THE PARASITES OF MAN

CAMERON

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IN TEMPERATE CLIMATES

By

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PREFACE

PARASITOLOGY is so old that no one knows its origin. At the same time it is only in the past few decades that it has become a science, but during these few decades no science has had a more spectacular advance. It has opened up the tropical continents to the European, it has shown how to conquer some of the most virulent and important diseases of mankind and it has had a very far-reaching effect on commerce and migration. So many of the parasitic diseases of man are most conspicuous in the tropics that until recently, parasitology as a subject commensurate with bacteriology, has been taught only in schools of tropical medicine. But parasites are not confined to the tropics; many are common in temperate and sub-tropical climates as well, while some are actually confined to the temperate zone. Many, common in the tropics, are common only because of a more primitive hygiene and not because of climate. Moreover, we no longer measure the distance from the equator in miles, but in hours; increasing numbers of persons visit the tropics yearly, and the medical man of north temperate climates can no longer afford to be wholly ignorant of diseases outside of his immediate zone of interest.

Accordingly, this work, based on the course of animal parasitology given to the Medical School of McGill University, has been prepared. It is intended for the medical man who practises in the English-speaking temperate and sub-tropical zones. Only parasites which actually occur in North America or Great Britain are discussed in detail; those which may be introduced in patients from the tropics, but which cannot become acclimatized, are dealt with as briefly as possible, while odd, aberrant, or doubtful forms are omitted entirely. This volume is not intended for the student of tropical medicine; his needs are amply catered to in other works. Nor is it intended for the parasitologist or the laboratory worker. Accordingly, the material has been selected to omit that which the practitioner does not require. References have been reduced to a minimum and only monographic or similar works have been included; full references can be traced through most of these or through the abstracts of the *Tropical Diseases Bulletin*, a publication with a scope much wider than its title.

Clinical and domestic data are expressed in grains, minims, feet, and degrees Fahrenheit, while laboratory data are given in metric measurements. However, some drugs, usually prepared commercially in metric doses, are so given.

I have to acknowledge the co-operation of numerous colleagues past and present in the preparation of this book.

PREFACE TO SECOND EDITION

IN addition to a revision of the text to bring the material up to date, this edition includes an appendix dealing with parasites and parasitic diseases of exotic origin, likely to be seen in north temperate climates as the result of the war. This appendix has been read by Sir Phillip Manson-Bahr, to whom I wish to express my sincere thanks; however, the responsibility for the statements therein is mine. A number of new illustrations and diagrams have been added to the text, including two sets of microphotographs of helminth eggs, prepared by Professor J. Max Miller, M.D.

I have to acknowledge the co-operation of numerous colleagues, past and present, in the preparation of this book, particularly my colleagues at the Institute of Parasitology.

INTRODUCTION

ALL animals are parasites, predators, or scavengers; they either live on or in other organisms, kill them to secure food, or feed on their leavings. Parasitism is a mode of life and a parasite is an organism with specialized requirements; it cannot alter these and should they be or become unobtainable it will die. Accordingly, parasites do not normally destroy the animal which they parasitize. Rather, they tend to strike a balance with the host as it is called, and the longer this relationship has been established the more perfect is the balance. Parasites do not normally cause disease in their normal host; it is only under abnormal conditions that parasitic disease occurs. This generalization is true only of parasites in the definitive host, i.e., in the host in which they reach maturity. It is not so true for the larval stages of parasites in the intermediate host; in fact it is often rather to the advantage of the parasite to damage this intermediate host, thus rendering it an easier prey to the definitive one. This rule is well exemplified in the Hydatid cyst.

When one examines the evolution of parasites, it becomes obvious that man acquired most of the forms which now habitually parasitize him from the animals with which he surrounded himself and relatively few from prehuman ancestors. This process is still continuing and he still shares many species with lower animals. When this occurs, the animal is referred to as a reservoir host and must be taken into consideration when preventive measures are undertaken.

