N. D. LEVINE

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by

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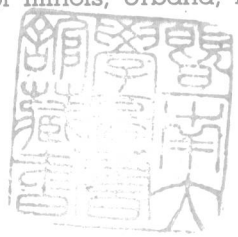

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Preface

The importance of protozoan parasites as causes of disease in domestic animals is well recognized, yet the literature on them is still widely scattered and the books now available provide little more than an introduction to the subject. The present book was written to serve as a text and reference work for veterinarians, protozoologists, parasitologists, zoologists and also for physicians. As our knowledge of the relations between human and animal parasites has increased, the list has also increased of parasites which were once thought to be confined to domestic and wild animals but which are now known to occur also in man. The area of overlap between the fields of human and animal disease is becoming continually greater, and the zoonoses are receiving more and more attention. For this reason, the protozoan parasites of man are included in this book, and their relations to those of lower animals are indicated.

When this book was begun, it was intended to be a revision of the pioneering Veterinary Protozoology by the late Banner Bill Morgan and the late Philip A. Hawkins, the second edition of which was published in 1952. However, it soon became apparent that far more than this was necessary, and the result has been an entirely new book.

It is planned to follow this volume with others on veterinary helminthology and entomology. The first chapter, therefore, deals with the general principles of parasitology, while the second is an introduction to protozoology. The different groups of protozoa are discussed in the succeeding chapters, and the final chapter deals with laboratory diagnostic technics. This systematic organization based on parasite groups is used rather than one based on host animals because it is more efficient, avoids repetition, and makes the subject easier to present and to understand. However, it is also useful to know which parasites one can expect to find in each host. Lists of parasites by host have therefore been prepared and are incorporated in the index. E. A. Benbrook (1958. Outline of parasites reported for domesticated animals in North America. 5th ed. Iowa State Univ. Press) has listed the parasites both by host and by location in the host.

The world today is too small to permit a provincial approach to parasitism and disease. Katanga and Uttar Pradesh, Kazakhstan and Luzon are only a step from New York and San Francisco, and their problems and their diseases are becoming more and more our concern. The scope of this book, therefore, is world-wide, and parasites are discussed regardless of where they occur. However, major attention is given to the parasites of those domestic animals which occur in the temperate zones, and relatively little is included on parasites of animals like the elephant, camel, llama, reindeer and yak, even tho they are important domestic animals in some regions.

When C. M. Wenyon wrote his classic Protozoology in 1926, he remarked that one of his chief difficulties had been that hardly a week passed without the publication of some paper of importance; that difficulty is far greater today than it was then. The number of published papers has been increasing exponentially, and there is no sign that the logarithmic phase of the curve is near its end. Even if one tries to read the current journals faithfully and to use the abstract journals assiduously, important papers may escape his notice. I am sure that some have escaped mine, and I should appreciate having them called to my attention. In addition, to help me in preparing future editions, I should appreciate receiving reprints of pertinent papers.

A favorite saying of Jean Baer is that textbooks perpetuate errors by copying them from one to another. I have tried to avoid this by going to the original papers as much as possible, but in so doing I may well have introduced some errors of my own. I should appreciate having these called to my attention also.

Papers are appearing so fast that, unless one is forced to it, he cannot take the time to read and ponder those outside his own immediate field of interest and to try to integrate them into a coherent whole. Writing this book has made me do so, and the process has taught me a great deal. Not only have I learned many things which I did not know, but I have come to realize more clearly how much information we still lack, even about parasites which have been studied extensively. This book reflects that situation. The reader will find at least one question, one gap in our knowledge on each page. Each is a challenge for future research which I hope will be accepted by many who read this book.

I should like to express appreciation to Drs. Carl A. Brandly, John O. Corliss, William R. Horsfall, Francis J. Kruidenier, R. N. Mohan and Miss Virginia Ivens for reading and commenting on various chapters; to Mrs. Helen V. Olson for preparing figures 6, 13A, 18, 19, 20K-1, 24 and 31; to Miss Ada Price for typing the greatest part of the manuscript; to Mesdames Marion Corzine, Mardell Harris, Kathryn Hill, Janet Manning, Lucille Rice, Carolyn Seets and Misses Eileen Bourgois and Beverly Seward for proof-reading the manuscript; to Drs. E. R. Becker, J. F. Christensen, H. Christl, F. P. Filice, D. M. Hammond, C. A. Hoare, R. R. Kudo, J. Ludvik, E. R. Noble, G. A. Noble, Muriel Robertson, R. M. Stabler, E. E. Tyzzer and D. H. Wenrich, to Ballière, Tindall & Cox, Cambridge University Press, Charles C Thomas, the Commonwealth Agricultural Bureaux, Gustav Fischer Verlag, Iowa State University Press, Johns Hopkins Press, Springer-Verlag, University of California Press, Williams & Wilkins Co., and The Wistar Institute, and to the Annals of Tropical Medicine and Parasitology, Iowa State Journal of Science, Journal of Morphology, Journal of Parasitology, Journal of Protozoology, Parasitology, Quarterly Journal of Microscopic Science, Quarterly Review of Biology, University of California Publications in Zoology, Veterinary Reviews and Annotations, Zeitschrift für Parasitenkunde, and Zentralblatt für Bakteriologie for permission to reproduce some of their illustrations; and, not the least, to the Burgess Publishing Company for its patience and understanding during the time this manuscript was in preparation.

