A Review

Relationship of Histoplasma Capsulatum to Avian Habitats

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FOLLOWING the discovery of the benign form of histoplasmosis by Christie and Peterson in 1945 (*1*), a great deal of progress has been made in gaining knowledge concerning this disease. In fact, so much has been learned that one of the greatest current difficulties is how to acquaint a larger number of physicians and diagnostic laboratories with the wealth of information on hand so that it can be used effectively.

Histoplasmosis is a pulmonary disease caused by the mold, *Histoplasma capsulatum*. This disease develops basically in one of four different forms (*2*). Its most prevalent manifestation is that of an asymptomatic, benign lung infection. Skin-test surveys have shown that in the principal endemic areas, such as the Mississippi-Ohio River basin of the United States, 80–90 percent of the population has been infected. A relatively smaller number of persons, about 10 percent, who inhale the airborne spores of *H. capsulatum*, develop a pulmonary infection that is not self-limited and which spreads internally. This disseminated form of histoplasmosis is potentially fatal unless it is promptly diagnosed and specific chemotherapy is initiated.

Chronic pulmonary histoplasmosis is a recently described clinical entity. It may be mistakenly diagnosed as tuberculosis unless appropriate laboratory tests are carried out. This form of histoplasmosis has been intensively studied by Furcolow and his co-workers and brought to the attention of the medical world (*3*). They found that as many as 6 percent of the patients in tuberculosis sanatoriums may be victims of *H. capsulatum* rather than *Mycobacterium tuberculosis*.

Acute pulmonary histoplasmosis is the most dramatic form of that disease. It occurs among individuals who have inhaled massive numbers of *H. capsulatum* spores. Although this form of the disease may be severe and disabling, most victims recover.

Diagnosis of all these forms of histoplasmosis is readily accomplished with the aid of appropriate serologic, histological, and mycological procedures. Skin tests and serologic techniques such as complement fixation, precipitin, and fluorescent antibody tests are quite specific and invaluable diagnostic tools. Selective isolation and growth media, as well as histological stains, have been developed for the definitive identification of *H. capsulatum* in clinical materials.

This paper summarizes the current status of information regarding one facet of our knowledge concerning *H. capsulatum*, namely the ecological relationship of this fungus to avian habitats.

H. capsulatum is a soil fungus with a wide geographic distribution (table 1). It has not only been isolated repeatedly from soil specimens, but both its microconidia and macroconidia have been demonstrated in soil, air, and water (*5, 12, 21, 26–31*). The occurrence of these

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spores in nature unequivocally proves that *H. capsulatum* actively grows and multiplies in soil as a saprophyte and does not merely reside there in a passive state. This conclusion is based on the fact that conidia are not produced by *H. capsulatum* in its parasitic phase.

Significantly, in Mexico (Coahuila, Colima, Nuevo Leon, Tamaulipas), the United States (New Mexico, Texas), and Venezuela (Lara, Zulia), *H. capsulatum* occurs in the *Coccidioides immitis* endemic areas. This does not imply that these completely different molds co-exist side by side in a given site. It does, however, indicate that ecological conditions suitable for each of them can and do occur within a particular region. These conditions are governed by such factors as altitude, rainfall, temperature, and soil types. The existence of *H. capsulatum* in the *C. immitis* endemic areas of the southwestern United States, based not only on soil findings but records of animal infections (*32*, *33*), probably accounts for most, if not all, of the high frequency of human reactions to histoplasmin found among lifetime residents of that area by Edwards and Palmer (*34*) and considered by them to be nonspecific. It seems reasonable to believe that those histoplasmin reactions were specific and not representative of infections by other fungi that cross-react with *H. capsulatum*.

Ecological studies carried out in Williamson County, Tenn., by Zeidberg and co-workers (*35–37*), during 1952–55, first revealed that *H. capsulatum* is not uniformly distributed in nature. It was found that chicken habitats favored the development of this mold. Soil samples collected at random yielded few isolates of *H. capsulatum*, but specimens collected in and around chicken yards, especially chicken coops, gave a higher percentage of positive cultures (tables 2–4).

Precisely what role chickens play in the occurrence and development in soil is unknown. It is known, however, that chickens are not carriers of the fungus. Naturally infected birds have not been discovered, and efforts to infect chickens with *H. capsulatum* have failed. I believe that chicken droppings may condition

Table 1. Areas endemic for Histoplasma capsulatum, confirmed by isolations from soil

Area	Area
North America	*Central America*
United States:	Republic of Panama (*13–15*)
Alabama (*4*)	*Caribbean Area*
Arkansas (*4*)	Trinidad (*16,17*)
Connecticut (*5*)	*South America*
District of Columbia (*6*)	Brazil:
Florida (*4*)	Bahia (*18*)
Georgia (*7*)	French Guiana (*4*)
Illinois (*4*)	Peru (*4,19*):
Indiana (*4*)	Cusco
Iowa (*4*)	Huanuco
Kansas (*4*)	Venezuela (*4,20–22*):
Kentucky (*4*)	Lara [1]
Maryland (*4*)	Merida
Michigan (*8*)	Miranda
Minnesota (*4*)	Monagas
Mississippi (*9*)	Zulia [1]
Missouri (*4*)	Federal District
New Mexico (*10*) [1]	*Africa*
New York (*4*)	Republic of the Congo (Leopoldville):
Ohio (*4*)	Katanga (*23*)
Oklahoma (*4*)	Tanganyika
Pennsylvania (*4*)	Amboni Caves (*24*)
Tennessee (*4*)	Union of South Africa
Texas (*4*) [1]	Transvaal (*4*)
Virginia (*4*)	*Asia*
West Virginia (*4*)	Malaya (*25*)
Wisconsin (*4*)	
Mexico:	
Coahuila (*11*) [1]	
Colima (*12*) [1]	
Nuevo Leon (*11*) [1]	
Tamaulipas (*11*) [1]	

[1] Also endemic for *Coccidioides immitis*.
NOTE: Numbers in parentheses are references.

