NATIONAL OPEN UNIVERSITY OF NIGERIA

SCHOOL OF SCIENCE AND TECHNOLOGY

COURSE CODE: BIO 212: HELMINTHOLOGY

COURSE TITLE: HELMINTHOLOGY

BIO 212: HELMINTHOLOGY

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COURSE GUIDE

CONTENTS   **PAGE**

Introduction

Parasitology to a lay man is the study of parasites. A parasite is an organism that lives in or on a host causing harm to the host. In addition, parasites are metabolically dependent on their hosts. A good knowledge on the biology of a parasite, transmission cycles and human behaviours that promote transmission are necessary to eliminate or reduce infection.

The course therefore introduces students to the biology of common parasites in the helminth category in tropical countries and some other countries of the world. To facilitate learning, diagrams on their morphologies and life cycles are included in the guide. For coherence, epidemiology and control are treated simultaneously.

The contents of this course therefore will serve as basis for studying parasites. Information on diagnosis of parasites will equip those willing to practice clinical parasitology with practical knowledge.

The Course

This course Guide tells you briefly what to expect from reading this material which bothers on the study of parasite "Parasitology".

The association 'Parasitism' is a harmful association in favour of the parasite which likely evolved from a non-harmful association called commensalism. The location and behaviour of parasites have enabled us to place them into different groups. Parasite can not survive without a host organism, therefore the study of host in parasite transmission is equally important.

Parasites can be grouped into those with zoological, veterinary and medical importance. Those invading livestock (veterinary parasites) and humans (human parasites) are touched in this course Guide. Generally, parasitic helminthes are grouped into trematodes, cestodes and nematodes. The digeneans are the only groups with medical importance in the class Trematoda, while nematodes form the majority of the helminthic infection. The entire course focuses on the following; lifecycle, morphology, diagnosis, epidemiology and control of the parasite.

Life cycle: this involves the routes of transmission and parasite development from

the immature to the adult stage. Some helminths have direct lifecycles involving no intermediate host. Examples in this category are *Ascaris lumbricoides,* hookworms and *Enterobius vermicularis*. On the other hand, some show indirect lifecycles which could either be homoxenous or heteroxenous. Homoxenous indirect life cycle utilizes a single intermediate host. e.g. *Schistosoma* spp that make use of specific snail intermediate hosts for intramolluscan development. In heteroxenous indirect lifecycle, two or more intermediate hosts are utilized in the transmission cycle. e.g. *Diphyllobothrium latum* that makes use of cyclops and fish as the first and second intermediate hosts respectively.

Morphology: this is gross anatomy. Each helminth group has generalized features. The morphologies of the adults are often different from the immature stages. The adult trematodes are often leaf-like with two suckers (the oral and ventral suckers). The reproductive system is often hermaphroditic. Cestode body is divided into head, neck and strobila. The integuments are metabolic in function. The distinctive morphological features of either the adult or the larval form are often used in the diagnosis of helminth infection.

Diagnosis: the methods used in the detection of parasite in the host is dependent on various factors. The site of infection of the parasite, morphological indifferences in some closely related species, cost effectiveness of different diagnostic methods and introduction of new technologies and proficiency of laboratory technologists to manage these new technologies. Parasites that inhabit the intestine will best be diagnosed through stool examination .e.g. *Ascaris lumbricoides*. In infection due to *Paragonimus* spp whose adult flukes reside in the lungs, diagnosis is done through sputum examination. Usually diagnosis of parasitic helminths is done by looking out for the striking feature(s) of the eggs laid by the adult parasites. However, when the eggs of two or more closely related species are morphologically indistinguishable, such as those of the two Hookworm species (*Necator americanus* and *Ancylostoma duodenales*) and the hexacanth eggs of *Taenia* spp., the adult parasites could be used.

The conventional method diagnosis (the use of microscope) can sometimes show limitation especially in the detection of parasites with very low intensity. Therefore, more sensitive methods like Enzyme Linked Immunosorbent Assay (ELISA) and parasite gene amplification using Polymerase Chain Reaction (PCR) are some of the modern technologies currently available. Nevertheless, these methods are under-used in Nigeria due to cost and lack of technological know-how.

Epidemiology and control – Epidemiology deals with the study of the cause of diseases and factors favouring transmission. In epidemiology, a good understanding of the transmission cycle is necessary. This will enable one to target control at stage(s) which will break the transmission cycle of the parasite. The killing of the snail intermediate hosts of Schistosomiasis will lead to a significant reduction in disease occurrence. In this case, the intramolluscan development which gives rise to the infective stage 'cercaria' will not occur. Epidemiology also offers knowledge on the behaviours that predispose people to infection.

Course Aim

This course aims at introducing the common parasites of man and livestock to students with a view to understanding their lifecycles, morphology, pathology, diagnosis, epidemiology and control.

Objectives

In addition to the aims above, this course set to achieve some objectives. After going through this course, you should be able to:

Get familiarised to some common parasites of man and animals
Know the striking morphological features of the parasites
Understand the transmission patterns of helminth parasite and transfer knowledge from the lifecycles to the control measures.
Diagnose some parasitic infections through their signs and symptoms of infection. e.g. bloody urine in a community that depends solely on natural flowing rivers for their water supply could be due to infection by *Schistosoma haematobium*

Working through the Course

A great effort was put into developing this course thereby enriching it with a lot of useful information. This accounts for why you would find it an irresistible companion both in this class and for field purposes. However, it requires that concerted effort is made in reading through this material for appreciating the effort in a commensurate manner so you would be required to devote some time to read it. You are also encouraged to work through and practice all assignments contained in this material.

Course Materials

You will be provided with the following materials

Course guide and study units. In addition, the course comes with a list of recommended textbooks which are necessary as supplements to the course material.

Study Units

The following table presents the study units contained in this course which is expected to be covered in 12 weeks:

Module 1

Unit 1   Association in organisms
Unit 2   Parasitic helminths, lifecycles, and the classification of the host organism

Textbooks and References

Brown, H.W. and Neva, F.N. (1983). Basic Clinical Parasitology (5th edition). pp1-17

Ukoli, F.M.A. (1990). Introduction of Parasitology in Tropical Africa. John Wiley and Sons Ltd., Chichester.

Hunter, G.W., Swartzwelder, J.C., Clyde, D.F (1976). A Manual of Tropical Medicine. 5th Ed. WB Saunders, Philadelphia.

Anuracpreeda, P., Wanichanon, C., Chawengkirtikul, R., Chaithirayanon, K. and Sobhon P. (2009). *Fasciola gigantica*: Immunodiagnosis of fasciolosis by detection of circulating 28.5kDa tegumental antigen. *Exp Parasitol.*, 123(4):334-40.

Echenique-Elizondo, M., Amondarain, J. and Liron de Robles, C. (2005). Fascioliasis: an exceptional cause of acute pancreatitis. *J.O.P.*6(1):36-9.

Massoud, A.A., Hussein, H.M., Reda, M.A., el-Wakil, H.S., Maher, K.M. and Mahmoud, F.S. (2000). Schistosoma mansoni egg specific antibodies and circulating antigens: assessment of their validity in immunodiagnosis of schistosomiasis. *J Egypt SocParasitol.*, 30(3):903-16

Parija SC. (2006). Protozoology and helminthology. In: *Textbook of Medical Parasitology: Textbook and Color Atlas*. 3rd ed. Chennai, India: AIPD, 237-80.

Ash, L. R. and Orihel, T.C. (1987). Parasites: a Guide to Laboratory Procedures and Identification, 1st ed. American Society of Clinical Pathologists Press, Chicago. 328 pp.

Baumeister, S., Pohlmeyer, K., Kuschfeldt, S., Stoye, M. (1997). Prevalence of *Echinococcus multilocularis* and other metacestodes and cestodes in the muskrat (*Ondatra zibethicus* LINK 1795) in Lower Saxony. Dtsch Tierarztl Wochenschr. 104(10):448-52.

Khalil, L., A. Jones, and R. A. Bray. eds. 1994. Keys to the Cestode Parasites of Vertebrates. CAB International, Wallingford, U.K. 751 pp.

Assessment

There are two components of assessment for this course. The Tutor Marked Assignment (TMA), and the end of course examination.

Tutor Marked Assignment

The TMA is the continuous assessment component of this course. It accounts for 30% of the total score. You will be given 4 TMA's to answer. Three of these must be answered before you are allowed to sit for the end of course examination. The TMA's would be given to you by your facilitator and returned after you have done the assignment.

Final Examination and Grading

This examination concludes the assessment for the course. It constitutes 70% of the whole course. You will be informed of the time for the examination. It may or not coincide with the university semester examination.

Unit	Title of Work	No. of Weeks to Complete Them	No. of Assignments
	Module 1		
1	Association in organisms	$^1/2$	2
2	Parasitic helminths, lifecycles, and the classification of the	$^1/4$	1
3	Human helminths infections	$^1/4$	2
	Module 2		
1	Digenetic Trematodes	1	2
2	Classification of digenetic tremaodes according to their habitat	**2**	4
	Module 3		
1	Basic body plan of a cestode	**1**	2
2	Tapeworms and Examples	**1**	2
3	Tapeworms of man and other human's cestodes	**2**	4
	Module 4		
1	General features and life cycles of nematodes	**¼**	1
2	Soil transmitted helminths	**1¼**	2
3	Blood and Tissue nematodes	**2¼**	5
4	Air-borne nematode	**¼**	1

How to Get the Most from this Course

In distance learning the study units replace the university lectures. This is one of the great advantages of distance learning: you can read and work through specially designed study materials at your own pace, and at a time and place that suits you best. Think of it as reading the lecture instead of listening to the lecturer. In the same way that a lecturer might give you some readings to do, the study units tell you when to read your set books or other materials. Just as a lecturer might give you an in-class exercise, your study units provide exercise for you to do at appropriate points.

Each of the study units follows a common format. The first item is introduction to the subject matter of the unit and how a particular unit is integrated with the other units and the course as a whole. Next is a set of learning objectives. These objectives let you know what you should be able to do by the time you have completed the unit. You should use these objectives to guide your study. When you have finished the unit you must go back and check whether you have achieved the objectives. If you make a habit of doing this you will significantly improve your chances of passing the course.

The main body of the unit guides you through the required reading from other sources. This will usually be either from your set books or reading section.

Self-tests are interspersed throughout the units, and answers are given at the ends of units. Working through these tests will help you come in the study units, work through these when you come to them, too.

When you need help, don't hesitate to call and ask your tutor to provide it.

1. Read the Course Guide thoroughly.

2. Organise a study schedule. Refer to the "Course Overview" for more details. Note the time you are expected to spend on each unit and how the assignments relate to the units. Important information details of your tutorials and the date of the first day of the semester is available at the National Open University, Study Centres.

   You need to gather together all this information in one place, such as your diary or a wall calendar. Whatever method you choose to use, you should decide on and write in your own dates for working on each unit.

3. Once you have created your own study schedule, do everything you can to stick to it. The major reason that students fail is that they get behind with their course work. If you get into difficulties with your schedule, please let your tutor know before it is late.

4. Turn to unit 1 and read the introduction and the objectives for the

unit.

5. Assemble the study materials. Information about what you need for a unit is given in the "Overview" at the beginning of each unit. You will almost always need both the study unit you are working on and one of your set books on your rest at the same time.

6. Work through the unit, the content of the unit itself has been arranged to provide a sequence for you to follow.

7. Keep in mind that you will learn a lot by doing the assignments carefully. They have been designed to help you meet the objectives of the course and, therefore will help pass the exam.

8. Review the objectives for each study unit to confirm that you have achieved them. If you feel unsure about any of the objectives, review the study material or consult your tutor.

9. When you are confident that you have achieved a unit's objectives, you can then start on the next unit. Proceed unit by unit through the course and try to pace your study so that you keep yourself on schedule.

10. When you have submitted an assignment to your tutor for marking, do not wait for its return before starting to work on the next unit. Keep to your schedule. When your assignments return, pay attention to your tutor's comments both on the tutor-marked assignment form and also written on the assignment. Consult your tutor as soon as possible if you have any question or problems.

11. After completing the last unit, review the course and prepare yourself for the final examination. Check that you have the unit objectives and the course objectives listed.

Facilitators/Tutors and Tutorials

There are some tutorials earmarked for this course. You will be notified later about the date, venue and time. You should try your best to attend the tutorials. This is the only chance to have face-to-face contact with your tutor. Prepare a question list before attending the tutorials. You will learn a lot from participating in discussions actively.

