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Institution: Oklahoma State University Location: Stillwater, Oklahoma

Title of Study: TWO FAMOUS PESTS, *PLASMODIUM VIVAX* AND *PHYTOPHTHORA INFESTANS*

Pages in Study: 56 Candidate for Degree of Master of Science

Major Field: Natural Science

Scope of Study: Parasites play a large role in shaping the destiny of mankind. One such parasite, *Plasmodium vivax*, has been and still is responsible for the deaths of thousands of people. This thesis points out the highlights of that life cycle, history of the disease, and gives a brief description of the phylum from which it comes. Another very important parasite, *Phytophthora infestans* is discussed with special emphasis on its destructiveness in Ireland in 1845 - 1846. Briefly the fungi are discussed to give a better understanding of this one particular Phycomycete.

Findings and Conclusions: Historians do not give enough credit to the effect that the destructive nature of certain parasites such as *Plasmodium vivax* and *Phytophthora infestans* have had on history. Neither do Americans, in general, have a healthy enough respect for the ability of parasites such as these to wreck our whole economy even though they may never reach our soil in epidemic proportions. They have brought death, disability, and deprivation to many of the underdeveloped countries, and in this way stand to weaken our economy due to our dependence on products from these countries.

ADVISER'S APPROVAL James H. Zant

TWO FAMOUS PESTS, *PLASMODIUM VIVAX* AND *PHYTOPHTHORA INFESTANS*

by

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Bachelor of Arts

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Abilene, Texas

1961

Submitted to the faculty of the Graduate School of
the Oklahoma State University in partial
fulfillment of the requirements
for the degree of
MASTER OF SCIENCE
May, 1962

TWO FAMOUS PESTS, PLASMODIUM VIVAX

AND PHYTOPHTHORA INFESTANS

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ACKNOWLEDGEMENT

Grateful indebtedness is acknowledged to the National Science Foundation for making this year of further academic work possible and to Dr. James H. Zant and Dr. L. Herbert Bruneau for their valuable guidance.

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CHAPTER ONE

INTRODUCTION

Man considers himself above the everyday struggle for survival that faces all other living creatures because of his superior intelligence. He defies the animals that might prey on him as well as the attacks of adverse climate and elements. He still faces one phase of this struggle, however, and that is in his fight against diseases caused by parasites.

His inventive genius won the fight over cholera, plague, smallpox, and yellow fever. Parasitic enemies depending on the slow process of evolution are no match for man's "scientific know-how." One disquieting aspect of this matter, however, is that most progress has depended on drugs, vaccines, and insecticides. These have been quite adequate to protect Americans in their better environment and social conditions. They have had much less success with millions of people living under less favored conditions. Therefore, in spite of some glowing victories as a whole the human race has a long way to go to free itself from parasitic disease.

Parasites seem to this author to be a fascinating subject, partly because of the intricacy of their life cycles and partly because of the immense role that some of them have played in shaping the destiny of mankind. For this reason, two of the most famous parasites, Plasmodium vivax(the malarial parasite) and Phytophthora infestans(the potatoe late blight parasite), have been chosen as the topic of this report.

An attempt has been made to increase the understanding of each parasite by discussing briefly the phylum of animals or plants in which it is placed. This discussion is then followed by details of the parasite's life cycle and importance to man.

CHAPTER TWO

PHYLUM PROTOZOA AT A GLANCE

Fully 30,000 kinds of Protozoa are known, and in numbers of individuals they far exceed all other animals.[1] The amazing Protozoans are morphologically a single cell which manifests all characteristics common to living things.[2] In the Animal Kingdom they comprise the lowest of all great groups, or phyla, in contrast to the multicellular tissue animals, or Metazoa. Many people maintain that Protozoans are non-cellular, since they are one-celled and a complete organism. They differ from the cells of a metazoan, each of which is dependent upon other cells and cannot live independently.[3] While some Protozoans are simple in structure; others have cell organs, or organelles, that are functionally analogous to the organ systems of multicellular animals.

The Protozoa are divided, according to the structures

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[1]Tracy Storer and Robert Usinger, Elements of Zoology (New York: McGraw-Hill Book Company, Inc., 1955), p. 267.

[2]L. A. Borradaile and F. A. Potts, The Invertebrata (Cambridge: The University Press, 1959), p. 10.

[3]Richard Kudo, Protozoology(Springfield, Illinois: Charles C. Thomas, Publisher, 1939), p. 3.

they possess for locomotion into five classes:

1. Mastigophora, or flagellates, with one or more whip-like flagella
2. Sarcodina, or rhizopods, with pseudopodia
3. Sporozoa, with no locomotor organelles
4. Ciliata, or ciliates, with cilia throughout life
5. Suctoria, with cilia in the young and tentacles in the adult.

MASTIGOPHORA

Morphology

This group is characterized by the presence of one or more whiplike locomotor organelles called flagella. The flagella are also used to capture food and may serve as sense receptors.[4] The cell body is usually of definite form, oval, long, or spherical, covered by a firm pellicle, and armored in some groups. The *Euglena* and certain other flagellates are of particular interest since they combine the characteristic of both plants and animals and are frequently claimed by botanists. Many species contain plastids, and those with chlorophyll can synthesize food by the aid of sunlight.[5]

The *Euglena viridis* is a common, solitary, free-living flagellate that contains chlorophyll. They are

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[4]Robert W. Hegner, *College Zoology* (New York: The Macmillan Co., 1955), p. 54.

[5]Storer and Usinger, op. cit., p. 274.

usually present in collections of pond weeds. The rather spindle-shaped body is .1 mm or less in length, blunt at the anterior end and pointed at the posterior end. Covering the peripheral layer of cytoplasm is a membrane or pellicle. A funnel-shaped depression called the cytostome is near the anterior end. This serves as the cell mouth and leads into the cell gullet or cytopharynx. Near the anterior end of the body is an orange red eye spot that is especially sensitive to light.[6] Toward the center of the Euglena is an oval nucleus containing the endosome, the function of which is not known.[7] The green color of the Euglena is due, of course, to a number of chloroplasts in the cytoplasm.

Locomotion

The flagellum beats back and forth to draw the Euglena through the water with a spiral rotation that enables the animal to follow a straight course. Euglena may also crawl by spiral movements of the cell body, and at times it performs worm-like "euglenoid movements" by local expansions and contractions that suggest the peristalsis in a vertebrate's intestine.[8]

Reproduction

In active cultures, Euglena reproduces frequently

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[6] Ibid., p. 275.

[7] Robert W. Hegner, Invertebrate Zoology (New York: The Macmillan Co., 1933), p.55.

[8] Storer and Usinger, op. cit., p. 276.

by longitudinal binary fission. The nucleus divides into two by mitosis, then the anterior organelles- flagellum, blepharoplast, cytopharynx, reservoir, and stigma- are duplicated, and the organism splits in two lengthwise.[9]

Nutrition

Photosynthesis largely provides for the nutriment of Euglena. They may also absorb food substances through the general body surfaces. Some of the group ingest small organisms by means of their cytopharynx.[10]

SARCODINA

Morphology

The Amoeba is a mass of clear, colorless, and jelly-like protoplasm. Two regions are distinguishable in the body -- an outer colorless layer of clear cytoplasm called ectoplasm, and an endoplasm which is a comparatively large central mass of granular cytoplasm. A clear round body usually lies near the end of the animal away from the direction of motion disappearing at intervals. This is the contractile vacuole. The cell membrane limits the protoplasm and permits the passage of water, oxygen, and carbon dioxide.[11] A nucleus controls the vital processes

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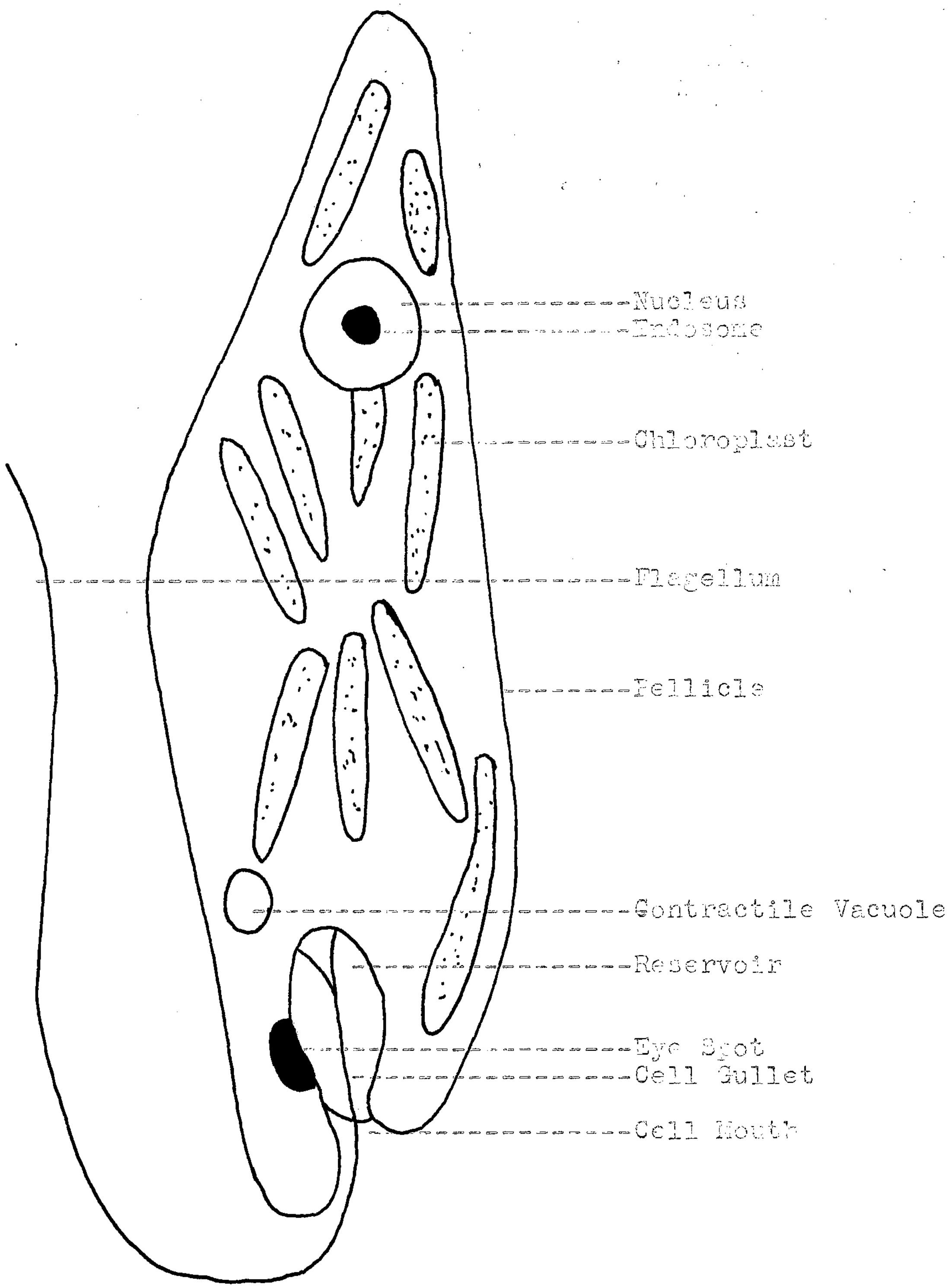
[9] Borradaile and Potts, op. cit., p. 33.