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Contents

Chapter 1

INTRODUCTION TO PARASITOLOGY

Parasitology is the science which treats of parasites. The word "parasite" is derived from the Greek and means, literally, "situated beside." It was used by the ancient Greeks originally for people who ate beside or at the tables of others, and referred both to sycophants or hangers-on and to priests who collected grain for their temples. While the social meaning of the term has been partially retained, it has been given a new connotation by scientists. *Parasites* are defined as organisms which live on or within some other living organism, which is known as the *host*. *Parasitism* is the association of two such organisms.

Parasites may be either animals or plants--viruses, rickettsiae, spirochetes, bacteria, yeasts, fungi, algae, mistletoe, dodder, protozoa, helminths, arthropods, molluscs, and even certain vertebrates such as the cuckoo. The general principles of parasitology apply to all. However, in this book we shall deal primarily with animal parasites, leaving the plant parasites to textbooks of microbiology.

In our everyday thinking we consider that animals can live in three main habitats--land, fresh water and sea water. A fourth habitat is the parasitic one, which is quite different from the other three. As a matter of fact, there are quite a few different parasitic habitats, each with its own characteristics. Parasites are found in the lumen of the intestinal tract, on the outside of the body, in the skin, in various tissues, in the blood plasma, inside different types of cells, and even inside cell nuclei.

Parasites have arisen from free-living animals. Some parasites closely resemble their free-living relatives, but others have undergone structural changes which make them more suited to their changed environment. Since these changes have in many cases been the loss of some power which their free-living relatives possess, parasites have some-

times been considered degenerate creatures. The opposite is true. Parasites are highly specialized organisms. Those powers which were unnecessary, they have lost. For instance, the adults of most parasitic worms have relatively little ability to move around. But they don't need it. Too much activity might even lead to their reaching the point of no return and being discharged from their host's body.

As another example, tapeworms have no intestinal tract. But, since they obtain their nourishment directly thru the body wall, an intestine would be superfluous. Thus, in the case of parasites as with all other animals and plants, the useless has been eliminated in the course of evolution.

In contrast, the reproductive system of parasites is often tremendously developed. Since the chances of an egg or larva leaving one host and infecting another are very small, the numbers of eggs produced must be very large. Many parasitic worms produce thousands of eggs a day. The female of *Ascaris suum*, the large roundworm of swine, lays about 1,400,000 eggs per day (Kelley and Smith, 1956). Assuming that she lives 200 days, which is not an excessive life, she will have laid 280 million eggs in her lifetime. Since the number of *Ascaris* in the world is staying more or less the same, we can conclude that on the average only two of these eggs will produce adult worms--a male and a female. The chance of any particular egg ever becoming a mature worm is thus about 1 in 140 million, which is much less than a man's chance of being struck by lightning.

The broad fish tapeworm of the dog, man and other animals, *Dibothriocephalus latus*, will produce over 4 miles of segments containing 2 billion eggs during a 10-year life span, and again the number of these tapeworms is not increasing. Since these tapeworms are hermaphrodites, each egg can become an egg-laying worm, but its chances of doing so are a hundred times less than those of the *Ascaris* egg. Parasites are continually being confronted by odds of this sort and are continually surmounting them.

Life of one sort or another seems to have flowed into every possible niche. Parasites live in some of the most difficult niches, and it is remarkable how they have succeeded in surviving in them. Parasites have tremendous problems to solve--problems of nutrition, of respiration, of excretion, of getting from one host to another--and the different and often ingenious ways in which different parasites have solved these problems are amazing. Some of their adjustments are almost perfect; others are less satisfactory. In general, we may say that the more satisfactory the solution, the more abundant are the parasites. The rare ones are the less successful ones.

We can think of parasitism as related basically to the solution of the problem of nutrition, and we can think of the other problems as somewhat secondary. This is obviously an incomplete and defective view, but nevertheless it has some value.

Living organisms have four general types of nutrition. *Holophytic* nutrition is typical of plants; it involves synthesis of carbohydrates by means of chlorophyll. *Holozoic* nutrition is animal-like; it involves ingestion of particulate food thru a permanent or temporary mouth. *Saprozoic* or *saprophytic* nutrition (the choice of term depending upon whether the organism is an animal or plant) involves absorption of nutrients in solution thru the body wall. The fourth type of nutrition is that employed by viruses, which synthesize their proteins directly from the host's amino acids and do not have a true body wall during their parasitic phase.

The terms *saprophyte* and *saprophytic* are often used by bacteriologists in another sense also, to refer to non-pathogenic, non-parasitic organisms. The terms *saprozoite* and *saprozoic* are also similarly used with reference to free-living animals, but much less frequently.

Coprozoic or *coprophilic* organisms are animals which live in feces. They

may be either saprozoic or holozoic or both, and are sometimes mistaken for true parasites.

Parasites resemble predators in some respects; indeed, one grades into the other. In general, we think of predators as larger than or as large as their prey, while we think of parasites as considerably smaller. A lion seizing an antelope is a predator, as is a spider capturing a fly. But there is a distinction only in size of prey between a predatory assassin bug capturing another insect and sucking out its juices and the closely related, parasitic kissing bug sucking blood out of a man. And a mosquito is just as much a predator as the kissing bug. The distinction is one of degree. As Elton (1935) put it, "The difference between a carnivore and a parasite is simply the difference between living upon capital and income, between the burglar and the blackmailer. The general result is the same although the methods employed are different."

