Parasites and the Germ Theory of Disease

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"THERE HAS ARISEN," NOTED DR. AUGUST HIRSCH (1885, vol. 2, 279) in his *Handbook of Geographical and Historical Pathology*, "a prospect of adding to this department of pathology." The department to which he referred was that of "parasitic diseases," and what was to be added to it, he thought, were "infectious diseases." Such diseases, he estimated, may well be of "a parasitic nature, or that there occur in them organisms of the lowest rank of organic development—the micrococci and bacilli." Hirsch, like many others at that time, stood on the brink of accepting a generalized parasitic theory of disease.

In this article I will examine the history of parasitology in the nineteenth and early twentieth century, and suggest ways in which models drawn from the study of parasitic organisms may have influenced debates over the etiology of infectious diseases. I shall argue that a generalized parasitic theory of disease only appeared very briefly, if at all, at the end of the nineteenth century. Both before and after this short period, those who studied parasites seemed to have little to offer those concerned with infectious diseases; parasites made minimal contributions to the modern germ theory of disease.

The professional study of parasitic organisms long predated the era of Pasteur and Koch; it began essentially in Germany at the beginning of the nineteenth century. In 1810, for example, Carl Rudolphi, who began his career at the University of Greifswald in Sweden after

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completing an M.D. thesis entitled "Observations on Intestinal Worms," moved to the University of Berlin where he acquired a reputation writing on the natural history of these strange creatures. With his publication of *Entozoorum Synopsis* in 1819, nearly 1,000 species of parasitic worms had been named (Müller 1838). These worms or Helminthes were usually divided into five groups: the nematodes, the acanthocephalans, the flukes, the tapeworms, and the so-called Cystica, later shown to be the bladder-like larval stages of some tapeworms.

In those early years certain pathologies were linked with the presence of these parasitic worms. But these worms were understood generally to be a symptom of the disease, not a cause of it. They were, in other words, assumed to have been spontaneously generated within the host. "What he [Rudolphi] says in favour of Generatio Aequivoca, is still," Johannes Müller (1838) noted, "almost the only recorded expression of opinion on which the defence of this doctrine can be made to rest."

As I have explained elsewhere (Farley 1972, 1977), the arguments in favor of the spontaneous generation of parasitic worms were very persuasive. Marcus Bloch (1782), for example, in his late-eighteenth-century prize essay awarded by the Danish Academy of Sciences, argued that parasitic worms were obviously destined to live only in very particular locations within a specific host organism. In the lawful, ordered, mechanical world of the eighteenth century, such parasites could not possibly arrive at these precise locations by chance, they had to be generated there. No other conclusion was possible.

But the most telling arguments in favor of spontaneous generation were published at the beginning of the nineteenth century by Johann Bremser (1819), a friend and colleague of Rudolphi. Parasitic worms, he argued, are members of a peculiar group of organisms which occur nowhere but inside the bodies of other animals. How then do they arrive at these locations? Are they produced inside the body, he asked, or do they or their eggs arrive there from outside? In his answers to these questions one is immediately struck by a parallel with debates over the contagious or noncontagious nature of some infectious diseases. Do these diseases, such as yellow fever and cholera (later in the century), likewise arise locally by a sort of spontaneous generation, or are they brought in from the outside by contagions? In both cases

a broad consensus favored the "spontaneous generation" of infectious diseases as well as of the parasites themselves.

As a general rule, most infectious diseases were known to remain localized within restricted areas of a city just as parasitic worms lived only in specific areas of the body. That both the diseases and the parasites arose in those locations rather than being carried there from the outside seemed to offer the best explanation for this distribution. Also, parasites, like certain infectious diseases, had a limited geographical range. Any explanation for this based on the distribution of contagions or eggs made little sense. Why would humans in eastern Europe, for example, be infected with the bothriocephalid tapeworm whilst those in the west carried taeniid tapeworms, if worm eggs were the source of infection? What would stop eggs, or contagions, being carried across Europe from one region to another? Likewise, the contagion theory offered no basis for understanding why some diseases were similarly limited in range, or why some seemed to arise "spontaneously" in certain areas with no known previous contact with a possible contagion. There were other problems also. If a parasitic worm arose from eggs passed from another worm of the same species in another host animal, Bremser asked, how are the eggs passed? People rarely ate food spoiled by the feces of a neighbor, he pointed out, and how would the eggs survive outside the body long enough to make contact? And how are eggs passed between animals that do not drink much, or do not eat each other, still less each other's feces? How are we to explain, Bremser wondered, the fact that herbivores carry as many worms as carnivores, and how are we to explain the occurrence of hydatid cysts within the muscles of vegetable-eating ruminants? In both cases the answer seemed obvious: both the diseases and the worms arose locally because of organic decay—whether in miasmatic swamps or in diseased body tissues. The following statement, made with reference to hydatid cysts, could equally well have been used to describe the origin of infectious diseases:

