History of Human Parasitic Infections

By ERNEST CARROL FAUST, Ph.D.

A RÉSUMÉ of human infections from parasitic organisms in the southeastern United States from the earliest records until recent times is logically divided into four periods: infections in the American Indian before the coming of the white man; from the earliest colonial period until about 1850; from 1850 until the end of the 19th century; and from 1900 until recent times.

Before the Coming of the White Man

Very little is known about parasitic infections among the Indians. There is no indication that malaria was present (*1*), but by inference intestinal protozoiases must have been relatively common since they have been found in all indigenous populations in regions where surveys have been made. The Indians must have had the common intestinal roundworms (*Ascaris* and *Trichocephalus*) for the removal of which the Cherokees employed crude oil of chenopodium, obtained from the seeds of the native plant *Chenopodium ambrosioides* var. *anthelminticum*, and possibly also the rhizomes of the pinkroot, *Spigelia marilandica* (*2*). Their dogs probably harbored *Ancylostoma braziliense* and possibly *Ancylostoma caninum*, but the human hookworms are believed to have been introduced later from the Eastern Hemisphere.

When serious epidemics developed in Indian communities the inhabitants burned their homes and resettled on uncontaminated soil (*3*).

From 1607 to 1850

During early colonial days the explorers and settlers who came from Europe brought with them the white man's common contagious diseases, notably smallpox, measles, scarlet fever and tuberculosis and, to a lesser extent, vivax malaria and hookworm infection (due to *Ancylostoma duodenale*). Much more serious parasitic diseases were those imported in African slaves, including virulent tropical strains of vivax malaria, malignant falciparum malaria, the subtly developing quartan malaria, tropical hookworm infection, Bancroft's filariasis, schistosomiasis, and probably other parasitic diseases indigenous to Africa (*4*). Among the latter it may be reasonably assumed that relatively virulent strains of *Entamoeba histolytica* were introduced.

Malaria

All three species of malaria parasites found an appropriate mosquito host in the native *Anopheles quadrimaculatus*, which bred more and more prolifically as virgin soil was broken for the cultivation of rice in the Carolina lowlands and for sugar cane in the Louisiana bayou country. The malaria-infected Negro slaves served as the reservoir for infection of the mosquito. The mosquito in turn transmitted the

Dr. Faust is the William Vincent professor of tropical diseases and hygiene and head of the graduate department of parasitology, Tulane University of Louisiana. His most recent book, just published, is entitled, "Animal Agents and Vectors of Human Disease."

parasites to other Negroes and to the white population. This situation caused rapid development of highly malarious communities. Later, with people settling in the fertile Ohio and Mississippi Valleys, malaria spread rapidly. By 1850 there were almost solid areas of intense malariousness from Baltimore south to Georgia, Alabama, and central Florida, inland to Cincinnati and St. Louis, and down to the Gulf of Mexico. From these highly endemic areas vivax malaria became established throughout almost the entire United States except for mountainous and desert regions, while the more tropical *falciparum* infection remained entrenched in the southeast (*1*).

Hookworm Infection

Human hookworm infection caused by the more tropical parasite *Necator americanus* became gradually disseminated throughout the moist sandy humus soils of the southeast, extending from Virginia to southern Illinois and down to the gulf, and as far westward as eastern Texas. Here again the Negro was the source of the infection as he polluted the soil with his excreta which contained the eggs of the parasite. After hatching and larval growth on the soil, the parasite was infective for all persons who stepped barefooted on the infested ground. Soon hookworm infection was contracted by the white laborers in areas adjacent to the bottom lands.

The earliest clinical records of hookworm disease in the United States were from Florida in 1845 and Louisiana in 1850, only a few years after Dubini described the Old World human hookworm (*A. duodenale*) from Italy and a half century before the tropical hookworm *N. americanus* was specifically described.

