The Glasgow Naturalist (2024) Volume 28, Supplement, 63-86 https://doi.org/10.37208/tgn28S22

Doc Flo's 1917 Lecture Notes on Protozoa

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ABSTRACT
Flora MacDonald (née Livingstone) was a first-year medical student in 1917 when she attended John Graham Kerr's lectures in zoology, starting with the protozoans. She made a clearly written, illustrated set of notes which remained among her papers for decades until their recent discovery by her descendants. The notes give an account of the state of knowledge of protozoa and other disease organisms at a time when many life cycles and transmission routes were being worked out, and some time before viruses could be visualised. Here we present a typed-up version of Flora's notes, a short biography of her by descendant Alasdair Whyte and a commentary on the state of knowledge demonstrated through the notes by protozoologist and medical educator Stephen Phillips.

EDITORIAL INTRODUCTION

This section is the result of a fortuitous interaction between zoology lecturer Anna McGregor and Celtic/Gaelic lecturer Alasdair Whyte. They are collaborating on a community development project related to the West Highlands oyster fishery. In conversation, Anna mentioned the Graham Kerr centenary events, and Alasdair revealed that his great grandmother Flora had been a Glasgow medical student. His family had recently discovered a jotter containing Flora's lecture notes from the start of her studies: would we like to see them? Alasdair made a scan from the jotter's original pages, and sent it to Roger Downie. The scan showed 125 clearly written pages, accompanied by many labelled drawings (Fig. 1). The notes cover the first 18 of Graham Kerr's lectures in the autumn of 1917, with the first 13 devoted to protozoans, mainly those of medical importance. The jotter is unlikely to contain precisely what Flora wrote down during the lectures: everything is so neat. More likely, as a conscientious student, Flora would have written up the jotter from the rough notes taken earlier in the day. Nathan Miller skilfully typed up the notes, and these are reproduced here, minus the drawings. Protozoologist Stephen Phillips, who lectured to the medical students of the 1970s to 1990s, kindly agreed to write a commentary on the notes. Alasdair Whyte wrote a short biographical note on his great grandmother, and Doc Flo's family has kindly allowed us to use the lecture notes and her story in this publication.

The notes provide a fascinating glimpse of what was taught to medical (and science) students about protozoa early in the 20th century: Stephen Phillips's commentary brings out the excitement of that time when much was being discovered about disease and its causation, and also summarises some of the advances since that time. We wonder how many other sets of early lecture notes remain hidden in attics.

DOC FLO: A BIOGRAPHICAL NOTE

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Dr Flora Livingstone MacDonald M.B.E., M.B., Ch.B. (1899–1982), my great-grandmother, graduated from the University of Glasgow in 1923 (Fig. 2). It is fair to say that medicine was in her blood. Dr Flora's maiden name – Nic an Lèigh in the original Gaelic (Livingstone in the anglicised form) – literally means "daughter of the physician". The Gaelic term lèigh is related to the English term leech. The clan, Clann an Lèigh, is a sept of the Beaton clan: hereditary physicians to the MacDonald Lords of the Isles.

Family lore holds that Dr Flora – or Doc Flo as she is still known on her native island of Muile (Mull) – was a keen scholar from an early age. One summer's day, she was sitting on a bridge over Abhainn Bhà (River Bà) near her childhood family home in Grùilinn (Gruline) when some well-to-do visitors enquired quizzically what picture-book the young girl was reading. "It's not a picture-book", replied young Flora politely, "it's Latin".

Young Flora's Latin reading would have stood her in good stead when she arrived at the University of Glasgow at 9 a.m. on 15th October 1917 for the first zoology lecture of her medical degree with Professor Graham Kerr. It was while the family was clearing the loft of Flora's family home – most recently the home of her eldest daughter, Mairi, my granny, and Cameron, my grampa – that we came across Doc Flo's jotter containing her immaculate lecture notes and illustrations from Professor Kerr's lectures.

After graduating in 1923, Dr Flora practised from An Sàilean (Salen) in Mull with her husband, Dr Reginald MacDonald, until Dr Reggie's death in 1953. Several

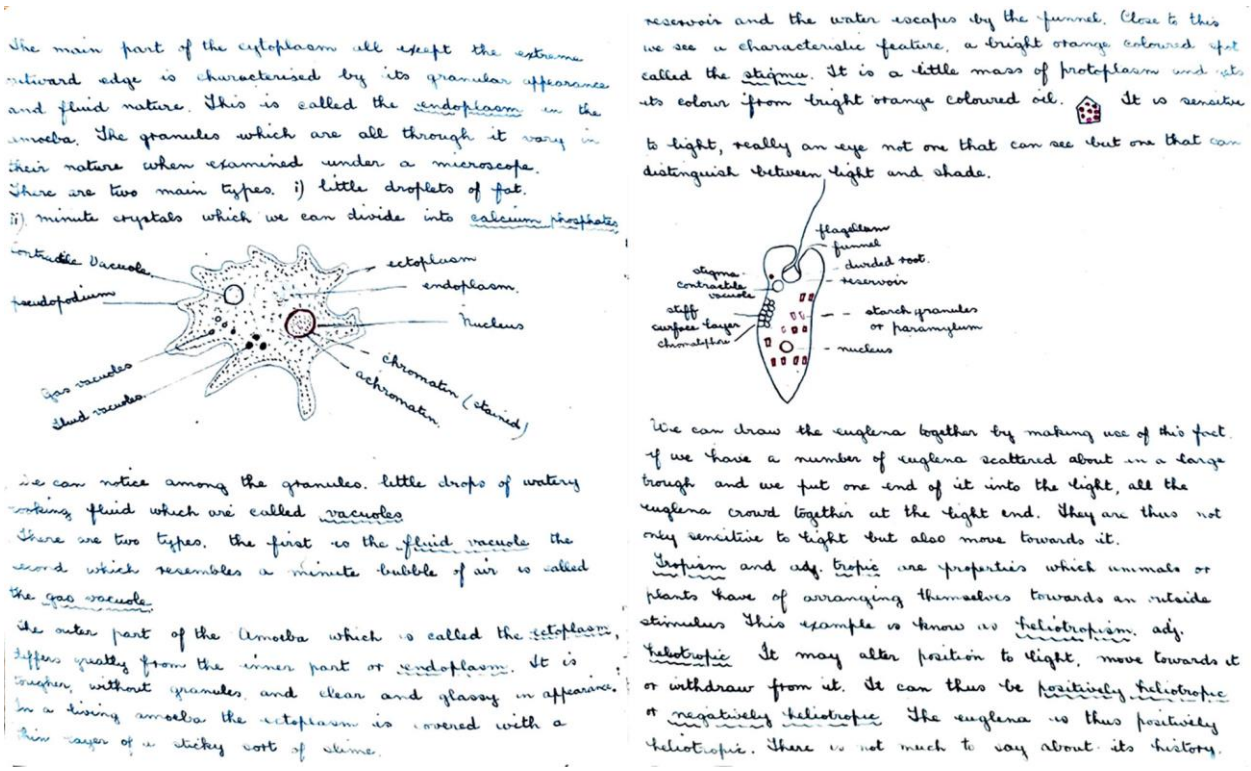

Fig. 1. Two pages from Flora's lecture notes. On the left, a page from lecture 1 on *Amoeba proteus*; on the right, a page from lecture 6 on *Euglena*.

Fig. 2. Flora in her graduation gown and carrying her graduation scroll. (Photo: A.C. Whyte family archive)

stories from that time, including tales of Doc Flo's daring dashes through the hills of Mull on her motorbike in response to medical emergencies, are still in circulation locally. Perhaps the most famous of these stories, though, relates to her actions on 1st to 2nd February 1945 when she was involved, along with several others, in the rescue of the crew of a plane which crashed on her native island. The plane was a Dakota KK194 which had taken off from Dorval near Montreal and crashed into Beinn Tealladh (Beinn Talaidh) in central Mull. Despite cutting her leg and being pulled out of waist-deep icy water in conditions described as "the most severe in living memory", Doc Flo led one of the rescue parties. The five occupants of the plane who had survived the impact were brought to safety. In October 1945, Dr Flora was awarded an M.B.E. for her part in the rescue.

Dr Flora and Dr Reginald had five children: Ian, Bill, Robin, Mairi and Mabel. Ian, the eldest, was in the second year of a medical degree at the University of Glasgow at the outbreak of WWII. He joined the R.A.F. and lost his life in April 1945. Robin worked for British Telecom, later BT, and lives in An t-Òban (Oban). Bill was a G.P. in Cinn Tìre (Kintyre). Mairi, my granny, and Mabel, who helped to write this piece, qualified as Registered General Nurses. Mabel met her husband, Roger Cannon, a medical graduate from the University of Glasgow, in Glasgow Royal Infirmary. Ian Cannon, Roger and Mabel's son, is also a University of Glasgow medical graduate. Roger and Ian practised together from Kirkintilloch for six months before Roger's retiral in September 1996. Ian retired from General Practice in March 2023.

DOC FLO'S LECTURE NOTES: COMMENTARY

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Introduction

As part of the celebrations to mark the Centenary of the opening of the Graham Kerr Building in 2024 I have been asked to cast my eyes over Doc Flo's "Zoology" notes from 13 lectures given by Professor Graham Kerr. My main qualification for doing so is that I taught and then was, for many years, Tutor-in-Charge of a two-term first year biology course given to medical and dental students when they started at the university. In the 1990s, entry qualifications for medicine changed, students were required to have taken Higher or A level Biology before coming into medicine which coincided with the introduction of "problem-based learning" in medicine, and an introductory biology course was no longer required.

Commentary

The first thing to note is that Doc Flo's notes were written in ink. My introduction to writing at primary school was with a simple ink nib and a desk ink well: a recipe for spilt ink and a mess. The pens seconded as darts. Fountain pens were invented in the 1880s and became popular in the 1920s up to the 1950s when affordable ballpoint pens became available: fountain pens could be considered messy to use and inconvenient. Is it likely that Doc Flo used a simple nib and an ink well? The notes have very few corrections and are sprinkled with ink drawn illustrations. She writes in full sentences rather than the key words and phrases which I noted down when I attended undergraduate lectures at University College London: possibly she made notes during the lecture and then wrote these up in more detail later in the day at her leisure. Her handwriting is clear and legible. In my days teaching first year classes, it was always said that when you went into the lecture theatre to give a lecture to a first-year biology class and said "Good morning" they responded with a verbal "Good morning". A first-year medical student would write down "Good morning".

Biological knowledge has expanded at an ever-increasing rate over the 100 years since Professor Kerr gave these lectures, as has the technology available for biological investigation. Perhaps this is best illustrated by Doc Flo's reference to the "invisible germs" which are the cause of infantile paralysis (polio) and dengue fever, although it was well known then that the latter was mosquito-transmitted, as she writes. The assumption that the former was also insect transmitted was not the case as we now know but is transmitted by the faecal/oral route or person to person transmission. The "invisible germs" or "filterable agents" became known as viruses and became visible with the invention of the electron microscope in the 1930s.

Three lectures on *Amoeba proteus* in the start of the course was an introduction to "the lowest form of life" and the structure and physiology of the cell before the advent of the electron microscope and an understanding of subcellular structures. Doc Flo reports Kerr's view on protoplasm, "the physical basis of life", that if we divide it into its parts for analysis, we will take away its most important qualities "and also life itself". This pessimistic view of developing an understanding of the molecular basis of life would soon be superseded, with work such as that of Szent-Gyorgi and Krebs in the 1930s on how cells process energy via the tricarboxylic acid cycle. Doc Flo would have seen *A. proteus* down the monocular compound microscope: the practical laboratory in the Graham Kerr Building, opened after Doc Flo graduated, had large windows to allow sufficient light in to illuminate the specimen viewed down the microscope. She writes that "under high power an amoeba with no nucleus cannot creep", suggesting her notes include the lectures and observations made in the practical classes. "Its living activity is controlled and governed by the nucleus" she writes. The fourth lecture covers the medically important amoeba causing amoebic dysentery - *Entamoeba histolytica* - and how to distinguish it from the harmless *E. coli*, information the modern medical student would be given but not the rest of the lecture on the Foraminifera, Heliozoa and other relatives of *A. proteus*. Let us remember that David Livingstone of this parish is reputed to have died in 1873 at the age of 60 in present day Zambia from a combination of amoebic dysentery and malaria, just 44 years before Doc Flo was learning about the parasitic protozoans.

Lecture 5 on the megalospheric type of amoebae and their complex life cycles would not be included in a medical student's curriculum today. At the end of this lecture is a list of the different types of protozoans which included the spirochaetes which we now know are bacteria. The next two lectures go into the detail of the flagellates with a detailed consideration of *Euglena* and then the colonial flagellate *Volvox*. These led into a lecture on what Doc Flo called "a family of deadly microbes", the genus *Trypanosoma*, in the lecture entitled "Parasitic germs of disease". Her illustrative example is *T. gambiense*, one of the causes of African sleeping sickness, which is very appropriate as this was the subject of the research of one of Graham Kerr's students, Muriel Robertson. Muriel Robertson first worked on reptilian trypanosomes in Sri Lanka 1907-1910, and then, as a member of the Lister Institute, spent three years in Uganda where there was a serious outbreak of sleeping sickness, working on *T. gambiense* and made major discoveries of the life cycle of the parasite in its human and insect host (tsetse fly – *Glossina* spp.). Half a century later, after Muriel Robertson and Doc Flo, the late Keith Vickerman, a past Regius Professor of Zoology in Glasgow, would describe the fine structure of the parasite and relate it to the life cycle described by Doc Flo in her notes from Graham Kerr's lectures. Doc Flo describes the relapsing nature of the human infection in the patient's blood which we now know reflects the parasite's ability to

undergo antigenic variation. Antigenic variation became a major focus of research at Glasgow University 50 years later and still continues. Doc Flo's notes on the variety of trypanosome species and their recent (to her) history, make interesting reading.