Summary

This course intends to provide you with some basic knowledge on the distribution, lifecycles, morphology, diagnosis and epidemiology of common helminths parasites. However, are expected to adequately answer the questions at the end of each unit of this study course.

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CONTENTS   **PAGE**

MODULE 1   EVOLUTION OF PARASITIC ASSOCIATION

Unit 1   Association in organisms

Unit 2   Parasitic helminths, lifecycles, and the classification of the host organism

Unit 3   Human helminths infections

MODULE 1

Unit 1   ASSOCIATION IN ORGANISMS

CONTENTS

1.0   Introduction
2.0   Objectives
3.0   Main contents
    3.1 Types of Association
        3.1.1   Symbiosis
        3.1.2   Mutualism
        3.1.3   Commensalism
        3.1.4   Parasitism
                3.1.4.1   Classification of the parasitic organisms
4.0   Conclusion
5.0   Summary
6.0   Tutor-Marked Assignment
7.0   References/Further Readings

1.0   INTRODUCTION

Organisms frequently associate together, often closely. There are a number of "motives" for these associations, including protection, nutrition, and as an aid to the dispersion (both geographically and temporally) of the organism. There are four main ways that animals of different species may be associated to one another; Symbiosis, Mutualism, Commensalism and Parasitism. These classifications however, on closer inspection, may become blurred, one type taking on the aspects of another, for example over time as the relationship evolves. However, as a general guide these terms are still very useful.

2.0   OBJECTIVES:

At the end of the study, students should be able to
1. Explain the various types of association existing between organisms
2. Give examples of various types of association

3.0   MAIN CONTENTS

3.1   Types of Association

3.1.1   Symbiosis

Here both organisms are dependent on each other. Examples being the association of flagellate protozoa in the gut of termites, where termites are dependent on the protozoa for breaking down their food stuffs, and the protozoa are dependent on the termites as host organisms. Another good example here which is often cited is the association between clown fish and anemones in tropical reefs; where the fish is dependent on anemone for protection and food while the anemone does not appear to gain anything from the association, except possibly cleaning. However, it has been observed that in some cases, in the absence of the fish partner the anemones tend to disappear from their reef home, indicating a true symbiotic rather than a mutualistic or commensal relationship.

Another well known example is found with the lichens, symbiotic association composed of fungi and algae. These associations may become very close, and it is thought that the eukaryotes as a group evolved as a result of such an association. Intracellular organelles such as the mitochondria and chloroplasts appear to have their origin as intracellular symbiotes of early eukaryotes, (some extremely primitive eukaryotes, such as the intestinal parasite *Giardia lamblia*, lack these organelles). Other forms of symbiosis may be much less close, for example an organism that uses another organism purely as a means of dispersal. For example bacterial or fungal spores on the legs of flies, or coelentrates and barnacles on the carapaces of marine crustaceans. This particular form of symbiosis is sometimes called Phoresis.

3.1.2 Mutualism

Here the associates may or may not be dependent on each other for their existence, but both benefit when they are associated. A good example of this occurs with the association of sea anemones on the backs of crabs. Both gain from the association

(the anemone providing some food for the crab, which in turn gives extra motility to the anemone), but both can survive on their own.

Another less well known example is found between certain species of ants and the caterpillars of some of the Lycaenidae butterflies (particularly the 'Blues'), where the caterpillar is protected by the ants within their nests, in return for which the caterpillar secretes a honeydew which the ants collect. In this case from the point of view of the ant, it benefits from the association, but does not appear to need it, (i.e. the association is facultative, or opportunistic). However, from the point of view of the caterpillar, this association is required for its survival (i.e. the association is obligatory). This illustrates that these definitions may become blurred, and, over time, one form of association may evolve into another.

3.1.3 Commensalism

Neither organism is dependent on the other for its existence, but in this case only one of the partners benefits from the association, the other being unaffected. An example of this, found in humans, are the non-pathogenic obligate commensal protozoa such as the amoebae *Entamoeba gingivalis*, commonly found in the mouth, feeding of bacteria, dead epithelial cells and food particles. Purely commensal relationships tend to be rather rare, as on a closer inspection element of mutualism or parasitism may become apparent.

3.1.4 Parasitism

Here one of the associates live either partly or wholly at the expense of the other associate, the other partner (the host organism) not gaining anything from the association. This association may give rise to extreme pathology in the host, or the parasitism may be generally not very pathogenic. Parasitism is carried out by many organisms, the main groups including viruses, bacteria, protozoa (these usually being endoparasitic), and various metazoan groups (multicellular eukaryotic animals), these being mostly groups of helminths (often endoparasitic), and arthropods (usually ectoparasitic), as well as some higher organisms, such as ectoparasitic lampreys and hagfish. Generally however, for partly historical reasons, the term parasitology generally only refers to the study of infection with eukaryotic protozoan, and invertebrate metazoan parasites, not bacteria, viruses or the higher chordate parasites, even though these are parasites in the true sense.

3.1.4.1   Classification of the parasitic organism

Organisms in these associations may either be on the outer surface of the host organism, (in which case the prefix **Ecto**- is used), or inside the host organism, (in which case the prefix **Endo**- is used). These prefixes may be used with any of the animal associations listed above. For example the flagellate protozoa in the termite guts are **Endosymbionts**, while the anemone can act as an **Ectocommensal** with the crab. Parasites may act as both **ecto**- and **endoparasites**. Parasites may also be classified according to the closeness of the relationship. For example **Facultative Parasites** (such as many bacteria) are those where the parasitic lifestyle is only taken up opportunistically, whereas **Obligate Parasites** (such as all viruses, and most of the helminth parasites described below) are those in which the organism must parasitise another organism. These parasites may often cause diseases, in which case they are referred to as **Pathogenic Parasites**.

In a somewhat wider interpretation of the term parasitism, some organisms exhibit parasitic behaviour only early in their life cycle, these being referred to as **Brood Parasites**. Examples of these include caterpillars of the Large Blue butterfly, which chemically mimic other caterpillars with mutualistic associations with ants (see above), but both fail to produce honeydew as compensation and consume ant grubs, and may in fact destroy the nest, (thereby acting as a pathogenic parasite for the ants). In this case, the parasitic lifestyle probably evolved from the mutualistic lifestyle of the other, related butterflies, again illustrating how one form of association may change into another. Another well known example of a brood parasite is a bird, the cuckoo.

Some parasites establish themselves in hosts in which they do not ordinarily live. These are called the **Incidental Parasites. A temporary parasite** is free-living during part of its existence and seeks its host intermittently to obtain its nourishment whereas **Permanent Parasite** remains on or in its host's body from early life until maturity, sometimes for its entire life. Parasite that has passed through the alimentary tract without infecting the host are called **Coprozoic or Spurious Parasite.**

Parasites often lack the necessary organs for assimilation of raw materials and depend upon the host for predigested food. An adequate supply of moisture is assured inside the host, but during the free-living existence of the parasite,

inadequate moisture may either prove fatal or prevent the larval development. Temperature is likewise important. Each species has an optimal temperature range for its existence and development. Both high and low temperatures are detrimental and even lethal.

4.0 CONCLUSION

In this unit you learnt:
the various types of association in organisms with focus on parasitism.

5.0 SUMMARY

Organisms interact with each other at different degrees. While some are solely dependent on each other, however, some are opportunistic and can adopt different means for their survival. One association can evolve into the other. An example is seen in commensalism which can evolve into mutualism or parasitism. Some parasites recide within their hosts while others are outsides. Obligate parasites live in living tissues whereas the facultative parasites are more successful in that they are capable of taking other sources of nutrition. Pathogenic parasites are of great medical concern because they cause diseases. The survival of a parasite in its habitat/microhabitat can be influenced by moisture and temperature.

6.0 TUTOR-MARKED ASSIGNMENT

1. What is/are the differences between symbiosis and mutualisms?
2. Give one example in each case of (a) Commensalism, (b) symbiosis (c) mutualism
3. Give examples in each case of obligate and falcutative parasites

7.0 REFERENCE/FURTHER READINGS

Brown, H.W. and Neva, F.A. (1983). Basic Clinical Parasitology (5[th]edition). pp1-17

**UNIT 2   PARASITIC HELMINTHS, LIFECYCLES, AND THE
CLASSIFICATION OF THE HOST ORGANISM**

CONTENTS

1.0   INTRODUCTION

Parasitic helminths may have either simple or complicated lifecycles. The terms used to describe the hosts harbouring different stages in these lifecycles are however the same. The degree of damage done to the hosts is however varied. For example in definitive host, the greatest harm is seen being the one the adult stage of the parasite is found. Sometimes, a host might assume dual functions, and therefore could be difficult to classify strictly into one type. Human host during infection by malarial parasite is one of such. Human could be classified as the definitive host being the one in which greatest harm is seen. Also, intermediate host because human harbours the asexual stages of the parasite (merozoites and trophozoites). A clear understanding of the relationship between host and parasite and function of host in survival and transmission of parasite is therefore necessary for a better classification.

2.0 OBJECTIVES:

At the end of this study, the students should be able to
1. Explain the different types of parasites' host
2. To distinguish between an intermediate host and a vector

3.1 Types of Host

3.1.1 Definitive Host

The adult parasites are found in the **definitive host**. This is where the parasite's sexual cycle usually takes place, with either cross or self fertilisation with hermaphroditic parasites, or sexual reproduction if the parasites have separate sexes, followed by production of eggs, or more rarely with viviparous helminths, larvae. The greatest harm is usually seen in this host.

3.1.2 Intermediate Host

In many cases the parasites larvae are found in different hosts, these are called the **Intermediate Hosts**. Parasitic helminth larvae may have one, two or more intermediate hosts in their lifecycles, or they may have no intermediate hosts. Often asexual stages of reproduction occur in these intermediate hosts, (for example with Platyhelminth parasites). Note that when describing hosts of parasitic protozoa, these terms are slightly different owing to the asexual characteristics of many of these organisms. With parasitic protozoa the vertebrate host is generally referred to as the definitive host, whilst the invertebrate is the intermediate host. Some parasitic nematodes (e.g. *Strongyloides stercoralis*) are Facultative parasites, having completely free living lifecycles in addition to parasitic ones. The two terms definitive and intermediate host are the most important in Parasitology when referring to the type of host. A **vector** however, should not be mistaken for intermediate host. A **vector** actively transmits infection to a host without necessarily harbouring the asexual stage of the parasite e.g. the vector of African trypanosomiasis *Glossina* spp. These groups of vectors pick up parasite (the infective stage) from the reservoir hosts during blood meal and transmit it to a susceptible definitive host. However, some vectors can still serve as intermediate hosts harbouring the asexual or the larval forms of the parasite e.g. certain *Anopheles* mosquitoes that harbour the microfilariae of filarial worms.

3.1.3 Accidental Host

Accidental hosts are those in which the parasite do not normally develop (due, for example to lack of exposure to infective forms of the parasite), but when occasionally chance infections occur, the parasite is able to complete its lifecycle. Hosts where the parasite can complete its lifecycle are called **Permissive hosts**, and include true definitive and intermediate hosts as well as many accidental hosts. Examples here include such parasites as *Fasciola hepatica*, where the normal definitive hosts are ruminants, but humans and other animals may also be infected and viable adult parasites develop. Another example is human infection with the nematode *Angiostrongylus cantonensis* in the far East.

In comparison another form of the accidental host is the **Non-Permissive host** where the parasite, although it may develop to some extent, reaches effectively a dead end, the parasite not being able to complete its lifecycle and eventually dying within the host. These forms of infection often occur where the parasite has intermediate hosts which may be accidentally ingested by animals other than the true definitive host. For example, with various marine ascarids of the family Anisakidae such as *Anisakis* sp., which give rise to the condition of 'Anisakiasis' on ingestion of raw infected fish.

3.1.4 Paratenic Host

Paratenic hosts may also be included in parasitic helminth lifecycles. In these forms of infection the parasites undergo an arrested development on infection, larval forms accumulating in these hosts until they have a chance of infecting the definitive host (e.g. in the Pseudophyllidean tapeworms). These hosts are therefore not essential to completion of the parasites lifecycle. This is in contrast to the case with true intermediate hosts whose ingestion is essential to the lifecycle, for example *Echinococcus* sp.

