Introduction to Parasitology

Parasitism is the most common way of life; more than 50% of all animal species are parasites. Parasites occur in all animal species, and they may have a profound effect on the health of people, domestic animals and wildlife. Parasitology is the study of parasitism; a multidisciplinary subject covering many topics including morphology, taxonomy, biology, behaviour, life-cycles, pathogenesis, epidemiology, ecology, physiology, biochemistry, genetics and molecular biology, as well as the diagnosis, immunology and treatment of infections.

Parasites live at the expense of their hosts whereas other symbiotes may be mutualists (living in mutual benefit with host) or commensals (living without benefit or detriment to host). Parasites may infect the gastrointestinal tracts or circulatory systems of their hosts; they may invade different tissues and organs or they may live on the external surfaces of their hosts. Many infections may be asymptomatic whereas others may cause acute (transient) or chronic (persistent) clinical diseases ranging markedly in severity (mild to fatal). Parasitic infections may cause mortality (foetal, neonatal, adult death), morbidity (disease manifest by enteritis, fever, anaemia, etc.), production losses (reduced meat, milk, fibre production), and tissue lesions (reduced marketability of product). Despite many advances in parasite treatment and control, infections still persist due to many factors, including urbanization (crowding together); more intensive farming systems, greater translocation of animals, further land and marine development, inadequate effluent disposal, emergence of parasite drug resistance, and spread of vector insecticide resistance.

Parasite assemblages

Many types of organisms have adopted a parasitic mode of existence; that is, they require a host for their own survival. Three major groups of parasites are recognized: protozoa (belonging to the kingdom Protista), and helminths and arthropods (belonging to the kingdom Animalia, or Metazoa).

Protozoa: Over 10,000 species of single-celled protozoa have been described in the gut, blood or tissues of vertebrate and invertebrate hosts. Parasitic flagellates cause enteric diseases such as giardiasis, urogenital diseases such as trichomoniasis, systemic diseases such as sleeping sickness, and tissue diseases such as Chagas' disease and kala azar. Parasitic amoebae cause dysentery, meningoencephalitis and corneal lesions. Spore-forming sporozoa cause many serious diseases: Apicomplexa cause coccidiosis, malaria and tick fevers; Microspora parasitize fish and insects; and Ascetospora cause seasonal mortalities in oysters. Parasitic ciliates cause diarrhoea or lesions in humans and animals while commensal species cause serious fouling problems in aquaculture.

Helminths: Around 50,000 species of multicellular helminths (worms) have been described from a wide range of hosts. Roundworms (nematodes) cause much morbidity and mortality in humans and animals throughout the world. Serious infections include filiariases, hookworm and threadworm diseases. Larval and adult tapeworms (cestodes) may be found in many vertebrate hosts. Some species do not cause clinical disease whereas others may cause severe weight loss, diarrhoea, abdominal pain or space-occupying lesions. Flukes (trematodes) include many important species such as sheep liver fluke and human schistosomes or blood flukes.

Arthropods: Thousands of arthropods are parasitic at some stage in their life-cycles. Many cause serious diseases and limit agricultural productivity. Parasitic insects include biting and sucking lice which may cause skin lesions or anaemia, fleas which may cause allergic dermatitis, and various flies which suck blood as adults or produce larvae which feed on host tissues. Parasitic arachnids include ticks which feed on blood and may cause anaemia or paralysis and mites which feed on skin and may cause mild itching, hair loss or severe mange.

Overview of parasitology

Parasitism

Three general environments are available for life as we know it: terrestrial, aquatic and biotic. By definition, parasites are those animals which occupy the last niche, i.e. live in or on another species, their host. Parasitism is a form of symbiosis, an intimate relationship between two different species. There is a biochemical interaction between host and parasite; i.e. they recognise each other, ultimately at the molecular level, and host tissues are stimulated to react in some way. This explains why parasitism may lead to disease, but not always. It is often a life-long relationship for the parasite, which cannot survive without its host. While it is often claimed (even by definition) that a parasite must damage its host in some way (to distinguish parasitism from commensalism and mutualism), in practice this can be impossible to establish, because we know so little about most symbiotic relationships; certainly, many human parasitic infections are asymptomatic (which is not the same as non-pathogenic).