Table 2. Results of examinations of soil samples collected at random from 112 premises, by source of sample, Williamson County, Tenn., July 1950 to March 1952

Source of sample	*Histoplasma capsulatum*		
	Samples examined	Isolations	
		Number	Percent
Total	493	28	5.7
Under dwelling	83	6	7.2
Near dwelling	136	[1] 1	.7
Inside chickenhouse	71	13	18.3
Chicken yard	32	8	25.0
Barnyard	44	0	
Inside barn	14	0	
Bank of watercourse	64	0	
In open	33	0	
Other	16	0	

[1] Grossly contaminated with chicken manure.
SOURCE: Reference 36.

soil in such a manner that *H. capsulatum* gains a differential advantage over other soil micro-organisms and thus is able to grow vigorously and compete successfully.

Chickens are not the sole species of birds associated with *H. capsulatum* habitats. This mold has been recovered from sites enriched with the dung of grackles, *Quiscalus quiscula* (*9*, *38*); oil birds, *Steatornis caripensis* (*16*, *39*, *40*); pigeons, *Columba livia* (*41*); and starlings, *Sturnus vulgaris* (*8*, *42*, *43*).

Avian habitats or soil obtained from such areas and transported elsewhere have been incriminated on numerous occasions as the source of outbreaks of acute pulmonary histoplasmosis (*8*, *42*, *43*). The public health importance of the ecological relationship between birds and *H. capsulatum* has been strongly documented. The ecology of *H. capsulatum*, however, is not simply explained on the basis of an avian-fungus relationship. A similar correlation exists between bats and *H. capsulatum* (*16*). On the other hand, negative correlations also occur. In many areas inhabited by birds and bats, *H. capsulatum* has never been isolated. Furthermore, cases of histoplasmosis have occurred in certain areas without apparent association with the birds and bats of the region (*7*, *44*). The biological and chemical properties of different soil types, climate, and the biochemical activities of soil micro-organisms also play an important role in determining where *H. capsulatum* can successfully grow (*45–47*).

Thus, although current knowledge on the ecology of *H. capsulatum* is impressive, it is still inadequate and a full understanding of the complex factors that govern the occurrence of *H. capsulatum* in soil has yet to be gained. Nevertheless, much of a practical and useful value may be accomplished with the available information. We have efficient techniques with which to discover new endemic areas for *H. capsulatum* and to pinpoint the source of outbreaks of infection. With adequate dissemination of information on the inherent risk of acquiring histoplasmosis while cleaning chicken coops, handling chicken manure, wrecking old buildings, and inhaling spore-laden dust from other avian habitats, the number of outbreaks of acute histoplasmosis that occur in many areas of the world may be reduced.

Table 3. Influence of habitat on occurrence of Histoplasma capsulatum in Williamson County, Tenn.

Habitat	Number of samples	Recovery of *H. capsulatum*	
		Isolations	
		Number	Percent
Chicken areas	54	21	38. 9
Other	46	6	13. 0
Total	100	27	27. 0

SOURCE: Reference 48.

Table 4. Influence of shelter on occurrence of Histoplasma capsulatum in soil

Source of sample	Number of samples	Isolations of *H. capsulatum*	
		Number	Percent
Total	100	27	27. 0
Chickenhouse	39	18	46. 2
Open chicken yards	15	3	20. 0
Under house	26	5	19. 2
In open	15	1	6. 7
Under barn	3	0	--------
Other	2	0	--------

SOURCE: Reference 48.

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Public Health Service Staff Appointments

Dr. Martin M. Cummings has been appointed director of the National Library of Medicine, the world's largest biomedical library and the nation's principal resource for published information in the biological and medical sciences.

Dr. Cummings, associate director for research grants, National Institutes of Health, since May 1963 and chief of NIH's Office of International Research since March 1961, succeeded Dr. Frank P. Rogers, who retired September 1, 1963.

Dr. Cummings, who was commissioned in the Public Health Service in 1946, came to NIH from the University of Oklahoma Medical School, where he had been chairman and professor of microbiology.

Born in Camden, N.J., in 1920, Dr. Cummings received a B.S. degree from Bucknell University in 1941 and his M.D. from Duke University in 1944. His special interests were chest diseases, including particularly tuberculosis and sarcoidosis. On these and other infectious diseases he has written more than 75 scientific papers, textbooks, and special publications.

Harry P. Kramer was appointed director of the Public Health Service's Robert A. Taft Sanitary Engineering Center in Cincinnati, Ohio, on October 1, 1963. He had been chief of the training program at the center since 1955.

Mr. Kramer came to the center in 1949 to help develop the training program. He had served as sanitary engineer and chemist with the City of Chicago.

Joseph E. Flanagan, Jr., long-time assistant director of the center, who had been serving as acting director, retired from the Public Health Service October 1 and is now associate director of the newly established department of environmental health of the American Medical Association in Chicago.