[10] Hegner, Invertebrate Zoology, op. cit., p. 33.

[11] Storer and Usinger, op. cit., p. 269.



<u>EUGLENA VIRIDIS</u>

of the organism while the contractile vacuole regulates water content. Food vacuoles are also contained in the cytoplasm, and they contain food in various stages of digestion.

Locomotion

Amoebae move from place to place by forming finger-like protrusions known as pseudopodia. The ectoplasm is pushed out and enlarged until a blunt projection is produced; the endoplasm then flows into it. The result is a movement of the entire animal in the direction of the pseudopodium. If more than one are formed at the same time, one usually survives while the others flow back and gradually disappear.[12]

Nutrition

The food of the Amoeba is usually small aquatic plants, diatoms, protozoa, bacteria, and other animal and vegetable matter. A certain amount of choice of food is exercised, or the Amoeba's body would become overloaded with particles of sand and other indigestible material. This apparent choice of food may be due to ordinary physical laws of fluids.[13]

Respiration

The contractile vacuole is also envolved in respiration, since carbon dioxide probably makes its way to the exterior by way of this organ. Oxygen dissolved in

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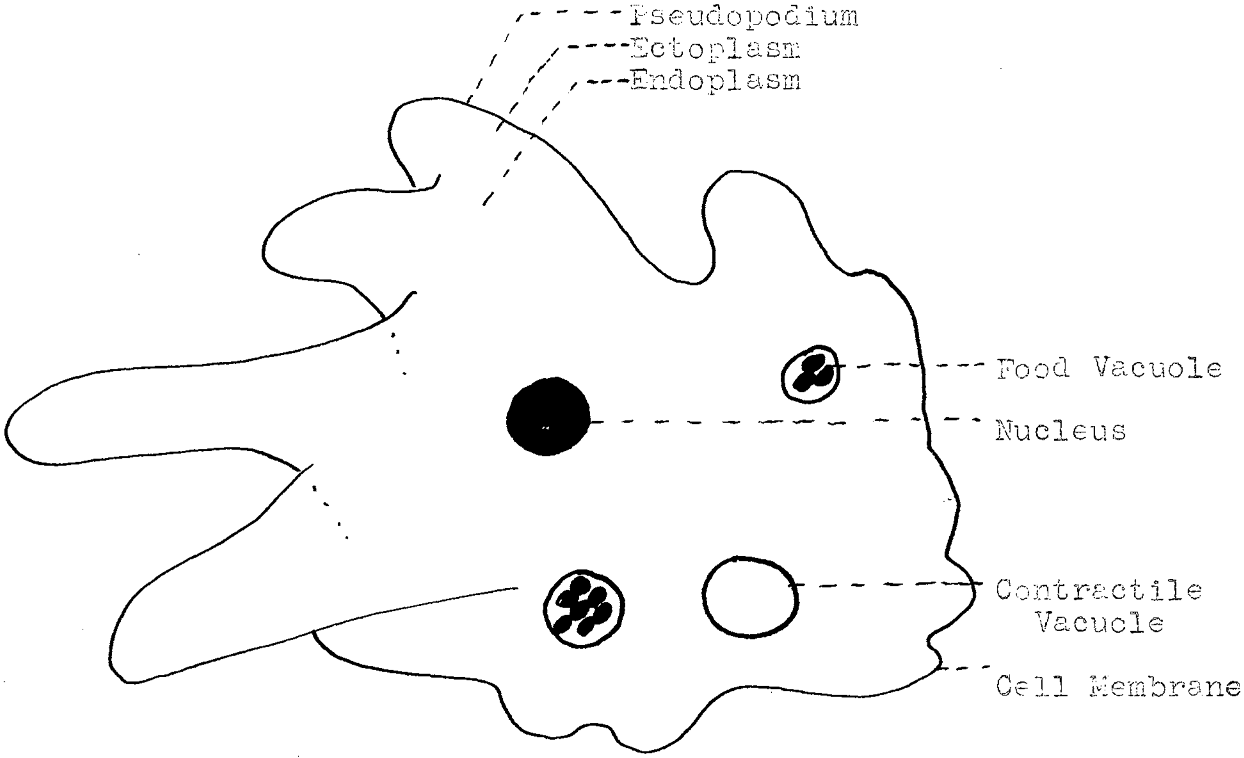
[12]Hegner, Invertebrate Zoology, op. cit., p. 6.

[13]Ibid., p. 11.



AMOEBA FORMING A FOOD VACUOLE

Amoeba encounters food and carbon particle	$2\frac{1}{2}$ minutes later a food cup is formed	$7\frac{1}{2}$ minutes later	8 minutes later

AMOEBA PROTEUS

water is taken in through the surface of the body. This gas is necessary for the life of the animal.[14]

Reproduction

There is a limit with regard to the size that may be attained by Amoeba proteus, as it rarely exceeds x25 mm in diameter. When this limit is reached, the animal divides into two parts. It is supposed that this division is started by some unkown change in the relations between the nucleus and the cytoplasm.[15]

SPOROZOA

Little will be said about the class Sporozoa at this point since Plasmodium vivax, a member of this class will be studied in detail. Suffice it to say that their body is rounded or elongate with one nucleus and no locomotor organelles or contractile vacuole. Food is absorbed directly from the host, and as in the Amoeba respiration and excretion are by diffusion.

Reproduction is carried on rapidly by a multiple asexual fission or schizogany in which the cell becomes multinucleate and then the cytoplasm divides.[16] Members of this class also produce sexual macro- and microgametes which join in pairs of opposite kind to form zygotes.

Sporozoa are possibly the most widely occuring of

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[14]Storer and Usinger, op. cit., p. 271

[15]Borradaile and Potts, op. cit., p. 33.

[16]Hegner, College Zoology, op. cit., p. 88.

animal parasites. Distinct species occur in various animals from protozoans to mammals. Some live within the host's cells and others in its body fluids or cavities. They inhabit variously the digestive tract, muscles, blood, kidneys, or other organs.[17]

CILIATA

Morphology

The Paramecium resembles a slipper or cigar in shape. A depression extends from the end directed forward in swimming, backward and toward the right, ending just posterior to the middle of the animal. This is the oral groove. The cytostome is near the end of the oral groove and opens into a funnel-shaped depression called the cytopharynx or gullet. Paramecium swims with the slender but blunt end foremost. The opposite end, which is thicker but more pointed, represents the posterior end. The motile organs are fine thread-like cilia regularaly arranged over the surface. Two layers of cytoplasm are visible, as in Amoeba, an outer comparatively thin clear area, the ectoplasm, and a central granular mass, the endoplasm.[18] Limiting the cytoplasm is the pellicle. One large contractile vacuole is situated near either end of the body. The nuclei are two in number, a large macronucleus and a smaller micronucleus.[19]

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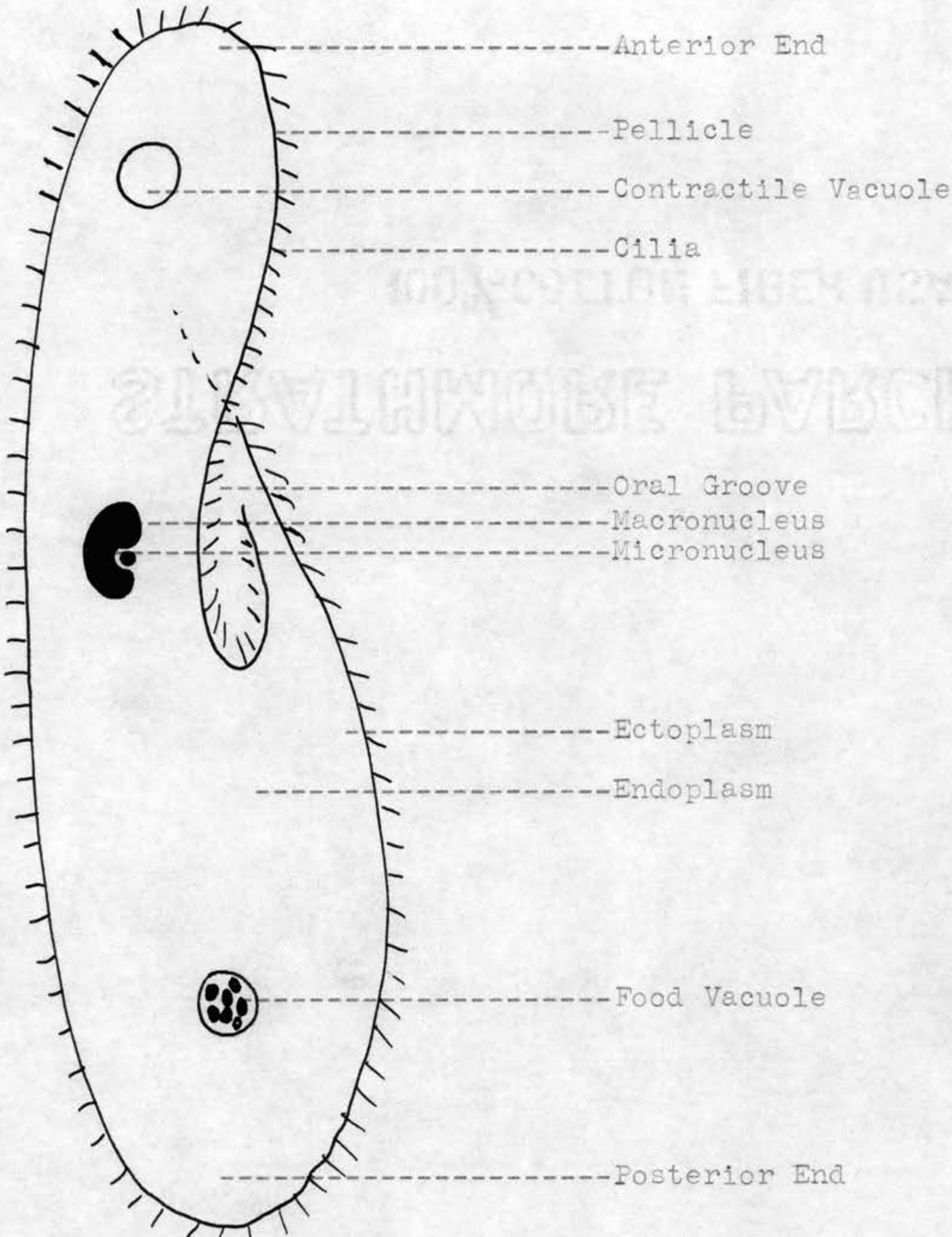
[17] Storer and Usinger, op. cit., p. 276.