Origins

Parasitism must have arisen very early in the history of life on Earth, when primordial microorganisms learnt to survive inside other cells which they had invaded either passively (e.g. by phagocytosis) or actively (e.g. by penetration). When multicellular organisms with alimentary tracts appeared, they would have inevitably (accidentally or intentionally) eaten free-living microorganisms (and, later, free-living helminths). Ingested animals that managed to survive in this new environment would have appreciated the nutrient-rich environment; energy saved in looking for food could then be diverted to proliferating and resisting the host's efforts to dislodge them. With time, these parasites became so adapted to life in the host; they "forgot" how to survive outside. However, to succeed, they still needed to produce offspring that could negotiate the outside world to find new hosts.

Not surprisingly, all parasitic animals have free-living counterparts to which they are clearly related, and the greatest diversity of parasites is still found within the alimentary tracts of "higher" animals. As host species diverged with evolution, they "carried" with them their parasites. It is virtually the rule today that parasitic protozoa and helminths found in any vertebrate species have almost identical relatives in related vertebrates, and most of them are exquisitely host-specific. For example, the two common amoebae of the human colon, *Entamoeba histolytica* and *E. coli*, have almost identical relatives within a wide range of vertebrate hosts. There is even *E. moshkovskii*, a species that has been found only in sewers, which probably evolved from parasitic species! *E. gingivalis* occurs only in the human mouth, and has lost its cystic stage, presumably because trophozoites are so efficient at transferring between hosts. The same occurs with helminths, e.g. the roundworm of the human small intestine, *Ascaris lumbricoides*, has counterparts in pigs, dogs, cats, flying foxes, elephants, dolphins and many other mammals.

Once established in the host intestine, some parasites "learned" to invade the gut mucosa and deeper tissues, or to survive in the guts of predators that consumed their original hosts. Involvement of invertebrate "micropredators" in such life-cycles could then have led to parasite transmission via blood or tissue ingestion. Other parasites, in their infective stages, developed the ability to invade via the skin. It is not too difficult to conceptualise how complex life-cycles, utilizing a range of different hosts, might have arisen. Many examples of "missing links" in parasite evolution can still be found today; although far more are well-and-truly extinct. It is misleading to think of extant protozoan or helminth species as "primitive", for they have been evolving as long as all other species, including *Homo sapiens*, and utilise sophisticated survival mechanisms that we are only beginning to understand.

Parasitism clearly has advantages over independent existence, for parasites greatly outnumber free-living animals, both in terms of individuals and species; from an evolutionary viewpoint, it is the ultimate life-style. The obvious benefit to the parasite is that its host provides, *gratis*, a relatively stable, nourishing home. The energy saved in seeking food, shelter and transport is then concentrated on reproducing and evading host defence mechanisms, which are provoked in virtually every case, although not always obviously.

Fields of study

Medical Parasitology is the study of those organisms which parasitise humans. According to the definition above, parasites could include the viruses, bacteria, fungi, protozoa and metazoans (multicellular organisms) which infect their host species. However, for historical reasons (and because they are NOT classed as animals), the first three have been incorporated into the discipline of Microbiology. Parasitology claims those protozoa (unicellular animals), helminths (worms) and arthropods (insects and arachnids) whose existence depends on the availability of host animals, i.e. they are obligate parasites. Some rare parasites are called facultative, because they can survive and reproduce without a host, but very few that infect humans belong to this group (e.g. free-living amoebae). While we could argue about whether certain insects and mites are "temporary parasites" or "micro-predators", insects as a group belong to the discipline of Entomology, while ticks and mites are the concern of Acarology. Another crude way of distinguishing these is to label them ectoparasites (living on the host body surface), in contrast to the endoparasites (which live inside the host). The major contribution of insects in Parasitology is as vectors of several infections, although several are true parasites in their own right.