3.1.5 Reservoir Host

These are accidental hosts and hosts of parasites which have zoonotic patterns of infection (i.e. normally infect a wide range of hosts), may act as **Reservoir Hosts** for the parasite. These are also a form of permissive hosts as fully viable infections develop, and a more accurate term would be alternative definitive hosts (though this is not in fact used). The term reservoir host is usually only used when describing the epidemiology of human infections. An example of parasites with zoonotic infections is *Schistosoma japonicum*. This parasite, as well as infected man, can also infect other mammals as definitive hosts, including rodents, cats, dogs, domesticated ruminants such as water buffalo and a wide range of other mammals. In Human African Trypanosomiasis (HAT), the reservoir host are cattle which serve as **sources of active infection** to man. The presence of these **Zoonoses** has implications for the control of the parasite in the field.

4.0 CONCLUSION

In this unit you learnt:
the various types of hosts of parasites, their distinctive characteristics and some relevant examples.

5.0 SUMMARY

The host of a parasite is the organism that harbours the parasite. The intermediate host is the one in which part of the developmental stages in the life cycle of the parasite takes place, usually the larval or the asexual stage. A vector actively transmits an infective stage of parasite harboured by the reservoir host which serves as source of active infection to the definitive host. The definitive host harbours the adult or the sexual stage of the parasite and often experiences the greatest harm during the life cycle of the parasite.

6.0 TUTOR-MARKED ASSIGNMENT

Briefly explain the following types of hosts; reservoir, accidental, definitive and intermediate hosts

7.0 REFERENCE/FURTHER READINGS

Brown, H.W. and Neva, F.A. (1983). Basic Clinical Parasitology (5[th]edition). pp1-17

UNIT 3　　HUMAN HELMINTH INFECTIONS

CONTENTS

1.0　Introduction
2.0　Objectives
3.0　Main contents
　　　3.1　　Nematode infections
　　　3.2　　Digenean trematode infections
　　　3.3　　Cestode (tapeworm) infections
4.0　Conclusion
5.0　Summary
6.0　Tutor-Marked Assignment
7.0　References/Further Readings

1.0　　INTRODUCTION

This unit aims to introduce the student to the diversity of helminth infections in man, and even more importantly, to the numbers of individuals that harbour these infections in all regions of the world. There are three major groups of helminths containing members that have man as their main hosts, these being the **Digenean Flukes**, the **Tapeworms (Cestodes)**, and the **Roundworms (Nematodes)**.

2.0　　OBJECTIVES

At the end of this unit, the student should be able to:

1.　　Get acquainted with the various helminth parasites and their common names
2.　　Give brief information about the distribution of their infections.

3.0   MAIN CONTENTS

3.1   NEMATODE INFECTIONS

Enterobius vermicularis **-** Pinworm, Threadworm.

E. vermicularis infection, an extremely common nematode infection, particularly in temperate areas such as Western Europe and North America, (being relatively rare in the tropics) and particularly in children. It has been estimated that the annual incidence of infection is over 200 million, this probably being a conservative figure.Samples of caucasian children in the U.S.A. and Canada have shown incidence of infection of from 30% to 80%, with similar levels in Europe.

Ascaris lumbricoides - The Large Human Roundworm.

Again the incidence rates for this parasite are very high with > 1500 million cases of infection annualy, of which about 210 million cases are symptomatic.

Trichuris trichiuria - The Large Human Roundworm.

The incidence rates for this parasite are also very high, with estimates of about 1300 million cases of infection annualy, of which >133 million cases are symptomatic.

The Hookworms.

These are represented by two parasites, *Necator americanus* in the tropics and sub tropics worldwide and the S. E. states of the U.S.A., and *Ancylostoma duodenale*, again with a worldwide distribution in the tropics and sub tropics as well as the Mediterranean region. There are > 1200 million cases of hookworm infection annually, of which about 100 million cases are symptomatic.

Lymphatic filariasis - Elephantiasis

18

This disease is caused principally by two parasites, *Wuchereria bancrofti* with an annual rate of infection of about 106 million cases, and *Brugia malayi* with an annual rate of infection of about 12.5 million. The total number of people infected with other types of lymphatic filarial worms is much smaller, at about 1.5 million cases. These lymphatic filarial worms, along with the related filarial parasite *Onchocerca volvulus*, are unusual among the nematodes in that they develop with, and are transmitted by insect vector intermediate hosts.

Onchocerca volvulus - River Blindness

The incidence rates for this parasite are not as high as some of the previously described parasites, with an annual rate of infection of about 18 million, however, due to the extreme pathology associated with this parasite, often with all adult members of affected villages losing their sight, along with severe skin conditions, the infection is significant.

Dracunculus medinensis - Guinea Worm

The incidence rates for this parasite are much lower, with an estimated annual rate of infection of about 100 000. This is much lower than in the recent past, when up to 50 million people were infected. This reduction in incidence illustrates how successful helminth control programmes can be effective in reducing the disease caused by these organisms.

Other important nematode infections include; **Trichinella spiralis**, **Strongyloides stercoralis**, and a number of more **rare infections**. Nematodes that normally infect other animals may still cause disease in man. These include **Toxocara canis** and a number of nematodes causing **anisakiasis**.

3.2   Digenean Trematode Infections

Schistosomiasis - Bilharzia.

Schistosomiasis is caused by *Schistosoma mansoni, S. haematobium, S. intercalatum, S. japonicum* and *S. mekongi*. This disease is the most important human helminthiasis in terms of morbidity and mortality. The numbers of people infected are lower than those of many of the nematode infections, with an estimated annual incidence of infection of > 200 million cases. In terms of active

disease however, the parasite is much more important, with an estimated annual mortality rate of about 1 million deaths directly due to infection with these parasites.

Opisthorchis sinensis - The Chinese Liver Fluke

This is also a very important trematode infection, with an estimated annual incidence of infection of about 20 - 30 million cases, mostly in the Far East, in Japan, China, Taiwan and South East Asia.

Paragonimus spp. - The Lung Fluke

This fluke causes a pulmonary disease, the adult parasites living in the lungs of their definitive hosts (e.g. man). There are a number of different species of this parasite, the most well documented being *P. westermani* in the Far East. It may however, be locally very common, with up to 40 to 50% of the population infected.

There are a number of other digenean trematode infections. These include various **Echinostome** infections as well as a number of other flukes. In addition, there are a number of these parasites that usually infect domesticated animals, but also cause well known human infections as well. These include ***Fasciola hepatica*** and ***Dicrocoelium dendriticum***.

3.3   Cestode (Tapeworm) Infections

Taenia saginata - The Beef Tapeworm

This only causes very limited pathology in man, but the annual incidence of infection is high, at an estimated 50 million cases.

Taenia solium - The Pork Tapeworm

This has a similar estimated annual incidence of infection of about 50 million cases. However, in this case the consequences may be more severe, due to the added risk of contracting infection with the larval metacestode, (cysticercosis).

This may have extreme consequences in terms of the pathology associated with infection, with an estimated annual mortality rate of about 50,000 deaths.

For the cestodes, these annual incidence rates are based on detection of infection with the adult parasite. This is achieved by examination of faeces, urine or sputum for **parasite eggs**. Diagnosis of infection with larval metacestode parasites, such as *Echinococcus* sp. is very difficult, due to the lack of non invasive diagnostic techniques. It is in consequence very difficult to estimate annual rates of infection, even though these metacestodes may be very important pathogens.

4.0   CONCLUSION

In this unit you learnt about:

The various parasitic helminths, their common names, distribution and number of cases (prevalence).

5.0   SUMMARY

Parasitic helminths are grouped into nematodes, cestodes and trematodes. The nematodes are the most diversified groups. Parasitic helminths infect a wide range of hosts, ranging between man, domestic animals and wild animals. Morbidity is often high leading to death of several thousands of people.

6.0   TUTOR-MARKED ASSIGNMENT

1. Give 3 examples in each case of parasitic human nematodes and cestodes.
2. Mention the 3 types of fluke with 2 examples of each species.

7.0   REFERENCE/FURTHER READINGS

Ukoli, F.M.A. (1990). Introduction of Parasitology in Tropical Africa. John Wiley and Sons Ltd., Chichester.

MODULE 2   TREMATODES

Unit 1   Digenetic Trematodes

Unit 2   Classification of digenetic trematodes according to their habitat

Unit 1   DIGENETIC TREMATODES

1.0   INTRODUCTION

The phylum Platyhelminthes comprises six classes which include the free living forms and those that are of zoological, medical and economic importance. The medically important groups are the trematodes and cestodes. Trematode also

called flukes cause various clinical infections in humans which occur worldwide. The parasites are so named because of their conspicuous suckers, the organs of attachment (*trematos* means "pierced with holes"). All the flukes that cause infections in humans belong to the group of digenetic trematodes.

The class Trematoda comprises 3 subclasses

Subclass 1 - Aspidogastrea
   They have large ventral adhesive organ subdivided by longitudinal and transverse septa into sucking discs. They are parasites of turtles, fishes and molluscs
Subclass 2 - Didymozoidea
   These are tissue-dwelling parasites of fish. They are greatly elongated, dioecious, with sexual dimorphism. No complete life cycle is known
Subclass 3 - Digenea
   This contains parasites of medical and economic importance to man and therefore will be dealt with more extensively.

2.0   OBJECTIVES

At the end of this unit, students should be able to
1.   Identify the striking features of digenetic trematodes
2.   Describe the morphology of the egg, larva, and adult stage of various digenetic trematodes
3.   Understand the general transmission patterns of some trematodes

3.0   MAIN CONTENTS

3.1   The Adult Digenean Fluke

The basic body form of the adult trematode takes a number of different forms, some of which are illustrated below;
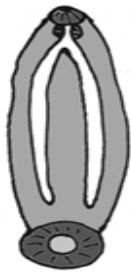
| Amphistome	Distome	Echinostome	Monostome
These have large fleshy bodies, with a prominent sucker at the posterior of the body (e.g. *Gastrodiscoides hominis*)	These are the most common type, with the mouth surrounded by the oral sucker and a ventral sucker, present anywhere on the ventral surface except the extreme posterior (e.g. *Fasciola hepatica*)	Similar to the distomes, except that the oral sucker is surrounded by a prominent collar, equiped with spines (e.g. *Echinostoma* sp.)	In these there is either only one sucker present (usually only the oral sucker), or there are two suckers, but one very reduced, or in some cases no suckers) (e.g. *Notocotylus attenuatus*)

Schistosome

Elongate trematodes, with separate sexes, the male generally larger, holding the female within a groove formed by a folding of the male body (the gynaecophoric canal). Found within the circulatory system. (e.g.*Schistosoma mansoni*)

There are other forms as well, for example the 'Holostome' type, where the body of the trematode is divided into two distinct regions, the anterior of which may hold an additional adhesive organ, (e.g. *Diplostomum* sp.), and the 'Gasterostome', where the gut is a very simple, sac like, structure, attached to a mouth situated near the centre of the body (reminiscent of the arrangement of some of the free living platyhelminthes).