[18] Hegner, Invertebrate Zoology, p. 66.

[19] Storer and Usinger, op. cit., p. 278



PARAMECIUM CAUDATUM

Locomotion

In a free field <u>Paramecium</u> swims by means of its cilia. These are usually directed backward, and their stroke then drives the animal forward. It can swim backward and will do so quickly if it meets some unfavorable chemical stimulus which is the well-known avoidance reaction.[20] In addition to its forward or backward movement <u>Paramecium</u> rotates on its long axis. This rotation is over to the left, both when the animal is swimming forward and when it is swimming backward. The body takes a spiral course because the cilia in the oral groove beat more effectively than those elsewhere. Rotation is thus effective in enabling an unsymmetrical animal to swim in a straight course through a medium which allows deviations to right or left, and up or down. It is well known that a human being cannot keep a straight course when lost in the woods, although he has a chance to err only to the right or left.[21]

Nutrition

An excellent four-course dinner for a <u>Paramecium</u> would be bacteria, small protozoans, algae, and yeasts. Cilia line the oral groove and beat the food toward the cytostome. A vacuole is formed at the end of the cytopharynx and floats through the cytoplasm until the nourish-

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[20]Borradaile and Potts, op. cit., p. 45.

[21]Hegner, <u>Invertebrate Zoology</u>, op. cit., p. 73.

ment is removed.[22]

Respiration

Respiration and excretion are carried on as usual by diffusion. Liquid waste is picked up in the radiating canals and discharged into the contractile vacuole.

Reproduction

Paramecium reproduces only by simple binary division. This process is interrupted occasionally by a temporary conjugation of two individuals. In binary fission the animal divides transversely. The first indication of a forth-coming division is seen in the micronucleus, which undergoes a sort of mitosis, its substance being equally divided between the two daughter nuclei. These separate and finally come to lie one near either end of the body. The macronucleus elongates and then divides transversely. While the organelles are duplicating themselves, a constriction appears near the middle of the longitudinal diameter of the body. This becomes deeper and deeper until only a slender thread of protoplasm holds the two halves of the body together. This connection is finally severed and the two daughter *Paramecia* are freed from each other.[23]

It should be mentioned that this group may be hard to define since many of the Sarcodina are flagellated

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[22]Storer and Usinger, op. cit., p. 283.

[23]Hegner, *Invertebrate Zoology*, op. cit., p. 76.

during some stages of their life cycles. If they are undoubtedly amoeboid during the greater part of their lives, they should be placed in the Sarcodina.[24]

SUCTORIA

Morphology

Their relation to Ciliata is shown by the production of free swimming ciliated larvae and the nuclear apparatus which consists of a micro- and a macronucleus.[25] Cilia are present only on young individuals and lost with the development of a stalk or attaching disc and tentacles. Therefore, an adult Suctorian is incapable of active movement. The body may be spheroidal, elliptical or dendritic. It is covered with a pellicle and occassionally possesses a lorica.[26] Some say the body is vase-like, with or without a stalk or peduncle. In sessile forms the body is attached with a broad base to the substratum. In stalked forms the body is raised up from the point of attachment on a straight, non-contractile stalk of secreted substance.[27] Some species attach themselves indifferently to a living or a lifeless object. Others are always attached to some particular animal, frequently to a particular organ of it. Very few species, however, are truly parasitic in the adult condition. Many of the larval stages are parasitic in the bodies

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[24]Robert Hegner, Human Protozology(New York: The Macmillan Co., 1925), p. 91.

[25]Hegner, Invertebrate Zoology, op. cit., p.97.

[26]Kudo, op. cit., p. 628.

[27]Ibid., p. 629.

of Ciliates.[28]

Nutrition

Suctorians have no cytostome and the food capturing is carried on exclusively by the tentacles. Tentacles are of two kinds: one is suctorial in function and bears a rounded knob on the extremity and the other is for piercing through the body of a prey and more or less sharply pointed. Tentacles may be confined to limited areas or may cover the entire body. Food organisms are usually small ciliates and nutrition is holozoic.[29]

Reproduction

Asexual reproduction is by binary fission or budding. The young swim around actively after leaving the parent eventually becoming attached to a suitable object. Sexual reproduction is by fusion.[30]

To say that the Phylum Protozoa is unique is not adequate. Their real complexity reaches its height, however, in the nature of some members of the Class Sporozoa.

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[28] E.A. Minchin, An Introduction to the Study of the Protozoa(London: Edward Arnold, 1922), p. 455.

[29] Kudo, op. cit., p. 628.

[30] Ibid., p. 629.

CHAPTER THREE

PUBLIC ENEMY NUMBER ONE

Malaria! - still alarms vast areas of our world. It is by far the most important of the tropical diseases because of its widespread distribution and high morbidity.[1] Inhabitants of highly humid, tropical areas carefully guard themselves and their children against possible infection through a mosquito bite of a tiny protozoan whose body processes are so uncomplicated that constant reproduction allows what was once a few individuals to become teeming thousands within the bictim's blood system. Safe in America's cooler climate and better living conditions we think that this disease as well as many others has been conquered. We are rather surprised to hear missionaries, members of our foreign services, travelers, and employees of various companies stationed in foreign lands speak of the hundreds of cases of malaria they see and how many cases die despite our drugs and modern medical knowledge. Just ten years ago more people were dying of malaria than all other diseases combined as will later be pointed out.

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[1] J. E. Ash and Sophie Spitz, Pathology of Tropical Diseases(Philidelphia: W. B. Saunders Co., 1945), p. 206.

History

How long has the world been under the scourge of malaria? Malaria's written history goes back to the Father of Medical Science, Hippocrates. In the 5th Century BC Hippocrates described several types of fever which must have been of malarial origin. It increased thereafter.[2] The decline of the civilization of ancient Greece between 500 and 300 BC and of Rome, at a later date, has been ascribed in large measure to malaria. Empedocles in 550 BC controlled an epidemic of malaria in Sicily by draining marshes and changing the courses of two rivers.[3]

Did this action indicate a basic understanding of the disease? No. Varro as late as the 2nd Century BC urged people not to build villas near marshes, "because small, living beings swarm there and are the cause of obstinate disease."[4] Malaria means "bad air" and was so named because of this association of the disease with the odorous air of swamps.

Julius Caesar himself is said to have suffered from intermittent fever (malaria) and the health of Caesar's

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[2]George Shattuck, Diseases of the Tropics(New York: Appleton-Century-Crofts, Inc., 1951), p. 1.

[3]Ibid., p. 2.

[4]Asa Chandler, Introduction to Parasitology(New York: John Wiley & Sons, Inc., 1955), p. 185.

army was shattered by this disease during the Civil wars of Rome.[5]

Not much is known about malaria in Europe during the 16th and 17th Centuries. Epidemic malaria is known to have been widespread in 1667 and 1558. Early in the 19th Century epidemics were reported in India.[6]

Laveran of Paris discovered the malarial parasite in 1880 only after the work of Leewvenhoek, Virchow, and Ehrlich. In 1898 Ross proved that the mosquito transmitted the disease. Some still didn't accept Laveran's discovery as they believed a bacterium might be causing the disease.[7]

There are some scientists who believe malaria did not exist in the Americas before the coming of Europeans.[8] Spanish conquerors no doubt brought the parasite with them as an unwelcome fellow traveler in their blood.

Importance to Our Generation

How many lives malaria is taking today, no one knows or can estimate accurately. In the 40's, however, there were certainly not less than three million malaria deaths - not cases - but deaths in the world, and at least 300 million cases of the malarial fever each year.[9]

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[5] Ibid., p. 2.

[6] Ibid., p. 3.

[7] Philip Manson-Bohr, Manson's Tropical Diseases (Baltimore: Williams and Wilkins Co., 1950), p.40.

[8] Shattuck, op. cit., p. 3.

[9] Ibid., p. 4.

India is one of malaria's strongholds and kills at least a million persons every normal year and more during epidemics. One author put it this way, "There is no aspect of life in that country which is not affected, either directly or indirectly, by this disease."[10]

> "It constitutes one of the most important causes of economic misfortune, engendering poverty, diminishing the quantity and quality of the food supply, lowering the physical and intellectual standard of the nation, and hampering increased prosperity and economic progress in every way," says another.[11]

Chandler states that at the publishing of his book, 1955, Malaria still ranked as the number one human disease from the point of prevalence and the mortality, sickness, and economic loss it produces. He points out, however, that battles were being won at that time and that some countries had actually rid themselves of the disease. Reduction has been complete enough in some, such as Ceylon, as to raise pessimistic warnings about overpopulation, unemployment, and Communism.[12]

During World War II malaria was the number one problem of nonimmune troops in the Mediterranean, India-Burna, and the South Pacific. Because it was impossible to avoid mosquito bites under campaign conditions, it was necessary to resort to suppressive medication.[13] This, of

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[10]Ibid., p. 5.