TYPES OF PARASITISM

There are several types of parasitism. *Parasitism* itself is defined as an association between two specifically distinct organisms in which one lives on or within the other in order to obtain sustenance.

Symbiosis is the permanent association of two specifically distinct organisms so dependent upon each other that life apart is impossible under natural conditions. The relation between many termites and their intestinal protozoa is symbiotic. The termites eat wood, but they cannot digest it; the protozoa can digest wood, turning it into glucose, but they have no way of obtaining it; working together, the termites ingest wood particles, the protozoa break the cellulose down to glucose, and the termites then digest the glucose. Lichens furnish another example of symbiosis. They are composed of certain species of algae and fungi living together.

Many insects, ticks and mites have symbiotic bacteria and rickettsiae. The symbiotic organisms are found either in special cells, the mycetocytes, in modified parts of the Malpighian tubules, or in special organs, the mycetomes. It is significant that, among blood-sucking arthropods, symbiosis occurs in those which live on blood thruout their life cycles (ticks, lice, bedbugs, kissing bugs, tsetse flies, hippoboscid flies) but not in those in which only the adults suck blood while the larvae are free-living (fleas, mosquitoes, phlebotomines, tabanids and stable flies). Blood lacks some metabolites which the arthropods are unable to synthesize themselves and for which they depend on their symbiotes. These metabolites appear to include vitamins of the B group and probably other substances as well (Buchner, 1953; Koch, 1956; Weyer, 1960).

Mutualism is an association of two organisms by which both are benefited. It differs from symbiosis in that it is not obligatory for both partners. One example often cited is that of a sea anemone living upon the back of a crab. The anemone is benefited by being moved to new hunting grounds and by obtaining morsels of food torn off by the crab, while the crab is protected by the bulk and stinging tentacles of the anemone. Another marine example is that of the scorpion fish of Indo-Malaya. It lives on the bottom of the sea, where it lies in wait for passing fish. It is covered with a crust of hydroids which camouflage it so that it can seize its unwary prey more easily. The hydroids presumably benefit by being moved to new sources of food and by being provided with a dwelling-place. However, since they can live other places beside the scorpion fish's back, their relation is mutualistic.

Another example of mutualism, and one closer to us, is the relationship between ruminants and the cellulose-digesting bacteria and other micro-organisms in their rumens. The latter are furnished a favorable home by their hosts and aid them by breaking down cellulose to usable compounds. The rumen-dwelling bacteria which produce B group vitamins and thus make an outside source of them unnecessary for ruminant nutrition probably

belong here too, altho they verge on the symbiotic. The bacteria which produce these vitamins in the large intestine of swine are more nearly mutualistic, since the pigs cannot absorb the vitamins thru the colon wall but must re-ingest their feces to obtain them. The same is true of rabbits, and is undoubtedly responsible for their coprophagy.

The bizarre protozoa which swarm in the rumen and reticulum are almost certainly mutualistic. Their host can get along without them, but they may benefit it by providing a better type of protein than it ingests. In addition, they are an important source of volatile fatty acids, and they smooth out the carbohydrate fermentation process.

Commensalism is an association between host and parasite in which one partner is benefited and the other is neither benefited nor harmed. Many intestinal bacteria such as *Escherichia coli* are normally commensals, as are many intestinal protozoa such as *Entamoeba coli* and *Trichomonas* spp.

The next two terms both refer to potentially pathogenic parasites. *Parasitosis* is the association between two organisms in which one injures the other, causing signs and lesions of disease. *Parasitiasis* is the association between two organisms in which one is potentially pathogenic but does not cause signs of disease.

The difference between parasitosis and parasitiasis is quantitative. In parasitiasis the host is able to repair the damage caused by the parasite without noticeable injury, while in parasitosis it cannot. As Whitlock (1955) put it, "Parasitiasis is a state of balance. Parasitosis is a state of imbalance." Applying the concept to ruminant helminths, Gordon (1957) said, "Helminthiasis is almost universal and continuous, helminthosis is more restricted and sporadic. However, one shades imperceptibly into the other in subclinical infestations." The same organism can cause either parasitosis or parasititiasis, depending upon the number present or upon the nutritional condition, age, sex, immune state, etc. of the host. Failure to recognize this distinction may cause many false diagnoses--the mere presence of a potentially pathogenic species of parasite does not necessarily mean that it is causing disease.

The *carrier state* furnishes a good example of parasitiasis. *Carriers* are animals which have a light infection with some parasite but are not harmed by it, usually due to immunity resulting from previous exposure, but which serve as a source of infection for susceptible animals. Thus, adult sheep and cattle may be lightly infected with gastrointestinal nematodes without noticeable effect, but their lambs and calves may become heavily parasitized from grazing with them. The condition in the adults is parasitiasis; that in the young is parasitosis. Adult chickens rarely suffer from coccidiosis because they have recovered from a clinical or subclinical attack when young. However, they are usually still lightly infected and continue to shed a few oocysts; they have coccidiasis. Cattle which have aborted as a result of *Brucella* infection may continue to shed the bacteria in their milk without ordinarily suffering further clinical attacks. The aborting cow has brucellosis, while the carrier has brucelliasis.