Lecture 9 starts with *T. cruzi* which had only been discovered eight years previously in the kissing bug (subfamily Triatominae) in Brazil, and is the cause of Chagas disease in South and Central America. Chagas disease is named after Carlos Chagas, a Brazilian physician and researcher who, on 14th April 1909, diagnosed the disease in a person for the first time. Lecture 9 continues with reference to *Leishmania* spp., named after another Glasgow graduate, William Leishman, who in 1901 discovered *L. donovani* in a post-mortem spleen specimen of a patient who had died from kala azar, or visceral leishmaniasis. Leishman published his findings in 1903. After leishmania her lectures reach the Sporozoa, which Doc Flo describes as a group "with great practical importance". As with me 50 years later in my undergraduate introduction to the Sporozoa, Doc Flo describes in detail the life cycle of the gregarine *Monocystis*, a common sporozoan found in the earthworm, *Lumbricus*. I wonder how many medical students today know the life cycle of *Monocystis*, or even of its existence.

Lectures 10 and 11 tell her about malaria and what was known then about human malaria parasites, the details of the clinical infections and what was known of the life cycle. She recounts the historical landmarks of the discovery of the malaria parasite by Laveran in the blood of a soldier with malaria, the role of Ronald Ross, another Scot, in 1898, in demonstrating the role of the mosquito, *Anopheles*, in the transmission of the parasite. She makes reference to the Italians, Grassi and colleagues, who confirmed the role of the mosquito in transmitting human malaria and who tried to take the credit for Ross's discovery. Doc Flo was clearly on the side of Ross in the celebrated dispute between Grassi and Ross. That there was a cycle in the liver in the mammalian malarias was not demonstrated until 1948 by Shortt and Garnham at the London School of Hygiene and Tropical Medicine (as we know it today).

Lecture 11 moves onto other Sporozoa including the tick-transmitted piroplasms, and in particular *Babesia bigemina* and *Theileria*. Finally, reference is made to Myxosporidia in fish and Microsporodia, mainly in insects, and the role of *Nosema bombyx* as an obligate intracellular parasite infecting *Bombyx mori* to cause pebrine disease in silkworms. This was first discovered by Louis Pasteur, and was the start of the "germ theory of disease". *Nosema* also infects bees. I can see no reference to *Toxoplasma gondii*, the ubiquitous sporozoan in almost all warm-blooded creatures, with a sexual cycle in felids. Its importance as a disease of people was not demonstrated until the late 1930s. In 1908, while working at the Pasteur Institute in Tunis, Charles Nicolle and Louis Manceaux discovered the parasite in the tissues of a hamster-like rodent known as the gundi (family Ctenodactylidae). The sexual cycle was demonstrated by the late Bill Hutcheson working at the University of Strathclyde.

Lecture 12, still entitled "Parasites", ranges from Sarcosporidia (*Sarcocystis* spp.), mostly a parasite of cattle, through an introduction to free living ciliates, such as *Paramecium* and *Vorticella*, and concluding with extensive reference to the spirochaetes which we know as bacteria, rather than protozoans. Doc Flo tells us their place is uncertain between "animals and plants". The importance of spirochaetes is seen in human diseases such as relapsing fever, transmitted by ticks and lice, and yaws and syphilis. As a medical practitioner, after qualification, Doc Flo would be familiar with syphilis and that treatment was not easy before the discovery of antibiotics, 20 years after she would have qualified.

Lecture 13 moves onto diseases, mostly caused by "invisible germs" or "filterable agents" which we know as viruses, such as dengue fever, sand-fly fever, small-pox, hydrophobia (rabies) and scarlet fever (a bacterium *Streptococcus*). There is mention of infantile paralysis (polio) which was thought then to have an insect vector. This lecture reminds us how much the discovery of antibiotics and the development of additional vaccines has transformed the work of a GP. She discusses the fact that some protozoan parasites and their hosts have evolved together, the hosts become "tolerant" and do not cause disease, while in other "susceptible" hosts infection can be disastrous. In 1917, Doc Flo notes that malaria or "ague", which was common in the U.K., has practically "died out" but there was a risk that soldiers returning from the (First World) war would bring malaria back and infect "English" mosquitoes. This, indeed, proved to be the case when soldiers infected with malaria were placed in demob camps in East Anglia, and from whom local *Anopheles* were infected and transmitted malaria to locals in East Anglia.

The remaining lectures in the recovered papers from Lecture 14 on 1st November, 1917, to Lecture 18 on 4th November, 1917 (not printed in this volume), progressed Doc Flo's knowledge from the protozoa to the simplest metazoan, *Hydra*, recognising two layers of cells, and cellular differentiation, through increasingly complex members of the Cnidaria (coelenterates) ending up with detailed structure and life history of corals, and concluding with the sponges, suggesting that there are more notes which have been lost with time.

With the passage of time within any subject in any discipline, the volume of knowledge expands relentlessly and choices have to be made as to what is essential knowledge to progress a student to the point when they have a sufficient grounding and practical experience to become a member of their chosen profession. In the case of the current medical students, I would guess that the only part of Doc Flo's notes which would be included in their lectures are the lectures on parasitology. I had a distinct advantage when I lectured to the first year medical students in that I gave them their introduction to parasites, and if you could not grab the

attention of an eager medical student with an illustrated lecture on a particularly gruesome parasite you were in the wrong profession. Some three weeks ago I had an occasion through a shared voluntary activity to meet a senior member of the medical profession who had studied medicine at Glasgow and had been in my first-year class. Having established who he thought I was, albeit decades older in appearance, he went on to say "I remember the lecture you gave on the guinea worm and winding the adult worm around a stick"… Success!!

JOHN GRAHAM KERR'S LECTURES ON PROTOZOA, AS RECORDED BY FLORA LIVINGSTONE, 1917

Lecture 1 *Amoeba proteus* 15th October 1917, 9-10 a.m.

The *Amoeba proteus* belongs to the lowest form of animal life. While all other creatures have gone through a process known as evolution, the amoeba has retained its original form. It is a little blob in that mixture called protoplasm which is the physical basis of life. We cannot determine the structure of protoplasm for if we divide it into its component parts for the purpose of analysis, we will find that we have taken away all the most important parts and also the life itself.

The dead remains of protoplasm

It is composed of a mixture of proteins which are again compounds of certain elements - carbon, oxygen, hydrogen, nitrogen and sulphur. Proportion – C52, O23, H4, N16, S2. The creature is composed of molecules the nature of which chemists cannot inform us. We can only use simple observation through a microscope in examining this creature.

Description: The amoeba is about half a millimetre in diameter. It is of absolutely irregular shape and there are endless varieties. It has when alive many rounded projections.

We can divide it into two distinct parts, a protoplasm rounded in shape called the nucleus and the remainder known as the cytoplasm.

The main part of the cytoplasm, all except the extreme outward edge, is characterised by its granular appearance and fluid nature. This is called the endoplasm in the amoeba. The granules which are all through it vary in their nature when examined under a microscope. There are two main types: i) Little droplets of fat; ii) Minute crystals which we can divide into calcium phosphates.

We can notice among the granules little drops of watery looking fluid which are called vacuoles. There are two types. The first is the fluid vacuole, the second, which resembles a minute bubble of air, is called the gas vacuole.

The outer part of the amoeba which is called the ectoplasm differs greatly from the inner part or endoplasm. It is tougher without granules and clear and glassy in appearance. In a living amoeba the ectoplasm is covered with a thin layer of a sticky sort of slime.

The nucleus has not the granular appearance of the rest, but it has a curious mottled look which is due to the fact that it is composed of two different materials. i.e. proteins which moreover has the addition of a considerable amount of phosphorus. If we kill an amoeba and dye or stain it, the nucleus takes on the stain in a certain way. Certain parts stand out distinctly while other parts between remain unchanged. The coloured portion is called the chromatin because it stains so readily while the unchanged portion is called the achromatin. There is usually only one nucleus in an amoeba but in some varieties there are more.

Physiology of the amoeba

Hitherto we have been considering the anatomy of the amoeba to study, to which life is not necessary. But we cannot study physiology except on a living body.

Its movement: The amoeba is in a state of continual movement. It is of a characteristic kind and is called the amoeboid movement. Bits of the surface push out to form projecting pieces called pseudopodia. They are rounded finger shaped lobes and are sometimes called lobopods. As it pushes forward, the endoplasm flows in a stream out to the end of the pseudopodium. The ectoplasm disappears and the endoplasm extends right to the point. If the ectoplasm is absent when forward movement ceases, the endoplasm becomes ectoplasm. These therefore are not fundamentally different. The ectoplasm is a portion of the cytoplasm which has taken on certain characteristics through its contact with water. We may find a peculiar type of amoeba which has a peculiarly blunt pseudopodium. In this the endoplasm twists through the ectoplasm and presently we have a big mass of the endoplasm outside the creature which loses its granular appearance and becomes ectoplasm; while pieces of ectoplasm shut off from the water turn into endoplasm.

Formation of pseudopodia

These push out more actively on one side than another and by them the amoeba gradually creeps along. It goes about an inch per day or 1mm per hour. It can only creep when its body is covered by the slime. Like the slug it makes a slimy path for itself. If it cannot form this slime, it may push out its pseudopodia in an aimless way but cannot travel.

The contractile vacuole: Has a sort of pulsation like the beating of the heart. We called this contraction and expansion systole and diastole. This vacuole is on the side opposite to the amoeba's line of travel. It appears like a fluid vacuole, but it increases in size and in 5-8 minutes it may be as big as the nucleus, then suddenly it vanishes. If we place some Indian ink in the water, we will notice that when the vacuole disappears the particles of ink are pushed out. In reality, it opens to the outside and pumps its fluid out. This cycle takes place every 5-8 min. It has two main functions. It pumps out watery fluid from the cytoplasm. Water is always soaking into the protoplasm, it accumulates and is then

passed out. The amoeba is thus constantly being flushed out with water, accomplishing two things: breathing or respiration and excretion.

Lecture 2 *Amoeba proteus* 16th Oct 1917
The amoeba feeding
We call taking food into the cytoplasm ingestion. The amoeba feeds upon plant organisms which are round shaped and microscopic. It goes up to one and pushes pseudopodia on each side of the organism and presently we find the plant is completely surrounded by the protoplasm of the amoeba.

The plant organism is now contained in a food vacuole. As water is also taken in, this forms a vacuole round the organism. The form of food may not be a large organism but tiny bacteria in the water. The watery fluid passed out by the contractile vacuole has a remarkable property called agglutination. It secretes agglutinate which has the power of making the bacteria form into solid lumps and thus they can ingest the solid mass. This is called the process of agglutination. An amoeba may feed on a different material, minute water plants in the form of delicate films. It creeps up to the film and surrounds a piece of it and it can exercise in some curious way a pull on the film and a little later we would see the film getting curled up inside the amoeba until it is all coiled up into a food vacuole.

What happens to the ingested food
If it is alive, for a little while it will move about trying to escape, but gradually the movements slacken and finally we find the organism is dead. If we apply a test to the amoeba, we will find the fluid in the vacuole is strongly acid. The cytoplasm has secreted this acid and then passed it into the cavity of the food vacuole. Its function is to kill the food organism.

The process of digestion
Digestion never affects living protoplasm, it must first be killed. We will notice that the outline of the food particle becomes blurred and indistinct. What really happens is that the cellulose wall round it becomes destroyed. Next, changes take place in the colour. It was at first bright green through the presence of chlorophyll, the colouring of all plants. It becomes darker then it gets a yellowish tinge, next yellow, then reddish, then browny and finally a dark reddish brown.

3rd step
The protoplasm of the food organism breaks up. This is called the process of disintegration. The dead remains of protoplasm are made up of proteins. This complicated mixture is now breaking up into comparatively simpler subjects. The object is to make the protoplasm so that it can be absorbed by the living protoplasm of the amoeba. Before they can absorb and use other protoplasm, it has to be broken into simpler compounds. If an amoeba met an amoeba, one would think it has its food ready-made but no. Living protoplasm cannot add living protoplasm to itself without it going through the aforesaid process. Then only can it absorb the simpler compounds and make new stuff for itself.

How this breaking up is brought about
It is brought about by the actions of ferments or enzymes. These bodies are protein in their nature but the curious fact is that they possess the remarkable power of causing or hastening chemical change in substances with which they come in contact, and it is remarkable that there are an enormous number of them and each can only change some particular type and always in the same way. The secretion of a digestive ferment or enzyme is powered by the amoeba into the food vacuole and the effect is the disintegration of the food organism.

By chemical tests during the period of actual digestion, the chemical reaction is found to be no longer acid but strongly alkaline. This indicates that we have at work a digestive ferment of the same type as in the human being, known as the tryptic ferment. The food particle undergoes this process of digestion, the amoeba absorbs all of the simpler substances that can be made use of. The useless parts remain in the food vacuole as so much debris or faecal material. Then when this stage the food vacuole rises to the surface, touches it and twists to the exterior and the granules of faecal matter are left in a little heap. Though the food vacuole is full of digestive ferment it does not damage the amoeba because it is alive. If we dissect a freshly killed rabbit and open it up, its parts are at first in a normal state but in two hours or so the walls of the stomach disintegrate; as soon as it is composed of dead protoplasm, the digestive ferments get to work on it.

The amoeba goes on feeding and increases in size, showing the process of growth. Its condition would soon because impossible, however, if it grew and grew. Its surface is very important for by the surface it takes in water and oxygen and as it grows bigger its surface will become relatively less, i.e. it will increase at a lesser rate than the bulk. We saw how cytoplasm is comparatively liquid and thus it would be quite impossible for the amoeba to keep its shape. It would flow out into a shapeless mass in which life would be quite impossible. Nature however always provides a corrective process to the process of growth. This is provided by a process known as the process of fission, a simple reproductive process. The first thing is that a change comes over the nucleus.