> Humidity, abundance and the bad or vegetable quality of the nourishment of an animal, are unequivocal means of producing acephalocysts (hydatid cysts) . . . irritation in fact, of a specific kind, has been excited by which a state favourable to their development has been produced (Phillips 1835).

In an earlier paper (Farley 1972) dealing with the origin of these parasitic worms, I remarked that without a vector or intermediate host concept, contagionists could never hope to explain the outbreak of some of these infectious diseases. Nor, without such a concept, could those opposed to spontaneous generation explain the origin of parasitic worms within the body. "The discovery of intermediate hosts for parasitic worms later in the century," I wrote, "was, then, of great significance for the understanding of contagious diseases." I no longer believe this last statement to be true; for most of the nineteenth century, concepts drawn from the study of parasites seemed totally inapplicable to the understanding of disease.

The Discovery of Intermediate Hosts

By the early nineteenth century, naturalists had come to realize that animals could develop in many different ways. Insect life cycles, involving nymphs or larvae and pupae, had long been known to differ from what was usually seen as the normal egg-to-adult pattern. By this time many benthic invertebrates were known also to produce larval stages which differed markedly from the adult animal, and which required complex metamorphoses in order to attain the adult form. Then, in 1842 the Danish naturalist Japetus Steenstrup described yet another developmental process: *Generationswechsel* or the alternation of generations. In this case, an immature stage, rather than behaving like a larva by transforming directly into a subsequent stage in the life cycle, as in insects, actually seemed to reproduce so as to generate more than one member of the next stage. The alternation of generations, Steenstrup wrote, occurs by "an animal giving birth to a progeny permanently dissimilar to its parent, but which itself produces a new generation, which either itself or in its offspring, returns to the form of the parent animal."

The jellyfishes provided the best-known example of such a life cycle. There, "the infusoria-like creatures proceeding from the ova" were not transformed directly into a jellyfish but developed instead into a small sessile "polypiform creature," quite unlike the parent jellyfish. This creature did not then transform into a jellyfish as a larval stage might have done, but instead budded off numerous minute medusa-like forms which detached from the polyp to grow eventually into

true jellyfishes. But the most important examples of such a life cycle, as far as this article is concerned, occurred in the trematodes or flukes. Flukes were then known to develop from free-living larval stages called "cercariae." But, Steenstrup asked, "Whence come then the free swimming cercariae?" They arose in large numbers, he concluded, from sac-like bodies within the tissues of snails. Thus, he noted, the trematode owes its origin to "animals, which in external form and partly in internal organization, differ from the animals into which that progeny is afterwards developed." Thus, Steenstrup concluded, "*all* cercariae originate in the sacciform bodies," and these bodies occur in the interior of snails. But Steenstrup, intent on interpreting the nature of this alternation of generations, was not interested in parasites *per se,* and remained unaware that he had introduced two key concepts into the world of parasitology: the *intermediate host* (a host in which a parasite develops but only to an immature stage) and the *parasitic life cycle* in which the parasite not only changes its host but also its form.

In the 1850s, a series of important feeding experiments with tapeworms, associated mainly with Friedrich Kuechenmeister, Carl von Siebold, and Pierre-Joseph van Beneden, revealed that the so-called Cystica (see above) was not a separate animal taxon, but consisted of the larval stages of terrestrial tapeworms (Farley 1972, 1977). This discovery was extremely significant, not only because it proved that tapeworms too had complex life cycles and intermediate hosts, but because the Cystica, being without any reproductive organs and found in muscles or the body cavity with no known outlet to the outside world, had traditionally been seen as the most obvious examples of spontaneously generated worms. Thus, what Steenstrup had described earlier in flukes was now seen to be true also for tapeworms: neither group arose by spontaneous generation, but passed from host to host in a complex life cycle involving great changes in body form.