3.1.1   The Basic Lifecycle of the Major Groups of the Digeneans

Most digeneans are hermaphroditic (the major exception being the schistosomes, and one other group). In the majority of these parasites self fertilization may occur, but cross fertilization between different individuals is more generally the rule. The sperm enter the female system, either via the Laurers canal or more commonly through the common genital atrium, which opens into the uterus.
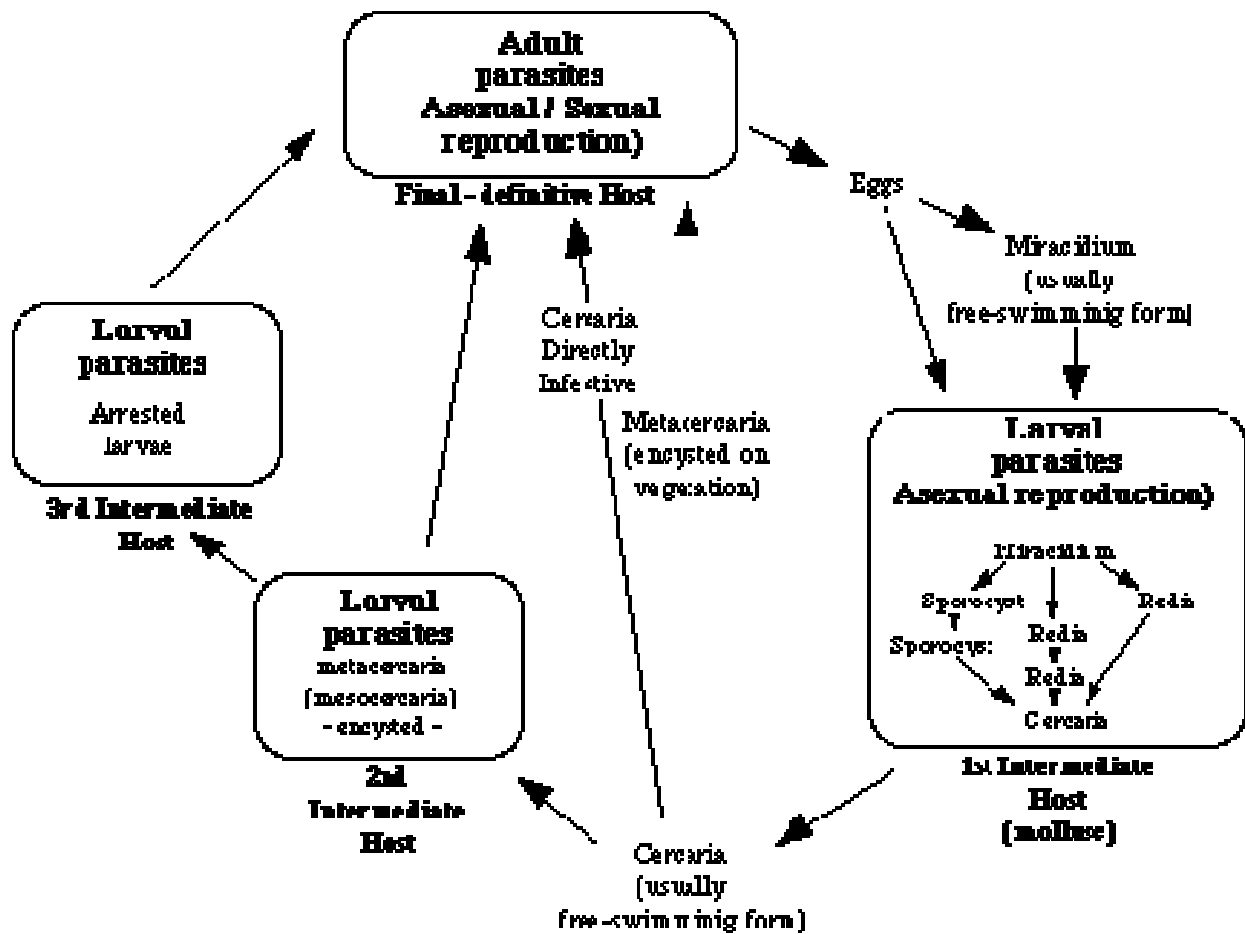
The generalised life cycle of digenetic trematodes

3.2   The Digenean Trematode Egg

The formation of the digenean egg follows that described for the platyhelminthes as a group. Briefly, as the egg enters the öotype of the fluke it becomes surrounded by a predetermined number of vitelline cells, the number of which will be specific for different parasites, which form the food reserve of the egg. These vitelline cells produce globules of a mixture of proteins and phenols, which are extruded to the outer surface of the developing egg. Here the phenols oxidise to form quinone, which then coalesces with the protein, reacting to form scleratin, a hard inert yellowish substance, making up the egg shell. As the eggs of different species may vary in thickness, their colours may vary from yellow, to a dark brown.The digenean egg is usually operculate, in common with other platyhelminthes. Exceptions to this may occur however, the most important being with the

schistosomes. Here the eggs are non-operculate, and are ornamented with spines, the appearance of which are characteristic for different species of schistosome.
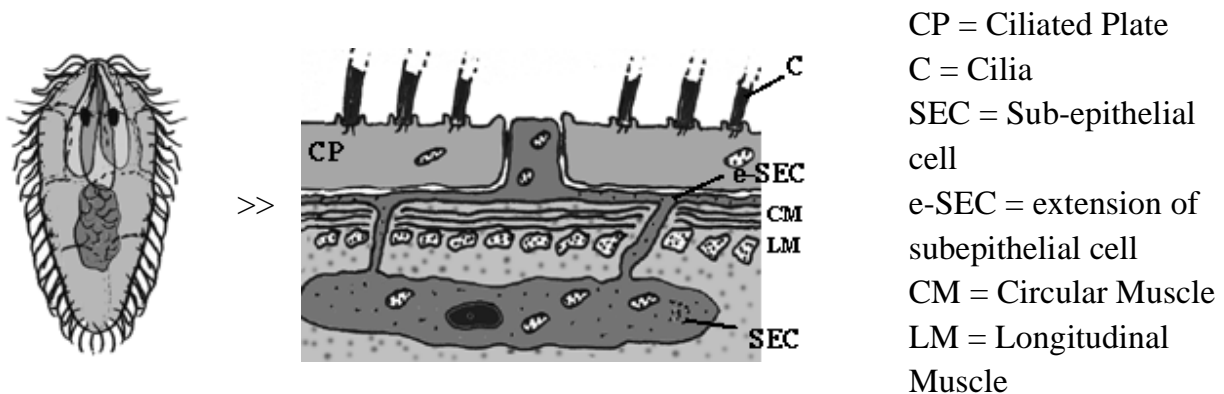

The eggs hatch of operculate eggs involves the release of the opercular cap. This takes place under a variety of conditions, modified according to the particular species of trematode. For example some trematode lifecycles involve the ingestion of the egg before hatching (e.g. *Dicrocoelium dendriticum*, the lancet fluke), whilst others such as those of *Fasciola hepatica*, (the liver fluke), hatch in water. For the eggs that hatch in the external environment, a number of factors may be important, for example light, temperature and changes in osmotic pressure. Again the exact details of these environmental requirements will be optimised for the particular conditions which will maximise the chances of completion of the parasite lifecycle. In all cases the egg hatches to release the miracidium.

3.3   The Larval Digeneans

3.3.1   The miracidium

The miracidium is the name of the ciliated larval stage that is hatched from the digenean egg. In comparison with the other larval platyhelminthes, it is very similar to the larvae of the monogeneans, (the oncomiracidium) and the larval cestodarian, or lycophore. In most cases the miracidium is usually a free swimming stage,that seeks out the primary, and in some cases only, intermediate hosts of these parasites. In all cases these primary, or 1st intermediate hosts are molluscs. In the few examples where the miracidium is not a free swimming stage the eggs are ingested, as with the lancet fluke *Dicrocoelium dendriticum*. Here the eggs hatch in the intestine of the mollusc liberating the miracidium, from where it immediately penetrates the intestinal wall to invade the molluscan tissues. In the free swimming miracidia the larval parasite exhibits distinct behavioural responses that enable it to enter the environment of or detect the presence of its hosts. These behavioural responses have principally been studied in the case of the schistosome miracidium. Morphologically, the surface of the miracidium is covered with a

series of ciliated plates, which may be clearly seen using electron microscopy after the removal of cilia. These ciliated epidermal plates (in some species the cilia being replaced by spines) are discontinuous, not being in contact with each other but being separated by extensions of the underlying subepidermal layer. The whole structure is being illustrated below.


CP = Ciliated Plate
C = Cilia
SEC = Sub-epithelial cell
e-SEC = extension of subepithelial cell
CM = Circular Muscle
LM = Longitudinal Muscle

The plates themselves show a definite arrangement, being placed in four to five transverse rows, the exact arrangement of which may vary between different trematodes. Beneath the plates are layers of muscle fibres. At the anterior end of the larvae is a non-ciliated conical projection, the terebratorium, (or anterior papillae), bearing apertures of the apical and penetration glands. These themselves are found at the anterior end of the body. Miracidia possess a number of sensory organs, the most important of which are the dorsaly situated eye spots, beneath

which is found the cerebral mass. Other sensory organs are situated within folds of the terebratorium. Below all of the structures is found the miracidium's large rounded germinal cells, which are often grouped in clusters called germ balls. Finally the miracidia possess a protonephridial excretory system, basically similar to that found in the adult parasites. On examination of eggs containing mature miracidia, it is clearly seen that flame cell activity is the first sign of the initiation of hatching of the egg. On invasion of the molluscan tissue the miracidium sheds its ciliated plates, in almost all cases rapidly transforming into an endoparasitic form, the sporocyst, although in a few unusual groups the miracidium may contain a fully developed redia.

3.3.2   The sporocyst

The sporocyst develops within the molluscan host as a hollow fluid filled germinal sac, into which protrude germinal masses. At the conical anterior of the sporocyst body a birth pore is located, from which subsequent generations of larvae emerge. The germinal masses develop internally into either daughter sporocysts, which are essentially the same as their parent sporocysts, or into a second larval stage, the redia.
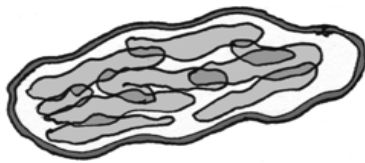
The sporocyst

3.3.3   The redia

The redia are the second larval form to develop within the molluscan host (but may be absent in some groups, such as the schistosomes). They are similar to sporocysts, containing germinal masses within a fluid filled sac, which may develop into either second generation daughter redia, or more commonly into the final larval stage within the mollusc, the cercaria.

They differ from the sporocysts however, in that they are a much more active form, and importantly they possess simple gut. The tissue they feed on is predominantly molluscan in origin, but the redia of some groups (e.g. those of the echinostomes) may actively seek out the developmental stages of other trematodes (e.g. schistosome sporocysts) within the same intermediate.

The gut itself consists of a mouth, opening into a large muscular pharynx, which in turn opens into a simple rhabdocoel like intestine. Externally, behind the mouth

many redia have a ridge-like collar, below which the birth canal opens and from which either cercariae or daughter redia emerge. Further along the body are lobe-like extensions of the body, which are thought to aid the movement of the parasite within its host's tissues.

An interesting exception to the general rule that cercaria are produced by the redia is found in a few tremadodes where the redia produce progenetic metacercaria, fully capable of producing viable eggs. In these few very unusual cases, the trematode may only have a single molluscan host, although the metacercaria may still be capable of developing in a second host as well. Exceptions such as these, and those described above involving miracidia containing fully developed redia is evidence of the evolutionary past of these organisms. It has been noted that the redia bears some resemblance to some of the more advanced turbellarians, and as described above, this stage is a very active form of the parasite, fully capable of actively ingesting host material, and in some cases even predation of competing parasites within their hosts. It has been postulated that the group as a whole emerged from an ancestral parasitic turbellarian, with a single molluscan host, after the development of internal division and asexual reproduction, later developing specialised forms to exploit the varying environments that these organisms have to cope with.

3.3.4 The cercaria

Some of the types of Cercariae
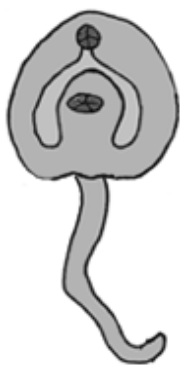
Gymnocephalus
(e.g. *Fasciola* sp.)
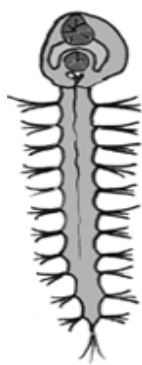
Trichocercous
(e.g. *Donax* sp.)

Furcocercous
(e.g. *Schistosoma* sp.)

Microcercous
(e.g. *Bithynia* sp.)

In almost all species of trematode, it is the cercarial stage that emerges from the mollusc, and is the infective form for the vertebrate host, although there may be exceptions to this general rule. For example in some cases a sporocyst, modified to have a thickened internal wall resistant to the environment, emerges, to be ingested by a second intermediate host, (e.g. as is the case in the trematode *Dicrocoeloides petiolatum*). Other exceptions, involving redia producing progenetic metacercaria, have already been described above.