These endings can also be applied to the names of the disease agents, as has already been done above. Thus, *Haemonchus contortus* may cause haemonchosis or haemonchiasis, *Taenia* may cause taeniosis or taeniasis, *Histomonas meleagridis* may cause histomonosis or histomoniasis, depending on the circumstances.

It was mentioned earlier that the solutions different parasites have made of their problems of living have varied in satisfactoriness. We might consider this in regard to type of parasitism. Symbiosis is a highly specialized type of association which occurs only in certain groups. Mutualism is a much looser association, also fairly uncommon. It could well be a step on the road to symbiosis. The most

common types of parasitism are the last three. Of these, commensalism is clearly the most desirable, both from the standpoint of the host (which isn't harmed) and of the parasite. Parasitosis, which harms the host, is in the long run harmful to the parasite also. By injuring their hosts, parasites harm their environment, and if they are so indiscreet as to kill their hosts, they die too. Parasitiasis is intermediate between parasitosis and commensalism in some cases, but not in all.

HOST-PARASITE RELATIONS

Depending on their species, parasites may live in any organ or tissue of the host; they may live on its surface, or they may spend most of their time away from it. Special terms have been applied to these relationships. An *endoparasite* is a parasite that lives within the host's body. An *ectoparasite* is one that lives on the outside of the body. An *erratic* (or *aberrant*) *parasite* is one that has wandered into an organ in which it does not ordinarily live. An *incidental parasite* is a parasite in a host in which it does not usually live. A *facultative parasite* is an organism that is capable of living either free or as a parasite. An *obligatory parasite* is an organism which must live a parasitic existence. A *periodic parasite* is one which makes short visits to its host to obtain nourishment or other benefits. A *pseudoparasite* is an object that is mistaken for a parasite. Parasites may themselves be parasitized by *hyperparasites*.

An organism which harbors a parasite is its *host*. There are several types of host. A *definitive host* is the host which harbors the adult stage of a parasite. An *intermediate host* is the host which harbors the larval stages of the parasite. A *first intermediate host* is the first host parasitized by the larval stages of the parasite. A *second intermediate host* is the host parasitized by the larval stages at a later period in the life cycle. A *paratenic* or *transport host* is a second (or third) intermediate host in which the parasite does not undergo any development but usually remains encysted until the definitive host eats the paratenic host.

The *vector* of a parasite or disease agent is an arthropod, mollusc or other agent which transmits the parasite from one vertebrate host to another. If the parasite develops or multiplies in the vector, it is called a *biological vector*. If the parasite does not develop or multiply in it, it is called a *mechanical vector*.

Intermediate hosts of helminths are biological vectors, but biological vectors are not necessarily intermediate hosts. Indeed, the latter term has no application to protozoa, bacteria, rickettsia or viruses, none of which have larvae. Mosquitoes are biological vectors of malaria and of yellow fever, and the tsetse fly is a biological vector of *Trypanosoma brucei*, for the parasites must develop in them to become infective for the next vertebrate host. However, tabanid flies are merely mechanical vectors of *Trypanosoma evansi*, since the parasites undergo no development in them.

The terms *infection* and *infestation* are used by different people in different ways. The former term originally referred to internal agents of disease, while the latter was used with reference to external harassing agents, including not only ectoparasites but also rodents, pirates and thieves. This usage was current during the latter part of the nineteenth century. Later on, it was felt desirable to distinguish between parasites which multiplied in their hosts and those which did not. "Infection" was then used for the former type of parasitism, and "infestation" for the latter. This usage was popular for a time, but it was never universally accepted. More recently there has been a trend toward the older usage. Most American parasitologists have accepted it, but most British ones prefer to speak of helminth infestations. In this book *infection* will be used to refer to parasitism by internal parasites, and

infestation to parasitism by external parasites.

The term *life cycle* refers to the development of a parasite thru its various forms. It may be simple, as in an organism which multiplies only by binary fission, or it may be extremely complex, involving alternation of sexual and asexual generations or development thru a series of different larval forms. A *monogenetic* parasite is one in which there is no alternation of generations. Examples of this type are bacteria, flagellate protozoa such as *Trichomonas,* nematodes such as *Ascaris* and *Ancylostoma,* and the ectoparasitic fish trematodes of the order Monogenorida (= Monogenea). A *heterogenetic* parasite is one in which there is alternation of generations. Examples of this type are malarial parasites and coccidia, in which sexual and asexual generations alternate, the endoparasitic trematodes of higher vertebrates of the order Digenorida (= Digenea), in which there may be several larval multiplicative stages before the adult, and the nematode, *Strongyloides,* in which one generation is parasitic and parthenogenetic while another is free-living and sexual.

Depending on their type, parasites may live in only one or in a number of different types of hosts during the course of their normal life cycles. A *monoxenous* parasite has only one type of host--the definitive host. Examples are coccidia, amoebae, hookworms, fish trematodes, horse bots, streptococci and most pox viruses. A *heteroxenous* parasite has two or more types of host in its life cycle. Examples are the malarial parasites, most trypanosomes, trematodes of higher vertebrates, filariae, tapeworms, the rickettsiae, yellow fever virus and various encephalitis viruses.

These two pairs of terms are independent of each other. Parasitic amoebae and hookworms are monogenetic and monoxenous. Filariid and spirurid nematodes are monogenetic and heteroxenous. *Strongyloides* and most coccidia are heterogenetic and monoxenous. Malarial parasites and trematodes of birds and mammals are heterogenetic and heteroxenous.