In theory, at least, the life-cycle and intermediate-host concepts could have proved valuable to contagionists as they struggled to understand the distribution and behavior of some of the infectious diseases; contagions, for example, could have been carried by host animals from place to place. But neither of these two key concepts were carried forward into the medical world of infectious diseases. Contagionists, on the defensive against the rising tide of mid-century anticontagionism, never, to my knowledge, suggested that either or

both of these concepts could be used to explain seeming anomalies to a contagionist interpretation. Of course, since few contagionists believed contagions to be living organisms, there was no reason why this connection should have been made. But there was another reason why ideas would be unlikely to flow from parasitology to medicine at this time. All parasitic animals were thought to belong to a *single* taxon: the Entozoa or Helminthes. The word "parasite" was not widely used; they were helminths or entozoans, and their study was termed "helminthology," and less often "entozoology" or "parasitology" (Cobbold 1864). Parasitism, therefore, was not seen as a lifestyle common to a wide variety of animal groups. The concepts of helminth life cycles and intermediate hosts were not transferable; they were only applicable to a single animal taxon.

The Beginning of Parasitology

This state of affairs was to last until the 1880s. By then, as Rudolf Leuckart had discovered, parasites included more than one animal taxon. Rudolf Leuckart can be seen as the "father of parasitology," as distinct from helminthology. He first became interested in helminths during the 1850s while on the medical faculty at the University of Giessen. In 1869 he moved to the University of Leipzig where, eleven years later, he opened perhaps the world's first parasite laboratory in a new zoological institute built for him (figure 1). Sometime during his career at Leipzig, he recognized the coccidians to be parasitic members of the unicellular protozoans and placed them in the class Sporozoa. As a result of this discovery Leuckart (1876) could claim that animal parasites consisted not only of helminths, "but numerous other creatures that sometime resemble so completely certain free-living animals . . that an independent mode of existence has been actually ascribed to them." They now included three groups of animals: the worms, protozoa, and arthropods. As the title of Leuckart's book illustrates, these discoveries resulted in the increasing use of the word "parasite" and "parasitology," to describe a way of life and a discipline common to a wide range of animals.

At approximately the same time as Leuckart expanded the field of parasitology, Patrick Manson (1879a), a relatively unknown physician working for the customs service in China, uncovered the role of blood-

FIG. 1. Blood and Entrails: Rudolf Leuckart and his assistants in the first parasitological laboratory, University of Leipzig,

sucking flies in the life cycle of filarial nematode worms. He almost, but not quite, discovered the role of biological vectors as distinct from intermediate hosts.

In 1872, Dr. Timothy Lewis ([1872] 1978), a member of the British Army Medical Service stationed in India, discovered six minute active "snake-like" nematode worms or filariae in a single droplet of human blood. "It is an almost universal law in the history of the more dangerous kinds of Entozoa," Manson (1879a) later remarked, "that the egg or embryo must escape from the host inhabited by the parent worm." After that, he noted, the embryo either lives independently for a short while or it is swallowed by another animal. Whig historians, who love to reconstruct great discoveries so as to show the application of the scientific method by great minds, have always assumed that Manson first suspected the mosquito to be this other animal after noting a periodicity of the filariae in the blood stream—they appear first at sunset, reach a peak about midnight and thereafter decrease so that by mid-morning few if any appear in the blood. But this periodicity was noted two years after he had discovered the role of the mosquitoes; initially he had merely noted the temporary absence of the filariae from the blood, but "was not aware . . of any law governing this" (Manson 1879b) Manson's discovery that female mosquitoes pick up the filariae with the blood of their human victims was an inspired guess. In the mosquito's stomach the filariae metamorphosed into what Manson considered to be the adult nematode worm. "There can be little doubt as to the subsequent history of the Filaria," he wrote. "Escaping into the water in which the mosquitoes died, it is through the medium of the fluid brought into contact with the tissues of man, and that, either piercing the integuments or, what is more probable, being swallowed, it works its way through the alimentary canal to its final resting place" (Manson 1879a). Manson had almost uncovered the role of vector hosts, but the model provided by cestodes (and trematodes to a lesser extent), which are transmitted from host to host through the food chain, had led him to the natural conclusion that humans became infected with these nematodes by eating them, not through reinfection by biting flies. His belief in this food-chain model was reinforced by the generally held assumption that a mosquito took only one blood meal before depositing her eggs, after which she died.