The trematode cercaria exhibits considerable variations in structure, which is very important taxonomically, and reflects in many cases adaptations to the specific lifecycle of the parasite involved. Because of this great diversity of form, a system of cercarial classification has evolved, based on the gross morphology of these larval forms. Firstly cercariae may be divided into three major groups;

i) **Monostome Cercariae** - These lack a ventral sucker, and have simple tails. These forms develop within rediae

ii) **Amphistome Cercariae** - In these the large ventral sucker is situated at the base of a slender unbranched tail. These forms develop within rediae.

iii) **Distome Cercariae** - This is the commonest cercarial form, with the ventral sucker
lying some distance from the posterior end, in roughly the anterior third of the body. These distome cercariae may themselves be divided into a large number of subgroups, based on other morphological features, particularly the form that the cercarial tail takes. Some of these forms are described below;

a) *Leptocercous Cercariae* - These cercariae have straight slender tails, which are much narrower than the cercarial body. This form is further subdivided into;
i) *Gymnocephalous Cercariae* - In these, the suckers are equal in size. This is a common form, represented within such species as *Fasciola hepatica*, and develop within rediae
ii) *Xiphidiocercariae* - These are similar to the gymnocephalous forms, but in these the oral sucker is equiped with a stylet, used in penetration of their next hosts, and they generally develop within sporocysts. iii) *Echinostome Cercariae* - In these there is a ring of spines at the anterior end of the larvae, as in adult forms of these parasites. These are found within trematodes of the genus *Echinostoma*, and develop within rediae.

b) *Trichocercous Cercariae* - These forms have long tails, equiped with rings of fine bristles. They are usually found in marine trematodes.

c) *Cystocercous Cercariae* - In these the end of the tail is highly enlarged, with a cavity into which the larval body may be retracted. These usually develop within sporocysts.

d) *Microcercous Cercariae* - Cercaria with vestigial tails, and which may develop within both rediae and sporocysts.

e) *Cercariaea Cercariae* - Cercaria with no tails, where the cercaria is not a free swimming form, and may develop within both rediae and sporocysts.

f) *Furcocercous Cercariae* - In these the tails are forked at the end. The cercaria of the most important group of trematodes, the schistosomes, have cercariae of this form. This form develops within sporocysts.

Otherwise, both externally and internally the structure of the body of the cercaria resembles that of the adult trematode into which they grow. For example, the ring of spines found at the anterior end of echinostome cercariae is also present in the adult flukes.

The outer surface of the cercaria is a tegument, which may however differ from that found in the adult form in a number of ways. For example in the schistosomes the tegument is covered with a trilaminate plasma membrane, (as opposed to the two bi-lipid membranes found in the adult), on the outer surface of which there is a glycocalyx, (absent in the adult). However, many other features of this tegument appear similar to that of the adult, the differences almost certainly being adaptations due to the differing environments that these two lifecycle stages experience. For example, spines found on the surface of both forms of tegument, and the overall structures of a syncytium conected to subtegumental cells are the same. Within the cercarial body a number of different types of gland cells may be found, including cystogenous gland cells, used by the larvae to secrete a cyst wall during the formation of the metacercarial stage, and penetration gland cells, used by the cercaria to penetrate its next host, either a second intermediate host, or in some groups the definitive host, (such as the schistosomes), where the cercaria is the final larval stage.

The cercariae released from their molluscan intermediate host are usually a free swimming form. These must then locate either their next, and usually final intermediate host, their definitive host which they actively penetrate (e.g. in

members of the family Schistosomatidae), or locate a suitable solid substrate to encyst upon, or be ingested by their definitive host (members of the family Azygiidae).

To locate these various targets the cercariae are equipped with a variety of sensory organs. These commonly include two or more eye spots, as well as touch receptors, and allow specialised cercarial behaviour, designed to bring the cercariae into an environment giving the maximum probability of infecting their next hosts. For example, the cercariae of the schistosomes exhibit negative phototrophy (swimming to the surface of the water), and positive thermotrophy and thigmotrophy, being attracted to warm objects moving in the water. As well as these behavioural responses within the free swimming cercariae, the parasite exhibits definite circadian rhythms in terms of shedding from the molluscan host, again being shed at times optimal for bringing them into contact with their next host. For example, the schistosome cercariae are generally shed during daylight, in the morning, whilst those of other species emerge only at night.

In a few groups, such as *Alaria* spp. However, the parasite employs three intermediate hosts. In these cases the cercaria penetrates the second intermediate host to form a resting stage, the mesocercaria described below. In these cases this second intermediate host is in turn ingested by a third intermediate host, where it encysts to form a metacercaria.

3.3.5 The mesocercaria

The mesocercaria is essentially a resting stage within the parasitic life cycle, employing a second intermediate host in a parasite lifecycle utilising four hosts.

The mesocercaria is a definite prolonged stage in the adult generation of strigeate trematodes, which closely resembles the cercarial body, from which it develops in the second intermediate host, and which does not possess metacercarial features; it develops in turn into the metacercaria in another host.

In parasites having this larval stage the mesocercaria are capable of infecting and surviving within a very wide range of paratenic hosts which may ingest the second intermediate host, thus in effect increasing the number of hosts which the parasite may use in its lifecycle. For example, amphibians infected with mesocercaria of

Alaria may themselves infect a wide variety of other amphibians, reptiles, birds and mammals if they are ingested by these animals.

3.3.6   The metacercaria

This is a much more common "resting" larval stage of the trematode parasitic lifecycle, formed either in a final intermediate host (when a mesocercaria, or more commonly a cercaria enters its body), or on a solid substrate in the external environment. The final intermediate host may be a fish (e.g. *Opisthorchis sinensis*), an arthropod (e.g. *Dicrocoelium dendtriticum*, employing an ant second intermediate host, and *Paragonimus westermani* employing a crustacean), or another mollusc, as with some of the echinostomes. As stated above, some trematodes however do not have second intermediate hosts, but either encyst as metacercariae on solid substrate's, such as aquatic vegetation or on shells of aquatic organisms, which will in turn be ingested by the parasites definitive host, or in some groups such as the schistosomes, as already described, the cercariae directly penetrate the skin of, and infect, the parasites definitive host. Although generally the metacercariae are inactive encysted forms, the metacercaria of some species do remain free and action. In most other metacercariae however, encystment does occur. The structure of the cyst wall itself varies considerably, though generally it is a complex mixture of tanned proteins, lipids and polysaccharides. Within the cyst wall the morphology of the larva usually closely resembles that of the cercarial body, although as described above, in some groups sexual maturation may occur either fully or partially. To continue further the metacercaria must be ingested, either along with the body of the intermediate host it inhabits by a carnivorous definitive host, or along with the vegetation it has encysted on by a herbivorous or omnivorous host.

3.3.7   The Larval Digeneans - the Juvenile Adult Stages

On ingestion the metacercaria (or cercaria) must transform into the adult form. The precise details of this process will vary considerably, depending on how the definitive host was infected. For example, in some species the adult flukes are found within the alimentary tract. In these cases the metacercarial cyst wall is broken down to release what is essentially a young fluke, which only has to migrate a short distance to reach their preferred site within the hosts body. In other groups however the adult forms are located in other sites within the body. In these

cases the liberated young fluke must penetrate the gut wall, or in the case of the schistosomes penetrate the hosts skin. Then they must undergo a migration through the hosts body. This is usually via the circulatory system, but again the precise details of the migratory path will vary considerably.

3.4 Features of digenetic trematodes

- Digenetic trematodes are unsegmented leaf-shaped worms that are flattened dorsoventrally.
- They bear 2 suckers, one surrounding the mouth (oral sucker) and another on the ventral surface of the body (ventral sucker). These serve as the organs of attachment.
- The sexes of the parasites are not separate (monoecious). An exception is schistosomes, which are diecious (unisexual).
- The alimentary canal is incomplete, and no anus is present.
- The excretory system is bilaterally symmetrical. It consists of flame cells and collecting tubes. These flame cells provide the basis for the identification of the species.
- The reproductive system consists of male and female reproductive organs and is complete in each fluke.
- The flukes are oviparous. They lay operculated eggs. An exception is schistosome eggs, which are not operculated.
- All have complicated life cycles, with alternating asexual and sexual developments in different hosts.

A.   B.
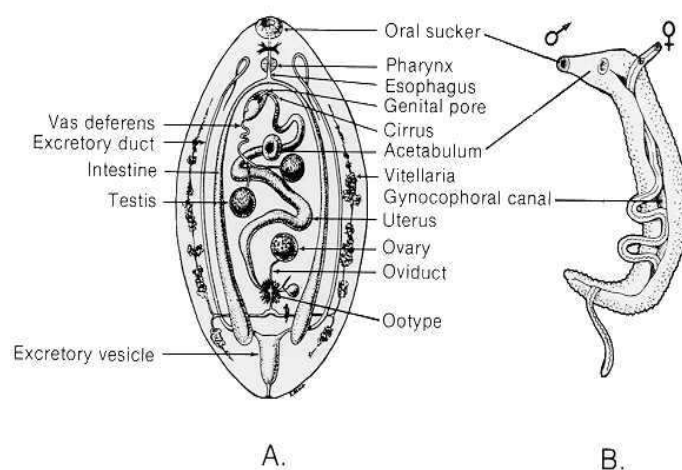
Structure of digenetic flukes. (A) Hermaphroditic fluke. (B) Bisexual fluke.

4.0   CONCLUSION

At the end of the unit you are expected to; know the various classes of the phylum Platylhelminthes and give examples of those of zoological and medical importance, know the various stages in the life cycles of digenetic trematodes and their striking morphological features.

5.0   SUMMARY

The medically important trematodes are the digineans.   The digenea are unsegmented leaf-shaped worms that are flattened dorsoventrally with two suckers (the oral sucker and ventral sucker). The digeneans have heteroxenous life cycles having one or more intermediate hosts. The adult worms lay eggs within the definitive host which hatch miracidia in water medium. Miracidia develop within the snail intermediate hosts of particular species. The life cycles continue following a specific pattern depending on the parasites' species giving rise to other larval stages like sporocysts, rediae, cercariae and metacercariae.

6.0   TUTOR-MARKED ASSIGNMENT

1. Describe the general life cycle of digenetic trematodes
2. Highlight the differences between gymnocephalus and furcocercus cercariae
3. Make a well labeled diagram of generalized morphological features of digenetic trematodes

7.0   REFERENCES/FURTHER READINGS

Hunter, G.W., Swartzwelder, J.C., Clyde, D.F (1976). A Manual of Tropical Medicine. 5th Ed. WB Saunders, Philadelphia.

Ukoli, F.M.A. (1990). Introduction of Parasitology in Tropical Africa. John Wiley and Sons Ltd., Chichester.

UNIT 2   CLASSIFICATION OF DIGENETIC TREMATODES ACCORDING TO THEIR HABITAT

CONTENTS

1.0   Introduction
2.0   Objectives
3.0   Main content

1.0   INTRODUCTION

The digeneans are a group of specialised endoparasitic platyhelminthes. A common feature is that all have complex lifecycles, involving one or more intermediate hosts, the first of which is always a mollusc, which is usually aquatic. As adults they are found in most vertebrates groups, including fish, amphibians, reptiles, birds, and mammals, acting as definitive hosts, where they may be highly pathogenic. They may be located in most of the internal organs of these definitive hosts, including the lungs, bladder and blood stream, although the majority are found in the gastrointestinal tract, or closely associated organs such as the bile duct and liver. They exhibit a flattened leaf-like body, structurally similar to many of the free living turbellarians. The digeneans are classified below based on their locations in the definitive hosts;

- **Blood flukes** - *Schistosoma haematobium, S. mansoni, S. japonicum, S. mekongi,* and *S. intercalatum*
- **Liver flukes** - *Fasciola hepatica, F. gigantica, C sinensis, Opisthorchis felineus, O viverrini, Dicrocoelium dendriticum,* and *D. hospes*

- **Pancreatic flukes** - *Eurytrema pacreaticum, E. coelomaticum,* and *E. ovis*
- **Lung flukes** -*Paragonimus westermani, P. mexicana,* and *P. skrjabini*
- **Intestinal flukes** – *Fasciolopsis buski, Metagonimus yokogawai, Echinostoma ilocanum, Watsonius watsoni, Heterophyes heterophyes,* and *Gastrodiscoides hominis*

2.0   OBJECTIVES

At the end of this unit, student should be able to;
1. Explain the transmission cycles of digenetic trematodes
2. Identify the factors responsible for transmission and highlight what control measure to be taken to prevent transmission
3. Identify each parasite using the diagnostic features of the eggs
4. Explain the pathology caused by parasite

3.0   MAIN CONTENTS

3.1   Blood flukes (*Schistosoma* species)

Schistosomiasis, or bilharzia, is a tropical parasitic disease caused by blood-dwelling fluke worms of the genus *Schistosoma*. Over 200 million people are infected in at least 75 countries with 600 million or more people at risk of infection. The main schistosomes that infect human beings include *S haematobium* (transmitted by *Bulinus* snails and causing urinary schistosomiasis in Africa and the Arabian peninsula), *S mansoni* (transmitted by *Biomphalaria* snails and causing intestinal and hepatic schistosomiasis in Africa, the Arabian peninsula, and South America), and *S japonicum* (transmitted by the amphibious snail *Oncomelania* and causing intestinal and hepatosplenic schistosomiasis in China, the Philippines, and Indonesia).