The disciplines also differ in ways other than taxonomic boundaries. In Microbiology, while morphology or staining properties (e.g. with Gram's stain) are important in the basic categorisation of the organisms, species identification generally depends on culturing and identifying specific enzymatic reactions, antigenic configurations or DNA sequences; i.e. the test-tube is important. In Parasitology, morphological recognition remains foremost, so that parasites (or their vectors) are still identified on characteristic shapes and sizes; i.e. the microscope rules supreme. Subspeciation or strain-typing is less well-developed, and may depend on molecular configurations or host-specificity. Culture has been a basic tool in Microbiology almost from its inception, and cell-culture is especially important in Virology (where viruses are not observed directly, but initially recognised by their effects on cultured cells). In Parasitology, culture was for a long time virtually impossible for most organisms, including protozoa. Nevertheless, in recent years, technical advances have allowed the *in vitro* cultivation of increasing numbers of parasite species, including even some helminths, although this is a procedure still in its infancy and used largely in research, rather than for routine clinical diagnosis. Advances in molecular biology are revolutionising all the biological sciences, including Parasitology. However, the organisms still must be identified initially on their morphology, and this is the basis of most parasite diagnoses made in clinical pathology laboratories.

Every known species (living and extinct) is assigned a unique combination of genus and species names which, by convention, are printed in italics or underlined. Infections with parasites are often indicated by the abbreviated genus name plus the suffix -osis. Some authorities use the suffix -iasis if the infection causes disease, but this distinction is often meaningless or impossible to establish. Purists argue that -osis belongs to species names derived from Greek, while those with Latin parentage deserve -iasis (it becomes tricky if you don't know the name's origins). Either can be used, depending on which sounds better (although a recent international convention aims to standardise all this), and we must be tolerant of the many exceptions, e.g. tuberculosis (mycobacteriosis), malaria (plasmodiosis), elephantiasis (lymphatic filariasis, or filariosis). If more than one parasite belongs in the genus, then the species name may be added to qualify the infection, *e.g.* schistosomiasis mansoni (not italicised).

Life-cycles

While parasites are adapted to living in or on their hosts, they can only survive by producing offspring capable of finding new hosts. The key to understanding their dispersal through the world is through knowledge of their life-cycles or modes of transmission, involving many aspects of parasite biology, reproduction and epidemiology.

Protozoa, in their motile, feeding, growing, asexually-multiplying forms are known as trophozoites (*trophe* = nutrition; *zooite* = minute animal). These are adapted for existence in the host and, generally, are unable to survive the rigours of life outside. Under appropriate conditions, which we do not yet understand, some trophozoites of gut protozoa coat themselves in a protective shell and shut down metabolically, to become cysts. These are designed to survive in the outside world long enough to reach new hosts. In the most highly-evolved protozoa (apicomplexans), which are obligate intracellular parasites, asexual division of the trophozoite (schizogony; *schizein* = to divide, or split; *-gony* = reproduction) leads to the generation of many merozoites (*meros* = piece, segment) which then invade other host cells. Eventually, instead of undergoing further schizogony, merozoites undergo sexual reproduction (gamogony) developing into either macrogametocytes (female) or microgametocytes (male). Fertilisation results in the formation of a zygote, termed an oocyst (= egg-cyst), which is designed to survive in the outside world so that it may infect another host. The ripe (sporulated) oocyst contains infective "seeds" known as sporozoites, which arise during its maturation (sporogony = generation of spores).

The metazoan parasites (multi-celled worms and arthropods) generally are dioecious, i.e. adults occur as separate males and females. Tapeworms and most flukes are the exceptions (hermaphrodites). After copulation, females produce fertile eggs, each containing an embryo. This undergoes embryonation developing into a juvenile or larva which will hatch out under suitable conditions. The egg may be the infective stage, or larvae may develop in the outside world to infectivity, or larvae may develop further in one or more intermediate hosts before they are able to reinfect their definitive hosts. Because their larvae must develop outside the host, adult helminths cannot multiply directly within a host (in stark contrast to protozoa which can proliferate to large numbers).

Many parasites complete their developmental cycle in a single host species (monoxenous life-cycles) while others require multiple host species (heteroxenous life-cycles). When multiple hosts are involved, the definitive host is that species in which the adult (or sexual) form of the parasite occurs, whereas the intermediate host is the species which supports the development and/or multiplication of the non-sexual, or larval (for helminths), stages of the parasite. Intermediate hosts which physically carry the infective stage from one host to another are also termed vectors; they are mechanical vectors if they simply transmit the parasite (unchanged and non-multiplied), and cyclical vectors if they also function as true intermediate hosts that support essential development and/or proliferation of the parasite. Intermediary hosts may be optional in some helminth life-cycles; the parasite might not undergo essential development in them, although it may increase in size. These paratenic hosts carry parasites through food chains to the definitive host, ensuring successful transmission even when the hosts are thinly dispersed through the environment. Some parasites exhibit low specificity for their definitive and/or intermediate hosts and so can develop in a range of animal species.