Other Infections

The colonists and those who later emigrated and settled inland from the Atlantic and gulf coasts were familiar with the cosmopolitan intestinal roundworms. Some species, like the pinworm *Enterobius vermicularis*, they brought with them from Europe; others, like *Ascaris lumbricoides* and *Trichocephalus trichiurus*, they may have acquired in part from the Indians, and in larger part from the Negroes who worked on the plantations. The evidence supporting these conclusions is indirect but relatively satisfactory, based on the descriptions of roundworms in the American medical texts of the late 18th and early 19th centuries and on the prescription of many native plants which were supposedly effective as intestinal vermifuges (*5*) and listed in the early editions of the United States Formulary (*Allium cepa*, *Allium sativum*, *Angelica archangelica*, *C. ambrosiodes* var. *anthelminticum*, *Convolvulus jalapa*, *Ferula assafoetida*, *Juglans regia*, *Laurus camphora*, *Melia azedarach*, *Spigelia anthelmia*, *S. marilandica*, *Tanacetum vulgare*, *Valeriana officinalis*, and *Veratrum sabadilla*). Moreover, persons who ate inadequately cooked beef must have acquired beef tapeworm (*Taenia saginata*) infection and, similarly, those who ate rare pork, infection with pork tapeworm (*Taenia solium*). Likewise, they probably became infected with *Trichinella spiralis*, which was first reported from hogs by Joseph Leidy of Philadelphia, in 1846.

Bancroft's filariasis, caused by *Wuchereria bancrofti*, was introduced in the African slaves (*4*) and became established in the domestic mosquito *Culex quinquefasciatus* as a biological vector. However, there is no information during this period as to the geographic distribution or prevalence of this disease.

Blood fluke infections, caused by *Schistosoma mansoni* and *Schistosoma haematobium*, which were likewise introduced into the Western Hemisphere in African slaves (*4*), failed to find sufficiently susceptible molluscan intermediate hosts in North America, never became established here, and did not survive the death of the human hosts who brought them into the country.

Primitive conditions for disposal of human excreta, contaminated water supplies, infrequent bathing among the laboring and even other classes, and various other defects in personal and community hygiene favored transmission and high prevalence of infections produced by the intestinal protozoa. Yet there is no direct evidence of their presence or frequency although it seems justifiable to assume that a considerable amount of the common dysentery of these decades, referred to as "bloody flux," was due to the pathogenic ameba, *E. histolytica*.

By 1850 most of the parasitic diseases of the southeast and those transmitted by arthropods or due to bacteria had reached their peak, had developed formidable plateaus of incidence, or were subject to serious periodic epidemics (*3*). At least two of our medical schools, the Medical College of South Carolina at Charleston and the Medical College of Louisiana, later named Tulane, at New Orleans, were established to train physicians to combat malaria, yellow fever, epidemic typhus fever, cholera, and endemic intestinal diseases.

Today it is readily understood that all of the environmental conditions which favor many of the infectious diseases were present during those decades in the southeastern States, namely (*a*) for malaria—newly cultivated, poorly drained land for the breeding of the anopheline mosquito vector; (*b*) for yellow fever—prevalence of *Aedes aegypti* transmitters in cisterns and other fresh water containers around the home; (*c*) for typhus fever—infrequent bathing and washing of clothes, resulting in body lice among both the poor classes and the socially elite; and (*d*) for the intestinal diseases—improper disposal of human excreta, contamination of food and drinking water, and carelessness in personal hygiene. While the slaves and poorer whites suffered most, the better educated and well-to-do persons were by no means exempt and at times had proportionately higher morbidity and mortality rates.

From 1850 to 1900

During the years 1862–65, when Northern soldiers were quartered in the South, there was a notable increase in malaria and dysentery, particularly in and around the Army camps. During this quadrennium the annual malaria morbidity rate in the Federal forces in North Carolina was 1,087 per 1,000 mean strength; in Tennessee, 848; and in the gulf area, 803 (*6*). Moreover, there were nearly 2 million reported cases of dysentery in the Northern Army, with 44,558 deaths attributed to this cause (*3*).

Following Appomatox, the South was deprived of its agricultural labor, the land remained untilled, and malaria mosquitoes bred without restraint (*1*). As late as 1881 the malaria death rate per 100,000 population was 428 in Shreveport, 318 in Vicksburg, 171 in Baton Rouge, 100 in New Orleans, and comparably high in other malarious areas of the southeast.

Epidemics of yellow fever of major or minor proportions were reported from one or more southern port cities annually from 1850 through 1900, except for 7 years—1861 (during blockade of southern ports by Federal forces), 1881, 1885, 1886, 1891, 1895, and 1896 (*7*). There were severe epidemics of dengue during 1849–60, 1873–76, 1880, and 1896–98. Between 1847 and 1853 there were about 1,200 cases of louse-borne typhus fever annually in the New Orleans Charity Hospital, originating from importation of the disease from Ireland and Mexico (*8*). Cholera arrived on boats from Europe. During 1849–53 and again in 1873, it caused heavy mortality at the port of New Orleans and was spread to communities far inland through the Mississippi, Ohio, Missouri, and Platte River Valleys (*9*).