By the 1880s, however, the parasitic web had extended to include

the protozoans, but the trematode-cestode life-cycle model remained in place. This model had acted as a guide for Manson's filarial work, and now this filarial work provided the model for his malarial theory, first proposed in 1894. Neither the malarial disease nor the malarial organism can be directly communicated from person to person, Manson noted; they "can be acquired only indirectly either through the air, the water, by food, or by another unknown way." Escape from the body being necessary for any parasite, and with no trace of the parasite in physiological or pathological discharges, Manson concluded that the same mechanism must exist in the malarial protozoa as it did earlier with filarial worms.

> If this be the case, the mosquitoes having been shown to be the agent by which the filaria is removed from the human blood vessels, this, or a similar suctorial insect must be the agent which removes from the human blood vessels those forms of malarial organism which are destined to continue the existence of this organism outside the body. It must, therefore, be in this or in a similar suctorial insect or insects that the first stages of the extracorporeal life of the malarial organism are passed (Manson 1894).

Manson constantly drew analogies with the helminths and the other sporozoans. He noted that the malarial parasite must escape from the body in the same fashion as the larval stages of tapeworms. Both must be eaten and, by analogy with filarial worms, mosquitoes seemed the most obvious villains. Likewise, he assumed that humans acquired the malarial parasites in the same manner as they acquired their parasitic worms—by eating them. The mosquitoes, Manson (1896, 716) argued, "seeks out some dark and sheltered spot near stagnant water. At the end of about six days she quits her shelter, and, alighting on the surface of the water, deposits her eggs thereon. She then dies, and, as a rule, falls into the water alongside her eggs." People became infected with the malarial parasite either by swallowing this water, or by inhaling dust which could include resistant cysts of the parasite, or dust from decomposed infected mosquitoes. Again, Manson's views reflected common knowledge about mosquitoes. In answer to Amico Bignami's (1898) claim that the parasite was passed to man through the bite of the mosquito, Manson once again pointed out that "the habit of the mosquito is to bite once only" (letter to Ronald Ross, October 12, 1896, reprinted in Manson–Bahr and Alcock 1927, 149).

Initially, as Theobald Smith and F. Kilborne noted in their famous paper on Texas cattle fever, the cattle tick was assumed also to pass the disease organism in a helminth-like manner. "Hitherto we had supposed," they wrote, "that the cattle tick acts as a carrier of the disease between the Southern cattle and the soil of the Northern pastures. It was believed that the tick obtained the parasite from the blood of its host, and in its dissolution on the pasture a certain resistant spore form was set free, which produced the disease when taken in with the food" (Smith and Kilborne [1893] 1937, 497).

The error was finally revealed in 1898 by Ronald Ross (Harrison 1978; Manson 1898). The mosquitoes, he found, not only removed the malarial parasite from the blood but acted as a true vector host. The parasites developed in the mosquitoes and eventually migrated to the salivary glands from where they could be injected back into the blood; people could acquire their parasites by inoculation as well as by feeding. Thomas Bancroft's discovery, a year later, that female mosquitoes produced more than one batch of eggs and took more than one blood meal added considerable credence to the vector theory (Manson-Bahr and Alcock 1927). Thus, arthropod vectors were a particular kind of intermediate host; the parasite developed in them and they carried the parasite both to and from the final host. Theobald Smith and Kilborne did not make that fundamental discovery as often claimed. They could not decide whether the protozoan parasite responsible for Texas cattle fever was accidentally carried in the spore state by the tick, or whether the tick was a necessary host in which the parasite developed and then became "localized in certain glands of the young tick," from where it would be discharged into the blood of the cattle. Being unable to locate the parasite inside the tick, they remained undecided. "Further investigations," they urged, "are necessary before the probable truth of one or the other of these hypotheses can be predicted with any degree of certainty" (Smith and Kilborne [1893] 1937, 514–15). Smith and Kilborne faced a difficulty similar to that of Manson. Just as mosquitoes were believed to bite only once, so the tick was believed to spend its life on a single host making improbable a host-to-host transmission by biting.