A zoonosis is a human infection caused by an organism which occurs naturally in other animals, known as reservoirs of infection. Most parasite life-cycles that are known have only been worked out quite recently; i.e. within the last 100 years. Information is therefore fragmentary, and many ambiguities exist. We could argue about whether the mosquito genus *Anopheles* or the primate species *Homo sapiens* is the definitive host for malaria parasites as gamogony in initiated in the human but culminates in fertilization in the mosquito.

Host specificity

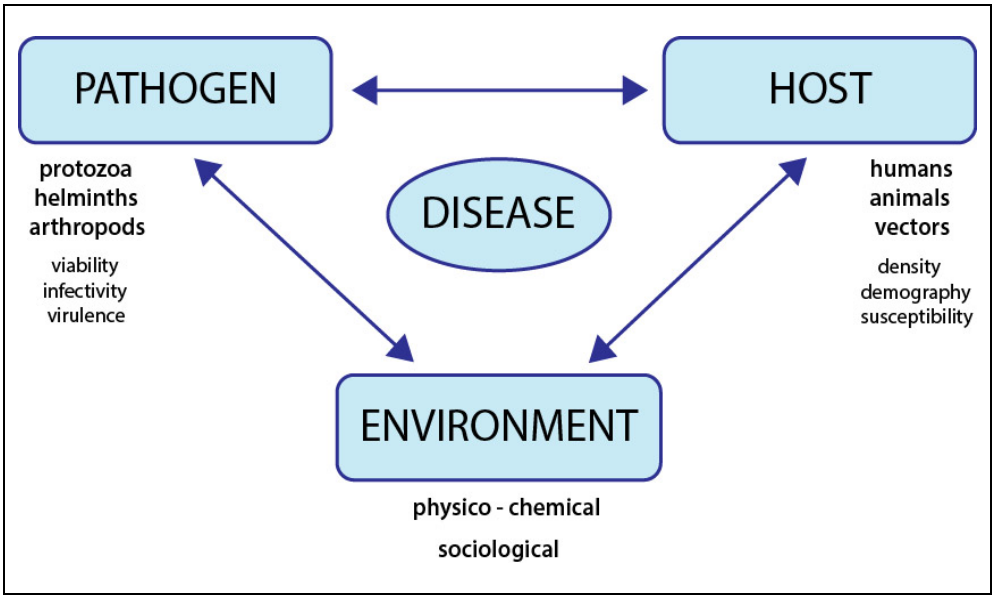
Parasites can be very particular about which host species they will use; this can apply to definitive as well as intermediate hosts. A parasite that is specific for a single host species is said to be oioxenous, one that parasitizes closely-related hosts is stenoxenous, while one that parasitizes unrelated hosts is euryxenous. Host-specificity is determined by a complex of factors, some obvious and others still obscure. The first requirement is that the prospective host shares its environment with the parasite (ecological specificity); e.g. parasites of dolphins might not have much luck infecting humans who don't live near the sea (although modern food transport networks have changed this!). Secondly, host behaviour must expose it to the parasite (ethological specificity); e.g. people who eat dolphin food (fish) may acquire parasites intended for dolphins. Finally, once the parasite comes into contact with the host, it must recognise appropriate cues and feel comfortable within its new surroundings (physiological specificity); e.g. if a parasite of dolphins thinks it is in a large fish or a dolphin when it arrives in the human gut, it may then behave accordingly. Obviously, this last determinant of host-specificity is the one we understand least.

Parasites interact with host secretions and surfaces and membranes: they must recognise and respond to molecular configurations (receptors/ligands). Detection of subtle variations in metabolites allows them to follow road-maps; they need to make critical changes in behaviour and development according to changes in host physiology/behaviour (neural/endocrine cues?); and they must be satisfied with their diet (host intestinal contents, blood and/or tissues). Clearly, all these combinations are unique for each host species, and vary even among individuals within a species, within an individual host throughout its own life-cycle and even throughout a 24-hour day. Likewise, each population of parasites is heterogeneous, so some individuals succumb very easily if in the wrong host ("losers") whereas others persist and may come close to full development ("pioneers"). This is the driving force of evolution, and parasites are the most rapidly evolving animals.