From 1900 to 1950

The etiology and natural history of many of the common parasitic and arthropod-transmitted diseases prevalent in the southeast became known during the 1890's and the following decade. These included the role of *Anopheles* in the malaria life cycle, of *A. aegypti* in urban yellow fever, and of the body louse in epidemic typhus fever. As early as 1876, Manson had demonstrated that the household mosquito *C. quinquefasciatus* was an intermediate host and vector of Bancroft's filariasis (*10*). By 1900 the life cycle of the human hookworms was likewise a matter of record (*11*), but it was not until the 1920's that specific information was provided on the epidemiology and life cycle of *Ascaris*, as well as the etiology of cutaneous larva migrans.

Amebiasis

Although *E. histolytica* had been described by Lösch in 1875 and its pathogenic role in the production of disease was conclusively demonstrated during the next 25 years by workers in Europe, Egypt, Philadelphia, Baltimore, and Texas, its life cycle involving man was first directly demonstrated in 1913 when Walker and

Sellards in the Philippines fed cysts of this ameba to 20 human volunteers and obtained infection in 18, with dysenteric symptoms in 4 (*12*). Epidemiological surveys were begun in 1913 to determine the prevalence of, and methods of exposure to, amebiasis in different groups of the population in the United States, that is, urban vs. rural, clinic patients vs. apparently well persons, children vs. adults, and institutionalized vs. noninstitutionalized groups. These studies demonstrated a higher average incidence among noninstitutionalized individuals in the southeast than in other areas, due probably to less careful personal and group hygiene (*13*).

Malaria

The first two decades of the 20th century saw considerable retreat of malaria along the northern border of the hyperendemic areas in the southeast, primarily as a result of better drainage of farmland. In contrast, there was no appreciable decrease in malariousness in the southeast (*14*). During the depression of the 1930's there were notable increases in morbidity and mortality from malaria in this area. Then widescale control was instituted by cooperation of Federal and State public health agencies, consisting of scientific drainage and other antilarval measures, screening of homes, chemotherapy, and more adequate diets among the poorer classes in the population.

Beginning in 1942 malaria vector control was carried out intensively within and around military training bases in the southeast, and, as soon as DDT was in supply, this insecticide was used effectively both as a larvicide (*15*) and an imagocide. More accurate blood film diagnosis of the malaria parasites so that malaria and typhoid fever were no longer confused (*16*), together with treatment of human carriers with quinacrine and later with chloroquine, practically terminated the chances for the remaining anophelines to pick up the infection and transmit it to other persons.

Although our local *A. quadrimaculatus* was shown to be readily susceptible to foreign strains of human plasmodia (*17*), as a result of effective control measures only a very few incidental transmission cycles developed in the United States following the return of many thousands of American troops from malarious areas during World War II, and more recently from Korea. Thus, by 1950 malaria had ceased to be a public health problem in any previously malarious area in the United States (*18*).

Hookworm Infection

Soon after the discovery and naming of the hookworm *N. americanus* in 1902 by Charles Wardell Stiles, surveys were undertaken to ascertain the clinical and public health importance of hookworm infection in the southeastern United States. It was discovered that this parasite had extensive distribution, especially in the nonmountainous areas, and was responsible for much serious illness, an appreciable number of deaths, and untold economic loss.

In 1915 the Rockefeller Foundation undertook studies in the southeast to obtain more exact epidemiological information, with the objective of developing effective control (*19*). In cooperation with State public health agencies all of the endemic areas were surveyed, after which anthelmintic treatment was administered to the hundreds of thousands of infected individuals (*20*). Sanitary methods for disposal of human excreta were instituted, and education concerning hookworm epidemiology and its prevention became a part of the public health programs of all communities in which the disease was prevalent.

These measures were successful in reducing the heavy hookworm burden in all but a few localities (*21*) although incidental hookworm infection remained throughout much of the previously heavily endemic area (*22*).

Cutaneous Larva Migrans

The disease, cutaneous larva migrans or creeping eruption, is restricted mostly to the South Atlantic and the gulf coasts, particularly on both coasts of northern Florida. Clinical and experimental studies of Kirby-Smith and associates between 1917 and 1927 demonstrated that the etiological agent is a nonhuman strain of *A. braziliense*, exposure to which occurs when persons lie on beaches or otherwise come in contact with the infective-stage larvae in places where dogs or cats harboring the adult worms have previously defecated (*23*).