S. intercalatum and *S. mekongi* are only of local importance. *S. japonicum* is a zoonotic parasite that infects a wide range of animals, including cattle, dogs, pigs, and rodents. *S. mansoni* also infects rodents and primates, but human beings are the main host. A dozen other schistosome species are animal parasites, some of which occasionally infect humans.

Unlike other trematodes, schistosomes have separate sexes, but males and females are found together. The male is short and stout and holds the relatively long female worm in its gynaecophoric canal, a groove like structure. With *S. haematobium,* both male and female live together in the veins that drain the urinary bladder, pelvis, and ureter, whereas *S. japonicum* and *S. mansoni* live in the inferior and superior mesenteric veins, respectively. Hence, these flukes are known as blood flukes. These species are distinguished from the other *Schistosoma* species based on the morphology of their eggs and their adult and cercarial forms. *S. haematobium* eggs have a terminal spine, whereas *S. mansoni* and *S. japonicum* eggs have lateral spines and central spines, respectively.

3.1.1 Morphology

The adult males measure up to 15 millimetres in length and females up to 10 mm. The schistosomes remain in copula throughout their life span, the uxorious male surrounding the female with his gynaecophoric canal. The male is actually flat but the sides roll up forming the groove. The cuticle of the male is covered with minute papillae. The female only possess these at the anterior and posterior end as the middle section being covered by the male body. Oral and ventral suckers are present, with the ventral one being lager serving to hold the worms in place, preventing them from being carried away by the circulatory current.

The ova of *S. mansoni* are 114-175 μm long by 45-68 μm wide. They are light yellowish brown, elongate and possess a lateral spine. The shell is acid fast when stained with modified Ziehl-Neelsen Stain.

Saline smear of *S. mansoni*, *S. japonicum* and *S. haematobium* ova showing their spine position; a good distinguishing feature when identifying Schistosome ova

Comparative Features of Major Human *Schistosoma* Species

Stages	*S. haematobium*	*S. mansoni*	*S. japonicum*
Adult			
Body surface of male	Finely tuberculate	Grossly tuberculate	Nontuberculate(smooth)
Testes	4-6, in a cluster	6-9, in a cluster	7, in a linear series
Position of ovary	Posterior to middle of body	Anterior to middle of body	Posterior to middle of body
Number of eggs in uterus	20-30	1-4	50-300
Egg			
Size and shape	110-170 µm long	114-175 µm long	70-100 µm long
	40-70 µm wide Terminal spine	45-68 µm wide Lateral spine	50-65 µm wide Central spine
Cercaria			
Cephalic glands	2 pairs, oxyphilic	2 pairs, basophilic	4 pairs, oxyphilic

### 3.1.2 Life Cycles and Transmission of Schistosomes

Once the eggs are laid by the adult female worms, the majority of them first pass through the veins of the blood vessel in which the worm is living, and then into the lumen of the intestine and are passed in the faeces (*S. mansoni* and *S. japonicum*) or into the lumen of the bladder, and are then passed in the urine (*S. haematobium).* Those eggs that reach fresh water hatch, releasing a miracidium which, to develop further must infect a specific snail species within 24 hours. The eggs of each species are markedly different but each produce virtually identical miracidium. A single miracidium can multiply in the snail to produce nearly 100,000 cercariae.

Asexual multiplication takes place in the snail, and results in the release of cercariae (minute in size with forked tails, 200mm long) into the water about 3 – 6 weeks later. Cercariae actively swim around and when they have located, or come into contact with a definitive host, they actively penetrate the skin. They can stay

active looking for a host for 24-48 hours after which if they don't find a host they will die.

The head of the cercariae migrates to the liver and develops into either adult male or female worms (flukes), where they pair up and then migrate to their region of the venous blood system (species specific sites). The females leave the males and moves to smaller venules closer to the lumen of the intestine or bladder to lay her eggs (about 6 weeks after infection). The majority of adult worms live from 2-4 years, but some can live considerably longer.
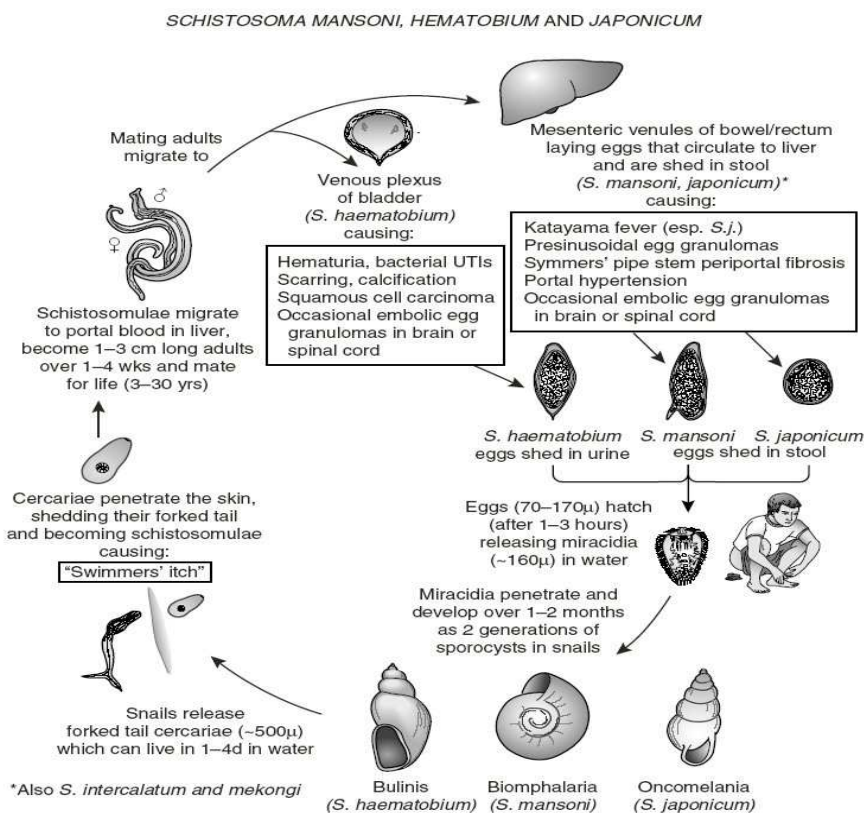
Life cycle of *Schistosoma* spp

Vectors and Geographical Areas Associated With Certain Trematode Types

Snail Host	Geographical Area	Trematode

Biomphalaria glabrata	Brazil	*S. mansoni*
Biomphalaria pfeifferi	Nigeria	*S. mansoni*
Bulinus globosus	Nigeria	*S. haematobium*
Bulinus truncates	Iran	*S. haematobium*
Oncomelania hupensis nosophora	Japan	*S. japonicum*
Thiara granifera	China	*Paragonimus westermani*
Semisulcospira libertine	China	*P. westermani*
Pirenella conica	Egypt	*Heterophyes heterophyes*
Lymnaea truncatula	England	*Fasciola hepatica*
Lymnaea natalensis	Nigeria	*Fasciola gigantic*

3.1.3 Pathology and clinical symptoms

- **Acute manifestations**
  a. Cercarial dermatitis, also known as swimmer's itch, is an allergic reaction caused by the penetration of cercariae in persons who have been exposed to cercariae in salt water or fresh water. Cercarial dermatitis manifests as petechial haemorrhages with oedema and pruritus, followed by maculopapular rash, which may become vesicular. The process is usually related to avian schistosomal species of the genera *Trichobilharzia, Gigantobilharzia,* and *Orientobilharzia,* which do not develop further in humans.
  b. Katayama syndrome corresponds to maturation of the fluke and the beginning of oviposition. This syndrome is caused by high worm load and egg antigen stimuli that result from immune complex formation and leads to a serum sickness –like illness. This is the most severe form and is most common in persons with *S mansoni* and *S japonicum* infections. Symptoms include high fever, chills, headache, hepatosplenomegaly, lymphadenopathy, eosinophilia, and dysentery. A history of travel in an endemic area provides a clue to the diagnosis.

- **Chronic manifestations**
  a. Symptoms depend on the *Schistosoma* species that causes the infection, the duration and severity of the infection, and the immune response of the host to the egg antigens.
  b. Terminal haematuria, dysuria, and frequent urination are the main clinical symptoms of urinary schistosomiasis.

c. The earliest bladder sign is pseudotubercle, but, in long-standing infection, radiography reveals nests of calcified ova (sandy patches) surrounded by fibrous tissue in the submucosa.

d. Dysentery, diarrhoea, weakness, and abdominal pain are the major symptoms of intestinal schistosomiasis.

e. A reaction to schistosomal eggs in the liver causes a periportal fibrotic reaction termed Symmers clay pipestem fibrosis.

f. Haemoptysis, palpitation, and dyspnea upon exertion are the symptoms of schistosomal cor pulmonale that develops as a complication of hepatic schistosomiasis.

g. Headache, seizures (both generalized and focal), myeloradiculopathy with lower limb and back pain, paresthesia, and urinary bladder dysfunction are the noted symptoms of CNS schistosomiasis due to *S. japonicum* infection.

3.1.4 Diagnosis

Intestinal schistosomes

- Laboratory confirmation of *S. mansoni* and *S. japonicum* infection can be made by finding the eggs in the faeces. When eggs cannot be found in the faeces, a rectal biopsy can be examined.

- Serological tests are of value in the diagnosis of schistosomiasis when eggs cannot be found. An enzyme linked immunosorbent assay (ELISA) using soluble egg antigen, is employed at HTD.

Urinary schistosome

- The definitive diagnosis of urinary schistosomiasis is made by finding the characteristic ova of *S. haematobium* in urine. Terminal urine should be collected as the terminal drops contain a large proportion of the eggs. The urine can then be centrifuged and the deposit examined microscopically for ova. Eggs can sometimes be found in seminal fluid in males.

- A bladder biopsy is seldom necessary to make the diagnosis. A rectal snip may show the presence of ova as they sometimes pass into the rectal mucosa.

- Serological tests can be of value when eggs cannot be found in clinical samples. An enzyme linked immunosorbent assay using soluble egg antigen to detect antischistosome antibody is most sensitive.

Note: There is a marked periodicity associated with the time when most eggs are passed out. Higher numbers of eggs are encountered in urine specimens passed between 10 am and 2pm, presumably as a result of changes in the host's metabolic and physical activities.

3.1.5 Epidemiology of Schistosomiasis

The following factors are of epidemiological importance in the transmission of schistosmiasis:

- The presence of water bodies such as rivers, streams, lakes, dams suitable for the breeding of the snail intermediate hosts
- Presence of appropriate snail hosts necessary for the developments of the asexual stages and transmission of the infective stage to the human definitive host
- Contamination of natural water bodies with infected human urine and faeces
- Human water contact activities including swimming, laundry and fetching
- Factors that promote intramolluscan development of parasite and subsequent transmission to man
- Socio-economic status of the people such as good sanitary system and water supply

3.1.6 Control

- Reduction of human-water contact
- Improved sanitation by proper waste disposal
- Attacking the adult forms of parasite through chemotherapy to reduce the worm burden or egg production
- Eradication or reduction of snail population through the use of molluscicides
- Development of vaccine to induce immunity
- Modification of the ecology of the snail habitat
- Biological control through the introduction of competitors snails into the snail habitat

- Education

3.2 Lung flukes (*Paragonimus* species)

The genus *Paragonimus* contains more than 30 species that have been reported to cause infections in animals and humans. Among these, approximately 10 species have been reported to cause infection in humans, of which *P. westermani* is the most important. *P. westermani*, also known as the Oriental lung fluke is most common in China, Korea, Thailand, Philippines, and Laos. Isolated endemic foci have also been reported from the states of Manipur, Nagaland, and Arunachal Pradesh in India. A low prevalence has been reported from African countries of Cameroon and Nigeria, where infections with *Paragonimus africanus* and *Paragonimus uterobilateralis* were reported. Humans are infected by eating raw or partially cooked crab or crayfish or crabs soaked in wine as a food delicacy or by drinking juice from raw crabs or crayfish as a part of a food habit.

It inhabits parenchyma of the lung close to bronchioles in humans, foxes, wolves, and various feline hosts (e.g, lions, leopards, tigers, cats).