Epidemiology

This is the descriptive and analytical study of how diseases or infective organisms are distributed in human populations. A parasite is endemic to a geographical region if it is sustained by transmission amongst people living there. An infection maintained in animal populations is enzootic (which must apply to all zoonoses), although this term is going out of fashion. An infection acquired locally (usually in an endemic region) is autochthonous. Infected people who bring an organism into a non-endemic area are labelled imported cases; should the parasite then transfer to another person in that region, the secondary case becomes an introduced infection. If the parasite establishes in the new population, it becomes newly-endemic.

Many workers recognize an 'epidemiological triad' of inter-connected factors required for infectious disease outbreaks: viz; the pathogen must be present; susceptible hosts must be present; and environmental conditions must support the viability of both. The manifold interactions between these major determinants influences disease prevalence, distribution, severity (morbidity and mortality) and transmission. Disease control programmes should address all arms of the triad to ensure interventions are integrated, appropriate and effective. For example, treating whole communities with drugs may seem effective over the short-term, but it does not provide the hosts with any lasting protection and does not decontaminate the environment so hosts become re-infected.



The level of infection in a population is measured by prevalence and incidence. Prevalence refers to the prevailing level of a condition within a defined population, and is best applied to conditions without a clearly identifiable onset, such as most helminthic infections, chronic toxoplasmosis, Chagas' disease (or malignant, degenerative or metabolic diseases). It is measured by a single study of a population over a brief time-period (cross-sectional survey). Incidence refers to the number of new cases acquired per unit of population per unit of time, and is more meaningful for acute, short events (incidents), with an identifiable beginning (or end!) e.g. many viral infections, acute malaria (or deaths, or accidents). It can be measured only by monitoring a population over a sufficient period or time (longitudinal study) and determining the rate increase or decrease. Obviously, the incidence, prevalence and duration of a particular condition are closely and simply inter-related. An epidemic occurs when the incidence of new cases significantly exceeds the usual rate; if the disease is protracted, this will be reflected by an increase in prevalence as well.