Parasitism, being a mode of life, is not confined to any one group of animals, and there is scarcely a phylum which does not contain some parasitic members. Most of those forms which attack man, however, belong to one of five main divisions of the animal kingdom—the protozoa, the threadworms, the flatworms, the annelid worms, and the arthropods. They will be discussed in that order.

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ILLUSTRATIONS

The frontispiece was painted by Professor Wynne Edwards, the microphotographs taken by Dr. H. Bruce Collier and Dr. M. J. Miller, and the line drawings made by Miss E. M. Johannsen, Mr. F. Clarke and the author.

PROTOZOA

PROTOZOA are single-celled or cell-less animals more closely akin to the bacteria in their methods of producing disease than are other parasitic animals. Indeed there is some difficulty in saying whether certain organisms are protozoa or protophyta and sometimes both groups are included in a super group, the *Protista*. The protozoan body consists of cytoplasm in which is embedded at least one nucleus. The cytoplasm is usually divided into ectoplasm and endoplasm, both layers possessing a number of organelles. The ectoplasm is the outer layer of the body and it performs the various functions of locomotion, excretion, respiration, feeding, and protection. The method of locomotion varies with the different groups of protozoa. It may consist in the extrusion of temporary processes of ectoplasm after which the body flows; these are called pseudopodia and engulf food particles as well as provide movement. In others, the ectoplasm possesses a few elongated thread-like processes called flagella, or numerous shorter threads which vibrate in unison, called cilia. Flagella may also possess undulating membranes to assist in movement.

The ectoplasm may also form a cyst or spore of varying degrees of thickness and rigidity. In addition, it may have various temporary or permanent openings. Vacuoles containing solid or fluid material for excretion are formed in some species in association with the ectoplasm and discharge through it. Some have a permanent anal pore.

Many protozoa produce secretions which must pass through the ectoplasm, even if produced in the endoplasm. These include haemolysins, cytolysins, and other toxic substances, digestive ferments, and enzymes.

The endoplasm is the internal part of the body and is more granular and fluid; it contains the nucleus and is mainly concerned with nutrition and reproduction. Food material is digested (often in vacuoles), absorbed, or stored in the form of glycogen, volutin, chromatoid bodies, and other substances. It may also contain skeletal structures called axostyles. In most parasitic protozoa, respiration seems to be anaerobic and is performed by the endoplasm splitting up complex compounds into more simple substances.

The nucleus is the most important structure in the endoplasm —a more or less spheroidal mass composed mainly of thymo-

nucleic acid, called chromatin because of its affinity for dyes. It consists essentially of a limiting, achromatic membrane, within which lies a linin network, the reticulum. The interstices of this network are filled with nuclear sap, and the chromatin lies mainly at the nodes but some of it may be concentrated into a more discrete mass called a karyosome. The nucleus may also contain a nucleolus (or nucleoli) consisting mainly of plastin and without chromatin.

In some protozoa nuclear structures occur without the nucleus. In the flagellates, especially in connection with the flagellum, is a kinetoplast; this consists of two parts—a larger, round, parabasal body and a smaller blepharoplast from which the flagellum arises.

(In older literature the kinetoplast is called the kineto-nucleus and the parabasal body the kinetoplast, while in foreign literature other names may be applied. In French the parabasal body is often called the centrosome, while in German it is referred to as the blepharoplast, the true blepharoplast being called the basal granule or end-bead.)

Reproduction is asexual or sexual or both. Asexual reproduction may be binary division (in which the organism splits into two new individuals) or schizogony (in which the organism splits into a number of new individuals).

Sexual reproduction or sporogamy is similar to schizogony but is preceded by the union of two cells—a process called syngamy. This consists in the complete union of special male and female cells (gametes) to form a zygote. A related process consists in the temporary association of two individuals in which nuclear material is exchanged. The latter process is called conjugation and is characteristic of the ciliates.