Ascariasis

Inquiry into the geographic distribution and epidemiology of ascariasis was undertaken in 1927 by Cort and his associates, a few years after the life cycle of the etiological agent, *A. lumbricoides*, had been elucidated by Ransom and his associates (*24*, *25*). It was found that in the southeastern United States this was fundamentally an infection of young children, who seeded the dooryard with *Ascaris* eggs (*26*). Later, the same children and their playmates became infected by getting the embryonated eggs in their mouths—eating contaminated soil on play objects or fingers and then swallowing the eggs. With a few notable exceptions, ascariasis in the southeast was found to be predominantly prevalent in the southern extensions of the Appalachian highlands. Headlee (*27*), who studied the epidemiology of ascariasis in New Orleans, emphasized that it is primarily a familial infection. Up to 1950 no sustained or extensive programs had been undertaken to control this infection.

Visceral Larva Migrans

The dog ascarid, *Toxocara canis*, has been found to produce a serious childhood disease designated as visceral larva migrans. When a child swallows fully embryonated eggs of this parasite, the eggs hatch in the child's duodenum and undertake a lung journey, just as occurs in human *Ascaris* infection. However, since man is not a particularly suitable host for the dog ascarid, granulomatous tissue reaction almost invariably traps the migrating larvae, most frequently in the liver, but at times in other organs and tissues. A pronounced sensitization reaction results, with prolonged high eosinophilia. The infection is incapacitating but not usually fatal. Discovery of the etiology of this disease (in New Orleans) has been so recent that its prevalence and geographic extent are essentially undetermined (*28*).

Strongyloidiasis

Strongyloides stercoralis is prevalent in warm moist climates; in the United States it occurs mostly in the gulf coast areas where there is a relatively high ground water level, as in the bayou country of Louisiana (*29*); elsewhere throughout most of the southeast the infection is relatively sporadic. This nematode is biologically interesting since under favorable conditions it can carry out one or more complete life cycles as a free-living organism on the soil, while at other times it propagates exclusively by internal autoinfection (*30*).

Whipworm Infection

In connection with epidemiological studies on hookworm and *Ascaris*, infection with the whipworm, *T. trichiurus*, has been found to be widely disseminated in the southeast (*31*, *22*). Most reports indicate that the average worm burden is relatively light, but young patients living in unsanitary rural environments occasionally have a high worm burden, with dysentery and other manifestations of severe colitis.

Enterobiasis

Infection with the pinworm, *E. vermicularis*, is common in children throughout the southeast, but is no more prevalent here than it is in cooler climates. The development of the NIH swab (cellophane) and more recently of the Graham swab (scotch tape) for recovery of the eggs deposited by the female worms migrating outside the anus, has provided much easier and more accurate diagnosis than fecal examination for eggs (*32*). In our area as elsewhere the infection is primarily familial or institutional.

Trichinosis

Surveys to determine the incidence of human infection with *T. spiralis* have been conducted in several localities in the southeast, usually employing digestion or pressed muscle examination of routine necropsies to demonstrate the larval stage. The percentage of positive cases has been relatively low (2.8 to 10.0) save for one report of 33.0 from Alabama. With few exceptions the larval count per gram of infected muscle has likewise been low (*33*, *34*). This corresponds to the clinical findings in the southeast where the infection rarely produces severe manifestations.

Tapeworm Infections

The beef tapeworm, *T. saginata*, has become considerably less prevalent than it was 25 years ago, and the pork tapeworm, *T. solium*, has

practically disappeared from the native population of the United States. In contrast, both of these infections are increasingly common in continental Latin America.

The dwarf tapeworm, *Hymenolepis nana*, which requires no intermediate host and results from poor personal hygiene and lack of environmental sanitation, is widely distributed in the southeast and is most often found in children (*35*). Occasionally patients are so heavily parasitized that they are seriously ill as a result of these worms.

Human infection with the fish tapeworm, *Diphyllobothrium latum*, has been demonstrated to be indigenous in only one locality in the southeast, a rural community in Florida, where Negro boys and their dogs harbor the adult worms (*36*). The larval stage (sparganum) of a related tapeworm, *Diphyllobothrium mansoni* complex, producing somatic infection in man (sparganosis) has been reported in earlier decades—once from Florida and once from Texas. Recently, eight new cases have been discovered in Louisiana, Mississippi, and eastern Texas. As in fish tapeworm infection, species of *Cyclops* are the first intermediate host of the parasite, while frogs, snakes, birds, and mammals, but never fishes, are the second larval-stage hosts, and cats or dogs are the natural definitive hosts of this tapeworm. There is no specific evidence as to how man acquires the infection, but it seems likely from the case histories that he drank unfiltered water containing infected *Cyclops*.