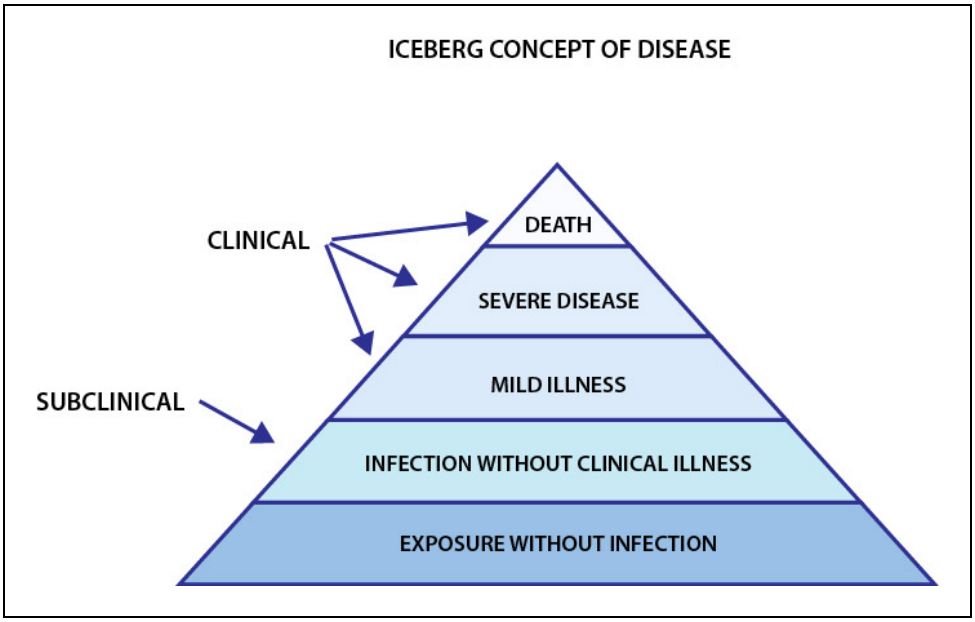
Quantitation of infection

Infective organisms have been categorized as either microparasites, which multiply directly within the host (all the microbes, plus protozoa) or macroparasites, which generally cannot multiply in the host; their numbers depend on how many infective eggs or larvae are taken on board. Ectoparasites do not happily fit into this classification, for they are clearly "macro", but often can multiply to huge populations on the one host. However, their development may be considered "external", as they usually reside outside the host on the surface. The term "infestation", sometimes used for macroparasitic infections, is going out of fashion, but can be applied to contaminated inanimate objects, e.g. a house infested with fleas, or bushland infested with ticks.

Infection with microparasites is an all-or-none situation; you either have measles, influenza, bubonic plague, toxoplasmosis, giardiasis, etc., or you don't. It is not often possible, or necessary, to quantify reliably the intensity of such an infection (number of organisms on board a host). In many instances, the severity of disease is not reliably related to the numbers of parasites detectable in blood, tissues or secretions (a notable exception is malaria, in which the percentage of infected red cells can be estimated and sometimes is important clinically). In the case of helminths and arthropods, which are generally visible macroscopically as discrete individuals, the number of organisms is meaningful, because it can be measured and does influence the likelihood or severity of disease. Therefore, in epidemiological studies of macroparasitic infections, their intensity becomes important, in addition to incidence and prevalence. Virtually all population studies have shown that the intensity of infection does not follow a normal distribution, but exhibits an "aggregated" or "over-dispersed" pattern: a small number of hosts harbour most of the parasites, whereas most individuals carry few or no parasites (characterised mathematically as a "negative binomial distribution").

Clinical and pathological considerations

While, by definition, a parasite should evoke a host reaction, there need not be any obvious adverse effects because, in the great majority of cases, infected individuals exhibit little evidence of disease. In many cases, it can be difficult or impossible to determine whether an organism is a parasite or commensal (e.g. many intestinal protozoa, and worms). However, other parasite infections do cause serious disease, to such an extent that they become major public health problems. It is generally assumed that the longer a parasite and its host species have co-evolved, i.e. have had time to adapt to each other, the less pathogenic the infection becomes. On the other hand, infections with parasites that are poorly adapted to humans, i.e. zoonoses, are more likely to cause serious disease. Some believe parasite virulence and host resistance tend towards enzootic stability over time as it is not in the best interest of either partner to kill the other, despite considerable evidence for the escalating 'arms race' observed between parasite aggressiveness and host immunity. More recently, others have noted a negative correlation between parasite virulence and transmission suggesting a 'trade-off hypothesis' between these factors (increased virulence results in decreased transmission). However, there are many exceptions to these "rules". Remember, clinicians generally only see those individuals who develop disease; there may be many more who remain well, even though infected. Clinicians often liken this scenario to observing an "iceberg" – they generally only see the tip-of-the-iceberg (hosts with disease) while the bulk remains hidden (hosts with subclinical infections, asymptomatic carriers).