The protozoa are divided into four major groups:

1. *Sarcodina*, or amoebae, in which movement is by means of temporary extrusions called pseudopodia.
2. *Infusoria*, or ciliates, in which movement is by means of permanent, numerous, short vibratile filaments called cilia.
3. *Mastigophora*, or flagellates, in which movement is by means of permanent, few, relatively long vibratile filaments called flagella.
4. *Sporozoa*, in which there are no organs of locomotion. This is probably not a natural group and the absence of these organs is the result of parasitism.

INTESTINAL PROTOZOA

The Intestinal Protozoa of man belong to all the four great divisions of protozoa—the amoebae, the flagellates, the ciliates, and the sporozoa; none of the latter is of importance and they do not require consideration. Only two species are definitely known to be pathogenic—*Entamoeba* (or *Endamoeba*) *histolytica* and *Balantidium coli*. The first species is somewhat common but relatively seldom pathogenic, whereas the second species, while common in pigs, is rare in man but when it does occur, often pathogenic.

Amoebae

Lewis, in 1870, first described an amoeba from man, although Lösch in 1873, was the first to show the pathogenic nature of *Entamoeba histolytica*, and Koch (1887) the first to note that it could be a tissue parasite. This parasite was first seen in America by Osler (1890) who discovered it in a liver abscess; this led to the important work by Councilman and Lafleur on the pathology of amoebiasis. However, it was not until 1913 that the pathogenic and non-pathogenic species were satisfactorily distinguished.

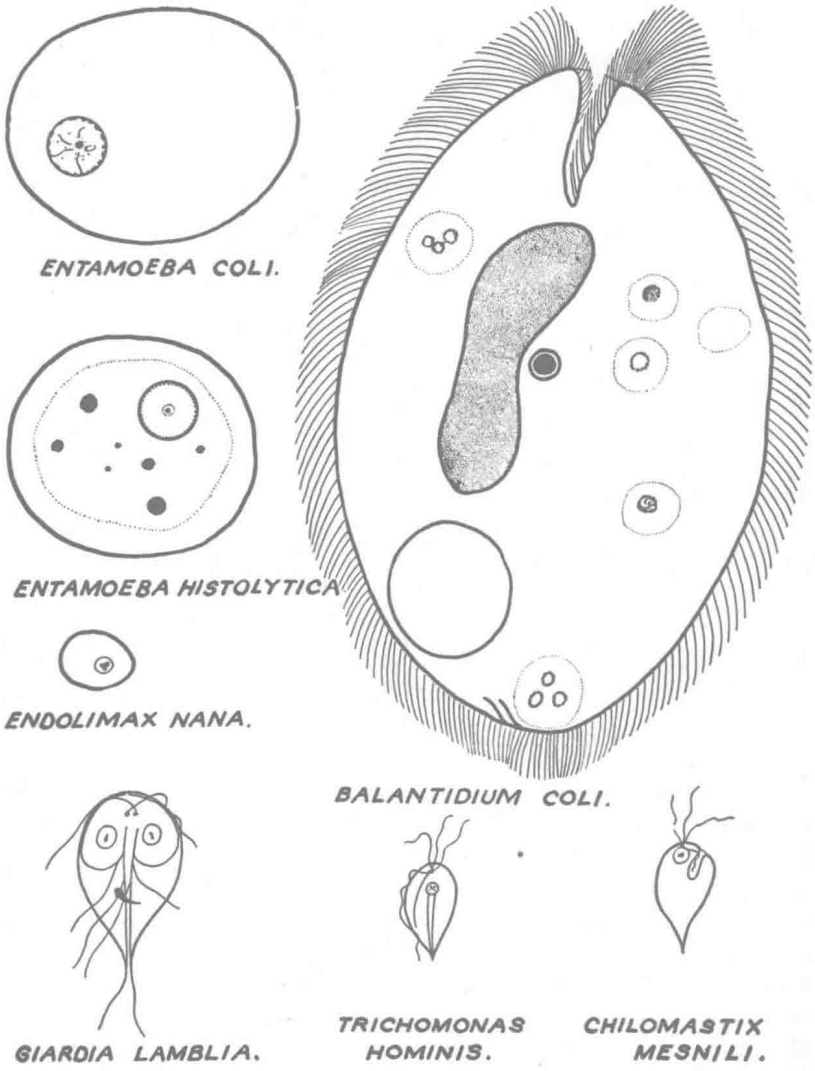
Entamoeba histolytica is the only amoeba known to be pathogenic; even in this case the great majority of infections never show any clinical symptoms and disease is exceptional.