Process of fission
If we stain the nucleus of an amoeba about to undergo fission, we will find that the chromatin is no longer evenly distributed. It collects in little pods known as chromosomes. These are seen to arrange themselves somewhere about the centre of the nucleus while the chromatin takes on a curious thread-like appearance all running in longitudinal lines. Later it will be seen to have changed its shape. The chromatin is much longer and the chromosomes have divided into two and the two sets have retreated from each other. This long double spindle nips across the middle and the two halves round themselves off and the chromatin returns to its normal shape and we have two ordinary nuclei. This is known as mitosis or karyokinesis. The amoeba becomes elongated more and more until it is like a dumbbell. The

isthmus becomes narrower and narrower and finally snaps and soon we have two amoebas.

Thus the amoeba counteracts too rapid growth and this is the simplest form of reproduction. We can add to our knowledge by the experimental method. We simply make a change in the surroundings of the creature and note what happens. We must first take care that when we make a change it must not be too violent, or the amoeba dies. The change must be very slight.

Mechanical Stimulus: First we can make a living amoeba respond to a mechanical stimulus. If we prod it with a needle the amoeba will creep away from the needle. Thus we know it is sensitive.

Chemical stimulus: There is another kind of amoeba called the *Amoeba limax* on account of its slug-like look. It has a single pseudopodium which it places in front. If we add a minute trace of alkali to a drop of water with an *Amoeba limax* in it, it forms a number of pseudopodia in the form of a number of long threads. One would now think it was an *Amoeba radiosa*.

It (*limax*) gives up its own characteristic appearance and takes on the characteristic of another species. If the water is warmed the amoeba becomes more active, but at 30ºC you find continuation of this activity disappears. Movements are sluggish and it soon dies. Lower temperatures cause it to become sluggish but do no apparent harm. Thus it is sensitive to change of temperature.

It is sensitive to light. If light shines up from below a slide with a dark patch on it and an amoeba is placed on the patch, it draws back whenever a part of it comes into the light. Their natural surroundings are in darkness because they live in the mud and debris at the bottom of ponds.

Lecture 3 *Amoeba proteus* 17th Oct 1917
Effect of electricity on the amoeba
If we take a drop of water containing amoeba and run a weak current of electricity through it, the amoeba at once changes its form. It is no longer like an *Amoeba proteus*, it has taken on the characteristics of an *Amoeba limax* and still more it creeps steadily in one direction, that is towards the negative pole. The two poles, negative and positive, are known as the cathode and the anode. If the current is rapidly reversed, the amoeba at once turns and goes the other way.

Effect of radium
If a piece of radium is placed near the water, the amoeba draws in all its pseudopodia. It takes on a spherical shape and if the radium remains longer near it, the protoplasm breaks down and the amoeba dies. Emanations from radium are hurtful to all living substances. Certain diseases such as malignant tumours are treated by radium, for the diseased cells are affected more easily and are destroyed before the healthy cells have time to get damaged.

Merotomy: This is an experiment. We divide a large amoeba into two by pressing on it with a needle. It usually happens that the nucleus slips to one side and the result is that we have two amoebas, one with a nucleus and the other merely cytoplasm. The one with the nucleus goes on existing as a perfectly normal amoeba: creeps, takes in food and digests it as usual, but there is one important change – the nucleus undergoes a gradually shrinking inside until it is in proportion to the rest of the amoeba. This brings out a general principle all through the animal kingdom. There is more or less a definite proportion between the nucleus and the cytoplasm. It is believed in certain disease conditions that the trouble is due to the loss of proportion between nucleus and cytoplasm. This affects the cytoplasm in a harmful way.

The amoeba without the nucleus: This amoeba is different from the other. It cannot creep but it pushes out its pseudopodia in an aimless way. Under high power we can see the reason, it has lost the power of producing the slime. You may also see it take in a particle of food. It takes it into the food vacuole but it remains undigested. The non-nucleated amoeba has lost the power of producing the digestive ferment. It is thus abnormal in its behaviour. Its activities become slowed down and in about a week it dies.

The chief details of interest are that the amoeba gives us an idea of a cell. A cell is a piece of protoplasm containing a single nucleus. The special interest is that the amoeba has only one cell, while the higher animals begin life as a single cell but do not remain so, they undergo fission many times. The great difference is that when an amoeba undergoes fission, the two amoebas go off independently in different directions. The animal cells undergo fission but remain together. This goes on many thousands of times and finally we have a huge body all united together.

The second interest: is that the amoeba has brought out an important point in the physiology of a cell. Its living activity is controlled and governed by the nucleus. If this is removed or becomes diseased or abnormal, the creature becomes helpless and dies. The study of movement brings us into touch with certain things. The most conspicuous movement, amoeboid movement, consists of pushing out pseudopodia. We can produce this movement artificially. Take a small quantity of rancid oils and put into slightly alkaline water and shake up. This is an emulsion. A drop of this placed under a cover slip shows us in many cases little droplets which do not remain circular but push out little pseudopodia from different parts. A droplet may travel for quite a long distance. The interesting point is we know the reason. Of two fluids, one inside another which do not mix, one becomes spherical in shape. In physics we call this surface tension. The surface later is in a state of tension like a small india-rubber balloon. A softened piece of the balloon would bulge out and become a sort of pseudopodium. The alkali outside diminishes the surface tension in various places and this forms pseudopodia. The living amoeba goes on in precisely the

same manner. The surface tension of the protoplasm is reduced, and the pseudopodia bulge out. We do not know however what diminishes the surface tension. We can also produce more complex movements artificially, such as the drawing in of a vegetable filament. We place some drops of chloroform in water and also some fine threads of shellac. If one of the chloroform drops touches the shellac it goes round it and spreads over the surface. It stretches out at the points where it touches the thread and we see there is a pulling action at work and soon the shellac filament is drawn into the centre of the drop. It is in fact ingested by the drop. This is another case of surface tension. The tension at the points of contact tends to draw in the filament.

Feeding and digestion of food in the amoeba: The great principle is this - whenever living substance is in a state of normal being, it takes into itself simpler dead substances and builds them up into new living protoplasm. But we saw that there was another process. The production of waste material, calcium phosphate and carbon dioxide. This is the breaking down of living material into simpler unliving substances. We get these two processes at work whenever there is life. This is called metabolism. The moment we try to study it, it stops so we cannot tell what it is. There are two other terms. The building up is called anabolism and the breaking down is called catabolism. We very often find these two phases practically balance one another but not always. Anabolism is sometimes predominant, then we get the phenomenon of growth. When catabolism predominates, the creature shrinks in size. But we don't always get this shrinkage because very often we get the products, instead of being thrown out, retained and stored up inside the body of the creature.

Another important point about the feeding: Ordinary green plants feed upon extremely simple chemical substances. They get nitrogen from simple substances, the various nitrates or the carbon from the air, carbon dioxide, CO_2. They divide this, let go the oxygen and use the carbon. An amoeba can't do this, we can't keep an amoeba going on nitrates and carbon dioxide, it must have protein material ready formed. The ultimate effect is that animal life is dependant on vegetable life because the plants make complex proteins from simple substances.

One more point: It is an absolute characteristic of metabolism that it involves the process of oxidation. The amoeba requires a constant supply of oxygen and as it rises it combines this oxygen with carbon in catabolism, which passes away in carbon dioxide. This is the same as the process of combustion. Carbon and oxygen together combine suddenly and produce heat. There is a slow process of combustion going on the whole time. It goes on quick enough in higher animals to warm the body until the temperature is much higher than surrounding objects. The amoeba has no noticeable temperature, but no doubt if we possessed a fine enough instrument, we would discover probably that it had a tiny temperature.

There are different kinds of amoeba, different species. We must be careful how we use the word 'species'. A group like cats and tigers is called a genus *Felis*. The different species have their own special name. *Felis leo* the lion, *Felis onca* the jaguar. Each is a distinct species. This is absolutely necessary because the popular names of animals are different in various countries. *Amoeba* is a genus and there are different species, i.e. *Amoeba proteus*, *Amoeba limax*, *Amoeba radiosa* etc. The important practical point is that certain species have taken on a parasitic form in the bodies of men or animals. One harmless parasite, the parasitic amoeba, is split up into a special genus called *Entamoeba*. The commonest is called *Entamoeba buccalis*. It is often found in the mouth among the teeth but is quite harmless.

Lecture 4 Parasitic amoebas 18th Oct 1917

Many of the parasitic forms of the amoeba are to be found in the intestines. It was not until 1903 that their importance was discovered. Schaudinn found that there were two different types of parasite. *Entamoeba coli*, a harmless little creature and *Entamoeba histolytica* or *tetragena*, a very dangerous parasite. It is the cause of the dangerous disease dysentery. This type is called amoebic dysentery to distinguish it from that caused by bacteria. This amoeba also causes another disease, liver abscess, a usually fatal, tropical disease.

Characteristics of E. histolytica: We must be able to distinguish this type from the harmless *E. coli*. This is a small amoeba, *E. coli* is larger. Therefore we judge by measurement. The unit is 1/1000 mm, known technically by the Greek letter Mu (μ). The amoeba's average size is 20-30μ in diameter. One of the most characteristic points is the way it moves. It has a single large pseudopodium which it pushes out in front and it moves in jerks. The pseudopodium is composed of pure ectoplasm.

When the patient is affected, these parasites burrow into the walls of the intestine, feed upon them, particularly on the red corpuscles of the blood. The result is that, as a rule, we find in the endoplasm the remains of the corpuscles. This is an infallible proof of the *E. histolytica*.

When the patient is recovering, the amoebas change. They leave the walls and creep about inside the cavity of the intestine. They alter in appearance. They are smaller, 12-16μ. The nucleus is larger in proportion. The endoplasm is full of vacuoles and the food particles are no longer corpuscles but little particles of debris they pick up in the intestine. This may go on for months, but as time goes on another change comes over the amoebas. They round off into a spherical shape (11-14μ diameter). They surround themselves all over their surface with a thin skin or shell, known as a cyst. It is a protective device. The amoebas pass away to the exterior and are easily damaged. They are easily destroyed by drought but if the encysted amoeba falls on moist ground or into water, it goes on living for a long period. When a new individual takes in these cysts in drinking water, they at

once start dysentery. They come out of the cyst, creep about and burrow into the intestinal walls. They multiply rapidly by fission and start a typical case. The cyst is 11-14μ in diameter. *E. coli* has also an encysted stage but it measures 15μ. If in doubt as to species, measure a number in order to obtain average.

An additional help in identification: When the amoeba is in the encysted stage the nucleus divides. *E. histolytica* divides twice and shows four nuclei. *E. coli* divides four times and shows eight nuclei. Thus, we see the amoeba is of great importance in relation to medicine. It is also important in relation to agriculture.

It has often been found that a soil normally extremely fertile suddenly loses its fertility. Chemically examined, it is as rich as ever but the agriculturalist says "it has turned sick". Green plants require to get their nitrogen supplied in the form of nitrates which are formed by bacteria called nitrifying bacteria. When a soil turns sick, it is due to a tremendous increase of amoebas and similar creatures which keep down the supply of bacteria and prevent the forming of sufficient nitrates. We have a striking bit of evidence, a sample of soil is baked or treated with chloroform, which kills the amoebas but not the bacteria. We now find the soil has lost its sickness and is quite fertile again.

We divide the animal kingdom into subdivisions called phyla. The division in which the amoeba is we call protozoa. The first animals belong to this class. The amoeba belongs to the first subdivision, Sarcodina, which are formed of a little mass of protoplasm which has no definite shape and is soft all over its surface. We find they feed and move by means of pseudopodia and are characterised by softness and pushing out of their surface. The subdivision of Sarcodina is into two types known under the heading Rhizopoda. The first group is Amoebea which includes the genus *Amoeba* and other forms such as *Arcella* and *Difflugia*. These differ from amoeba in having a shell dome shaped in the *Arcella* composed of horny matter secreted by the protoplasm. On the concave side there is an opening through which the pseudopodia come out. As it is burdened with this shell, it is apt to fall and would be unable to right itself were it not for the fact that it can form gas vacuoles. A capsized one will form these on one side only, these buoy it up and give it a decided tilt until it can finally touch the ground with its pseudopodia and thus right itself. *Difflugia* has a shell but of a very different kind. Instead of a continuous horny substance it is made of grains of sand, which the creature ingests and passes out on to the surface, on which there is a sticky sort of cement in which they form the shell. The other group is the Foraminifera, to which belongs the common marine form *Polystomella*.

Main features of Polystomella: It is common on our own coasts creeping about on the seaweed. In its living condition it is a round disc-shaped organism and all around it are numerous pseudopodia quite unlike those of the amoeba, being thin threads. These are not always separate. Sometimes they fuse together and form a network. It has a skeleton in the form of a shell composed of calcium carbonate. In order to examine the creature, we have to dissolve the shell with corrosive sublimate and acetic acid. Then we can examine it. The protoplasm has a very curious and typical appearance. The whole creature is composed of pieces of protoplasm. The successive pieces are joined together at their inner ends. These segments live in successive chambers and they are filled with the mass. It is just ordinary looking protoplasm. This is not confined to the cavities. The wall of the chambers is perforated with myriads of little holes or Foramina, through which the protoplasm extends and forms a layer in the outside of the shell. This is called the external layer. It is this that extends out to form the pseudopodia. A common mistake is to imagine that the pseudopodia came out through the little holes in the shell.

The pseudopodia of the Polystomella: are used, not only for movement, but also for feeding. If it comes to a plant the pseudopodia stick to it and there are active flowing movements from the body to the pseudopodia. The particle is soon surrounded by a mass where it is digested. The products flow back to the body. There is a continual flowing of protoplasm back and forward along the pseudopodia. If we examine a number of *Polystomella*, we will find they are not all the same, there are two distinct types. The difference is seen particularly in the middle part. In one, the large sphere of protoplasm in the centre is found to be 60-100μ in diameter. In the other the centre is only 10μ. From this difference they derive their names. The first is the megalospheric type, the second the microspheric type. The formation of the second chamber is different. The chamber in the latter goes off into a horn like projection, the other is round.