Another group of terms deals with *host range,* i.e., the number of host species in which a particular parasite may occur. These parasites can be either monoxenous or heteroxenous, monogenetic or digenetic. Indeed, there may be a difference in host-restriction between the definitive and intermediate hosts of the same parasite. For example, the blood fluke, *Schistosoma japonicum,* can become adult in a rather wide range of mammals, but its larval stages will develop in only a few closely related species of snails.

The term, monoxenous parasite, is used by some authors for a parasite which is restricted to a single host species. Such parasites undoubtedly exist, but they are fewer than our present records indicate. The human malarial parasites were once thought to be monoxenous in this sense of the word, but they have more recently been found capable of infecting apes, and it is now known that chimpanzees in West Africa are naturally infected with *P. malariae,* the cause of quartan malaria in man (Garnham, 1958). Many species of coccidia are also known from but a single host, but for the most part closely related wild hosts have not been examined nor have cross transmission experiments been attempted with them. Because of this and because of the confusion arising between this usage of monoxenous and the one defined above, this usage should be avoided.

A *stenoxenous* parasite is one which has a narrow host range. Among the coccidia, members of the genus *Eimeria* are generally stenoxenous, as are the human malaria parasites and cyclophyllidorid tapeworms. Many nematodes such as the hookworms, nodular worms, filariids and spirurids tend to be stenoxenous. Both biting and sucking lice are stenoxenous, and many are even limited to specific areas on their host. Relatively few bacteria are stenoxenous, but *Streptococcus agalactiae, Mycobacterium leprae, Vibrio, Mycoplasma,* the spiro-

chete, *Treponema,* the rickettsiae, *Anaplasma, Eperythrozoon, Haemobartonella* and *Cowdria,* and the viruses of hog cholera, duck hepatitis and yellow fever are stenoxenous.

An *euryxenous* parasite is one which has a broad host range. Among the coccidia, members of the genus *Isospora* are often euryxenous. So are most trypanosomes, most *Plasmodium* species (but not those affecting man), and many species of *Trichomonas*. Most trematodes are euryxenous, as are *Trichinella, Dracunculus* and *Dioctophyma* among the nematodes. Fleas, chiggers and many ticks are euryxenous. Most parasitic bacteria are euryxenous; examples are most species of *Salmonella, Escherichia, Brucella, Erysipelothrix* and *Listeria.* Among euryxenous rickettsiae are *Rickettsia, Coxiella* and *Miyagawanella psittacii.* Among euryxenous viruses are those of rabies and many encephalitides. *Leptospira* and *Borrelia* are euryxenous spirochetes.

The use of these two terms, however, may be deceptive. There exist in nature all intergrades between them, and all we have done has been to pick out the two extremes of a continuum and give them names.

Actually, the host range of most parasites is broader than generally supposed. The fact is that most animal species have not been examined for parasites. For example, the genus *Eimeria* is one of the commonest and best known among parasitic protozoa. Becker (1956) listed 403 species, of which 394 were from chordates and 202 from mammals. This is quite impressive, especially to someone who wishes to study their taxonomy. However, according to Muller and Campbell (1954), there are 33,640 known living species of chordates and 3552 of mammals. Some hosts have more than one species of *Eimeria,* but some coccidian species occur in more than one host. Assuming that these more or less cancel out, we can calculate that *Eimeria* has been described from only 1.17% of the world's chordates and from 5.7% of its mammals. If all these possible hosts were to be examined, one might expect to find some 3500 species of *Eimeria* in mammals and 34,000 in chordates.

So far only the qualitative aspect of the host range has been discussed. However, altho a parasite may be capable of living in more than one host, it is much more common in some hosts than in others. The *principal hosts* of a parasite are those hosts in which it is most commonly found. The *supplementary hosts* are those of secondary importance, and the *incidental hosts* are those which are infected only occasionally under natural conditions. To these should be added *experimental hosts,* which do not normally become infected under natural conditions but which can be infected in the laboratory. This last category may include both incidental and supplementary hosts and also hosts never infected in nature.

In order to take into account this quantitative aspect of the host-parasite relationship, the terms *quantitative host spectrum* or *quantitative host range* are used. These give the amount of infection present in each infected species.

Several factors affect the quantitative host spectrum. One is geographic distribution. The natural quantitative spectrum may be quite different in one locality than in another. The species of animals present may be different, or the incidence of infection may be different. For example, a number of nematodes parasitize both domestic and wild ruminants. However, since the wild ruminants of North America and Africa are not the same, the quantitative host spectra of the same parasites on the two continents are different. The spectrum is still different in Australia, where there are no wild ruminants but where wild rabbits are susceptible to infection with a few ruminant nematodes.

A second factor is climate. Many of the same host species may be present in different areas but climatic conditions in one area may prevent or favor a para-

site's transmission. For instance, the common dog hookworm in most parts of the United States is *Ancylostoma caninum*, but in Canada it is *Uncinaria stenocephala*. This is due to a difference in temperature tolerance of the free-living larval stages.

Local conditions such as ground cover are also important. If the vegetation is open so that the sunlight can get down to the surface of the soil where a parasite's eggs, cysts or free-living stages are found, survival will be much less, transmission will be reduced and the numbers of affected hosts will be fewer than if the vegetation is thick and protective. Or the kinds and numbers of parasites in a herd of animals confined to a low, moist pasture may be quite different from those in a herd kept on a hill pasture or on drylot.