Entamoeba histolytica

E. histolytica lives on the mucosa or in the sub-mucosa of the large intestine and may be seen in the faeces in particles of blood-stained mucus; outside of the body, however, the amoebae (i.e., the trophozoites) quickly die. They vary greatly in shape and in size, measuring when more or less rounded up, from 10μ to 40μ, the average size is about 20μ (18μ–25μ). While the variations in size are often due to growth, size races also appear to exist

The body consists of a finely granular central endoplasm surrounded by a clear transparent ectoplasm, which forms the large, thin, blade-like pseudopodia which enable it to move with some rapidity in the host.

The endoplasm contains the nucleus and food vacuoles. The nucleus is spherical (3–7μ in diameter), the chromatin being distributed in the form of minute dots around the limiting membrane



Outline drawings of the intestinal protozoa: all magnified ×1500.

and in the form of a fine central karyosome. The nucleus can be seen only in stained preparations, although its position can sometimes be detected in living specimens.

The food vacuoles contain red blood-cells in various stages of digestion, as well as tissue cells. Solid food is absent in those amoebae which live in the tissue; amoebae containing red blood-cells are seen only in dysenteric stools. As these amoebae must die, such stools are non-infective, unless they also contain cysts.

Amoebae multiply by simple fission, first the nucleus, then the cytoplasm, dividing into two.

Vegetative amoebae are never infective and are killed by the gastric juice. Under certain conditions, probably those adverse to the vegetative stage, such as gradual dehydration in the large intestine, cysts are formed; these are essential for the continuance of the species and develop into the only infective stages.

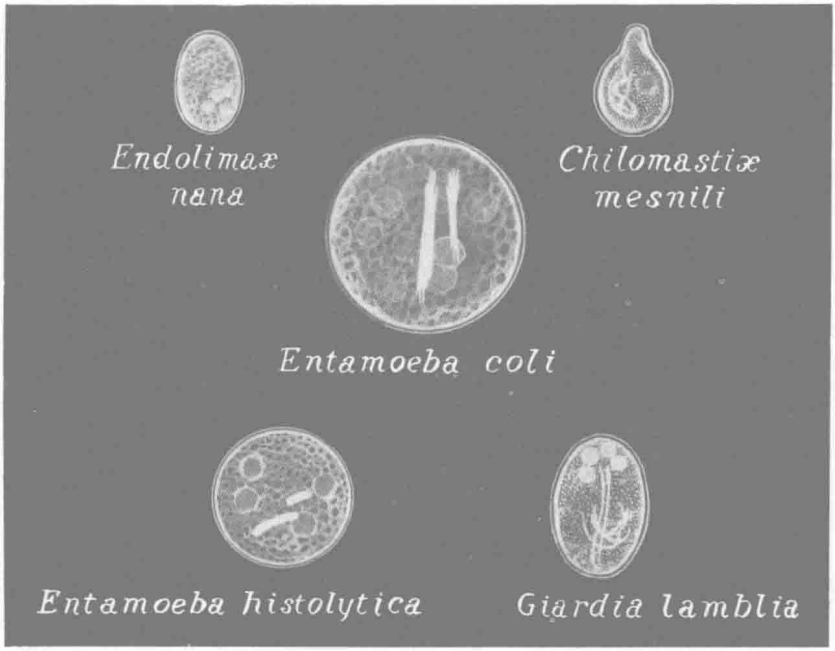
The cysts arise from smaller individuals from which all food vacuoles have been extruded. These then secrete a spherical cyst wall, clear and hyaline in appearance, but very thin. This young cyst which measures 5–20μ (average 10–14μ) has a single nucleus similar to the vegetative form, a diffuse mass of glycogen and one, two, or more chromatoid bodies. These are longish, wide rods with rounded ends (bacilliform), easily visible in unstained specimens and staining deeply with haematoxylin, but not staining at all with iodine. Both the glycogen mass and these rods gradually disappear and are possibly food reserves; their exact function, however, is unknown.