If we stain two specimens, we find a difference in the nuclei. In the first this is always present about the centre of the mass that is in the spiral. It is in the middle of the bulk of protoplasm. The nuclear material is not confined to the nucleus. If we stain the specimen we find shreds of chromatin scattered through the whole protoplasm, namely nuclear material lying outside the nucleus. These are called chromidia. Thus it has a principal nucleus and in addition chromidia. A stained microspheric type is different. It has no big nucleus but a number of small nuclei scattered about.

If we examine a large number of *Polystomella*, we will find about 30 megalospheric to one microspheric. We find the meaning of this curious fact in the life history of the creature. The micropsheric after a time reproduces by fission, not into two organisms but into many. This is known as schizogony. In this process the pseudopodia seem to become more numerous, increase in size and presently the whole cytoplasm has crept out of the shell into the surrounding waters and taken the place of the pseudopodia. If we stain the specimen we will find that the nuclei have broken up and there are only chromidia left. Later we find new nuclei have made their appearance. The chromidia have become condensed to form these new nuclei. When this comes about, the

cytoplasm begins to show signs of breaking up. It divides up and we have now an empty shell and in its neighbourhood, pieces of its cytoplasm which are rounded off. Each piece has a nucleus and chromidia, slender pseudopodia and is able to creep. Each surrounds itself with a thin shell. The microspheric has given rise, through schizogony, to myriads of new ones. They are about 60-100μ in diameter. Another clue to their identity, the protoplasm begins to bulge out and gives rise to a second cell joined to the first. This second rounded piece is really the beginning of a young megalospheric.

Lecture 5 Megalospheric type 19th Oct 1917

The megalospheric also reproduces but in a very different way. The chromidia undergo a great increase in number. The protoplasm is full of it while the large nucleus breaks down and disappears, probably into chromidia. A very large number of small round nuclei make their appearance, probably formed by the chromidia. The cytoplasm concentrates round these small nuclei, so that the whole substance is divided into these little pieces of protoplasm each containing a little nucleus. We will notice a curious quivering movement throughout the whole *Polystomella* and then a swarm of minute individuals make their way out. Each consists of a little rounded bit of protoplasm with a nucleus. These can swim with great rapidity. They have no pseudopodia, but instead they have two thread-like projections called flagella. These are quite different from pseudopodia. They can be bent rapidly backwards and by their lashing movement they swim. These are really protoplasmic threads. If we saw this process, we would see the creatures swimming away. If they wander about they soon die, but if there was another *Polystomella* undergoing fission in the same waters, each pair of individuals - one from each *Polystomella* – would undergo a process of fusion together. Their cytoplasm as well as the two nuclei would become as one creature.

The flagella disappears and this new individual remains motionless for a time. By measurement, we might obtain a clue, it measures 10μ. This is the centre portion of a new microspheric individual. It soon buds off again and again until it is a characteristic microspheric.

The important thing, fusion, is found all through the animal kingdom. The cells that undergo fusion are what are known as gametes. The cell formed by their fusion is called a zygote. The process is known as syngamy.

This *Polystomella* example of foraminifera has a special characteristic in its shell or skeleton, a kind of horny material or flinty or siliceous material, or solid grains of sand, but in the great majority of cases, calcium carbonate. The characteristic pseudopodia are long and thread-like and have a tendency to become fused with one another. Another great characteristic feature is the dimorphism, i.e. division into the two types, megalospheric and microspheric.

These foraminifera are marine, some creep about on seaweed on mud or on rocks, but not in fresh water. A vast number are called pelagic. They inhabit the open sea, drift about in the waters and form what is called the plankton or drifting population of the sea. This term is only applied to those carried hither and thither by the water. In many parts we find these very numerous, untold myriads floating about. Individuals are continually dying and sinking down. When alive, they float freely because they are always forming vacuoles full of fluid jelly substance, lighter than water. In certain parts, particularly in warm places, there is a continual rain of dead bodies from the surface to the depths. They sink very slowly, in fact take years in deep water. The limy skeleton undergoes a process of solution, it dissolves gradually in the sea water. In the deepest parts they are dissolved long before they can reach the bottom. In parts not so deep, the shells reach the bottom, accumulate, they no longer dissolve because there are no currents, and the water is completely saturated with calcium carbonate. Thus, the shells no longer dissolve. In these ocean parts we have a great deposit of foraminifera ooze. This is a greyish ooze, sometimes called globigerina ooze: from another species which also lives among the polystomellas. One day this ooze will form rock composed entirely of foraminifera shell. In past times, in the old genealogical periods, this process also went on and in the course of ages, turned into rock made of chalk. If we broke up real chalk and examined it under the microscope, we would find it was made up mostly of foraminifera shells. It was concluded that chalk had been formed at great depths because near the shore the ordinary mud from the rivers etc. would prevent this process taking effect. However, when it was investigated, it was found the foraminifera in chalk are closely allied to the modern ones found in shallow water. Thus we concluded, the chalk was formed in comparatively shallow water. There is a peculiar limestone in Egypt, on the shores of the Mediterranean and goes in a band from Persia through Asia to Japan. This is called nummulite limestone because it is composed of curious flat discs shaped like coins. When examined, these are really shells full of chambers much larger than those of the *Polystomella*. If we examined sections we would find some were megalospheric and some microspheric.

In another subdivision come the Actinopoda, characterised by the fact that their pseudopodia are stiff, project stiffly from surface like spines. These are again subdivided into Heliozoa of which a particular example is the *Actinosphaerium*. This creature lives in fresh water and when looked at under the low power, it is approximately spherical in shape. Its pseudopodia are thin and tapering, it resembles a sun and is thus called the Heliozoa or sun animalcule.

If we examine it under a higher power, we see that the cytoplasm contains an enormous number of vacuoles, spongy in character because of their being filled with fluid. The vacuoles are arranged round the surface of the creature in a row. These are larger than the inner vacuoles and this is its ectoplasm. The endoplasm constitutes all the rest of the creature which is full of

much smaller vacuoles. The ectoplasm is prolonged out into pseudopodia, each of which is a long tapering structure, fairly stiff. Under very high power we see that the stiffness is due to the fact that up the centre there is a rod like structure called the axial filament. It supports and gives stiffness. It goes down as far as the edge of the endoplasm.

It uses these pseudopodia for feeding not for moving. If an organism comes up against one, it becomes paralyzed. There seems to be a poison about them. The organism sticks on pseudopodium and the neighbouring ones lean over and become attached to it. They gradually thicken and shorten and gradually as they shorten, the animal is pulled down and is ingested in a food vacuole, like in an amoeba. If we watch this, we see an interesting detail as the pseudopodia shorten, the axial filament vanishes completely. It has become resolved into ordinary protoplasm. Although it looks so distinct, it is not really a thing by itself but just a piece of protoplasm which has undergone a temporary modification like the ectoplasm and endoplasm of the amoeba, not due to the fundamental difference but changes through circumstances.

The endoplasm is nucleated, a large number from one to several thousands. There are contractile vacuoles, we see a bit of the surface bulging out and then collapsing. This is an example of Actinopoda and subdivision Heliozoa. Its essential characteristics are: First, it is a freshwater creature, Second, it has an axial filament in pseudopodia. Sometimes there is a siliceous skeleton, but not in the *Actinosphaerium*.

Another interesting group allied to this is called Radiolaria, differing in habit inasmuch that it is marine instead of freshwater.

Radiolaria type-features: It is spherical in shape more than the former and it has a simple nucleus about the centre. It is very characteristic. Cytoplasm is divided into two distinct regions separated by a membrane which has large openings. It is called the central capsule. It is pretty granular, full of cytoplasm. Outside is another mass differing in the fact that it is full of vacuoles. They contain a watery jelly which buoys up the creature. They are pelagic and float on the ocean.

The outer protoplasm extends into pseudopodia, radiating like those of Heliozoa. This differs as a rule in having no axial filament and also the individuals are apt to get fused together. This tendency is very slight and not like foraminifera. Then we come to the most characteristic point. The outer protoplasm secretes just below the surface a layer of glassy transparent silica. There are breaks in it and thus it forms a perforated shell. After it has formed a skeleton, it goes on growing, becomes larger. The protoplasm extends beyond skeleton and in a short time a second skeleton may be formed. This may be repeated until we have a series of concentric spheres. They assume all sorts of wonderful shapes and are most beautiful things. The interesting points in physiology are that in the outer protoplasm with vacuoles, dotted about are little round bodies. If they are stained each has a nucleus, yellowish green colour. These are little plants of a particular genus called Zooxanthella which live in protoplasm. We must now remember how to distinguish between this type symbiosis and the parasites. Parasiticism is applied to creatures living on another. A creature is called a parasite when the living is entirely to the advantage of one individual, i.e. the parasite. When it is mutual, when each helps the other, they are in symbiosis. These cells produce oxygen and this is of great advantage to Radiolaria (Table 1).

Lecture 6 Flagellata - *Euglena, Volvox* 22nd Oct 1917

The second main division of Protozoa is known as Flagellata. They swim by means of flagella. This is a quite regular thing all through their life. It includes in its species the most deadly microbes and that makes it doubly interesting.

Euglena: This is a fairly common freshwater creature and when in large numbers, gives that bright green colour we see on the top of ponds. This colour is due to chlorophyll. The shape is like a spindle. It swims actively by means of a long flagellum which projects from the blunt end. To make out the details, we require to magnify very highly. We will notice it is composed of a mass of protoplasm. There are no distinct divisions (ectoplasm and endoplasm). The surface later is rather denser and stiffer than the rest. This gives it a more or less definite shape. Close to the blunt end the surface turns inwards forming a depression called a funnel. This opens into a chambe r called the reservoir. A flagellum

I.	Sarcodina
	(A) Rhizopoda
	(1) Amoebea – *Amoeba, Arcella difflugia*
	(2) Foraminifera – *Polystomella* (megalospheric and microspheric)
	(B) Actinopoda
	(1) Heliozoa – *Actinosphaerium*
	(2) Radiolaria
II.	Flagellata – *Euglena, Volvox, Trypanosoma*
III.	Sporozoa – *Monocystis, Plasmodium*
IV.	Ciliata – *Paramecium* (Holotricha), *Vorticella* (Peritricha), *Stentor, Stylonchia* (Hypotricha)
V.	Suctoria - *Acimeta*
VI.	Spirochaeta

Table 1. Divisions of Phylum Protozoa.

proceeds from this and if we follow it to its base, it divides. These branches are on the inner sides of the funnel. The creature is green in colour, it gets this from germs called chromatophore. These in *Euglena* are rather rounded discs of protoplasm packed together close to the surface. There is a large rounded nucleus situated towards the pointed end of the creature. The contractile vacuole lies close to the reservoir between it and the surface. It bursts inwards into the reservoir and the water escapes by the funnel. Close to this we see a characteristic feature, a bright orange coloured spot called the stigma. It is a little mass of protoplasm and gets its colour from bright orange coloured oil. It is sensitive to light, really an eye, not one that can see but one that can distinguish between light and shade.

We can draw the *Euglena* together by making use of this fact. If we have a number of *Euglena* scattered about in a large trough and we put one end of it into the light, all the *Euglena* crowd together at the light end. They are thus not only sensitive to light, but also move towards it.

Tropism (adj. tropic): properties which animals or plants have of arranging themselves towards an outside stimulus. This example is known as heliotropism (adj. heliotropic). It may alter position to light, move towards it or withdraw from it. It can thus be positively heliotropic or negatively heliotropic. The *Euglena* is thus positively heliotropic. There is not much to say about its history. If the euglenas are kept in favourable surroundings, they undergo fission in a longitudinal direction and finally split in two. When conditions are unfavourable, it encysts. It surrounds itself with a protective cyst of a jelly like material. We often find fission taking place while *Euglena* is in a cyst in a condition of rest. Other times it undergoes fission while swimming about. We often get a characteristic appearance of this. They divide over and over again in the cyst and each makes a new cyst for itself. This extends the original cyst until it is of enormous size. If we examine the thick green scum on a pond, we will find it is composed of them. They do not ingest particles of food but feed like green plants. This mode of nutrition is called holophytic. It is able to build up protein out of comparatively simple chemical materials. Particularly it is able to build up starch-like materials through the activity of its chlorophyll under the influence of light. When we examine euglenas that have been under a bright light, we will see all through the protoplasm bright shining glassy looking rods. They are composed of a substance called paramylum, in reality grains of starch. Chemically it is like ordinary vegetable starch, a little different from animal starch. This is produced by the action of light on the chlorophyll. In the case of extended cysts, the giving off of oxygen causes bubbles and makes them float. We must note an interesting point. The *Euglena* is not absolutely holophytic. If we take two glass jars full of euglenas, keep both equally in the light, and to one jar add some organic material, a few drops of animal or vegetable matter, the euglenas will be found to flourish more than those in the other jar. They will increase more rapidly and are generally better in every way. Thus to a certain extent, the *Euglena* can absorb food material through its surface in solution. The last point to notice in the physiology is its movement. There are two distinct types of movements. One is the ordinary swimming movement in a very characteristic fashion. It stretches flagella out straight and the tip twirls round with a screw like motion. The body is thus towed along. The second type is so characteristic that it is called euglenoid movement. This is seen in a stationary *Euglena*. Its body swells out at one end. This swollen portion gradually travels back to the end. Thus, it goes through a sort of writhing movement.