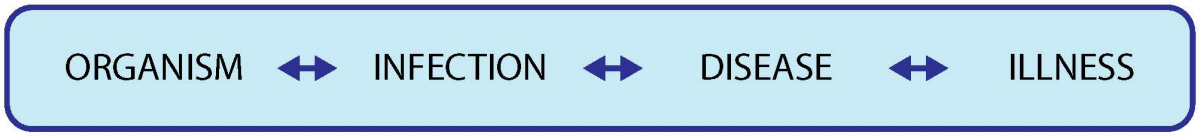


The development of disease depends on both host factors **(**susceptibility/resistance**)** and parasite factors (pathogenicity/virulence). Hosts have been shown to be more susceptible to disease by virtue of their age (neonates and geriatrics), gender (females, mainly during pregnancy and lactation), nutritional state (malnourished), physiological state ('stressed' hosts) and immunological competency (greater in immuno-suppressed or immuno-compromised hosts). From the parasite perspective, the major important determinant for the development of disease is parasite pathogenicity (or virulence), i.e. capacity to induce disease, including the inter-related factors of invasiveness (motility, enzyme secretion, presence of specific tissue receptors, induction of phagocytosis), fecundity (rate of producing offspring), means of egress from host, stimulation and/or suppression of immunity and inflammation, production of exo- and endo-toxins and resistance to host defences. Such virulence-determinants often correlate directly with the parasite's capacity to survive and reproduce, and they also may adversely affect host survival and fecundity. There often appears to be a negative correlation between parasite virulence and parasite transmission, where increasing virulence comes at the cost of decreasing parasite survival and thus transmission (Trade-off Hypothesis). Parasitism applies a pressure on host populations that selects out more resistant individuals; it has even been argued that parasites serve to improve the fitness of their host species (Red Queen Hypothesis), and were a major influence in the evolution of sex! However, genetic changes that increase resistance to infection often handicap the host in other ways, generating a dynamic equilibrium between protection against infection and susceptibility to other diseases (balanced polymorphism). There is no doubt that infectious organisms exert a powerful and continuous evolutionary pressure on host populations (and vice versa).

Parasitic diseases

In the field of infectious diseases, it is conceptually important not to confuse aetiological agents with their effects on the host. An infection occurs when an organism, i.e. the parasite, is found in its host. Some experts don't like to label this an infection, unless there is evidence of a response in host tissues; this applies particularly to commensal organisms, which normally occur on human skin or in the gastrointestinal tract, but which cause disease only when they breach the surface barriers. Infection is a host-organism interaction; it cannot exist without a host. Presence of infective organisms in the environment, e.g. in food, on fomites or in water, is not infection, but contamination (or "infestation"). For instance, we should not talk about "infected water supplies".

Moreover, an infection is not the same as a disease, which is a pathological change in the host, i.e. abnormalities induced in tissues by direct mechano-chemical damage and/or release of toxins and/or inflammatory mediators. Illness occurs when the host suffers the effects of the disease and becomes a patient, i.e. complains of symptoms (subjective, felt by the patient) which interfere with normal life, and perhaps manifests clinical signs (objective, detectable by the doctor), always with psychosocial undertones and ramifications. This is summarised as follows:



Where you start in the above sequence depends on whether you are the parasite or the patient! The illness is what the patient complains about to the doctor (often with judicious prompting), the disease is what the clinician may detect on physical examination (and the pathologist confirms in laboratory tests or at autopsy), and the causative organism (or its products, or antibodies to it) is what the diagnostic laboratory usually seeks and identifies. In any particular patient, all of these apparent components might be totally unrelated, so that linking them together becomes a major and still unresolved difficulty, even in some very common infections. Such distinctions may seem pedantic, but their appreciation helps in understanding stages in the evolution of an infectious disease, and is important to minimise confusion. Many people are infected; in fact, every one of us has at some time harboured at least one parasite species, and most of the world's people carry many parasites most of the time. However, relatively few are diseased, and not all of them suffer illness. Infections without illness are called subclinical or asymptomatic. Note that this does not mean being free of disease.

The interval between exposure to infection and the onset of illness is known as the incubation, latent or pre-patent period or phase. Some authorities define this period as the time from exposure to the time of becoming infective to others, but not all agree with this. Others define the latent period as the time from exposure to the first occurrence of recognisable specific manifestations, be they symptoms, signs, positive serology or other laboratory findings; if for symptoms, then it is called the incubation period. With many parasitic infections in endemic areas, these definitions may be of little use clinically, because people are repeatedly being exposed to infection.

An infection is patent when direct evidence of the organism can be detected, e.g. in the patient's faeces, blood or secretions, regardless of whether symptoms have appeared. Some infections may be patent but subclinical; others may cause illness, yet not be patent. However, the individual who has patent infection is essential to transmission of the organism, because it can then be transferred directly to other hosts, to vectors, or into the environment, where it may need to develop through stages to infectivity. Obviously, the detection of patency depends on the sensitivity of the test being used to identify the organism.

Often, indirect evidence of infection is the best that can be offered by the diagnostic laboratory, in the form of circulating antibodies generated against antigens of the infecting organism. Apart from the issues of specificity and sensitivity, another difficulty common to all antibody tests is distinguishing between ongoing, active infection and recently resolved or past infection. In other cases, serology may be even less adequate, for the simple reason that a test has not been developed, and the infection is known as cryptic. In some infections, specific monoclonal antibodies can be used to identify parasite antigens, and the polymerase chain reaction is becoming increasingly available to identify nucleotide sequences from infective organisms, although the limitations of these technological advances have not been well-established.