Paragonimus species belong to the family 'Troglotrematidae' and they possess the following striking features:

- The adult worm is reddish brown fluke.
- The body of adult worm is thick, fleshy, and ovoid in shape
- The tegument is spiny or scaly
- Have weak suckers
- Testes lie side by side
- Uterus is short with a few tight uterine coils forming a 'rosette'
- Extensive vitellaria in lateral fields
- Cercariae are microcercous xiphidiocercariae
- The eggs are ovoid, brownish yellow, thick shelled and operculated.

3.2.1 Life cycle and Transmission

The infection is typically transmitted via ingestion of metacercariae contained in raw freshwater crabs or crayfish. Additionally, consumption of the raw meat of paratenic hosts (e.g, omnivorous mammals) may also contribute to human

infection. Freshwater snails and crabs are first and second intermediate hosts of *Paragonimus* species, respectively. In the duodenum, the cyst wall is dissolved, and the metacercariae are released. The metacercariae migrate by penetrating through the intestinal wall, peritoneal cavity, and, finally, through the abdominal wall and diaphragm into the lungs. There, the immature worms finally settle close to the bronchi, grow, and develop to become sexually mature hermaphrodite worms.

Adult worms begin to lay the eggs, which are unembryonated and are passed out in the sputum. However, if they are swallowed, they are excreted in the feces. The eggs develop further in the water. In each egg, a ciliated miracidium develops during a period of 2-3 weeks. The miracidium escapes from the egg and penetrates a suitable species of snail (first intermediate host), in which it goes through a generation of sporocysts and 2 generations of rediae to form the cercariae. The cercariae come out of the snail, invade a freshwater crustacean (crayfish or crab), and encyst to form metacercariae. When ingested, these cause the infection, and the cycle is repeated.
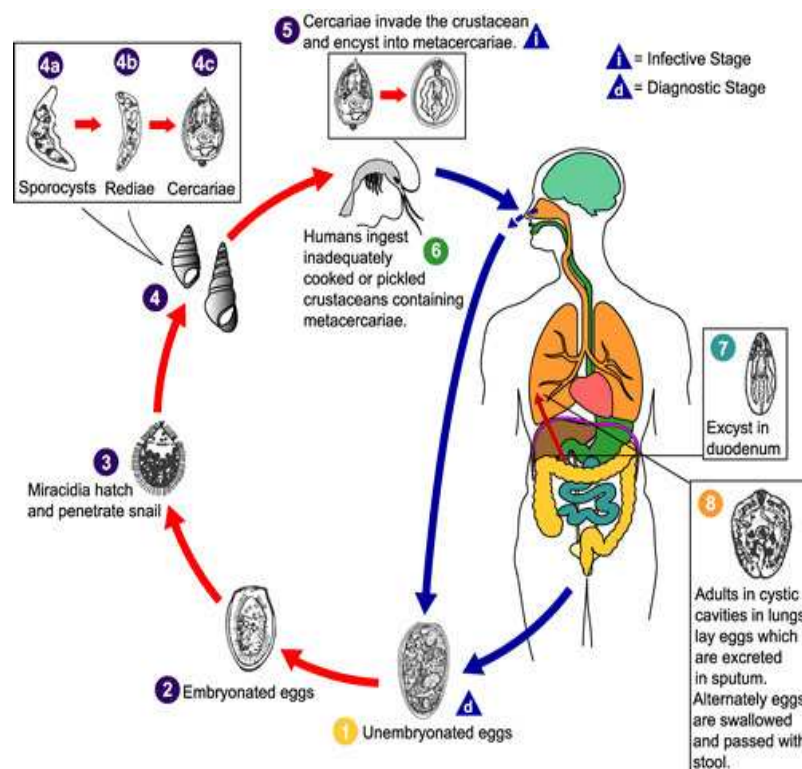
Life cycle of *Paragonimus* spp

3.2.2 Pathology and clinical symptoms

a. **Acute manifestations**: Acute pulmonary infection is characterized by low-grade fever, cough, night sweats, chest pain, and blood-stained rusty-brown sputum.

b. **Chronic manifestations**: Lung abscess or pleural effusion develops in individuals with chronic infections. Fever, haemoptysis, pleurisy pain, dyspnea and recurrent attacks of bacterial pneumonia are the common symptoms. The condition mimics pulmonary tuberculosis.

c. Fever, headache, nausea, vomiting, visual disturbances, motor weakness, and localized or generalized paralysis are the symptoms of cerebral paragonimiasis.

3.2.3 Diagnosis

Diagnosis is based on finding the characteristic eggs in brown sputum. The eggs can also be found in the faeces due to swallowing sputum. A chest x-ray may show cystic shadows and calcification. Serological tests, in particular, the ELISA method, are useful diagnostic tests.

Saline smear of *Paragonimus westermanni* egg. The egg shells are thick and operculated

3.2.4 Epidemiology

The epidemiology of the disease (Paragonimiasis) depends on one of the following

- Presence of appropriate snail, crab and mammalian reservoir hosts in the area
- Pollution of snail habitats with sputum and faeces of man as well as natural mammalian reservoir hosts infected with the parasite

- Consumption of metacercariae through eating of raw or undercooked crabs or through contamination of the fingers and cooking utensils with metacercariae while cleaning

3.2.5 Control

- Proper cooking of crabs before consumptions
- Proper waste disposal

3.3 Liver fluke (*Fasciola hepatica* and *F. gigantica*)

Fascioliasis is a a zoonotic disease caused by infection with *F. hepatica*. It is a major disease of livestock that is associated with important economic losses due to mortality; liver condemnation; reduced production of meat, milk, and wool; and expenditures for anthelmintics. The disease has a cosmopolitan distribution, with cases reported from Scandinavia to New Zealand and southern Argentina to Mexico. Also of importance is the West Africa species of *Fasciola* (*F. gigantica*). The two share similar morphology, life cycle and pathogenicity. They belong to the family 'Fasciolidae' having the following major features;

- They are large with flattened leaf-like forms
- They have ramifying and complicated digestive and reproductive systems
- Most members of the family inhabit the liver and the bile duct. However, *Fasciolopsis buski* inhabits the intestine
- Cercariae are gymnocephalous
- Metacercariae encyst on vegetation thus establishing a two-host cycle

3.3.1 Morphology of the Adult
- They are leaf-like with oral cone and shoulder at anterior end
- The intestinal caeca, testes and ovary are branched
- Tight and relatively short uterus is opposite to the ovary at the anterior end
- Vitellaria are extensive and are laterally distributed

Distinctions between *F. hepatica* and *F. gigantica*
- *F. gigantica* is larger (75 by 12mm) while is smaller (30 by 13mm)
- *F. gigantica* is oblong with prolonged posterior end while *F. hepatica* is more or less triangular in shape
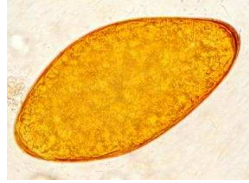
- The eggs of *F. gigantica* is also larger

Figure showing the egg of *Fasciola;* ova of *Fasciola* are ovoid in shape, quinone colour and often showing an inconspicuous operculum. *Fasciola hepatica* ova measure 130 - 150μm by 63 - 90μm. There is much cross-over in ova size between all of the *Fasciola* species.

3.3.2 Life cycle and Transmission

Opercular eggs are passed out from the faeces of the infected animal (cattle or sheep).The eggs embryonate in the presence of light as stimulus and hatch into miracidia which locate an appropriate snail intermediate host by a chemical response called **chemotaxis**. The snail host of *F. hepatica* is *Lymnaea trucantula* while that of *F. gigantica* is *L. natalensis.* The intramolluscan development of miracidium produces sporocysts which in turn develop rediae. The mother rediae produce second generation of rediae (daughter rediae) which later give rise to gynocephalous cercariae. This crawls out of the snail, locates submerged plant, loses its tail and encysts into metacercariae. Infection occurs when sheep and cattle ingest plant with metacercariae during grazing. Metacercariae excyst in the duodenum and the emerging young adult punctures its way into the body cavity and wanders around until it locates the liver capsule. Its burrows into the tissues, feeding on the cells until it gets to the bile duct where it eventually attains maturity.

3.3.3 Pathology

- Pathology depends on the intensity of infection and duration of the disease.

- Fluke causes bilary obstruction. Because of pressure, toxic metabolic products, and feeding habits, the worms provoke inflammatory, adenomatous, and fibrotic changes of the bilary tract.
- Parenchymal atrophy and periportal cirrhosis develop.
- Severe headache, chills, fever, urticaria, a stabbing substernal pain, and right upper quadrant pains that radiate to the back and shoulders may be the first evidence of infection.
- As infection progresses, an enlarged tender liver, jaundice, digestive disturbance, diarrhoea and anaemia develops.

3.3.4 Laboratory diagnosis

- Definitive diagnosis is made by observing the ova in faeces.
- Where identification cannot be made from the size of the ova, clinical information and the source of infection may help to provide a diagnosis. This includes an enlarged tender liver and a febrile eosinophilic syndrome
- Positive complement-fixation test and intracutaneous reactions with *Fasciola* antigens are used when direct faecal examination fails to reveal the eggs.

3.3.5 Epidemiology and Control

Fascioliasis is prevalent in areas where cattle or sheep graze and in areas where appropriate lymnaeid snail hosts flourish. Therefore control measures involves

- Treatment of animals to improve general condition and reduce egg output
- Breaking of transmission cycle by eradicating the snail hosts. However, this is difficult to achieve on the field
- Infection in humans can be prevented by eliminating raw water cress and other uncooked green vegetable from the diets.
- A safe water supply is also necessary

3.4   Intestinal flukes (*Fasciolopsis. buski, Heterophyes heterophyes, Metagonimus yokogawai*)

F. buski is the most common intestinal nematode that causes infections in humans. It is widely distributed in Asia and the Indian subcontinent, especially in areas where humans raise pigs and consume freshwater plants.The trematodes *H. heterophyes* and *M. yokogawai* are less-common causes of human infection.

3.4.1 Life cycle

F. buski, known as the giant intestinal fluke, is found in the duodenum and jejunum of pigs and humans and is the largest intestinal fluke to parasitize humans. Humans are infected by eating freshwater aquatic plants such as water caltrops, water chestnuts, and water bamboo, which can harbour the metacercariae. In the intestine, the metacercariae excyst, attach to the duodenum or jejunum, develop, and grow into adult worms. They lay unembryonated eggs, which are excreted in the faeces.

In water, inside the egg, a ciliated miracidium develops, comes out, and penetrates a suitable snail host. Inside the snail, after several stages of asexual multiplication, large numbers of cercariae are produced. The latter emerge from the snail and encyst on the surface of aquatic plants to metacercariae. Ingestion of these plants causes infection in humans, and the cycle is repeated.
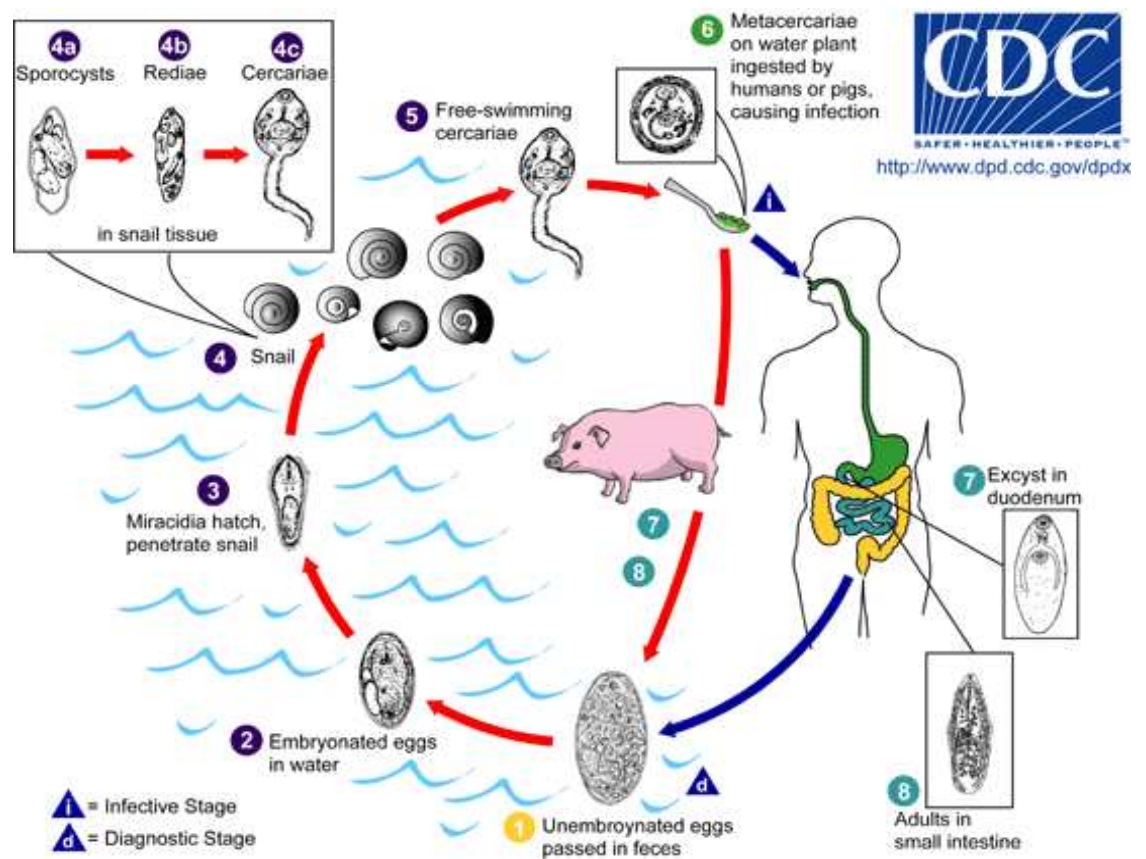

Life cycle of *F.buski*

3.4.2 Morphology

Eggs of *Fasciolopsis buski* are broadly ellipsoidal, operculated and measure 130-150 µm long by 60-90 µm wide. The eggs are unembryonated when passed in feces. The eggs of *F. buski* can be difficult to distinguish from *Fasciola hepatica*, although the abopercular end of the latter often has a roughened or irregular area.