A fourth factor is that of the distribution of acceptable intermediate hosts. *Trypanosoma brucei* occurs only in Africa because its tsetse fly intermediate hosts occur only there. The fringed tapeworm of sheep, *Thysanosoma actinioides*, is found in the western United States but not in the east despite the fact that infected sheep have repeatedly been introduced onto eastern pastures. A suitable intermediate host does not occur on these pastures, so the parasite cannot be transmitted.

A fifth factor is that of chronologic time. The quantitative host spectrum may be quite different in the same locality at different periods, particularly if an eradication campaign has been carried out in the interim. Echinococcosis is a case in point. At one time it was extremely common in the dogs, sheep and people in Iceland, but it has now been eradicated. Gapeworms were once common in poultry in the United States, but as the result of modern poultry management practices they are now exceedingly rare in chickens and turkeys, altho they are not uncommon in pheasants.

A sixth factor is that of the ethology or habits of the host. A species may be highly susceptible to infection with a particular parasite, yet natural infections may seldom or never occur. The habits of the host may be such that it rarely comes in contact with a source of infection even tho both exist in the same locality. For example, wild mink in the midwestern United States are not infrequently infected with the lung fluke, *Paragonimus kellicotti*. It is easy to infect dogs with this fluke experimentally, yet it is extremely rare in midwestern dogs. The reason is that dogs rarely eat the crayfish which are the fluke's intermediate host.

Because of these factors, we must speak of *natural* and *potential* host spectra. The latter term refers to the absolute infectability of potential hosts and not to the natural situation. The natural host spectrum is an expression of the actual situation at a particular time and place. The two spectra may be quite different, and of course the natural one will vary considerably, depending on the circumstances. The complete host spectrum has not been worked out for any parasite, and to do so would be a very time-consuming process. However, it will have to be done, at least for the more important parasites, before we can fully understand their ecology and the epidemiology of the diseases they cause.

Certain parasites and diseases occur in man alone, others in domestic animals alone, and others in wild animals alone. Still others, including some important ones, occur in both man and domestic animals, man and wild animals, domestic and wild animals, or in all three. A knowledge of their host relations is important in understanding their ecology and epidemiology.

A disease which is common to man and lower animals is known as a *zoonosis*. Zoonoses were redefined in 1958 by the Joint WHO/FAO Expert Committee on Zoonoses as "those diseases and infections which are naturally transmitted between vertebrate animals and man" (World Health Organization, 1959). Less than 20 years ago it was said that there were 50 zoonoses, but in the above report the World Health Organization listed more

than 100, of which 23 were considered of major importance. Many more are certain to be revealed by future investigations.

Our thinking about parasites and diseases is ordinarily oriented toward either man or domestic animals. In this context, it is convenient to have a special term for hosts other than those with which we are primarily concerned. A *reservoir host* is a vertebrate host in which a parasite or disease occurs naturally and which is a source of infection for man or domestic animals, as the case may be. Wild animals are reservoirs of infection for man of relapsing fever, yellow fever and moist Oriental sore, while domestic animals are reservoirs for man of trichinosis and classical Oriental sore. Wild animals are reservoirs of infection for domestic animals of many trypanosomes, while man is a reservoir for domestic animals of *Entamoeba histolytica.*

Parasites and diseases may continue to exist indefinitely in their reservoir hosts, and man or domestic animals may become infected when they enter the locality where the parasites or diseases exist. Such a locality is known as a *nidus* (literally, "nest"). This term is used primarily in connection with vector-borne diseases, altho it need not be restricted to them.

Natural nidi may be elementary or diffuse (Palovsky, 1957). An *elementary nidus* is confined within narrow limits. A rodent burrow containing rodents, argasid ticks and relapsing fever spirochetes or a woodrat nest containing woodrats, kissing bugs and *Trypanosoma cruzi* is an elementary nidus. In a *diffuse nidus* the donors, vectors and recipients are distributed more widely over the landscape. A wooded region in which ticks circulate *Rickettsia rickettsii* among the rodents and lagomorphs is a diffuse nidus of Rocky Mountain spotted fever, as is an area where tsetse flies transmit trypanosomes among wild game. The *nidality* of a disease refers to the distribution and characteristics of its nidi.

The concept of the *deme* is useful in discussing host-parasite relationships, epidemiology, taxonomy, evolution, etc. (see Hoare, 1955). A *deme* is a natural population within a species. It lies more or less below the subspecies level, but it is not a formal taxon and is not given a Latin name. There are different types of deme. *Nosodemes* differ in their clinical manifestations. One example is *Leishmania donovani,* which has five nosodemes, Indian, Mediterranean, Sudanese, Chinese and South American, which produce different types of disease. *Serodemes* differ serologically. These are best known among the bacteria and viruses, but also occur among the animal parasites. *Tritrichomonas foetus,* for example, has several serological types or serodemes. *Xenodemes* differ in their hosts, and *topodemes* differ in geographic distribution.

There are also other types of demes. The population of a parasite species within a single host animal is a *monodeme,* and that in a single flock or herd is an *agelededeme.* Thus, a population of the stomach worm, *Haemonchus contortus,* in a single sheep is a monodeme, the population in all the sheep of a single flock is an agelededeme, that in all sheep is a xenodeme. The population in all cattle is another xenodeme and that in all goats is a third, the population of *H. contortus* in all hosts in North America is a topodeme, etc.