Usually, disease results predominantly from the host's efforts to deal with the parasite, involving immunological and other less well understood responses to an organism which refuses to go away, and which utilises effective strategies to avoid being damaged. The principles (and even details) of host responses to infecting organisms apply equally to microbial and parasitic infections and, as we learn more about the precise mechanisms, the more difficult it becomes, in the clinical context, to justify the separation of these groups of pathogens. Obviously, viruses may succumb more readily than worm larvae to protective mechanisms involving antibodies, complement, lymphocytes, phagocytes and other effector cells and molecules, but all infections initially

trigger similar basic repertoires of responses. A major discriminating influence is whether the organisms are intra- or extra-cellular, which partly determines the class of MHC molecules with which they interact. The minute parasitic protozoa that multiply in host cells have much in common with viruses, so that host responses to these infections and the resulting diseases can be so similar that, clinically, they may be indistinguishable.

Furthermore, patients have only a limited range of symptoms to complain about, so that generally it is impossible to diagnose the causative organism from the clinical features. However, a careful history, taken to evaluate the likelihood of exposure to specific parasites, often narrows the range of options (differential diagnosis), and indicates the specimens which should be sent to the laboratory for definitive diagnosis. This can be much cheaper (and much more satisfying for the clinician - and the patient) than running a battery of tests blindly, and is essential for effective treatment and suitable preventive measures.

Parasitological parameters

Parasitic infections can be studied from many angles: we can focus on the parasites, their hosts, the environments they share and the ways in which they interact. People working in this field come from numerous backgrounds, including zoology, physiology, biochemistry, immunology, molecular biology, pharmacology, ecology, economics, anthropology, sociology, engineering, agriculture, education, mathematics and, of course, human and veterinary medicine. Specific disciplines focus on specific aspects, thus parasitological knowledge may be fragmentary. In order to obtain (retain) a holistic overview, many parasitologists use a parametric approach to organize information. The following headings have proven useful:

- AETIOLOGY (study of causative agent: in this case, the parasitic organism): colloquial and scientific (binomial) species name, broad group e.g. amoeba, nematode; stages occurring in humans (larva, cyst, trophozoite etc.); approximate size/shape.

- LIFE-CYCLE (summary of biology): Hosts – definitive, intermediate, paratenic; anatomical locations and sites of multiplication; development and survival in intermediate host/environment.

- EPIDEMIOLOGY (dispersal in populations): Distribution, worldwide and in endemic areas; prevalence, by age, sex, occupation (demographics); transmission into and out of hosts; ecological determinants, i.e. geography, climate, vectors, human behaviour and resources (housing, hygiene, sanitation, nutrition, occupation, animal reservoirs, medical facilities, preventive and control measures).

- PATHOGENESIS (dispersal within host; mechanisms of disease): sites affected; mechanical and/or chemical damage; local and systemic host responses (acute and chronic); effectiveness of immunity; effects on transmission; host resistance/susceptibility.

- CLINICAL MANIFESTATIONS (how patient affected): logical extension of knowledge on pathogenesis; know mainly which organ system(s) involved and how manifests; symptoms and signs.

- DIAGNOSIS (how detected): specimens required; how and when collected, preserved, transported; how examined in laboratory; reliability of results (sensitivity, specificity, predictive values); safety aspects (i.e. infectivity of clinical specimens, their handling and disposal).

- TREATMENT (therapy): Is it necessary? effective? safe? Types of drugs, dosages, spectrum of activity, modes of action; contra-indications, side-effects; compliance; susceptibility/resistance

- PREVENTION/CONTROL (prophylaxis/intervention/management): public health concerns (individual, family, community: who is at risk?); chemoprophylaxis (necessary under what conditions?); interruption of transmission; behaviour modification; patient/community education; prospective/retrospective screening programmes, vaccination (availability, strategies); environment/food/water contamination, purification/disinfection.

Students and practitioners should always remember that parasitology demands good comprehensive knowledge of at least two biological entities, the parasites and their hosts. Only by studying both in concert can we begin to understand the complex interactions between them, particularly those resulting in disease.