23	
CADDO					5	2	1			1								201	1
CALCASIEU						3	3											95	1
CALDWELL					1													1	
CAMERON					2										2		1	1	
CATAHOULA							1											1	
CLAIBORNE						1												1	
CONCORDIA																		2	1
DESOTO																		6	
EAST BATON ROUGE					1	1	7	1			9		2				37	170	9
EAST CARROLL																		4	1
EAST FELICIANA							1										2	1	
EVANGELINE						1	1										2	2	
FRANKLIN																		2	
GRANT																		2	1
IBERIA							1				2					1	3	7	1
IBERVILLE									1									11	6
JACKSON																		3	
JEFFERSON					35	1	1	1			65							82	3
JEFFERSON DAVIS											1						1	7	
LAFAYETTE								1									1	55	2
LAFOURCHE					2						1							10	
LASALLE																		1	
LINCOLN							1											33	
LIVINGSTON							1										2	2	
MADISON					1													11	
MOREHOUSE																		10	
NATCHITOCHE											10						2	3	
ORLEANS	1				24	6	11	2			1		2					729	10
OUACHITA							3				21						5	102	1
PLAQUEMINES															1			4	
POINTE COUPEE							1											2	
RAPIDES						1	4	1			5						35	82	9
RED RIVER																			
RICHLAND							2											3	2
SABINE																		4	
ST. BERNARD					1													2	
ST. CHARLES																		5	
ST. HELENA																		2	
ST. JAMES											1							6	
ST. JOHN							2											6	
ST. LANDRY											3							9	
ST. MARTIN					1		1										8	9	
ST. MARY																	2	1	4
ST. TAMMANY					8													18	1
TANGIPAHOA																		27	4
TENSAS																		1	
TERREBONNE							2										1	12	
UNION							1										1	12	
VERMILION											23						29	4	
VERNON																		6	
WASHINGTON							1	1			1							30	
WEBSTER							3				1							5	
WEST BATON ROUGE							1											9	
WEST CARROLL																		2	
WEST FELICIANA															1			1	
WINN						1													
OUT OF STATE																		4	

\* Includes Rubella, Congenital Syndrome

From January 1 through March 31, 1978, the following cases were also reported: 1 - Brucellosis; 3 - Malaria (Contracted outside the U.S.A.); 1 - Psittacosis.