But whether we ascribe the final breakthrough to Ross or to the Italian workers, it certainly came. By 1900 parasitic helminths and protozoa were known to develop in intermediate hosts or vector hosts

and to be transmitted to humans either through infected food or by the bites of blood-sucking arthropods.

The period between Manson's "half-vector" filarial-worm discovery in the late 1870s and the realization at the very end of the century that a vector was a true biological host that both picks up and transmits the parasite, and in which the parasite must pass though some developmental stages, coincided, of course, with Koch's famous discoveries of the bacterial cause of anthrax, tuberculosis, and cholera. It witnessed the growing belief, by such eminences as Hirsch, that many infectious diseases were caused by bacteria, and that bacteria could be added to the list of parasitic organisms. A generalized parasitic theory of disease seemed imminent.

But this never occurred. Instead, the belief grew that there were two sorts of disease: those caused by bacteria and those caused by parasites. The latter were never contagious; their life cycle always involved passage through an intermediate or vector host in which obligatory stages of development took place. Bacterial diseases, on the other hand, were usually contagious although they might also be transmitted by arthropods in a purely accidental and mechanical fashion. As Victoria Harden (1985) has explained, the association between obligatory vector hosts and parasitic organisms was so strong that once a vector was discovered, the disease was assumed to have a protozoal (or helminth) cause, and, vice versa, once bacterial organisms were suspected, vector hosts were discounted. As noted by Charles Stiles (1901), who had been trained in parasitology in Rudolf Leuckart's laboratory:

> We may lay down two general biologic rules. . . : The first rule, to which at present a few exceptions are known, is that diseases which are accidentally spread by insects are caused by parasitic plants, particularly by bacteria. The second, to which no exceptions are as yet known, is that those diseases which are dependent upon insects or other arthropods for their dissemination and transmission are caused by parasitic animals, particularly by sporozoa and worms.

The implications of this "law" were profound. Yellow fever, shown in 1900 to be transmitted by the bite of mosquitoes and long thought to have a bacterial cause, was now suspected of being a protozoan disease (Harden 1985). Similarly, the plague, being caused by bacteria

and harbored by the rat, could not be transmitted to humans by the bite of infected fleas. Fleas may carry the bacteria, according to Stiles, but these organisms could infect people only through a wound or scratch.

Patrick Manson (1899) also made a sharp distinction between bacterial and parasitic diseases in his address at the opening of the London School of Tropical Medicine. The tropical external climate, Manson argued, influences the distribution of pathogens; it limits many pathogens to the tropics and brings about diseases with "a limited climatic range." But, he added, coming to the crux of his argument, such climatic rules do not affect bacterial pathogens. They are cosmopolitan precisely because they live in the human body and rarely come under the influence of the external climate. Instead, "transmitted directly from host to host, they can be acquired in any climate when suitable social conditions occur." On the other hand, parasitic diseases had a limited range. They were often so limited because they usually passed through intermediate or vector hosts (mosquitoes, flies, snails, etc.) that were native to the tropics, not to temperate zones.

According to Harden (1985), parasitologists seemed to have been the most fervent supporters of the two-disease theory. This probably reflected the growth of parasitology as a discipline in twentieth century Britain and the United States. In both countries parasitology and bacteriology pulled apart; the latter became essentially a medical discipline while parasitology occupied niches outside the mainstream of medicine. Intermediate and vector hosts were a unique part of the parasitologist's field; to claim that parasitic diseases differed significantly from bacterial diseases may well have been part of an attempt to delineate and legitimate a new discipline. And so, once again, as in the nineteenth century, parasitologists had little to offer those investigating infectious diseases.