[11]Chandler, op. cit., p. 186.

[12]Ibid., p. 187.

[13]Ash and Spitz, op. cit., p. 206.

course, does not prevent infection. It simply kept the infection subclinical so that the soldiers could delay their illness until a more strategic time. At the fall of Battan, when quinine ran out, 85% of every regiment had acute malaria. In the South Pacific campaigns, malaria caused more than five times as many casualties as did combat. Such ignorant attitudes as displayed by one high ranking officer could well be a lesson for all, "We are out here to fight Japs, and to hell with mosquitoes."[14] It took efforts of entomologists, engineers, physicians, and occasional court martials to bring the malaria rates down.

Its presence is still made known occasionally in this country as the author's father had malaria as a child in Southern Kansas. Admittedly, this parasite has not had the devastating effect in the United States, however, that it has had in other parts of the world. Chandler has this to say,

> "Although historians and economists have largely failed to recognize it, malaria must have played a large part in the history of the world and the progress of nations."[15]

Distribution

Malaria is distributed in the area lying between 63° N and 35° S wherever there is a sufficiency of mosquitoes hospitable to the protozoan, together with con-

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[14] Chandler, op. cit., p. 188.

[15] Ibid., p. 185.

dition of temperature and humidity which permit the complete development of the parasite in the infected mosquito.[16] Plasmodium vivax is more widely distributed than the other types. It is the prevalent infection in most areas within the temperate zones, but is widespread throughout the tropics as well.[17]

Etiology

The parasites causing malaria are protozoa belonging to the genus Plasmodium. It has been demonstrated beyond a doubt that the human malarial parasite will not develop in any but mosquitoes of the Anopheles genus.[18]

Life Cycle

Four species of the genus Plasmodium can cause malaria in man. Plasmodium vivax is the most widely spread and Plasmodium falciparum results in death most often. Plasmodium vivax has a 48 hour cycle and is particularly likely to cause relapses. This cycle as described in Chandler's book is as follows:[19]

I. Asexual Phase

A. Exo-erythrocytic Stage - This is the stage in which the Plasmodium vivax enters the

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[16] C.M. Wenyon, ed., Memoranda on Medical Disease (Chemical Publishing Co.,Inc., 1942), p. 134.

[17] Thomas Mackie, George Hunter and C. Brooke Worth, A Manual of Tropical Medicine(Philadelphia: W. B. Saunders Co., 1945), p. 215.

[18] C.M. Wenyon, Protozology(New York: William Wood & Co., 1926), p. 962.

[19] Chandler, op. cit., pp. 188-197.

human body as a result of a mosquito bite is called the sporozoite, a slender, spindle shaped, one-celled individual. The sporozoites make their way through the blood stream to the spleen and/or liver. Usually they enter these cells and go through at least two schizogony cycles - cycles in which they are asexually multiplying. This stage may continue indefinitely. The parasite then invades the blood stream and enters erythrocytes (red blood cells).

B. Erythrocytic Stage - A week or ten days after infection, the parasite invades the red blood cells and begins a growing process. The earliest form of the parasite seen in the corpuscles is the ring stage. It looks like a signet or class ring caused by a transparent area in the center of the parasite. As the parasite grows large it becomes rounded or irregular in shape. The nucleus divided into two, four, and eventually more parts. The nucleus now moves away from the center of the body, and small amounts of the cytoplasm concentrate around each. These segment allowing the individual to reproduce again asexually and each segment -- now a new individual -- is called a merozoite. They break free from the corpuscle and each merozoite is fully capable of attacking new corpuscles repeating the process. This cycle is in Plasmodium vivax and takes 48 hours, and as many of these corpuscles erupt releasing the merozoites, the individual becomes fevered. Although this liberation does not take place at all hours, certain broods mature at certain hours and thus spells of high fever are a few hours apart.

C. Gametes - After a few generations of these merozoites have been produced, some grow more slowly, producing more pigment, and developing into larger single-nucleated organisms. These are the gametocytes which may circulate in the blood for several weeks but will undergo no further development.

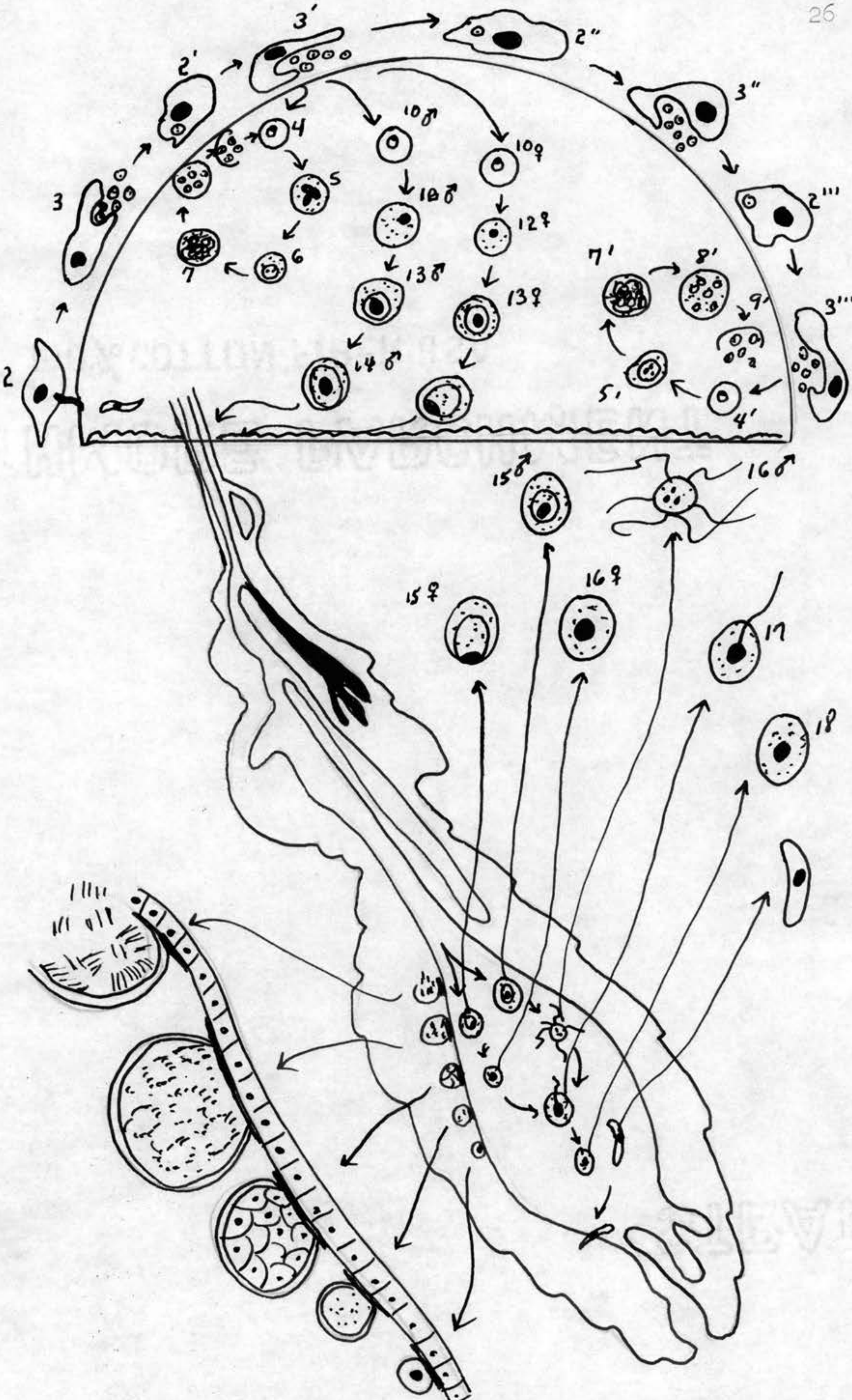
II. Sexual Phase

A. Gametes - When removed from the warm blood by being sucked up by a mosquito, or even placed on a microscope slide, the microgametocytes (male gametes), undergo a striking development. The nucleus quickly divides and within 10-15 minutes 6-8 long flagella-like structures are extruded and the parasite is known as the flagellated body. This process actually forms the male microgamete (sperm). These slender structures are free to swim about in search of a macrogamete which meanwhile has undergone little change.

B. Zygote - The result of the union of the filament from the flagellated body with the inactive female gamete is a zygote. This individual, the beginning of a new generation, grows, elongates, and becomes quite like a little worm. It penetrates the stomach wall of the mosquito, lodging itself under the outer limiting membrane. Rapid growth takes place here and a cyst wall develops called an oocyst.

C. Oocyst - The nuclei divide repeatedly and a number of fairly outlined cells are formed. These cells enlarge and eventually sporozoites, each about 15 microns in length, break loose. Such an oocyst may contain more than 10,000 sporozoites, and there may be as many as 50 oocysts on one mosquito's stomach. These sporozoites are released into the mosquitoes body cavity from which they make their way to the 3-lobed salivary gland lying in the front part of the thorax and connecting with the proboscis. The sporozoites are discharged with the saliva when the mosquito bites.

As this cycle is very difficult to understand, the following diagram may help. It is copied from Chandler's, Introduction to Parasitology.



Life Cycle of the

Malarial Parasite

KEY TO ILLUSTRATION

1. Sporozoites injected
2. Sporozoites entering liver cell
3. Exo-erythrocytic schizogony

4-9. Erythrocytic schizogony, repeated indefinitely

10-14. Development of the male and female gametes

15. Male and female gametocytes in stomach of *Anopheles*
16. Formation of male gametes by exflagellation
17. Fertilization
18. Zygote
19. Ookinete

21-25. Development of oocyst

Immunity

It has been generally accepted that malaria confers a certain degree of immunity on its host after the acute attack has subsided.[20] The mechanism accociated with or responsible for this conversion of an acute attack into a chronic form is of considerable controversy.