The nucleus divides twice to form four smaller nuclei of the same shape as the vegetative stage, and these nuclei often lie in pairs at the opposite poles of the cyst. The nuclei are poorly seen in living specimens. Stages with less than four nuclei are common.

The cysts appear to live for only a few days at laboratory temperatures, but they will live for several weeks in moist stools or in water at ordinary temperatures. In water free from bacteria, they will live much longer—up to 5-7 months in distilled water. They are quickly killed by drying and by temperatures of 120° F. Chlorine of the strength used to sterilize drinking water has no effect; at least twice the usual concentration is necessary.

The cysts re-enter the host by the mouth—generally on food or drink polluted by contaminated water or by food handlers who

are "carriers," or by insects. They must pass through the gastric juice before the four-nucleated amoeba escapes from the cyst wall (generally in the small intestine). The nuclei divide, and the body splits up into eight small amoebae which grow to full size in the large intestine, and there multiply. Of these, some penetrate the mucosa, while others are passed into the bowel lumen and give rise



The cysts of the common intestinal protozoa of man, unstained, as seen with a dark-ground microscope.

to new cysts. The number of these cysts in the stool, in consequence, varies greatly from day to day.

The amoebae which have invaded the tissue, move to the bottom of the glands of the large intestine and multiply; partly mechanically and partly by lysis, the gland cells separate and degenerate and block up the tubule. If a number close together are affected, a slightly raised, yellowish nodule results. The amoebae continue into the interglandular tissue, and so produce a small abscess of the mucosa. This ruptures and opens to form a small ulcer and discharges some of the amoebae to spread the

infection to adjacent glands; others are passed in blood and mucus in the stool and perish. Those which remain in the ulcer continue to destroy the edges, break through into the submucosa and produce large ulcers. All these ulcers have undercut edges and contain a yellow gelatinous mixture of tissue debris, mucus, and parasites. These "plugs" are often discharged as "sago-grains" in the stool and may contain enormous numbers of amoebae. Secondary bacterial invasion of the ulcers may result, or ultimately they may heal to form scar tissue free from parasites. All these stages may be found simultaneously in the same host. In most cases, however, the host repair processes prevent the formation of large abscesses; dysentery is absent and the carrier condition results. In this case the amoebae are often smaller than in the acute cases.

The ulcers are very characteristic in appearance, looking like "button-holes." They may completely surround the large intestine and are most common where the greatest stasis occurs. The overhanging edges seen in acute cases are not so obvious in chronic cases; in these the edge of the ulcer may be merely thickened.

The characteristic lesions are ulcers with undermined edges, covered with a plug of cytolized necrosed tissue, nodular thickenings at the summits of the folds of mucosa, flask-shaped on section, with gelatinous contents, and sinuses below the mucosa connecting ulcerations and filled with the same material. All these lesions may contain amoebae.

Sometimes the amoebae reach the liver via the portal system and cause a miliary hepatitis or amoebic abscesses. The cystic stages of the amoeba are not formed in the liver. The abscesses may be single or multiple; early ones show as greyish brown areas of necrosed and dissolved tissue; larger ones have a definite wall, often with strands of connective tissue in the abscess. They occasionally occur in other organs.

Although believed to be always a tissue parasite, dysentery or other serious symptoms are relatively uncommon in temperate climates. Clinically six types occur:

(1) Latent or asymptomatic: the commonest form.
(2) Amoebic diarrhoea: the most common clinical form in temperate climates.
(3) Chronic amoebic dysentery, including amoebic appendicitis, typhlitis, granulomata, and analagous symptoms.