The Parting of the Ways

Johannes Muller (1838), writing about his predecessor at the faculty of medicine in Berlin, made the extraordinary claim that the success of Germans in helminthology was a reflection of a lack of overseas possessions; that whereas the British looked outward to the flora and fauna of empire, the Germans were forced to look inwards. "The

limitation caused by our geographical position," he wrote, "has imparted to our spirit a certain direction towards what is concealed, and has made us much the greater in the investigation of a world of concealed inhabitants of our native creatures, viz. the Entozoa."

He turned out to be quite wrong; it was, in fact, the expansion of the British Empire at the very end of the nineteenth century which moved the British into helminthology. It, together with protozoology and medical entomology, became synonymous with a new postgraduate medical field of study—tropical medicine.

In March 1898, the Colonial Office forwarded a memo to the War Office, Foreign Office, India Office, the General Medical Council, the Seamen's Hospital Society, and all 26 British medical schools. Joseph Chamberlain, the Colonial Secretary, was, according to this memo, "anxious to do anything in his power to extend the benefits of medical science to the natives of tropical colonies and protectorates, and to diminish the risk to the lives and health of those Europeans who . . . are called upon to serve in unhealthy climates." He requested, therefore, that all British medical schools provide instruction in tropical medicine (Colonial Office, 1898a). Three months later, however, Chamberlain seems to have acquiesced to Patrick Manson's view that only one school of tropical medicine was needed and that the Seamen's Hospital at Greenwich should house that school. By July 1898, the foundations of the new school at Greenwich were being laid.

Tropical medicine became the main impetus for the emergence of parasitology as a discipline in Britain (Worboys 1976, 1983). The London School of Tropical Medicine established lectureships in helminthology and protozoology, and the Liverpool School of Tropical Medicine, which opened at the same time, established lectureships and chairs in parasitology and entomology. Bacteriology was ignored.

Interuniversity jealousies were mainly responsible for the rather curious curriculum of the London School of Tropical Medicine. In November 1898, two weeks after the medical schools had been apprised of the decision to build a separate postgraduate school at Greenwich, King's College Medical School forwarded its belated reply to the original Colonial Office memo. Basically, the letter extolled the virtues of its bacteriological laboratory and supported the idea of special training in tropical medicine, which must, they noted, "of necessity include a thorough practical training in bacteriological methods."

> The council trust that the Government would be willing, in any arrangements they may eventually make, to recognize instruction given in the bacteriological laboratory of King's College as a qualifying course in Tropical Medicine (Colonial Office, 1898b).

Bacteriology now became a thorny issue. In a letter to Chamberlain, Manson argued that, if the King's College proposal to recognize its course as a qualification in tropical medicine were agreed to, then "every medical school or bacteriological laboratory in London and throughout the country would have an equal claim to be regarded as affording a qualifying course of study" (Colonial Office, 1898c). To justify the existence of a single special school, through which all medical officers had to pass, it was clearly necessary to avoid duplication with the curricula of British medical schools. What better way than to omit bacteriology and hygiene from the curriculum of the new school? By 1900, training in bacteriology was available at most British medical schools; indeed, it was the most obvious sign that a school was progressive and promoting the spirit of modern medical science. Thus, the London School of Tropical Medicine put its greatest emphasis on protozoology and helminthology while downplaying bacteriology and hygiene. Michael Worboys (1983) expressed it best: "In an important sense tropical medicine was defined initially by what an orthodox medical degree left out." It had left out parasitology.

But this decision also gave institutional meaning to the growing idea that, indeed, bacterial and parasitic diseases were not alike. Manson's address at the opening of the school (Manson 1899) not only distinguished one from the other but claimed also that the study of parasitic diseases should be the sole function of parasitologists working in the new field of tropical medicine. The presence of vector and intermediate hosts produced diseases with limited geographical range as opposed to those produced by the cosmopolitan bacteria. Those parasitic diseases limited to the tropics became the focus of tropical medicine in the British Empire. Indeed, apart from the rather unsuccessful Molteno Institute for Research in Parasitology at the University of Cambridge, the only institute of parasitology in the British Empire that was not linked to tropical medicine was founded in 1932 at MacDonald College of McGill University in Montreal. Supported by the Canadian National Research Council, the Quebec Department of Agriculture, and the Empire Marketing Board in Lon-

don, it was founded to investigate parasites of economic importance to the swine, sheep, and poultry industries (Cameron 1940), although during the Second World War the faculty of the institute was called upon to offer classes toward a McGill diploma in tropical medicine. But Canada was not only a member of the British Empire; it shared the North American continent with the United States where parasitology, as at McGill, often had a veterinary and agricultural focus.