Specific protective antibodies have been demonstrated in the serum of monkeys during the stages of chronic infection. The concentration of these protective antibodies varies during the course of the disease.[21] These findings probably represent the first direct experimental evidence of extracellular immunity in malaria.

The colored race has a relative racial immunity to malaria which is particularly effective against Plasmodium vivax. The development of immunity is first characterized by the acquisition of tolerance to the infection.[22]

Epidemiology

Conditions which favour the presence and breeding of Anopheles mosquitoes tend to also favour the increase of malaria.[23]

Factors which determine the level of transmission of malaria in any region are as follows:

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[20] Z. Taylor Bercovitz, ed., Clinical Tropical Medicine (New York: Paul B. Haeber, Inc., 1944), p. 157.

[21] Ibid., p. 158.

[22] Mackie, Hunter, and Worth, op. cit., p. 229.

[23] Manson-Bahr, op. cit., p. 41.

1. The degree of infection in man
2. The species of the *Anopheles* mosquitoes, their abundance, feeding and resting behaviors, and their susceptibility to *Plasmodium* - the vector
3. The presence of a susceptible human population
4. Local climatic conditions.
5. Local geographic conditions which may be potential *Anopheles* breeding areas[24]

Clinical Characteristics

There may or may not be a stage wherein the patient feels upset, is tired, has an ache in his bones, perhaps a headache, loses his apetite, possibly vomits, and suffers from chilly sensations. At this period his temperature may already have begun to rise. In many cases, however, he finds himself suddenly in the grip of ague, suffering from a definite rigor and such an intense feeling of cold that his teeth chatter and he shivers and shakes. Very often the patient begins to vomit violently. His temperature elevates, the sensation of cold being entirely subjective. Then comes the stage of heat and febrile distress, with flushed face, rapid pulse, intense headache, frequent vomiting, quick breathing and dry burning skin, during which the temperature often runs up to 105°F.[25]

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[24]Mackie, Hunter, and Worth, op. cit., p. 230.

[25]Wenyon, *Memoranda on Medical Disease*, op. cit.,p.138.

There may be a slight delirium. Then comes the sweaty stages, the perspiration pouring from the patient and soaking everything on and about him. The fever rapidly declines and comfort takes the place of acute misery. Then, according to the nature of the infection, one or two days later the fever recurs. It lasts as a rule from 6-10 hours, one hour for the cold stage, 3-4 hours for the defervescence, and 2-4 hours for the hot period.[26] The most striking macroscopic change in the body, due to the presence of malarial parasites, is a marked enlargement of the spleen , which in some cases reaches enormous dimensions. The liver is also enlarged, but to a lesser extent.[27]

Diagnosis

Diagnosis of malaria is frequently difficult. It may be confused with many diseases, both cosmopolitan and tropical. Among the tropical diseases it may be confused with are kalaazar, amoebic liver abscess, relapsing fever and yellow fever. Among the cosmopolitan diseases it may frequently simulate are typhoid fever, tuberculosis, brucellosis, influenza, and pyelitis.[28]

Definite diagnosis depends upon demonstration of the parasites. For this purpose the thick blood film is far

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[26]Ibid., p. 139.

[27]Wenyon, Protozology, op. cit., p. 955.

[28]Mackie, Hunter, Worth, op. cit., p. 245.

superior to the thin film technic since in light infection it may be impossible to find Plasmodia in the thin film. It may be necessary to examine stained thin blood films for positive identification of the particular species present.[29]

Treatment

Three drugs have been used in the treatment of malaria - quinine, atabrine and plasmochin.

Quinine is a general protoplasmic poison and in sufficient concentration is lethal to all cells. It has little effect on the circulatory system in therapeutic doses. In excessive dosage it produces an initial rise in pulse rate and blood pressure followed by a depression of both. When given intravenously in too large a dosage or too quickly, rapid progressive fall of blood pressure occurs with the appearance of circulatory collapse due to cardiac depression and vasodilation.[30]

Atabrine is a yellow dye. In certain individuals atabrine taken by mouth acts as a gastro-intestinal irritant producing epigastric pain, nausea, vomiting and diarrhea.[31] These side effects can be controlled to a large extent by giving the drug with food or with heavily sweetened fluids.

Plasmochin is a toxic drug with little margin of

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[29] Ibid., p. 245.

[30] Ibid., p. 246.

[31] Ibid., p. 247.

safety between the therapeutic and the toxic doses. It should never be given to ambulatory patients.[32]

[32]Ibid., p. 247.

CHAPTER FOUR

OVERLOOKED AND UNDERSTUDIED EUMYCOPHYTA

Just what is a fungus? One author states that fungi are...

> "parasitic or saprophytic Thallophyta entirely destitute of chlorophyll, and possessing in the very large majority of cases a vegetative portion, the mycelium, made up of filaments or haphae."[1]

One could easily read this definition and still not know what a fungus is. To paraphrase Bessey's definition, a fungus is a plant without chlorophyll typically consisting of filaments that may or may not be divided into cells. Fungi can also include encysted or amoeboid one-celled organisms which reproduce by some type of motile or non-motile spore.[2]

Now, both of these definitions speak out definitely as to the plant nature of the fungus. However, many people question even this seemingly obvious characteristic of the organism. One author consistently concludes that the fungi are not plants but a third kingdom of organisms

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[1]Helen Gynne-Vaughan, Fungi(Cambridge: The University Press, 1922), p. 1.

[2]Ernst Bessey, A Text Book of Mycology(Philadelphia: P . Blakiston's Son and Co., Inc., 1935), p. 2.

parallel to the Animal and Plant Kingdoms.[3] Still another comes out and boldly states that fungi are unmistakably plants of a low organization developed from germs somewhat but not wholly homologous to seeds of higher orders.[4] Nevertheless, knowing the exact relation of the fungi to other main groups of organisms is not necessary to become acquainted with them and their characteristics as a group.

To bring this group closer to home, one can easily recognize certain fungi if they realize that the mold that grows on jam, the mildew that disfigures the rosebud, the yeast that ferments the wine, the rust that reddens the wheat fields, the ringworm that blemishes the skin, the mushroom in the cellophane package, and the toadstool in the front yard are the fungi.[5]

These examples of fungi seem very different in shape. What generalities can be made as to their structure?

Morphology

As has already been pointed out, the fungi constitute a group of organisms devoid of chlorophyll. They resemble green plants in that they have definite cell walls; they are usually non-motile; and they are usually filamentous and multicellular.[6] Their nuclei can be demonstrated

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[3]Ibid., p. 1.

[4]M. C. Cooke, Fungi: Their Nature and Uses (New York: D. Appleton and Co., 1891), p. 5.

[5]John Alexopoulos, Introductory Mycology (London: John Wiley and Sons, Inc., 1952), p. 1.

[6]Ibid., p. 3.

rather easily.

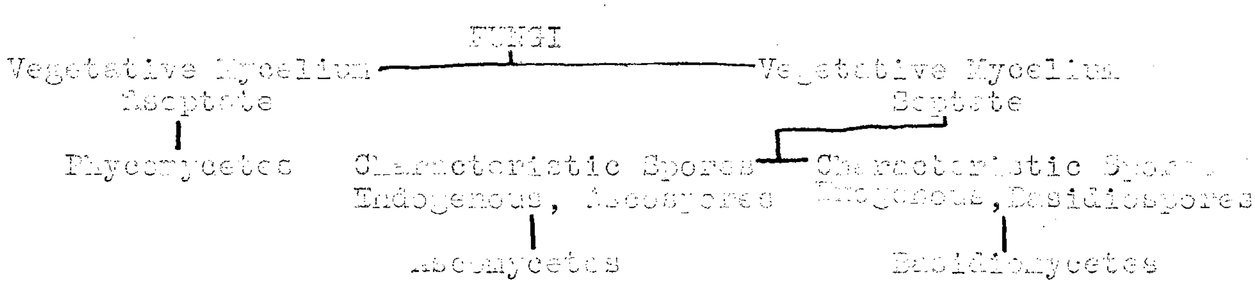
Since fungi have no chlorophyll, they are definitely different from green plants. They also differ from them in that they have no roots, stems, or leaves.

Nutrition

Lacking chlorophyll, sunlight is unnecessary to sustain their life. They live just as easily in the light as in the dark.[7] They must as a result, however, obtain organic foods from plants or animals for their sustenance. They must either infect living organisms as parasites or attack dead organic matter as saprobes. A very large percentage of all plant diseases is caused by fungi. If it weren't for this negative feature of the fungi, they would probably never have been studied to the extent which they have been however meager.

Classification

The fungi are divided into three main groups as follows:[8]



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[7] F. L. Stevens, Plant Disease Fungi (New York: Macmillan Co., 1925), p. 1.

[8] Gynne-Vaughan, op. cit., p. 5.

PHYCOMYCETES

The Phycomycetes are true fungi which reproduce by means of sporangia, an asexual fruiting body. They are commonly called algal fungi because they resemble somewhat the green algae in structure and in this method of reproduction.

Most of the Phycomycetes live in the water and soil. The purely aquatic Phycomycetes are of little direct economic importance. Many of the terrestrial Phycomycetes, however, are among the most destructive parasites of crop plants, causing mildews and blights.[9] It is, in fact, from this group that an example, Phytophthora infestans, has been taken for further elaboration due to its highly destructive nature.