Volvox: This is a freshwater creature, it lives in ponds and lakes. It has an appearance never to be mistaken. A small sphere which rolls along. It goes through the water in a certain direction and rotates along at the same time at right angles to its course. A student told the examiner "The *Volvox* is a creature which goes at right angles to its line of travel". Yet this Irishism is correct.

The first thing we see is that it is composed not of a single cell, but commonly about 10,000 cells, which under low power show as little green dots in the wall. Under very high power we find each cell is oval shaped. A little mass of protoplasm. At outer or broad end the protoplasm extends itself through the surface into two fine threads and it is by means of these flagella that the volvox as a whole is carried along. In the centre we see the nucleus and a pair of contractile vacuoles which contract alternately.

The creature is green in colour but the chlorophyll, instead of being contained in chromatophore, there is a large chromatophore which sheathes the whole creature but top end. The *Volvox* is thus composed of many cells which have a slender bridge between them. There is an orange red stigma present at one side of cell. These stigma are well developed in one hemisphere only, the part in front as the creature moves. They are also only at one end and this particular side is again the one that goes in front. Thus all the so-called eyes look forward. The individual cell is encysted, it has round itself a layer of transparent jelly or cyst, like the sphere as a whole. Each individual is really embedded in a cyst or jelly of its own which comes in contact with the cyst of neighbour. The outlines are difficult to distinguish but each has a hexagonal shape.

Life History: If we start a *Volvox* in favourable water, it will reproduce in a characteristic fashion. In the hinder hemisphere there are a certain number of cell individuals, considerably larger than the rest, connected to each other by several threads called parthenogonidia. There are as many as 35 of these cells in one *Volvox* colony. A noticeable point is that only 8 out of the 35 become functionally reproductive. What happens is they gradually increase in size, undergo a process of fission and divide into two. Then each divides again, making four cells, again into eight cells. A plate or rather saucer-like arrangement is formed. As they go on dividing, the rim curls inwards and eventually becomes a sphere. We have now a sphere with thousands of cells. Each individual cell sets to work to form a jelly-like cyst. The

result is they get pushed apart and the sphere undergoes an enormous increase in size and eventually we find it is a young *Volvox*. Each cell has flagella. Swimming motion begins and it breaks away from the parent and starts life independently. This mode of reproduction goes on for months creating large numbers of colonies. Then a new reproductive process begins, induced by unfavourable conditions such as the coming of autumn. In this new process, gametes are produced which are not exactly alike. One is very small, very active and is called a microgamete. The other is larger, non-motile and is called a macrogamete or egg. This is the first differentiation of the two sexes, for the macrogamete is female and the microgamete male.

Lecture 7 The *Volvox* 23rd Oct 1917

The cells which give rise to gametes are called gametocytes. First, we will look at the male colonies. In a young male colony, what seem to be cells are really microgametocytes. These are like ordinary cells and take on a yellowish-brown colour. They undergo fission into two, four, eight, etc. and eventually form an almost flattened saucer shaped plate of cells which have a long spindle shape. Each cell becomes a microgamete. When developed it is elongated in form and within are all the details of structure as in ordinary cell. At one end are longer and more powerful flagella. In centre, there is a nucleus and a particularly well-developed stigma. There is a chromatophore and a pair of contractile vacuoles at one side. This mass of microgametes move in a colony, soon break out and separate, swimming away in different directions.

The eggs of macrogametes: These look just like the parthenogonidia. They consist of a spherical cell which grows larger and larger. It becomes dark in colour and if a section is made we see that the cytoplasm has become loaded up with reserve protein forming yolk – reserve food material stored in an egg – All we can see in the way of change. A process of syngamy takes place. A microgamete comes across a macrogamete and burrows into it thus fusing with it. We have now a zygote or fertilised egg. This single cell is formed by two separate gametes. The zygote is very like a macrogamete. We can distinguish by this point – after syngamy the cell forms round it a clear protective cyst. These processes come about when external conditions are unfavourable such as cold weather or drought. As the unfavourable conditions continue, *Volvox* colonies are killed and disintegrate but the zygotes drop to the bottom and lie in the mud all through the winter or dry season. They then wake up and become active. They leave the cyst and divide like parthenogonidia until we have a new colony. One important point – In ordinary protozoa, the individual was a single cell. In course of time fission gave rise to two independent cells which were distinct new individuals, each of one cell. In the *Volvox* we have a new type of a higher order, it does not consist of one cell but a colony of cells which have undergone fission but remain together and co-operate to form an individual. The *Volvox* foreshadows more complex animals. Ordinarily we seldom think of life without also thinking of death. Both seem necessarily to follow each other but *Volvox* teaches us the inaccuracy of this. In the later part of the life history, we saw that a particular part of a whole individual became converted into gametes. Thus the living substance does not die a natural death because through syngamy it is given a new lease of life. The living substance is not necessarily mortal, it is potentially immortal. The substance goes on living indefinitely. The living substance of the gametes agrees with the whole substance of protozoa. *Polystomella* does not really die. The microspherics divide and go on living as well as the megalospheric, provided of course that they undergo syngamy. Thus, we see death is not inseparable with life. All the earliest types did not die, they went on living in other forms. Of course, a lot die accidentally. Sexual colonies produce gametes, the rest necessarily die. We have thus the phenomenon of natural death. The division of this higher order of individuals is as follows. There is the gonad or immortal cells and the larger portion composed of soma or mortal cells. We have also in Volvox, appearance of sexuality. The division into two types – microgamete (male) and macrogamete (egg or female).

The next group coming under heading Flagellata are a family of deadly microbes, Genus *Trypanosoma*.

Structure and character of a trypanosome

This microbe was first seen in 1841 by Valentin, living in the blood of an ordinary trout. In 1843 the name was given by Gruby to a similar creature in the blood of a frog. Various people came across them and paid little attention to them, until it became apparent that they were not rare but very common and caused serious diseases. Tremendous research gives us its structure and life history. It is thrown into bends or folds and swims like an eel. When fixed, it retains this shape.

It is composed of a soft mass of protoplasm with no distinct dimensions. The surface layer is a little stiffer than the rest. It is rather pointed at one end. It has a long flagellum which has a curious feature, it does not begin in the pointed end but at the blunt end. It passes along the edge and is joined to the body like membrane, like a fin or frill like arrangement. The creature swims by movement of this apparatus and proceeds in the same way as *Euglena*, i.e. follows flagellum. The extreme characteristic is the nuclear apparatus. It is divided into two portions. About the middle of the body is a big nucleus called the trophonucleus, which controls and superintends metabolism of the creature. It has a very characteristic structure. Chromatin in centre called karyosome and radiating out are small threads of chromatin which swell out at the end into little pieces.

This is only one part because the trypanosome has another nucleus, either round or rod shaped near the blunt end. This is called the kinetonucleus because it is believed to govern the movement. Close to this is a very small part which stains very deeply called the basal granule. It is connected with the end of the flagellum at the root.

There is sometimes at the broad end a distinct vacuole.

This creature reproduces by fission longitudinally and is like *Euglena*. In the process the various parts divide in a very definite order. First, the basal granule divides into two and apparently also the flagellum. We say apparently because although it has the appearance of splitting, extending right along, we don't know if there is a break or not.

Some people think it is a new one sprouting, probably however it is splitting. After the granule the kinetonucleus divides, then the trophonucleus and finally the whole protoplasmic body. These trypanosomes are typically blood parasites and swim and wriggle in the blood. The way we locate them is by noticing how the blood corpuscles are knocked about. Look for this movement to find the germ. They live in the blood of vertebrates, from the fishes up to man and they are spread from one to another by the bite of a blood sucking animal such as flies and bugs. In the case of fishes, it is done by leeches. The agent does not do this is a mechanical way, drawing a mouthful of infected blood from one person and squirting it into another like a hypodermic syringe. It is more complicated a matter. The trypanosome undergoes a special phase of its life history in the transmitting agent. First part of life in the blood of a vertebrate. The other in the body of an agent. As an example, we cannot do better than take the germ which causes sleeping sickness, *Trypanosoma gambiense*, which was examined carefully by Miss Robertson, a former student in G.U. It is found in the blood of a human being. In an infected individual the trypanosomes live in the blood and increase in number. If we take drops of blood and examine day by day, we find they increase and diminish, increase and diminish, in a sort of cycle. They reproduce by fission and if we examine blood during one of these periods, we will find the trypanosomes not only increase in number but undergo a change in form. In particular, there appear among them many individuals which differ in this very elongated shape, they are of a long slender type. Here and there among them are short stumpy ones. An important point is that it is only when stumpy ones are present that the blood can infect a blood sucking fly. If it sucks in only the slender ones, these are killed and ingested but the short ones infect the fly. They go on living and in a certain proportion of cases infect it with the parasites. The particular type of fly which becomes infected is of the genus *Glossina* and is called the *Glossina palpalis* or Tsetse Fly. With any ordinary fly nothing happens. The process is this – the short stumpy type, when taken into the alimentary canal, i.e. the stomach and intestines, become very active and undergo rapid processes of fission until the whole intestine is a seething mass of trypanosomes. Then in a week or so, from the 8th-18th day after the fly has fed, you find long slender ones make their appearance. These become more and more numerous and presently they work forwards into the stomach of the fly. Some of them go still further into the salivary glands where we find them on the 16th-30th day. In these glands these long slender ones multiply rapidly under fission and the trypanosomes produced differ from the ordinary type. They are of the *Crithidium* type resembling another creature *Crithidium* in the positions of the two nuclei. In the ordinary creature kinetonucleus was at the blunt end on the side of the trypanosome away from point. Crithidial type.

Presently we find many have the appearance of short stumpy ones, the same as those originally swallowed. Only now is the Tsetse Fly able to infect. As it bites the saliva is injected into the wound and with it is carried the germs. These proceed to multiply rapidly in the blood and soon the individual is stricken with sleeping sickness.

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Mode of infection of T. gambiense (continued): The special point to notice is that between the time that it is taken in by fly and the injection into a new being, a definite cycle takes place. There is another possible type of infection that takes place without this cycle. When the fly bites an infected individual, the proboscis become contaminated with the affected blood and some trypanosomes may stick to it. In such a case it is quite possible if fly bit someone else soon after, the germs would be introduced directly into the blood. Thus, there are two great types of infection with regards to germs spread by a blood sucking insect.

The types are (i) cyclical and (ii) direct. The type in which germs undergo a part of their life history in a second host is the cyclical. The type in which germs are injected directly into the blood from the contaminated proboscis is the direct. It is important to distinguish the two types. The first is the one of practical importance because direct infection is theoretically possible and sometimes takes place but is comparatively rare (Table 2).

Trypanosoma	**Host 1**	**Host 2**
T. brucei (Nagana)	Horse, Ox, etc.	*Glossina morsitans*
T. evansi (Surra)	Horse, Camel, etc.	*Stomoxys? Tabanus?*
T. equinum (Mal-de-Caderas)	Horse, etc.	?
T. equiperdum (Dourine)	Horse	------
T. lewisi	Rat	Rat flea
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T. gambiense	Man	*Glossina palpalis*
T. rhodesiense	Man	*Glossina morsitans*
T. cruzi	Man	*Conorhinus* (Benchuca)

Table 2. Trypanosomes and their hosts.

1. T. brucei: This is named after Bruce, its discoverer. It is the germ of a disease common to domesticated animals called the tsetse fly disease. The native of Africa call it Nagana. It has been known ever since the first days of the African explorers. As far as Zululand there are tracts of country called fly belts. If horses, mules, donkeys or oxen are taken into these tracts they are all certain to die. The early explorers learned from the natives that the death was associated with the bite of a particular type of fly called tsetse or *Glossina morsitans*. In these belts this type was abundant and the animals contracted the disease Nagana. At first, the fly was supposed to be very poisonous and the poison caused death, but in 1895 David Bruce investigated the disease and he was able to find out the real cause. He found that the tsetse, before it could cause the disease in an animal, must first have bitten a diseased animal. It is not in itself poisonous, but it carries poison from infected animals and inoculates the healthy. More than this, it must bite the second individual within two or three days. Thus, the poison it carries only remains potent for a short time, which suggests a living microbe which could only live that time. Bruce sought for a living microbe and he discovered that infected blood was swarming with *T. brucei*. In medicine these diseases are called Trypanosomiasis. Since this discovery, we got to know still more in particular, not only that there is direct infection in the first two or three days, but after the eleventh day it again becomes infectious. It has now developed cyclical infection which remains with it permanently. The details of the life history have not yet been discovered.

2. T. evansi: This germ causes a disease among horses and camels called Surra and it is particularly common in India, although sometimes found in Asia and Arabia. It is caused by a trypanosome, but it is not quite clear what insect carries the germs. Two insects are suspected – *Stomoxys*, which is rather like a house fly in appearance but it bites. It is characterised by biting proboscis. The other is a horse fly of the Genus *Tabanus*.

3. T. equinum: This germ is the cause of a destructive disease of horses in South America called Mal-de-Caderas. When an epidemic sweeps over the country it usually exterminates every horse. It is produced by a trypanosome but so far the carrier is unknown. It has been noticed that the epidemic will come to a stock farm, right to the fence and come no further. Thus it is most improbable that the carrier is a fly.

4. T. equiperdum: This germ causes a disease in horses, particularly in breeding ones. The disease is called Dourine. It is common round the shores of the Mediterranean and it differs in that it is a contagious disease and not an infectious one. The germ is spread by contact.

5. T. lewisi: This germ is common in rats, particularly in the young ones. The adults seem to develop a sort of immunity to the disease. The carrier is the rat flea. When it has taken in diseased blood, it becomes infectious in about a week. This is cyclical infection. It is found that in the interior of the flea, the trypanosomes are found not only in the cavity of the intestine, but they bore into its walls and are for a long time intracellular in their habits. They multiply in these cells and then make their way back into the cavity of the intestine.