In the United States, land grant colleges, not schools of tropical medicine, became the major forum for parasitology; its practitioners were mainly zoologists, not physicians. In these land grant colleges parasitology had either a veterinary and agricultural emphasis (Schwabe 1981) or, as "medical zoology," was tailored to the needs of a new and increasingly important set of undergraduate students called "pre-meds." American parasitology was nurtured in Nebraska and Illinois; its father was Henry B. Ward, who spent two years in Germany studying at Gottingen, Freiburg, and eventually under Rudolf Leuckart at Leipzig. In 1893 he moved to the University of Nebraska at Lincoln, where he began to include the study of parasites in his undergraduate zoology classes. The University of Nebraska catalogues show that by 1904 Ward was presenting a class called "Parasites of Man." It was clearly aimed at pre-medical students who were required to spend their first two years at Lincoln before transferring to the Omaha Medical College. Reflecting the move of American medical education away from proprietary schools toward university-based schools of medicine, the Omaha College had become the College of Medicine of the University of Nebraska. At the same time, the introductory zoology class, now also increasingly geared for pre-medical students, was retitled "Introduction to Animal Biology and Medical Zoology." By 1910 the division between medicine and agriculture had become complete with two parasitology classes being offered: "Medical Zoology" for pre-medical students and "Animal Parasites" for zoology students. By then, however, Ward had gone. Denied the deanship in medicine at Omaha, he had accepted the chair of zoology at Illinois where he built up the first American graduate school in parasitology, and founded the *Journal of Parasitology* (van Cleave 1947).

But, as in Britain, American parasitology also served the needs of tropical medicine. The department of medical zoology in the Rockefeller-funded Johns Hopkins University School of Hygiene and Public Health became one of the most active research centers in American

tropical medicine (Fee 1987). Divided into the three classical divisions of protozoology, helminthology, and entomology, it became the department with the strongest links with the International Health Board or Division, which dominated tropical medicine between the two world wars, and whose members often circulated through the department to gain necessary expertise in tropical diseases. As in the London School of Tropical Medicine, parasitology at Johns Hopkins became primarily a postgraduate medical study, although some of their degrees were also available to science graduates.

For most of the nineteenth century, the world of parasites was quite separate from the world of infectious diseases. Diseases were not deemed to be caused by organisms, and parasites were restricted to a single and very peculiar taxon of parasitic worms. Thus, the discovery that these worms were not spontaneously generated and that their life cycles involved passage through intermediate hosts played no role in the debate over the contagious or noncontagious nature of infectious diseases. In the last two decades of the nineteenth century, however, the barrier between the two seems to have been broken. Parasitism seemed to be a way of life common to a wide variety of organisms, among which could be numbered the bacteria, now viewed to be the cause of many infectious diseases. In the words of Hirsch, all belonged to a "single department of pathology." But no fruitful interchange seems to have taken place, and no such single department of pathology was ever born. Initially, indeed, the only interchange that did occur took place within the field of parasitology; it resulted in those working on protozoan vectors being partially misled by the model provided by parasitic worms.

Before the 1880s, intellectual differences had kept parasitology isolated from medicine, but by the turn of the century institutional differences came to play the most significant role. Parasitology in Britain and the United States became established as a discipline outside the mainstream of medicine. It became segregated from the modern medical field of bacteriology; it concentrated on naming and describing nonbacterial parasites and their life cycles and became increasingly irrelevant to work on bacterial diseases. Twentieth-century parasitology came to resemble nineteenth-century helminthology; neither had much to offer those concerned with infectious diseases.

Only within the last few years has parasitology moved back into the mainstream of medical research. Those who call themselves par-

asitologists have changed very little, but their discipline is being taken over by a new breed of aggressive, "high-tech" researchers. Armed with far larger research grants than traditional parasitologists could ever hope to acquire, these newcomers have transformed the field out of all recognition. Today, mathematical modelling, epidemiology, and vaccine studies are replacing the older emphasis on descriptive and life cycle studies.

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