The characteristics of the Phycomycetes which distinguish them from other fungi are:

1. A typically coenocytic mycelium (one without cell walls
2. The production of spores within sporangia
3. The formation of resting spores or resting sporangia as a result of sexual reproduction.[10]

The spores spoken of in the second difference give the basis for further subdivision of this class. If the spore

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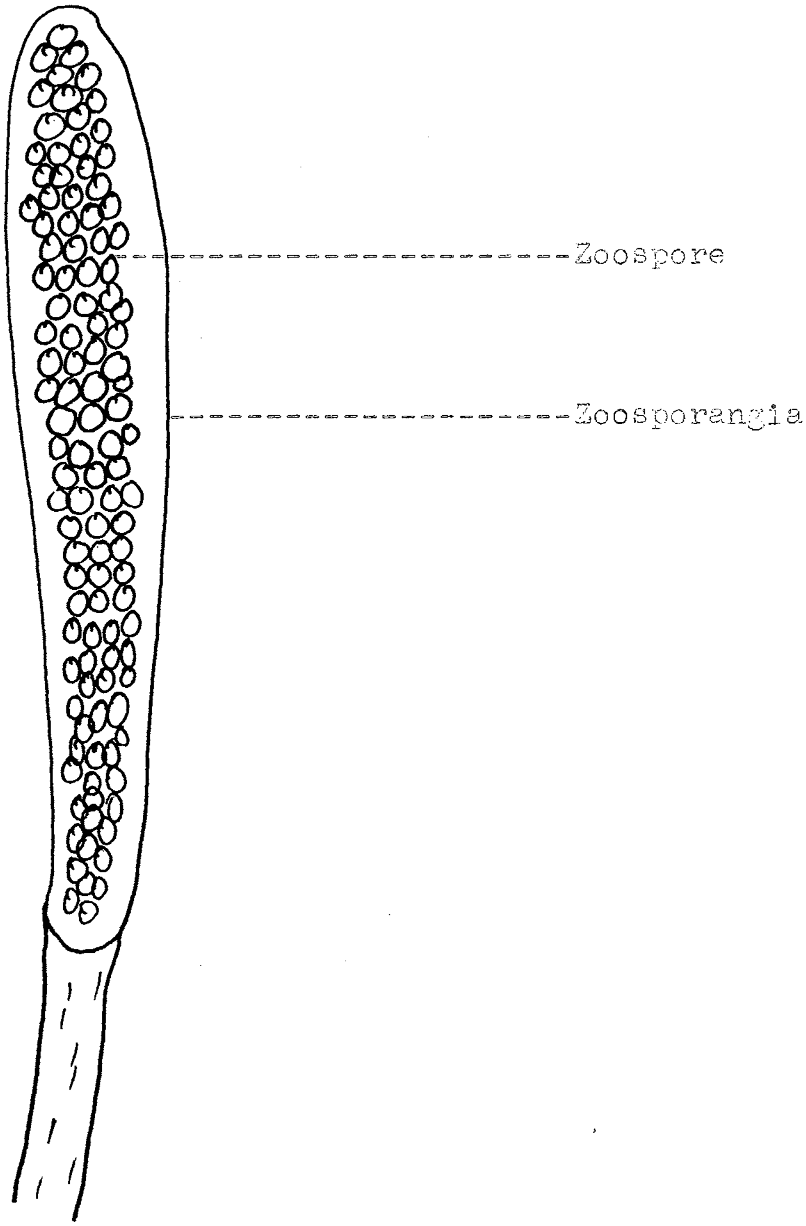
[9]Alexopoulos, op. cit., p. 99

[10]Ibid., p. 100.



SAPROLEGNIA FERAX

has one flagellum, two flagella, or none, it is placed into its respective series. Potatoe blight spores have two flagella, and common bread mold has none.

ASCOMYCETES

The Ascomycetes are sometimes referred to as one of the higher groups of fungi. They are more advanced than the Phycomycetes as can be seen in the complexity of their structure. Examples of the Ascomycetes are the yeasts, black molds, and common green molds. Others are the powdery mildews, cup fungi, morels, and the truffles.[11]

Just as the sporangium is the characteristic structure of the Phycomycetes, so the ascus is the chief distinguishing feature of the Ascomycetes. The ascus is a sac-like structure containing a definite number of ascospores resulting from sexual reproduction. Other characteristics of the Ascomycetes are: a septate mycelium(which means the filaments of the fungus have cell walls); the production by most species of a fruiting body enclosing the asci; and the complete absence of any type of motile structure.[12]

The Ascomycetes in general have two distinct reproductive phases, the ascus or sexual stage, and the conidial or asexual stage. Asexual reproduction may be carried on by fission, budding, fragmentation, oidia, or conidia,

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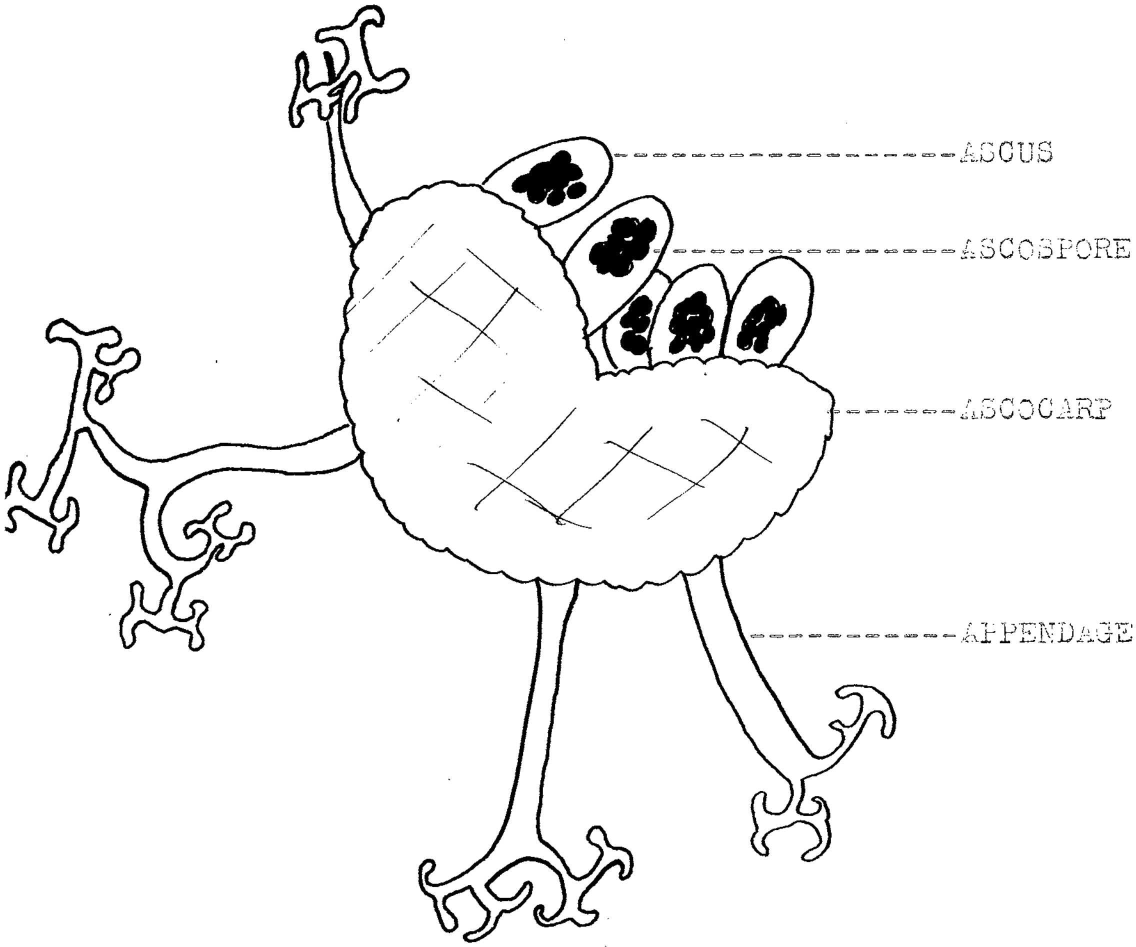
[11] Ibid., p. 182.

[12] Ibid., p. 182.



SPOROCARP OF THE ASCOMYCETE,
MICROSPHAERA

according to the species. Sexual reproduction aims at the union of two compatible nuclei.[13] These two nuclei do not fuse immediately as we are accustomed to thinking of as in the human. They remain rather in close association, forming a functional pair called a dikaryon.

The mycelium or main body of the Ascomycete is composed of septate hyphae (filaments) the walls of which contain a large proportion of chitin.[14] The hyphae are well developed and profusely branched.

BASIDIOMYCETES

The most advanced of all fungal classes is the Basidiomycetes. They consist of such forms as the mushrooms, toadstools, puffballs, and stinkhorns. Smuts, rusts, and the jelly fungi are also Basidiomycetes.

Basidiomycetes differ from all other fungi in that they produce their spores, called basidiospores, on the outside of a specialized, spore-producing body, the basidium. The mycelium of the Basidiomycetes consists of well-developed, septate hyphae which penetrate into the substratum and absorb nourishment.[15] You can find basidiomycete mycelium if you look in moist places in the woods, on rotten logs -- mostly under the bark -- on wet, dead leaves, or on other organic matter. It may be white, bright yellow, or orange, and often spreads out in a fan-shaped growth.

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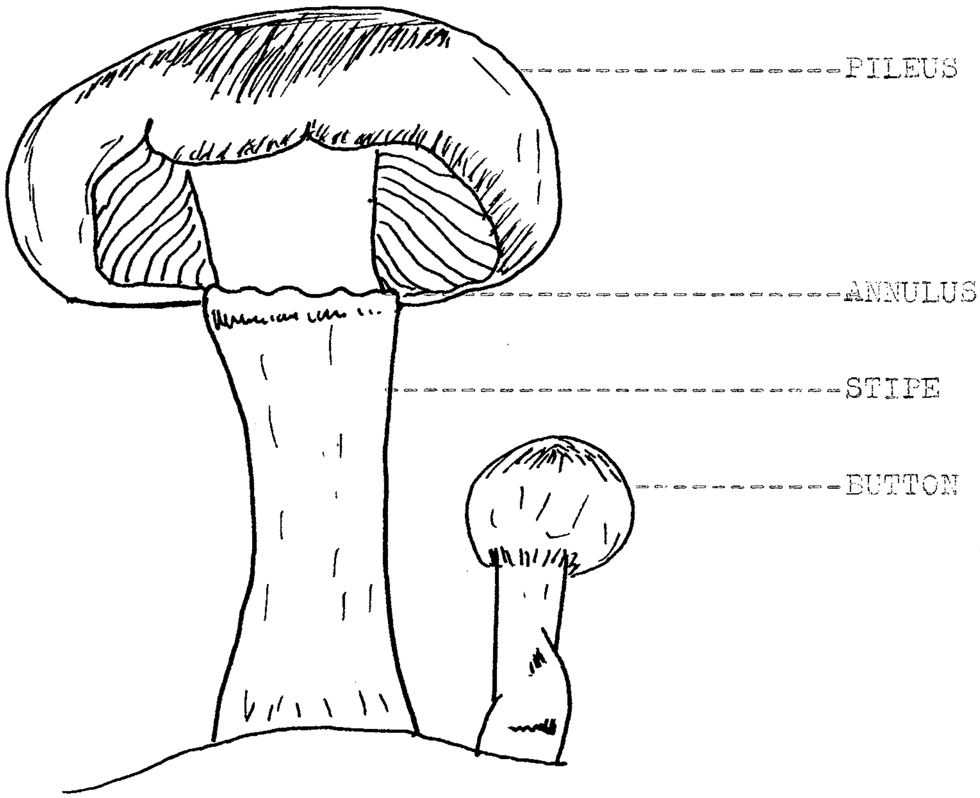
[13] Ibid.,p. 183.