Each of these demes may differ morphologically and physiologically, and a large part of the taxonomist's work consists in determining the limits of their variation and deciding whether they are really demes or different species. Since the judgments of all taxonomists do not agree, there is some variation in the names which different parasitologists use. Demes are advance guards in the march of evolution, and no sharp line can be drawn beyond which they become subspecies or species. Taxonomists have been able to arrive at no better statement of how species are defined than to say that a species is what a specialist on its group says it is. And since some scientists are splitters and others are lumpers, their definitions vary with their temperaments. For most of us, the best rule is the pragmatic one of using those names which

make for the greatest understanding of the organisms we study and of their relations with each other and with their hosts.

Parasite evolution: Parasites have evolved along with their hosts, and as a consequence the relationships between the parasites of different hosts often give valuable clues to the relationships of the hosts themselves. Certain major groups of parasites are confined to certain groups of hosts. Sucking lice are found only on mammals. Biting lice occur primarily on birds, but a few species are found on mammals. The monogenetic trematodes are found almost without exception on fish; some of the more highly evolved digenetic trematodes are found in fish, but more occur in higher vertebrates. There is a tendency, too, for the more advanced digenetic trematodes to occur in the higher host groups.

One would expect that, as evolution progressed in different host groups, there would develop in each one its own group of parasites. This has often occurred. Thus, of the 48 families of digenetic trematodes listed by Dawes (1956), 17 occur only in fish, 8 only in birds, 3 only in mammals, 2 in fish and amphibia, 3 in reptiles and birds, 6 in birds and mammals, 1 in fish, amphibia and reptiles, 2 in reptiles, birds and mammals, 1 in amphibia, reptiles and birds, 3 in all but fish, and 2 in all five classes of vertebrates. Of the 11 classes of tapeworms recognized by Wardle and McLeod (1952), 4 are found only in elasmobranch fish, 3 only in teleosts, 1 only in birds, 1 in teleosts, amphibia and reptiles, 1 in teleosts, birds and mammals, and 1 in amphibia, reptiles, birds and mammals.

This same tendency is apparent even in parasitic groups which are quite widely distributed. For example, many reptiles and mammals (but not birds) have pinworms of the family Oxyuridae, but each group has its own genera. Iguanas have *Ozolaimus* and *Macracis*, other reptiles have *Thelandros*, *Pharyngodon* and several other genera, rodents have *Aspiculuris*, *Syphacia* and *Wellcomia*, rabbits have *Passalurus*, equids have *Oxyuris*, ruminants have *Skrjabinema*, and man and other primates have *Enterobius*.

On the other hand, there are many exceptions to this general rule, and it cannot be used without corroboration as the sole criterion of host relationship. Many fish-eating birds and mammals have the same species of trematodes for which fish act as intermediate hosts. And the fact that the pig and man share a surprising number of parasites is no proof of their close relationship despite their similarity of character and personality; it simply reflects their omnivorous habits and close association.

Adaptation to parasitism: Adaptation to a parasitic existence has required many modifications, both morphological and physiological. Locomotion, at least of the parasitic stages, has often become restricted. Certain organs and organ systems may be lost. Tapeworms lack an intestine altho their ancestors presumably had one, and adult trematodes have no eyespots altho their turbellarian ancestors and many of their larvae have them. Parasitic amoebae have no contractile vacuoles altho their free-living relatives do.

In contrast, many structures are modified or hypertrophied for the parasitic life. Many helminths have hooks and suckers to help them hold their position. The protozoon, *Giardia*, has turned most of its ventral surface into a sucking disc. The mouthparts of many insects and mites have become highly efficient instruments for tapping their hosts' blood supply. The chigger, which does not suck blood, has developed a method of liquefying its hosts' tissues. The food storage organs of many parasites have been enlarged. Many blood sucking arthropods which are unable to obtain all the nutrients they need from blood, have established symbiotic relationships with various microorganisms and have formed special organs for them.

The reproductive system of many parasites has been hypertrophied to produce tremendous numbers of eggs. Other parasites, such as the trematodes, have

developed life cycles in which the larvae also multiply.

In the parasites with high reproductive rates, infection is left largely to chance. Many other parasites, however, have developed life cycles in which chance is more or less eliminated. In these, the reproductive rate is low. The larva of the sheep ked, *Melophagus ovinus*, develops to maturity in the body of its mother and pupates immediately after emerging. The pupa remains in its host's wool. The female tsetse fly, too, produces fully developed larvae. The tropical American botfly, *Dermatobia hominis*, captures a mosquito and lays her eggs on it. These hatch when the mosquito lights to suck blood, and the larvae enter the host.

Morphological and developmental modifications are the most obvious ones, but biochemical ones are even more important. How do parasites survive in their hosts without destruction? What keeps those which live in the intestine from being digested along with the host's food? Why is it that morphologically similar species are restricted to different hosts which themselves may be morphologically quite similar?