Trypanosomes which affect man

1. T. gambiense: We have already discussed this type which was discovered by Ford in 1901 on the banks of the Gambesi in West Africa. He was examining the blood of a patient supposed to have malaria and instead of malaria germs he found this trypanosome. Various other cases were found in West Africa in people supposed to be suffering from malarial fever. The fever was then distinguished as trypanosomic fever, which is very like malaria and often mistaken for it.

About the beginning of this century, sleeping sickness, a disease long known in West Tropical Africa and described in chronicles of early explorers of that region, made its appearance as far north as the head-waters of the Congo. Stanley's exploring expedition is supposed to have had something to do with it, his native porters probably carried the germs. It spread in Uganda and all the districts near Lake Victoria Nyansa. It was a tremendous epidemic and carried off hundreds of thousands. The Royal Society sent a mission to investigate but for a long time they were baffled. In April 1903, Dr. Castellani an Italian, while examining the cerebro-spinal fluid of a typical case of sleeping sickness – i.e. fluid in the cavities of the brain – saw to his astonishment a trypanosome wriggling through it. He naturally suspected trypanosomes to have something to do with the disease. Later in the same year, David Bruce took up the question, he who had worked out the causation of Nagana. As soon as he learned of the discovery, his interest was aroused and he wondered if it was not a human tsetse fly disease. He and his colleagues set to work to test this idea. They examined the cerebro-spinal fluid and blood of a large number of persons and in every case this trypanosome was present in each. It became apparent that this was really the cause of the disease and that trypanosome fever was only the early stage of sleeping sickness.

They next tested question of transmission. The probability was a blood sucking fly. They caused extensive collections to be made of all the blood sucking flies of the regions. These were identified and maps were formed showing localities in which each fly was found. On another map was planned out the districts where sleeping sickness was prevalent. When they came to compare the maps, one fly map agreed exactly with the sleeping sickness map. The fly was *Glossina palpalis*. Wherever sleeping sickness was found, these flies abounded. This was nearly a proof that the creature was concerned with the inoculation of the germs. They set to work to prove this. They took a number of flies, allowed them to become infected and then let the flies bite monkeys. It was found that in a certain proportion of cases, the bite caused typical sleeping sickness. This was a complete demonstration.

At first it was thought transmission was direct, some took place in 2 or 3 days time, but they found there were two types of infection. One in 2-3 days and one 20-30 days later. After the second phase the fly remained infectious for months and probably to the end of its life. If we feed a number of flies on infected blood, not all develop infection. Only a few do, the others seem to be immune and the germ is killed. It is worth noting that there is some reason to suspect that occasionally, sleeping sickness is contagious. By various experiments it is seen that the creature can burrow through thin parts of the skin on the body without being actually injected. This however is not of any real practical importance. The *Glossina* is the main point. Practical importance is how to combat these epidemics of at least limit their ravages. In some parasitic diseases a number of things have been done, especially in the case of malaria. It is hoped to be able to do the same with this type. Unfortunately the problem is more difficult. In malaria, the insect carrier has a young stage spent in pools of water and it can be exterminated by treating the pools with oil. The *Glossina* however passes a long stage of its life inside the body of a parent. Instead of laying eggs it produces young at a late stage of development. This is a sort of grub which the parent deposits in the earth and as these are scattered about over regions, they cannot be destroyed. It was found however that there are two factors essential to life of *Glossina*, viz damp and shade. They live near water and deposit young within 30 yards of the banks of a stream or lake; further, they always place them in the shade of bushes or trees. We can do something therefore in a particular neighbourhood by cutting down the brush within 30 yards of water, and this greatly diminishes *Glossina* and thus copes with an epidemic.

Another difficulty is that *T. gambiense* is not naturally a parasite of human beings, but of wild animals. These animals are immune to the disease but the germ lives in their bodies and all these wild creatures which harbour it act as a kind of reservoir in which a supply is always available to infect the *Glossina*. We could kill off the wild game, but the trouble is there is a very great variety of wild animals, not only antelopes but many others such as wild rats and mice and it would be quite impossible to exterminate all those. Within very recent years, a new sleeping sickness appeared in Nyasaland and Northern Rhodesia but it is gradually spreading to Portuguese East Africa (once German East Africa). It is spreading and is extraordinarily virile, just as deadly as the old one and more rapid. It is a great danger of the future for it will spread over the whole continent of Africa. This is caused by the *T. rhodesiense* and is found to be carried by *Glossina morsitans*, the ordinary tsetse fly, which is found over all Africa, except the extreme north and south. Hence the great danger.

Lecture 9 *Trypanosoma cruzi* 25th Oct 1917

In 1909, *T. cruzi* was discovered in Brazil, not as a human parasite but as an insect parasite in a large bug called *Conorhinus*, called Benchuca in Argentinian. Dr. Chagas was examining the alimentary canal of Benchuca and he found a trypanosome like a common parasite. He thought it was not a parasite of a bug and so he took some infected bugs and allowed them to bite animals which then became infected. He wondered if they bit human beings. In the regions of Brazil where they came from, there were very common cases of a malarial disease, especially in young children. These were found to be the cause. The infection is cyclical. Chagas found that when a *Conorhinus* was fed infected blood, it took 7-8 days to become infected.

Set of important diseases produced by parasites

Leishmania, after Leishmann (a graduate of Glasgow) is a disease common in various parts of India, in Siam and Lower Bengal. It is a severe and malarial-like disease called Kala-azar. It is found also in the west of Asia. The cause was found to be in the presence of a curious parasite called *Leishmania*. It was found in 1900. Leishmann was examining cells from the spleen of patients suffering from the disease. He found imbedded in the cells little rounded oval bodies which were scattered about in the cytoplasm. On staining the cells, he found these bodies had two deeply staining parts. One round and the other rod shaped. They were found always in the patients and were considered parasites.

A little consideration suggests what parasites. They are arranged in the same way as the tropho and kineto nuclei of *Trypanosoma*. This was one which had gone through a peculiar metamorphosis. Rogers found these were the rounded off stage of trypanosomes. He could cultivate them in nuclear fluid, particularly if acid and in a warm temperature. They changed and lost rounded form and became elongated in shape with a yellow flagellum at blunt end. The two nuclei were still present. They were like trypanosomes of a genus called *Leptomonas.*

This parasite is often found in the body of insects but changed in the body of a human being. It is probably conveyed by bite of insects. It has been found in bugs and in the alimentary canal. They assume the *Leptomonas* form. This points to the bug as carrier. It is also common in dogs, therefore it may be a parasite of dogs and foxes etc. naturally. From this natural host, it may have passed secondarily to man. The order is *Leishmania donovani*.

There are two more species known. *L. tropica* and *L. infantum*. The latter causes disease like Kala-azar, especially in children of Algiers and Tunis. It is also found recently in Italy. *L. tropica* is found in the tropical ulcers of skin and is common in the East. It only causes a local sore, not a general disease. These ulcers occur on exposed parts of the body and suggest an insect is responsible, which is not a creeping but a flying creature. A fourth species not yet named is found in Brazil and Paraguay, it causes sores on the surface of the body, bad ulcers supposed to be syphilitic in nature, but which we now known to be caused by a species of *Leishmania*. The great characteristic of Flagellata is that they swim by means of flagella for the main part of their life.

Sporozoa

This is a group of great practical importance. It has important types of parasites and it is a characteristic of the whole group that they all live as parasites in the bodies of other animals. It is easy to obtain suitable material in which to trace out without difficulty the life history of one genus. This is *Monocystis*. It is found as a parasite in the ordinary earthworm. When we dissect an earthworm, towards the front end there are conspicuous organs, whitish or pale yellow in colour called Seminal Vesicles. These are little hollow organs in which the microgametes of the worm develop. They are filled with a whitish material which under the microscope consists of microgametes in various stages of development. The most conspicuous stage has the form of a spherical mass of protoplasm, the surface of which grows out into numerous rounded projections like a raspberry. This is the Sperm Morula, a stage in the development of microgametes.

Each rounded projection will gradually take on a pear shape and later it would become drawn out into a long thread like structure or microgamete. These break off and mass disintegrates. With an infected worm, however, occasionally there is present inside protoplasm of sperm morula a little elliptic shaped body with a nucleus. This is the young stage of parasite.

The monocystis goes on living and absorbing nourishment from the sperm morula. It increases in size, becomes more elongated in shape and as it does so the sperm morula becomes distorted and loses spherical shape although the microgametes go on developing. The monocystis increases in size and assumes pear shaped form. The sperm morula is stretched out all over its surface until it is only a thin film around parasite. The microgametes have still gone on developing and have reached hair-like forms. The monocystis seems to be ensheathed in a furry coat, but each hair is a microgamete. All this part of the life history is known. It is the trophozoite stage. If asked to define meaning, it is the stage of life history of Protozoa during which it is absorbing nourishment and increasing in size. When it reaches full size it bursts its way out of the remainder of the sperm morula, loses its furry covering and lies free in the cavity of the seminal vesicle. It is a single cell composed of nucleus and cytoplasm. It has a definite cuticle. The main part of cytoplasm is crammed with shining granules. These granules are reserve food material which the monocystis has been storing up.

During the trophozoite stage there is no reproductive process but now there comes about a new part of life history called Sporogony, which proceeds as follows: two of these completely formed trophozoites take on a rounded form and surround themselves with a spherical cell or cyst. These two individuals which are associated together in cyst are gametocytes. If we watch them, we find nucleus of each divides over and over again and the small nuclei pass out to the surface of cytoplasm and each becomes contained in a little projecting part of the cytoplasm. Then projecting pieces become ripped off from the rest and these pieces are the gametes of *Monocystis*. The rest of the cytoplasm simply disintegrates and disappears. It is known as residual protoplasm.

Interesting point in form: the two lots of gametes are different in size. Gametes from one gametocyte are smaller than the others. These gametes begin to move about in the cyst, become mixed up and a process of syngamy takes place. Then they become completely fused. The nuclei fuse and now we have a zygote formed by the union. The interesting point is that in the cyst a large and small gamete fuse together. Thus the zygotes formed are produced by a gamete from each original gametocyte. The zygote when formed, proceeds to surround itself with a shell as is usual with zygotes. It is canoe-shaped and called Pseudonavicella. This name comes from a plant diatom which was boat shaped and called navicella. This being of a similar shape, it got its name.

In this shell of cyst the zygote undergoes a characteristic change. The nucleus divides up three times in succession. We have now 8 nuclei and the protoplasm divides up also into eight sausage-shaped pieces called sporozoites. If a bird eats the worm or it dies, the cysts break up and the Pseudonavicella get scattered through the earth. Garden soil is often full of them. These serve as infection for other worms. When worm swallows infected earth, the wall of the Pseudonavicella is digested and the sporozoites are set free in the alimentary canal. They burrow this way into its walls and begin life history afresh.

Another sporozoite which causes malarial fever, the most deadly of all diseases and marked by more suffering and higher death rate than any other, is called *Plasmodium.* As we all know, malarial fever is characterised by high temperature which is not continuous but comes in successive attacks. These come at fixed intervals. There are three types (Table 3).

If we examine the blood of a malaria patient just after a fever attack, the blood gets its red colour from the red blood corpuscles, circular discs containing red colouring matter or haemoglobin. This important substance in the body has the power of uniting readily with oxygen. It forms a compound which however is easily split up. This fact is made use of it serves as vehicle which carries oxygen through the body and hands it over to various parts where it is required. We will find some of the red corpuscles have got within them a little amoeba-like parasite called an amoebula. This is the very young trophozoite of *Plasmodium*. It lives at the expense of

Tertian malaria	Period 48 hours	Two attacks in 4 days
Quartan fever	Period 42 hours	Two attacks in 6 days

Table 3. Types of malarial fever in humans.

the blood corpuscle, increases in size and develops a large vacuole. This gives it the appearance of a signet ring. It is called the ring stage.

Not only does amoebula increase in size, but the cytoplasm deposits little granules of dark brown, nearly black pigment, called melanin. It is really the product of digestion of haemoglobin. This is characteristic of malaria, the formation of dark brown pigment all through the tissues of the body.

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The trophozoite of *Plasmodium* goes on growing in the corpuscle until the parasite comes to occupy the whole interior. This full grown creature becomes what is called a Schizont i.e. it produces by Schizogony. The nucleus divides several times over and we have a number of nuclei scattered about the protoplasm. This divides into as many pieces as there are nuclei. Each piece has a small nucleus. During this process the dark brown pigment is not divided up but is left out and ties between them.

Soon the blood corpuscle bursts and in its place is a group of more or less amoeboid looking creatures with a heap of melanin pigment amongst them. These are called Merozoites. The group becomes scattered and individuals creep away into the blood. Their fate is to bore into blood corpuscles and become a young amoebula and thus begin cycle afresh. This is a Schizogony cycle.

At the moment when the blood corpuscle bursts, the merozoites are set free in the blood and there goes with them a virulent poison. It may be the melanin itself or some invisible companion. This is important for all the parasites keep time in this process and the whole cycle occupies a definite number of hours: 48 hours in one type, 42 hours in other. At the end of every period, hours as case may be, the considerable quantities of the virulent poison send up temperature and give rise to fever attack. This process goes on over and over again. Effect is that a greater and greater proportion of the blood corpuscles become affected. Thus spreads infection in the body. After a time there commences a new part of the cycle. The Sporogony part begins like Schizogony. Merozoites bore into corpuscles and become amoebulas. These grow inside until they occupy the whole cavity. They don't become Schizonts or break into merozoites. There are two different kinds and as they increase in size, differ slightly when free from corpuscle. One type is larger.

These have a large nucleus rather towards surface of parasite and the cytoplasm takes on certain stains. A very deep colour for its cytoplasm is crowded with granules of reserve food material or a kind of yolk.

The other type are much smaller, the protoplasm has no deeply staining granules. The nucleus is not at one side but in the centre. One rich in chromatin and thus staining deeply.