[14] Ibid., p. 183.

[15] Ibid., p. 339.



TYPICAL MUSHROOM

Most Basidiomycetes have no sex organs at all. The somatic hyphae and detached somatic cells take over the sexual function.[16]

The particular fungus to be used as an example of this phylum as has been stated is the potatoe blight fungus which falls in the oomycetes of the Phycomycetes. The mycelium of this fungus commonly enters the cells of the host. This organism is what is known as a facultative parasite. It was shown by de Bary that such fungi possess the power of disintigrating and killing tissues in advance, so that they are not parasitic in the strict sense, but first kill the cells of the host and then live saprophytically upon the dead remains. In this particualr fungus disease the walls and contents of parasitized cells are browned, and this fungus alone on tubers induces dry rot.[18] Invasion of numerous saprophytic fungi and bacteria usually turns this into a disagreeable wet rot, however, which became responsible for the deaths of many thousands of people.

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[16] Ibid., p. 340.

[17] Gynne-Vaughan, op. cit., p. 6.

[18] Stevens, op. cit., p. 67.

CHAPTER FIVE

"AND A FAMINE CAME OVER THE LAND"

A hundred years ago people would have laughed at the idea that these minute fungi, which the microscope had revealed, could be of any importance to mankind. But ideas changed when the English clergyman Berkeley proved that one of these had been the effective agent in the terrible famine that ravaged Ireland in 1845-6.[1]

The fungus rèsponsible for late blight was named _Botrytis infestans_ by Montagne in 1845, but was transferred to the genus, _Phytophthora_ in 1876.[2]

Life Cycle

The life history of the potatoe blight fungus may be described as follows. The spore germinates on a leaf of a potatoe, or on the tuber, and puts forth a narrow germ tube in much the same way as the spores of toadstools do. The germ tube enters the tissues of the potatoe, which it kills, and branching rapidly gives rise to the mycelium of the fungus. This mycelium differs from that of the Basidiomycetes and the Ascomycetes, however, because the hyphae do not have crosswalls, except in the special branches on which the spores are borne. These grow out

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[1]A.F. Parker Rhodes, _Fungi, Friends and Foes_(London: Paul Elek Publishers, 1950), p. 52

[2]Ibid., p. 53.

from the surface of the leaves, or on rotten tubers. They are called sporangiophores. The reproductive bodies, properly called zoosporangia, which they bear are not the same sort of thing as the imperfect spores or conidia of Ascomycetes. If there is not much water about, as on the surface of a potatoe leaf as described above, they may germinate by means of a germ tube. There is another thing they can do. They can give out their contents in the form of a number of very small bodies, having each two fine hairs attached, by means of which they swim through the water. Because they resemble little animals they are called zoospores. They are very frequent among the Oomycetes, to which group the blight fungus belongs, but the sporangia can usually behave as spores under dry conditions, and some species have lost the power to produce zoospores at all.[3]

The main incentive to studying the life history of a fungus which is a pest of crops is to find out how best to kill it and prevent the damage it does.

Symptoms

The disease consists in a gradual decay of the leaves and stems, which become a putrid mass, and the tubers are affected by degrees in a similar way. The symptoms of

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[3] B.B. Mundkur, Fungi and Plant Disease (London: Macmillan and Co., 1949), pp. 54-55.

the disease were described as follows:

> "The first obvious sign is the appearance on the edge of the leaf of a black spot which gradually spreads; the gangrene then attacks the haulms, and in a few days the latter are decayed, emitting a peculiar and rather offensive odour. When the attack is severe, the tubers also decay." [4]

Control

Despite the fact it was the first plant disease at all clearly understood, it was not the occassion of the first successful efforts to apply preventive measures. Indeed it was long before effective measures came to be used against the blight, either in Ireland or elsewhere. In those days the vine was reckoned as a more important crop than the staple food of an unimportant colonial people, and these fungicides were used in viticulture before potatoe-growing.

At first the greatest success was accomplished in securing comparatively resistant sorts. Soon after was the discovery of Bordeaux mixture, used effectively as a preventive of the late blight.[5] Bordeaux mixture was discovered by Millardet in France about 1883.[6] Nowadays, among all farmers who can afford it, it is a regular practice to spray potatoes several times in a season with

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[4] E. C. Large, The Advance of the Fungi (New York: Henry Holt and Co., 1940), p. 13.

[5] Benjamin Duggar, Fungous Diseases of Plants (Boston: Ginn and Co., 1909), p. 170

[6] Ibid, p. 3.

Bordeaux mixture (a mixture of lime and Copper Sulfate), or some similar fungicide. Recently in some countries, first in Holland, farmers have been issued regular predictions of the times when a blight epidemic is most likely to start, this being usually in warm muggy weather of a kind fairly easy for meteorologists to forecast.[7] By taking note of these predictions, a considerable saving of fungicide can be made and the cost of production of is thus reduced. Many other diseases of crops are tied up with the weather, but as yet neither plant pathology nor meteorology are sufficiently advanced for much use to be made of what knowledge we have.

History and Importance to Man

A great deal of space has been given to this phase because of its great interest and importance.

The introduction of the potatoe into Europe had done an immense service to its inhabitants in affording them a staple food which would be worked into the rotation of crops so as to replace the unproductive fallow or the relatively unnutritious turnip by a crop which gave as much food as the cereals. Especially this was so in Ireland, where the climate was more favorable to growing potatoes than corn, and where the standards of life were depressed owing to the colonial status of the country.

But every crop has its pests, and the fungus, which

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[7]Ibid., p. 171.

is now known as the causative agent of the destructive blight of potatoes must have existed among them for some time, though in a non-epidemic form. What were the immediate causes which led to the Irish disaster in 1845? We may never exactly know since so little was then known about the nature and cause of plant diseases that not much can be deduced from the casual reports which came from various European countries. The most probable conjecture is that the fungus lived in the Andes on the wild and cultivated potatoes, but had never done much damage.[8] There is, for example, no record of serious potatoe failure in the Inca Empire, whose subjects would certainly have taken note of anything approaching the Irish outbreak.

But on their introduction into Europe, the potatoes were selected for increased yield, without any suspicion of the existence of the disease. At length some spores got carried over the Atlantic by chance, and at the first occurrence of favorable weather conditions, started an epidemic.

> "A fatal malady has broken out amongst the potatoe crop. On all sides we hear of the destruction. In Belgium the fields are said to have been completely desolated. There is hardly a sound sample in Covent Garden Market.[9]

So began the first warning of a calamity, in the editorial column of the Gardeners' Chronicle and Agriculture

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[8] Ibid., p. 13.

[9] Rhodes, op. cit., p. 53.

Gazette, on August 23rd, 1845. The potatoes had suffered from disease in the past: from scab, from a malady called the curl, from drought, and from too much rain in bad seasons, but nothing quite so destructive as this new murrain had ever been seen before. It struck down the growing plants like a bad frost. It spread faster than the cholera amongst men.[10]

At the prospect of losing the country's food supply for the coming winter, the editor took the easy and obviously false way out by saying,

> "As to cure for this distemper there is none. One of our correspondents is already angry with us for not telling the public how to stop it; but he ought to consider that Man has no power to arrest the dispensations of Providence. We are visited by a great calamity which we must bear."[11]

When the potatoes were dug from the ground they were found marked with the dark patches, symptomatic of the disease. The colors of these patches were that of bruised flesh. Its tints were like those accompanying a black eye. Potatoes left on the floor of a barn for a week were found worse than when they were lifted.[12] The disease was spreading among the potatoes in the ground and in store, and it was thought that every tuber, no matter how slightly affected, would be lost. "A kind of mouldiness which the Rev. M. J. Berkeley had observed to appear

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[10]Large, op. cit., p. 13.

[11]Ibid., p. 14.

[12]Ibid., p. 15.

on the diseased tubers would add to the mischief by hastening decay."[13]

It was apparent that the peculiar changes of weather which had occurred during the summer of 1845 had much to do with the outbreadk and the spread of the Potatoe Murrain. It changed from quite favorable weather to approximately three weeks of one continuous gloom. Tha sun scarcely was visible during the time, with a succession of most chilling rains and some fog, and for six weeks the temperature was from 1½ to 7 degrees below the average for the previous 19 years. One reference stated that Phytophthora infestans would seen to be most effective when the host is growing vigorously as was true in the case of the potatoes before the temperature change.[14] De Bary records that Phytophthora infestans germinates with difficulty in daylight and not at all in sunlight.[15]

Many thought that the plants had been unable to get rid of the excess of water in their usual way, and so they had contracted a kind of dropsy and wet putrification had set in. The Rev. Berkeley, "a gentleman eminent above all other naturalists of the United Kingdom in his knowledge of the habits of fungi," was of contrary opinion.[16]

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[13]Ibid., p. 14.

[14]Dugger, op. cit., p. 65.

[15]Ibid., p. 72.

[16]Large, op. cit., p. 15.