The second question has been answered by saying that the same mechanism operates which prevents the hosts from digesting themselves, that the parasites protect themselves by producing mucus or that mucoproteins in their integument protect them, that they secrete antienzymes, or that the surface membrane of living organisms is impermeable to proteolytic enzymes. However, much more research must be done before a satisfactory answer can be given. Answers given to the first and third questions are vague. Compatibility of host and parasite protoplasm is invoked, but all this does is put a name to the beast. The question of how this compatibility is brought about remains unanswered, and a great deal of biochemical and immunochemical research must be done before it can be answered (see Becker, 1953; Read, 1950; von Brand, 1952).

Injurious effects of parasites on their hosts. Parasites may injure their hosts in several ways:

1. They may suck blood (mosquitoes, hookworms), lymph (midges) or exudates (lungworms).
2. They may feed on solid tissues, either directly (giant kidney worms, liver flukes) or after first liquefying them (chiggers).
3. They may compete with the host for the food it has ingested, either by ingesting the intestinal contents (ascarids) or by absorbing them thru the body wall (tapeworms). In some cases they may take up large amounts of certain vitamins selectively, as the broad fish tapeworm does with Vitamin B_{12}.
4. They may cause mechanical obstruction of the intestine (ascarids), bile ducts (ascarids, fringed tapeworm), blood vessels (dog heartworm), lymphatics (filariids), bronchi (lungworms) or other body channels.
5. They may cause pressure atrophy (hydatid cysts).
6. They may destroy host cells by growing in them (coccidia, malaria parasites).
7. They may produce various toxic substances such as hemolysins, histolysins, anticoagulants, and toxic products of metabolism.
8. They may cause allergic reactions.
9. They may cause various host reactions such as inflammation, hypertrophy, hyperplasia, nodule formation, etc.
10. They may carry diseases and parasites, including malaria (mosquitoes), trypanosomosis (tsetse flies), swine influenza (lungworms), salmon poisoning of dogs (flukes), heartworms (mosquitoes) and onchocercosis (blackflies).
11. They may reduce their hosts' resistance to other diseases and parasites.

A great deal more could be said about this subject. Additional information is given in the symposium on mechanisms of microbial pathogenicity of the Society for General Microbiology (Howie and O'Hea, 1955).

Resistance and Immunity to Parasites. This is such a tremendous subject that its facets can only be hinted at. The general principles of immunology apply to animal parasites as much as they do to bacteria, viruses and other microorganisms. However, since the association of many of the larger parasites with their hosts is not as intimate as that of microorganisms, the hosts' immune responses may not be as great. This is especially true with regard to the formation of circulating antibodies.

Immunity or resistance may be either *natural* (*innate*) or *acquired*. Natural resistance is the basis of host-parasite specificity, but, as mentioned above, little is known of its mechanism. Acquired immunity may be either *active* or *passive*. Active immunity results from the body's own action. It follows exposure to living or dead disease agents, and can result from natural infection or artificial administration of virulent, attenuated or killed organisms.

One type of active immunity is *premunition*. This is immunity due to the continued presence of the disease agent. It occurs in such diseases as babesiosis and anaplasmosis.

Passive immunity results from the introduction of antibodies produced by some other animal. It may be acquired naturally, thru the colostrum or milk in mammals or thru the egg yolk in birds, or artificially by injection of antiserum. Passive immunity is seldom as long-lasting as active immunity.

Immunity against parasites and disease agents generally increases with age. There are exceptions, however. Young cattle, for instance, are more resistant to *Babesia* and *Anaplasma* than are adults. Age immunity may be either developed as the result of previous exposure or it may be natural. Not all the factors operating in the latter case are known. An important one is that very young animals cannot mobilize their body defenses against invasion as efficiently as adults. For instance, they do not produce antibodies at first, depending on those acquired from their mothers. Another factor, discovered by Ackert and his co-workers (cf. Ackert, Edgar and Frick, 1939) to explain the relative resistance of older chickens to *Ascaridia galli*, is that these birds have more intestinal goblet cells than do young birds. The goblet cells secrete mucus which inhibits the development of the worms. For further information on immunity in parasitic infections, see Taliaferro (1929), Culbertson (1941) and Soulsby (1960).

Genetic constitution is also important in determining resistance to parasites. For instance, Ackert *et al.* (1935) showed that Rhode Island Red and Plymouth Rock chickens are more resistant to *Ascaridia galli* than are Buff Orpingtons, Minorcas and White Leghorns. Cameron (1935) found that in a mixed flock of sheep, Cheviots were less heavily parasitized with gastrointestinal nematodes than Shetlands and Scottish Blackface, and that these in turn were less heavily parasitized than Border Leicesters. Stewart, Miller and Douglas (1937) found that Romney sheep were markedly resistant to infection with *Ostertagia circumcincta*, while Rambouillets were less so and Southdowns, Shropshires and Hampshires were least resistant. Certain individuals among the more susceptible breeds, however, were just as resistant as the Romneys. Whitlock (1958) has studied genetic resistance to trichostrongylidosis in sheep in some detail.

The nutritional status of the host may affect its resistance. Poorly nourished animals are usually more susceptible to infection and suffer more severely from its effects. Protein depletion or protein starvation is particularly important. Lack of specific vitamins and minerals generally decreases resistance, but there are cases in which lack of a certain vitamin which the parasite requires may affect the parasite adversely. Thus, Becker and Smith (1942) found that when calcium pantothenate was added to a ration containing restricted vitamins B_1, B_6 and pantothenate, the number of oocysts produced by *Eimeria nieschulzi* infections in the rat was increased.