A patient with this stage of parasite has blood, which if examined under microscope while alive in about half an hour, will show an extraordinary change coming over parasite. The larger type, its nucleus divides and one half is passed out to the exterior. That nucleus is now a polar body.

In the other type it is different. The nucleus divides up to 2 or 3 times, at least twice. Small nuclei are produced which rise towards the surface of the parasite. Suddenly its cytoplasm shoots out into 6 long slender filaments. Each has a small nucleus drawn out into a thread-like shape.

These filaments lash violently about from which fact this is called the flagellated stage. These are not flagella they are microgametes. After end of half an hour, these wave so violently they break off from centre piece and if another type is about the filament swims towards it, comes in contact and is drawn into interior.

Now we have a spherical cell with a single nucleus formed by fusion of its own and the thread one. Thus, thread is a microgamete, other is a macrogamete and the product is a zygote. These previous stages after bursting are either macrogametocytes or microgametocytes. This is the meaning. When the blood cooled, the cooling made this process of syngamy take place. The next phase in life history is passed in a cold-blooded creature, i.e. a mosquito in which the above process takes place. Mosquito means little fly but used in England we mean a particular kind – a gnat – common British gnat, Genus *Culex*. It is also common in tropics where it is known as a mosquito. If we watch how they bite:

SPOROZOA	**HOST**
Telosporidia	
Gregarinida *Monocystis*	*Lumbricus*
Coccidia	Arthropoda
Haemosphoridia, *Plasmodium, Babesia*	1. Man 2. Cattle
Neosporidia	
Cnidosporidia	
Myxosporidia	Fishes
Microsporidia, *Nosema*	Bees, Silkworms
Sarcosporidia	Sheep and mice
Haplosporidia - *Rhinosporidium*	Nose of man

Table 4. Types of Sporozoa and their Hosts.

Culex: It is hunch backed, its proboscis is bent downwards. There is another kind *Anopheles*, when it bites it is in line of proboscis and appears to be standing on its head.

Anopheles: This is the malaria carrier. *Culex* is not. If a culex ate gametocytes they are killed and digested but in *Anopheles* this process would begin in the alimentary canal and zygotes would be present. All the other stages, schizonts or amoebulas, are killed and digested, only the gametocytes live. The zygotes alter in shape, from spherical to worm shaped. This creature creeps actively about, an unusual feature as zygotes are nearly always motionless. This is an ookinete. It creeps about and comes to wall of alimentary canal, it proceeds to burrow through it and finally arrives on the outer side where it assumes its spherical shape.

It absorbs nourishment from the blood and grows in size, its nucleus divides several times over and then protoplasm divides into pieces each with its own nucleus. Each is a sporoblast. The spherical zygote has increased in size and divided into these. It goes on increasing and we find each sporoblast undergoes characteristic change. Nucleus divides over and over again into thread-like nuclei and these pass out into thread-like pieces of protoplasm which develop on the surface of sporoblast. These thread-like things are sporozoites. Residual protoplasm, the centre part is left, only the surface part is used.

A sphere may contain many thousands and this mass bursts and myriads are set free in the mosquito's blood. These make their way forward and eventually penetrate into salivary glands.

Hollow-shaped radiating cells surround tubular cavity or duct of a gland, tube by which saliva passes out. They bore in, crowds of them. Some don't stop there, a number get into the cavity of duct. This completes cycle. From gametocytes to sporozoites takes 10-12 days. When the cycle is completed the mosquito has these in the duct and when it bites, saliva is injected into the wound, slushes gland and sweeps parasites into the bitten person. If they succeed in reaching a blood corpuscle, they burrow in and become amoebula, starting history all over again.

A curious fact about malaria is that if you have it and recover, you are liable to attacks after intervals even as long as five years. Somehow or other the parasite remains in the system although there is no disease. It is an interesting problem how the parasite remains there. We have two distinct views. The general one is that as you recover and kill off parasites, one particular phase is resistant and not killed, the macrogametocytes. These have great powers of resistance for long periods. If you get below "par" and system is run down, these parasites are stored up and behave like Schizonts, produce merozoites and the whole concern starts again.

Another view is that not only macrogametes, but practically any stage survives. The idea is that parasite goes on living in small numbers, reproduces but never sufficiently to cause fever. With system below par, they start again. We must notice three distinct types of parasite which differ in small details and are therefore distinct species.

Plasmodium vivax	Tertian Fever
P. falciparum	Malignant tertian or tropical fever
P. malariae	Quartan fever

Table 5. Species of *Plasmodium* and fevers in humans.

How to identify: All agree with general life history, but important difference is that *P. falciparum* has got a very characteristic feature, the shape of the gametocyte is crescent-shaped. It is called the crescent stage. In other cases it is round.

Thus these crescents in malarial blood point to malignant tertian. The other is the number and arrangement of merozoites. *P. malariae* has this characteristic in it, there are 6-12 merozoites in each group and these are regularly placed in a little rosette. In *P. vivax* the merozoites are more numerous, 15-20 in a group. In *P. falciparum*, 8-32 and in this case, merozoites have no regular arrangement. The clinical difference is the time between successive attacks, 48 hours in tertian, 72 hours in quartan and in malignant tertian, 48 hours or less. It is not so regular.

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We have this knowledge: it begins with Sir Ray Lankester in 1871, in the blood of the frog he found a parasite *Drepanidium* (*Lankesterella*). It is the first example of this group of parasites to which *Plasmodium* belongs. Next advance, Laveran, a medical officer in the French army in 1880, described various parasitic creatures found in malarial blood, no one believed then but now we recognise that he discovered the parasite of malaria as well as the various stages: amoebula, merozoites, gametocytes, the male producing projections round the body, flagellated stage of parasite, but Laveran made tremendous advances in our knowledge.

Next McCallum, a Canadian working at Baltimore in 1898 found malarial parasite in birds, the male gametocyte, he saw flagella break off, swim and undergo syngamy with another parasite. He at once saw the meaning, the flagella were microgametes and the spherical ones macrogametes. Ronald Ross in India also in 1898 found connection of malaria with mosquitoes (known before but not investigated): he tested this parasite in birds and he was able to show how the gametocytes were taken in with the blood of the bird by *Culex*. This gave rise to zygotes which again gave rise to sporozoites. He was able to demonstrate that *Culex* infected a new bird.

Shortly after, the Italians, 1901. Grassi found bird malaria and history of parasite was same as human. In case of birds the carrier was the *Culex* but in humans

Anopheles. Although the Italians get the credit, Ross was really the discoverer: it was a dappled winged mosquito which carried human parasite. This was really the *Anopheles*. The final touch was Schaudinn who completed the business by observing the penetration of sporozoites into the blood corpuscles.

In Telosporidia, the reproductive processes are concentrated together and occur after the period of growth of trophozoite (after process of feeding and growing).

First subdivision: Gregarinida which includes *Monocystis*. The characteristic of trophozoite is that it is intracellular. It is in substance of single cell but this cell is never a blood corpuscle. Later on it becomes free from the cell and is found living in some cavity of the host's body. In Gregarinida there is no schizogony process but only sporogony. This results in formation of sporozoites usually many in number, enclosed in a special little cyst. The trophozoite has a characteristic movement, neither by cilia or flagella but by euglenoid movement. It also shows a curious gliding movement which is produced in a remarkable way. The creature secretes a jelly like material at one end. This pushes it forward. They are common parasites in the lower forms of animals.

The second group: is Coccidia which are again parasites in a great variety of the lower animals. We get them partly in the class of animals known as Arthropoda (insects, shrimps, centipedes), partly in rabbits in which it often causes destructive epidemics. The parasite is again intracellular but in this case it stays in the cell, it is not free like Gregarinida. It increases in size, spherical shaped and it destroys and kills the host's cell.

The danger in this process of schizogony is that the crowds of merozoites are set free to penetrate new cells. Thus enormous numbers of cells are completely destroyed finally causing death of host. It is succeeded by sporogony, zygotes are produced and these are surrounded by a cyst. These parasites are spread by sporogony in the cyst which is swallowed like *Monocystis* by a new individual. The cyst is dissolved and the parasites take back their abode again.

The third group: Haemosporidia, to which malarial parasite *Plasmodium* belongs, has the following special characteristics. The trophozoite as a rule is intracellular, in this case amoeboid in form. The host cell is typically a blood corpuscle. The process of schizogony and sporogony culminates in the formation of sporozoites but these move about freely instead of being confined in a cyst or shell. In this case also sporozoite phase is spent in a different animal to schizogony which is in vertebrates but the former is usually in a blood-sucking insect.

Besides *Plasmodium*, there is *Babesia* or *Piroplasma*. This genus has for an example the best known *B. bigeminium* causes Texas Fever, a destructive disease of cattle in the southern states of North and South America where it is called Tristeza. In South Africa and Australia it is called Red Water Fever. Sporozoite stage is in blood corpuscle as a small oval-shaped parasite. This trophozoite undergoes schizogony in corpuscles and we often find two parasites produced by schizogony or fission. This suggested the name bi-geminium. There are sometimes four or even eight. Whether there is sporogony has not yet been discovered. *Babesia* is transmitted by ticks belonging to genus *Boophilus*. They attach themselves to the body and hang on for about 3 weeks, sucking in blood and eventually they drop off. The parasite is transmitted only by adult female ticks. If a fully developed female takes in infected blood, parasites are set free in the alimentary canal. They undergo change in shape, develop rod-like pseudopodia and creep away into various organs until the whole body is infested. Among other parts, the ovaries in which they burrow into substance of eggs. When the tick drops off the animal, it deposits its eggs and after a time (20-45 days) these hatch out and young tick is infected and can convey infection. This is a type of cyclical infection not met before, the descendants spread the infection. This explains a curious point about Texas Fever – When herds of cattle were driven north from the southern states where the parasite was common, they infected pasture lands through which they passed. These epidemics did not appear at once, only about a month after infected herd had passed through. This gap was very puzzling but now we know the reason.

Similar species of *Babesia* occur in Europe and Northern Africa. *Ixodes* or common European tick is the carrier. This causes another destructive disease of stock. East Coast Fever in South and East Africa and Asia. The parasite is a little smaller, Genus *Theileria.* The great characteristic of all diseases caused by *Babesia* and its allies is that the animals infected show tremendous destruction of red corpuscles. The result is that the haemoglobin is discharged into blood, passes to kidneys and thence to urine hence the name and destructive nature.

The second subdivision: Neosporidia differs in the fact that the reproductive processes instead of being concentrated are scattered all through life history. When the creature is a trophozoite it goes on with reproduction. These are carried on by reproductive cells called spores.

i) Cnidosporidia: these above all are characterised by spores, each is a rounded or oval body but special feature is near one side it has two little bodies, Polar Capsules. Examined under high power these show curious spinal markings. These are very interesting for if we put a spore where it is subject to digestive ferment, an explosion takes place and these shoot out from the end of a thin tube. This was originally coiled up, outside in, within the capsule causing spiral marking. When the spore is subject to digestive ferment, the capsule explodes and shoots out this tube to anchor spore in position in the alimentary canal of the animal that swallowed it. It shoots into substance of wall and anchors spore securely. There are a number of parasites characterised by spores and polar capsules.

1st Myxosporidia: common in fishes, it causes destructive epidemics. In this case parasite is amoeba-like with distinct ectoplasm and endoplasm. The ectoplasm shoots out into pseudopodia in front. These are only used for creeping and holding on. It absorbs its nourishment all over the surface of the body. Within protoplasm are numbers of nuclei. It is common, especially in winter for spores to be produced in endoplasm while the creature is still growing.

2nd Microsporidia: These are found sometimes in fishes but more commonly in insects and at least two are of great practical importance. Two species of the Genus *Nosema*: one causes a destructive epidemic in silk worms- Pebrine disease, the cause of which was among the great works of the Frenchman Pasteur who prepared the way for Lister in antiseptic surgery. The other causes a disease of bees, Isle of Wight disease, this is not quite certain however for a diseased bee has *Nosema* and also bacteria in the weakened body. Which is which?

3rd Sarcosporidia: is a parasite of vertebrates such as sheep, as far as is known they do no harm. But in the mouse it is a destructive disease. These have the characteristic appearance being of enormous size compared with most Sporozoa. We find them in the muscles of a mouse, heavily infected, running through the muscles is a long whitish thread which is a single parasite.

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The muscle fibre of vertebrates is elongated in shape and the parasite burrows into it as a small amoebula. It penetrates muscle fibre and increases in size, eventually increases in length to form a thread visible to the naked eye. It is granular in appearance and has a number of nuclei. The whole protoplasm of parasite divides into little crescentic shaped spores.

The Sarcosporidia are not as a rule dangerous only occasionally the parasite becomes ruptured and the broken up contents are set free and with them a virulent poison. This does no harm when shut up in living parasite. This causes a pathological disease.

The life history is not known or the means of infection or spreading from host to host. If a mouse eats an infected mouse it in turn becomes infected but sheep do not eat each other so there must be some other means.

Haplosporidia are fairly common parasites in fish such as plaice. One has been discovered in the human body causing a curious tumour formation but these cases are extremely rare such as *Rhinosporidium* in the nose. All these creatures go into group Sporozoa which is characteristically parasitic. Again, they are characterised by the fact they never have cilia or flagella when fully developed. They are covered with continuous cuticle without an opening and feed by absorbing nourishment through whole of general surface.