He had at once connected the potatoe disease with the prevalence of a kind of mold on the affected tissues, but in Dr. Lindley's view (author of the quoted editorial) the eminent Mr. Berkeley preoccupied with toadstools and mushrooms and molds and mildews, all the greater and lesser fungi, was attaching far too much importance to a little growth of mold on the diseased potatoe plants. It was only to be expected that as soon as living matter lost its force, all sorts of parasitic types would acquire power and contend for its destruction. Berkeley insisted otherwise. He not only insisted that the growth of mold on the potatoe plants was a highly significant phenomenon, but as soon as he had seen the diseased foliage himself, in his parish, he put forward the revolutionary idea that the mold might be the cause and not the consequence of the Potatoe Murrain.[17]

Thus began an argument between two learned men, each firm in his own conviction and with weight of reason to support it, on an issue that was in fact much deeper that that of attributing the Potatoe Murrain to its most probable cause. The issue was the establishment or the rejection of a new conception of the whole field of disease, not only in plants, but ultimately in all living things. A grand philosophical controversy was beginning, in which

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[17] Ibid., p. 16.

nearly every scientist or natural philosopher in the world would soon be taking sides. In advancing the hypothesis that a living parasitic organism on the potatoe foliage was the cause and not the consequence of the Potatoe Murrain, the Rev. Berkeley was anticipating the germ theory of Pasteur by nearly a quarter of a century.

It seemed a wild notion that a mere mold could be the cause of disease in a living plant. And in truth Berkeley could bring forth little evidence that would be required for rigorous proof of his contention. It was one thing to state that the mold caused the disease and quite another to show how it could do so.

Other hypotheses about the possible cause of the disease were put forth. It was suggested that the rot might be caused by static electricity -- generated in the atmosphere by the issuing puffs of smoke and steam from hundreds of railway locomotives that had recently come into use. Others supposed it to be due to vapors rising from blind volcanoes in the interior of the earth.[18]

On September 13, there was another dramatic paragraph in <u>The Gardener's Chronicle</u>:

> "We stop the press, with very great regret, to announce that the Potatoe Murrain has declared itself in Ireland. The crops about Dublin are suddenly perishing.[19]

In Ireland the cottiers lived almost exclusively on potatoes.

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[18]Ibid., p. 20.

[19]Ibid., p. 21.

If the Potatoe Murrain spread through the small holdings of Ireland there would be millions of men, women, and children, who would not merely suffer acute privation, but who would starve to death. More than humanitarian reasons caused John Lindley to stop the presses with this announcement. The Irish had never been tame and could be expected to put up a certain fight before they died. There would be rebellions, lootings of landowners' property, even more assassinations and a general disturbance.[20]

Thomas Foster, special commissioner for the Times in London, was in Ireland the summer before the outbreak of blight and described the already terrible conditions existing there. His description was one of a parasitism in Ireland before the blight.[21] He spoke of a horrible rent-collecting system that reached into every cabin and drained the substance of the people and the land. The landowners had used every means to raise money, cutting down most of the trees in Ireland and selling the timber, so that the land was a treeless waste. Then, they had granted long term leases of their land to middlemen evading all feudal responsibility for the welfare of their tenants and handing the management over to pure mercenaries. These men in turn subdivided the land, and at every sub-division the rent went up. The smallest farmer, forced to pay these increased rents, learned from his superiors, and again sub-let his land into quarter acres to his laborers or

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[20] Ibid., p. 22.

[21] Ibid., p. 23.

cottiers, who were the ultimate cells of this social organism. On these small bits of ground the cottiers kept body and soul together with from 8-14 pounds of potatoes a day.

As news was received of the spreading of the Potatoe Murrain throughout the length and breadth of Ireland, the cottiers, who had paid little attention to the blighted condition of the foliage, dug nervously in their plots and found the potatoes rotting in the ground. Mr. Foster had been present in October, at the opening of a pit in which some sixty barrels of potatoes -- five months' provision for a family -- had been put down a few weeks before. On sorting the good from the bad less than a single barrel were found to be sound.[22]

Within 7 days so great a part of the potatoe crop was destroyed, that, as a result of actual deaths from famine and still more from subsequent emigration, the population of Ireland was reduced to less than half what it had been, and has never since recovered.[23]

Dr. C. Montagne described the species of fungus causing the disease and called it Botrytis infestans. He sent his sketches and descriptions to his friend Rev. Berkeley who formally published them in the first number of the Journal of the Horticulture Society in January, 1846.[24] It was noteworthy that in this connection it was

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[22]Ibid., p. 27.

[23]Parker-Rhodes, op. cit., p. 53.

[24]Large, op. cit., p. 29.

the Rev. Berkeley, a man accustomed by the exercise of his clerical profession to a certain amount of Christian protestation, who rejected all the nebulous, transcendental, and spiritual explanations, while it was the more materialistically minded scientists who most eagerly used them.

While botanists wrangled and compared notes about the fungus, it was winter in Ireland. As the small supplies of sound or only partially-blighted potatoes rapidly diminished, some four and a half million cottiers and poor farmers faced starvation. One or two of the landowners, who had managed to retain personal control of their estates forgave their tenants the payment of rent for a year, which meant that they could keep their corn for themselves and use it for food. But these humane concessions were very rare. The majority of the landowners, through their agents and collectors, pressed harder than ever for rents, to make sure of them while the getting was good.[25]

The government did not assist emigration, contending that the proper criterion of fitness to emigrate was the possession by the applicant of the initiative, the means, and the courage to do so. There was mass emigration nevertheless. Passage money was sent by relatives who had already escaped to the New World, and all over Ireland the lamentation of departure mingled with those for the dead.

In 1847, ninety thousand set out for Canada alone;

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[25]Ibid., p. 34.

and of this ninety thousand, 2000 died of fever before they reached Dublin or Kingstown, while 13,000 more died in Liverpool or during the passage. Those who survived spread a trail of typhoid about them as they penetrated into the Canadian interior.[26]

The potatoe blight was in Ireland forever; it would become epidemic and ravage the crop again, whenever weather conditions favored its development. In the years from 1845-1860 a million people died in Ireland alone as a direct consequence of the famine and one and a half million emigrated.[27]

The potatoe blight fungus, Phytophthora infestans Mont., had revealed itself as a new and formidable enemy of mankind. By destroying the stable food supply of a human society already very sick from economic causes, it brought about more of death and suffering than any other disaster since the Napoleonic Wars.[28] The little fungus, only to be fairly seen in the strangely lighted field of the microscope, with its filamentous spawn and its translucent spores, not only brought famine to Ireland, but shed new light on the nature of disease, awakened the natural philosopher to the significance of those living things that God created small, and called into being a new branch of applied science -- the science which was to have for its province the defence of the health of the crops.

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[26]Ibid., p. 38.

[27]Ibid., p. 39.

[28]Ibid., p. 40

CHAPTER SIX

CONCLUSION

Because of their way of life, Americans have never suffered seriously from what they consider the minor plagues of the world. Despite the fact that the two parasites dealt with in this study are almost eliminated in the United States, they are still important to Americans.

Isolationism is gone. The world is fast becoming an economic unit, and a disease that affects the production of rice in Burma or meat in Argentina or coffee in Brazil inevitably affects us economically.

We have less than 10 per cent of the world's population and 8 percent of its area, but we use 50 per cent of the produce of the Free World. We depend on foreign sources for over 40 per cent of our minerals, and 10 per cent of our other raw materials -- soon it will be 20 per cent. Undeveloped areas of the world -- the areas principally affected by parasitic diseases -- supply60 per cent of our imports and 40 per cent of our exports.[1] Obviously, the, the diseases that profoundly affect the

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[1]Chandler, op. cit., p. 4.

health and productivity of these areas are of very real concern to us.

A healthy respect for the small organisms which cause big problems has been gained from this study.

SELECTED BIBLIOGRAPHY

Alexopoulos, John. Introductory Mycology, London: John Wiley and Sons, Inc., 1952.

Ash, J. E. and Spitz, Sophie. Pathology of Tropical Disease, Philidelphia: W. B. Saunders Co., 1945.

Bercovitz, Z. Taylor, ed., Clinical Tropical Medicine, New York: Paul B. Haeber, Inc., 1944.

Bessey, Ernst. A Text Book of Mycology, Philidelphia: P. Blakiston's Son & Co., 1935.

Borradaile, L. A. and Potts, F. A. The Invertebrata, Cambridge: The university Press, 1959.

Chandler, Asa. Introduction to Parasitology, New York: John Wiley and Sons, Inc., 1955.

Cooke, M. C. Fungi: Their Nature and Uses, New York: D. Appleton and Co., 1891.

Dugger, Benjamin. Fungous Diseases of Plants, Boston: Ginn and Co., 1909.

Hegner, Robert W. College Zoology, New york: The Macmillan Co., 1955.

Hegner, Robert W. Invertebrate Zoology, New York: The Macmillan Co., 1933.

Kudo, Richard. Protozoology, Springfield, Ill.: Charles C. Thomas, Publishers, 1939.

Large, E. C. The Advance of the Fungi, New York: Henry Holt and Co., 1942.

Mackie, Thomas; Hunter, George; and Worth, C. Brooke. A Manual of Tropical Medicine, Phildelphia: W. B. Saunders Co., 1945.

Manson, Philip. Manson's Tropical Diseases, Baltimore: William and Wilkins Co., 1950

Mundkur, B. B. Fungi and Plant Disease, London: The Macmillan Co., 1949.

Rhodes, A. F. Parker. Fungi, Friends and Foes, London: Paul Elek Publishers, 1950.

Shattuck, George. Diseases of the Tropics, New York: Appleton-Century-Crofts, Inc., 1951.

Stevens, F. L. Plant Disease Fungi, New York: The Macmillan Co., 1925.

Storer, Tracy and Usinger, Robert. Elements of Zoology, New York: McGraw-Hill Book Co., Inc., 1955.

Vaughan, Helen Gynne. Fungi, Cambridge: The University Press, 1922.

Wenyon, C. M., ed. Memoranda on Medical Disease, New York: Chemical Publishing Co., Inc., 1942.

Wenyon, C. M. Protozology, New York: William Wood and Co., 1926.

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