Hydatid disease, produced by the larval stage of *Echinococcus granulosus*, has been demonstrated to be an autochthonous human infection in Virginia, Tennessee, Mississippi, and Louisiana.

Filariasis

During the 18th and 19th centuries Bancroft's filariasis may have been widely distributed throughout the southeast although there is no authentic record of the demonstration of the etiological agent, *W. bancrofti*. Charleston, S. C., was found to be an endemic center during blood surveys conducted in 1886, 1890, and 1915. Between 1915 and 1918 Edward Francis of the Public Health Service examined human blood films in 10 southern cities (Charleston, Columbia, Beaufort, and Georgetown, S. C.; Savannah and Milledgeville, Ga.; Jacksonville and Tampa, Fla.; Mobile, Ala.; and New Orleans, La.) to determine the extent of the infection. Only in Charleston were positive individuals discovered (13 to 35 percent of inmates of the old folks' home) who had maintained residence exclusively in the United States.

Since the publication of Francis' report (*37*), the writer has learned of only one authentic diagnosis of an autochthonous case—in 1930 in a native of Georgia who was for a time a patient of the Public Health Service Hospital at Carville, La. The lack of any subsequent reliable reports of cases suggests that Bancroft's filariasis has disappeared from the southeast. Meanwhile there have been three recent findings of immature female filariae from subcutaneous nodules of white persons with long residence in Florida. While these worms conform to the criteria for *Dirofilaria conjunctivae*, it seems reasonable to believe that they are in reality young specimens of the dog filaria, *Dirofilaria immitis*, which were unable to complete their development in man (*38*).

Summary and Conclusions

The parasite problems of the southeastern United States have been presented from the perspective of history and epidemiology. Some of the common intestinal roundworms and probably most of the intestinal protozoa were indigenous in the American Indian. Other animal parasites were introduced from Europe by the explorers and colonists although diseases among them were relatively unimportant compared with the contagious diseases of viral and bacterial origin which were brought to the Americas. Many serious problems developed with the importation of slaves from Africa and the consequent propagation in the favorable environment of the southeast of tropical strains of malaria parasites and the hookworm *Necator americanus*. As families from the Atlantic and gulf coast areas settled in the fertile inland valleys, these and other parasitic diseases became more extensively distributed. Poor conditions of sanitation and primitive personal hygiene made of the entire southeast a hyperen-

demic area for a number of diseases of parasitic origin, of others transmitted by arthropods, and those caused by enteric bacteria.

With the discovery of the etiological agents and knowledge of their life cycles and their methods of transmission, came improvement in water supplies, drainage, and disposal of human excreta. Public health programs were instituted to control these diseases. Parasite control in the southeast presents less of a problem now than it did in 1850 or 1900.

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EDITOR'S NOTE: The term, *"Trichocephalus trichiurus,"* used in the above paper is synonymous with the term, *"Trichuris trichiura,"* in the other papers of this symposium.

Special Committee on Medical Research

A Special Committee on Medical Research has been named by the National Science Foundation to review and evaluate the medical research program of the Department of Health, Education, and Welfare.

In requesting the review, the Secretary of Health, Education, and Welfare asked that the committee consider the rate of growth of the Department's health, medical, and related research program in the light of Federal responsibilities and appraise its present level of medical research support. Careful consideration was also requested of the proper balance of support for basic and applied research and the relative distribution of effort among the major special fields of health research.

The special committee is headed by Dr. C. N. H. Long, chairman, department of physiology, Yale University School of Medicine. Other members are Dr. E. A. Doisy, professor of biochemistry, St. Louis University School of Medicine; Dr. Ernest W. Goodpasture, Armed Forces Institute of Pathology, Walter Reed Army Medical Center; Dr. A. B. Hastings, department of biological chemistry, Harvard Medical School; Dr. Charles Huggins, director, the Ben May laboratory for cancer research, University of Chicago; Dr. Colin M. MacLeod, department of microbiology, New York University School of Medicine; Dr. C. Phillip Miller, department of medicine, University of Chicago; Dr. W. M. Stanley, director, virus laboratory, University of California. Dr. Joseph W. Pisani, on leave of absence from the State University College of Medicine at New York City, where he is assistant dean, is serving as executive secretary of the committee.