4th Subdivision: Ciliata – *Paramecium*

These ciliata have a definite shape owing to fairly stiff cuticle. Although shape is definite in one type, there are a great variety of forms in the various groups. These ciliata show the greatest complexity in structure found in any creature. The most complicated individual cells in animal or vegetable kingdom belong to ciliata. An individual of one cell with different parts showing in a wonderful way organs in miniature. The body is bounded by distinct outer layers which may be very complicated in parasite. There is the cuticle, a thin bounding membrane. Then layer of trichocysts, then deepest layer of ectoplasm characterised by the fact that protoplasm differentiates (under high power) into little bands, myonema. The interesting point is myoneme is the beginning of muscle. It is a strand of protoplasm which has derived the property of contracting in length owing to their presence. The ciliata can distort their form from time to time. Sometimes these are very well developed. There is Ciliata *Vorticella*, a bell-shaped creature on end of long stalk. It can contract into a spherical shape and shoot down to plant to which it adheres. Inside the stalk we can see under high power a well-developed myoneme running in a spiral strand round stalk. This pulls it down. The endoplasm is very ordinary. The ciliata has usually a distinct mouth opening. The cuticle is continued through mouth opening into a tube or oesophagus. The nuclear appearance is very striking characteristic. In all typical examples it is in two portions, macro- and micro- nuclei. The macro-nucleus looks after the metabolism while the micro-nucleus is purely reproductive in function. We find these vary in shape in different members of the group. In *Paramecium* the macro-nucleus is of a large kidney shape and micro-nucleus is small and lies near the former. In *Stentor* the macro-nucleus is like a string of beads and micro-nucleus is a small sphere close by.

Stylonychie has a dumbbell-shaped macro-nucleus with a small pair of micro-nuclei. In *Vorticella* former is horseshoe-shaped while latter is small and spherical.

These get the name ciliata because they possess cilia by which they move. In *Paramecium* in sub-division (a) Holotricha the cilia are scattered about body in a uniform coating-the simplest condition. If we look at *Stylonychie* in sub-division (b) Hypotricha the cilia have disappeared while on other parts they are fused together into bunches or clumps, solid leg-like structures and it can creep about on these on smooth places. In *Stentor* the cilia are all over the body. They differ in characteristic coating of small cilia, but round the peristome they are clumped together: instead of legs, they form flat plates. The object is to make a whirlpool and bring down the food.

Peritricha cilia: *Vorticella* has none save round mouth. Closely allied to ciliata is the group Suctoria, commonest genus *Acineta*. This specimen has a triangular shaped body borne on a kind of stalk. The lower part is sheathed in clear stiff material which is prolonged into stalk. It fastens on water plants. On the upper end are minute things like little pins, slender structures with heads on end. Under high power each pin-like structure is a long slender tube with trumpet-

shaped mouth. Its function is that if small cilia are touched by a particle of food, movement stops and the protoplasm is drawn inward through the tube by which it feeds. It has a nuclear apparatus like ciliata with macronucleus and micronucleus and contractile vacuole.

It swam in early stages before the tubes had grown and it had adhered to anything. It swam like a small ciliate. They are really ciliates which have diverged.

There is another interesting parasite of the genus *Spirochaeta*. It is an organism of extremely minute size and details are difficult to make out. It is a puzzle which of the other protozoa they come nearest to and whether it is more correct to look on them as small animals or as plants. It is thus a doubtful creature and can be properly regarded as a rather intermediate stage than as belonging to either class. They have a very definite characteristic shape. They are in the form of spiral coil or corkscrew coil. Their cytoplasm is full of granules and it has recently been discovered the spiral is prolonged into flagella. By means of these the creature swims. It has a characteristic movement, shoots in one direction then movement is reversed and it goes in the other direction. Straight lines first one way and then the other. These spirochaetae are common in various animals such as fresh-water mussel in its alimentary canal.

Diseases such as relapsing fever are caused by spirochaetae. The relapsing fever of tropical Africa is caused by a spirochaeta called *S. duttoni*. It measures 16-24μ in length. The particular feature of these fevers is that fever comes in successive attacks. The spirochaeatae show a periodic increase in numbers for they reproduce by fission at certain times with tremendous activity until the whole body is swarming. The numbers gradually die away and fever disappears then shortly they start again.

These parasites are conveyed by individual to individual by the bite of ticks. In Tropical African fever the tick is *Ornithodorus*. It takes in infected blood, then spirochaetae reproduce with great activity and the whole body is infected. The tick is now capable of conveying infection. It inoculated germs and infects the new individual. It resembles the *Babesia* parasite in the fact that the ovaries and hence the next generation are infected. In *Babesia* only the progeny are capable of conveying infection but in this type not only the progeny but also original adult can do so.

Besides this fever there is also a type once common in this country but which has now disappeared, still found however in eastern parts of Europe. This is European Relapsing Fever, *S. recurrentis*. It differs in size, being 4-10μ in length. In determining the type of fever we can easily tell by size of spirochaetae. In European type, infection is conveyed by lice but not by the bite however. It takes in infected blood and its own body gets swarming – By the way, for a little while afterwards it is impossible to find any spirochaetae, they cannot be traced. In one examined after 24 hours from time of infection, not a single parasite was to be seen, but in a week they made their appearance and from 8th-19th day the insect was swarming. This is the period of ability to transfer infection. After 19th day parasites diminish in number and disappear apparently finally – It is not conveyed by bite but if an infected insect is crushed on the body of a new individual and the contents get on to the skin, or places like the eyeball, where skin is thin or where there are cuts or scratches, the parasites find their way into the blood and cause infection.

Parasite of Syphilis is also of this species, *S. pallida*. Yaws, a tropical disease, too has the parasite *S. pertenus*. Infectious jaundice has parasite *Icterohaemorrhagiae*. Relapsing fever, *S. dulloni*. European R.F., *S. recurrentis*.

Rat-bite fever: in Japan supposed to be conveyed by the bite of rats is also caused by a parasite which is minute in size and multiplies by fission. In some spirochaetae, especially in mosquitoes, under certain circumstances division by fission is so rapid that as they divide in halves they get smaller and smaller until they get so small they pass beyond the limit of our highest powers on the microscope. Thus we get a case of invisible germs. A microbe too small to be seen. There are a number of diseases due to germs but in which there has been complete failure to see a germ. Such an example is Yellow Fever: we know there is such a germ or microbe and that it is carried by a mosquito called *Stegomyia*. We also know the size although it has never been seen. Filters are used to filter bacteria and are made in different degrees of fineness. The infected fluid is filtered through finer and finer pores. They start with coarse filter and germs come through, for fluid is still infectious. Take it on the to the next filter until finally in some one of the series the germ is stopped, thus we have a rough measurement. Apparently the germ is present in the blood of a yellow fever patient during first three days. During that period if a mosquito bites the patient, it becomes infected and after 12 days it can infect a new individual. Thus it has cyclical infection so there must be a life history going on. *Spirochaeta* has given us a definite known example of invisible germ and yellow fever suggests that germ may be simply a very minute spirochaeta.

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There are one or two other diseases caused by invisible germs.

Dengue fever: Common in warm countries: has never revealed its germ to us, but these epidemics of fever occur only where a certain species of mosquito is found. *Culex fatigans*. Places where it has been exterminated are free from fever.

Sand Fly Fever: Conveyed by *Phlebotomus*, a small midge, is common in Mediterranean countries and eastwards to India. It is clearly due to the bite of the sand fly which can convey infection only about 7 days after taking in infected blood. Thus the infection is cyclical.

A third disease, although evidence is not so clear, is Infantile Paralysis which often occurs as an epidemic. Again a fly, *Stomoxys* is blamed. In recent years it has turned out that a kind of Protozoa is responsible for various diseases grouped under the name of Chlamydozoa. Among these are smallpox, scarlet fever, hydrophobia, trachoma and also a disease of sheep called foot and mouth disease. In the cells of an infected body were seen curious little rounded bodies which when first discovered were supposed to be parasites. However these are not in themselves parasites, they are a kind of reaction on the part of the cell to the presence of the real parasite. If these little bodies are examined under the highest power, in the centre will be seen an extremely minute, deeply stained dot. These have a dumbbell shape as if divided into two or undergoing fission. These chlamydozoa are the parasites and protoplasm surrounds them with a cyst.

Points about protozoan parasites: Large numbers have taken a parasitic existence, some cause diseases of a severe kind but the production of disease is not a normal characteristic of a protozoan parasite. In any one particular locality in the world there is a definite set of animals and these have a definite set of protozoan parasites. Normally these two sets are associated for long periods of time and they get accustomed to each other, so no disease is produced. A great struggle for existence goes on in nature and not only is a handicapped individual apt to be exterminated but also all the strain which it represents. Thus strains with a particular weakness tend to be eliminated, so with parasites, where a particular type of parasite is in a particular type of animal. Animals liable to disease are handicapped in struggle and so are weeded out. Species as a whole is not liable to disease. Look at the relation of parasite and host. If host animal is inoculated it may behave towards parasite in three different ways.

1. Repellent: If introduced into blood of host it is at once killed off.
2. Tolerant: In this case parasite goes on living and multiplying but never sufficiently to cause disease. Living activities of host keep it under control
3. Susceptible: In this case, parasite not only goes on living but it multiplies so greatly that it overruns the body of the host, interferes with activities and produces symptoms of disease.

Evolution: Susceptible individuals in any neighbourhood are cleared out. When we do get disease produced is when a particular kind of host and parasite are brought together which have not had time to get accustomed to each other, that is before susceptibility has been eliminated. Thus when brought together for the first time, disease is generally produced through the actions of human beings, hosts are introduced to some part of the world where they meet local parasites to which they are not accustomed. Take the case of Malaria: the white man goes to the tropics where the natives are tolerant while he is still susceptible, not having had time to get weeded out, he meets the parasite, falls a victim and develops disease.

Nagana is another example. Cattle in fly belt regions meet with the trypanosomes and instantly disease is produced. Disease is also caused by migration of hosts.

In other cases, epidemics are caused by movements of parasites and not hosts. The yellow fever parasite is carried to some place strange to it and then violent disease results. Sleeping sickness is the same. *T. gambiense* was transported up to Congo probably by Stanley's men. Thus there is always the dangerous possibilities of disease being carried into unknown regions with fatal results. Yellow fever is a disease found particularly on east coasts of tropical America and west coasts of tropical Africa. Its prevalence and conveyance depend on presence of the mosquito *Stegomyia*, one which is widely distributed throughout the tropics of the world: East Africa, India, Malay Peninsula, Islands of Malay Peninsula, Queensland etc. Thus if this germ is carried east, for *Stegomyia* is transferred in ships, all these districts may be infected. Malaria called by us 'ague' was once quite common in this country; it has practically disappeared because English mosquitoes were no longer infected. But, at present, numbers of men in the war are coming home with malaria; there is the possibility of our mosquitoes again being infected and it is perfectly possible malaria will become prevalent again.

Phylum Protozoa: This group is one of the main groups of the animal kingdom, its special features are: first, most usually it is composed of unicellular individuals. That cell may undergo fission and divide up but in such a case these separate and lead their own lives independently. These protozoa probably represent the most ancient type of animal life. In the earliest days of evolution, first animal was probably protozoan. But although protozoa are extremely ancient and simple, we may find individuals showing very great complexity in structure such as Ciliata. In feeding, the protozoa digest food in the body of the cell, intracellular digestion. They reproduce by fission if an individual protozoan is kept under favourable conditions such as a *Paramecium*. In due course it undergoes fission and new individuals again divide up. As time goes on the activity of this process slackens off and takes place more rarely until it finally stops. *Paramecium* might divide 200-300 times. It doesn't begin again unless by some stimulus, which is normally afforded by process of syngamy. The zygote has powers of fission renewed and it starts again for 200-300 times, and so on. All remainder of animal kingdom we class together in the set Metazoa. When metazoa begins life history, like the protozoa it consists of a single cell, the difference comes out later. This zygote undergoes fission, 2-4-8 etc. It goes on dividing for a definite number of generations but here the produced cells instead of separating, remain united to form a single individual. Owing to this fact about the process of fission, normally the individuals of a particular kind are approximately all of the same size for the process goes on for some time. The first result is that in the point of size, metazoa are enormously larger than protozoa. Correlated with these cells we see a wonderful division of labour, different sets and different tissues. Each tissue

or set of cells having to perform some particular function.

1st Division: Gonad and Soma: The gonads are made of the cells which retain the power of undergoing syngamy. The soma or greater part are cells which have lost that power and so perish. It is the soma which forms the great bulk of the body, it is of enormous size, its cells undergo differentiation into different groups for different functions.

One other point: the protozoa are creatures which all live in watery fluid, if dried they at once die. Thus the general feature of living protoplasm is that it can only live if the surface is bathed in fluid. In metazoa therefore, the whole body is permeated with watery fluid lymph, the medium in which cells live. It is sometimes called the internal medium. The external medium is either air or water. This lymph is very important. It is not pure water but fluid of great complexity. Into it diffuses constantly the various products of the metabolism of the body. Each particular part gives off definite fluid which passes into the lymph.

The point is this – that every cell in the body does not give exactly the same substance. Every type of cell has its own peculiarities in the particular substance which it gives off and the result is that the internal medium is not only tremendously complex but the composition is definitely made up of contributions of a particular type from each particular organ. If the balance gets upset and one set of cells pay out too much or not enough, it means that the composition of the medium becomes abnormal and disease is produced.

Skeleton	As the body is composed of semi-fluid protoplasm, it would be unable to retain its shape without supporting tissue.
Muscles (Muscular system)	Cells which have especially developed the power of contraction. These are bands of cells attached to skeleton by their ends and so move parts of the skeleton and hence move the body.
Nerves (Nervous system)	These are for the purpose of receiving impulses and impressions from the outer world and for the purpose of transmitting impressions from one part of the body to another. Impulses to the muscles and finally keeping up general control over living activities. Besides these, those connections with metabolism.
Alimentary system	Function is the taking in of food, digestion, absorption of products.
Blood vessels (Transport system)	Consists of cells arranged in the form or tubes, i.e. blood vessels, containing other cells which float free in liquid, the blood. The tubes are muscular in character whose pieces can contract and pump blood through system of vessels.
Renal system (Renal organs)	The function of these is to extract the more or less poisonous waste material from the blood and pass it to exterior of the body.

Table 6. General types of tissue in Metazoa.