The adults of *F. buski* measure 20-75 mm long and have poorly-developed oral and ventral suckers. Adults reside in the intestine of the mammalian host.
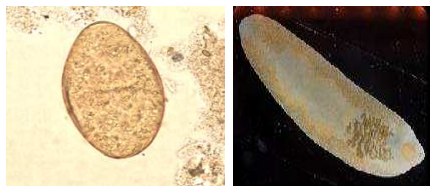
The egg and the adult forms of *F. buski*

3.4.3 Clinical Features

Most infections are light and asymptomatic. In heavier infections, symptoms include diarrhoea, abdominal pain, fever, ascites, anasarca and intestinal obstruction.

3.4.4 Laboratory Diagnosis

Microscopic identification of eggs, or more rarely of the adult flukes, in the stool or vomitus is the basis of specific diagnosis. The eggs are indistinguishable from those of *Fasciola hepatica*.

3.5 Pancreatic flukes (*Eurytrema pancreaticum, E. coelomaticum, E. ovis*)

These are widely distributed in China, Korea, Japan, Hong Kong, South America, etc. *E. pacreaticum* is a common parasite of pancreatic (or rarely bile) ducts of herbivorous mammals, i.e., cattle, sheeps, goats, monkeys, and camels.

3.5.1 Life cycle

The adult flukes live in the pancreatic passages of the herbivores. Eggs are passed in the faeces and ingested by land snail, which is the first intermediate host (snail). The cercariae develop into infective metacercariae only if ingested by grasshoppers, the second intermediate host. The life cycle is completed when the infected insects are eaten by grazing herbivores. The metacercariae excyst and migrate to the pancreatic passage, where they develop into adults. Humans become infected when they accidentally swallow infected grasshoppers.

3.5.2 Morphology

The parasite (10~18 x 5~9 mm in size) is broad, flat, and oval to fusiform. The suckers are large, the oral sucker is larger than the ventral sucker. The eggs (50~80 x 35~40 μm) are embryonated in the uterus.

E. pacreaticum : from pancreas of cattle, Acetocarmine stain, X40

3.5.3 Pathology and clinical symptoms

Eurytremiasis is usually characterised by mild symptoms. Heavy infections, however, may be marked by gastrointestinal disturbances, including abdominal distress, flatulence, vomiting, diarrhoea or constipation. jaundice, an enlarged liver, and systemic symptoms. Eosinophilia is rare.

3.5.4 Diagnosis

Diagnosis is made by finding the characteristic eggs in faeces. Spurious infection must be ruled out by repeated examination. Eggs of the *Dicrocoelium dendriticum* and *E. pancreatum* are almost indistinguishable. Definitive diagnosis can be made by recovery of adult flukes at surgery or autopsy.

3.5.5 Prevention

Human infections are generally accidental.

4.0   CONCLUSION

In this unit, you learnt the morphology, life cycles, pathology, the diagnostic techniques and epidemiology of the various digenetic trematodes.

5.0   SUMMARY

Digenetic trematodes are the medically important groups of trematodes that inhabit different tissues and organs of their hosts. Hence, they are named according to their various locations in the parasitised hosts. Diagnosis is dependent on the route through which parasites' eggs are voided out of the host. Therefore, the faecal, urine and sputum samples are examined microscopically to identify the characteristic eggs. The pathological effects vary from mild to severe due to the parasite burden in the host.   Proper waste disposal, and proper cooking of crabs, fishes and land snails which act as intermediate host are some of the control measures.

6.0   TUTOR-MARKED ASSIGNMENT

1. Hightlight the differences in the mode of transmission cycles of *S. haematobium* and *H. heterophyes*
2. What are the morphological differences in the species of schistosomes?
3. Discuss the life cycle of a named digenea trematode of livestock importance.

7.0   REFERENCES/FURTHER READINGS

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MODULE 3 CESTODES

Unit 1   Basic body plan of a cestode

Unit 2   Tapeworms and Examples

Unit 3   Tapeworms of man and other human's cestodes

UNIT 1   BASIC BODY PLAN OF A CESTODE

CONTENT

1.0   Introduction
2.0   Objectives
3.0   Main contents
     3.1   The adult parasite
         3.1.1   Scolex
         3.1.2   The neck
         3.1.3   The strobila
     3.2   The cestode integument
     3.3   Larval metacercaria

1.0   INTRODUCTION

The cestodes consist of two separate subclasses, the Cestodarians, parasites of fish and other cold blooded vertebrates. These are non-segmented parasites, with only a single set of sexual organs. In contrast, the more well known members of the Subclass Eucestoda are parasites of both warm and cold blooded vertebrates, including mammals such as man. They resemble a colony of individual animals in that their bodies are divided into a series of segments (the proglotids), each with their own complete set of internal organs. There may be many hundreds of these proglotids, resulting in the complete parasite having a long, ribbon like body. The appearance of this long body is the origin for the common name for these parasites, the tapeworms. The common names of these parasites are often derived from their intermediate hosts, ingestion of which results in their infection, e.g. the Fish, Beef and Pork Tapeworms. Alternatively they may be named after the definitive host that the adult parasites are normally found in. For example the Rat tapeworm *H. diminuta* and the Dog Tapeworm *Dipylidium caninum*. The study of the morphology of the cestode body may be divided into two distinct areas. Firstly, the morphology of the adult cestode (the tapeworm) and secondly the morphology of the cestode larvae, or Metacestode.

2.0   OBJECTIVES

At the end of this unit, students should be able to
1.   Identify the striking features of cestodes
2.   Describe the egg, larva and adult stage morphology of common cestodes

3.0   MAIN CONTENTS

3.1   The Adult Parasite

The body of the adult tapeworm may be divided into three region

3.1.1 The Scolex

This is the "head" and attachment organ of the parasite. There are four main types of scolex, by which the tapeworm may be taxonomically classified.

a) No special attachment organs

The scolices of some tapeworms of the order Caryophyllidea (parasites of freshwater fish) have no special attachment organs. (NB. Some authors do not recognise this taxonomic order, placing these parasites within the Pseudophyllidea).

b) A Bothria - This is composed of a pair of shallow, elongated, weakly muscular grooves. Tapeworms of the order Pseudophyllidea are equipped with bothria on their scolices.
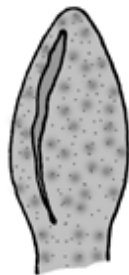
The bothria of Pseudophyllidea

c) A Bothridia - These are broad, leaflike muscular structure, exhibiting a large degree of variation. Some bothridia are sessile, some are stalked, whilst others are hooked with accessory suckers. Tapeworms of the order Tetraphyllidea and others are equipped with bothridia.
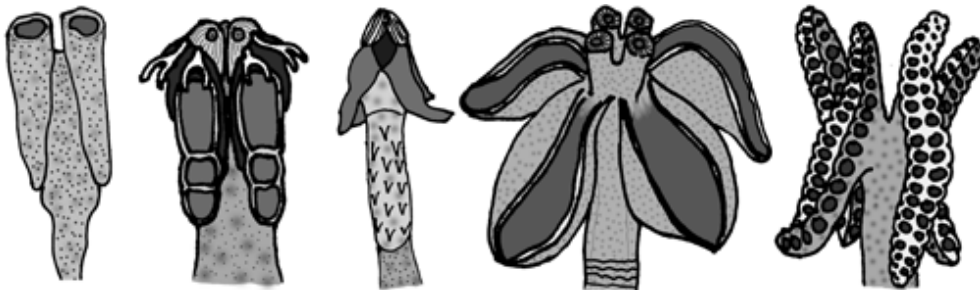
d) Acetabulate Suckers - Tapeworms of the order Cyclophyllidea are equipped with four acetabulate suckers. Parasites in this order may also have additional features at the apex of the scolex such as;

- Glandular areas
- Protrusible suckers
- Suckers armed with hooks
- Hooks (e.g. Taenia)
- A rostellum, an eversible muscular proboscide, often covered with hooks (e.g. *Hymenolepis, Echinococcus, Dipylidium*)
- A Myzorhynchus (a protrusible muscular mass).

3.1.2 The Neck

This is the area of proliferation of the parasite, from which the proglottids of the strobila grow.

3.1.3 The Strobila

This is composed of a series of proglottids. Each proglottid contains a complete set of male and female reproductive organs, although these organs usually mature at different rates. Usually the male organs develop before the female organs, and degenerate before the female organs mature. The large, gravid proglottids at the posterior end of the tapeworm are full of developing, or in the extreme terminal proglottids, mature eggs.

3.2 The Cestode Tegument

The related cestodarians that also belong within the cestodes, have a tegument that appears to be intermediate with that of the eucestodes and monogeneans. This is another piece of evolutionary evidence that indicates a monogenean origin for the tapeworms. In this case the surface of the cestodarian tegument is covered with numerous microvilli, similar in form to the eucestode microtriche (see below), but lacking the electron dense cap seen in these parasites.

The cesode tegument is a syncytial layer, showing many features typical of that found in other parasitic platyhelminthes.
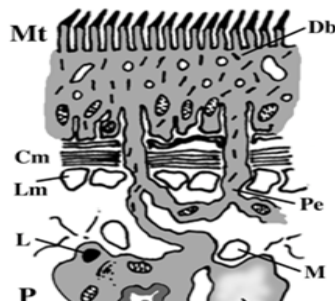
Diagram showing eucestode tegument

There are however, a number of distinguishing features present in these parasites. On the very outer surface of the tegument a surface glycocalyx is seen to cover the outer plasma membrane. Below this glycocalyx, a characteristic feature of the eucestode tegument is the presence of numerous microtriches (**Mt**), long spine like processes that are in fact a highly modified form of micovilli. Each microthrix has a hard, pointed, electron dense cap which is seperated from the rest of the microthrix by a crescent shaped membranous cap. The mirotriches are thought to serve two functions. Firstly, the tapeworms do not possess a gut and must absorb all of their nutrients across the surface tegument. The microtiches greatly increase the surface area of the parasite, and can be seen as an adaptation to maximise the amounts of nutrients available to the parasite. This is supported by the finding of microtubles in the shaft of the microtriches. Secondly the spine like character of the microtriches probably help the parasite maintain its position in the gut. This can be more clearly seen by comparing the microtriches found in different regions of the parasite's body. It has been noted in many species that the microtriches found covering the scolex, the attachment organ of the parasite, were much longer than those covering the strobila, and in some species show special adaptations. For example the microtriches covering the strobila of *E. granulosus* have been found to show curved hooks or sometimes even barbs. Below the layer of microtriches the main syncytial layer of the tegument is found. This has been seen to contain numerous vesicles and membrane bound, electron dense rod-like structures, refered to as disc-shaped bodies (**Db**). Finally numerous mitochondria, mainly in the distal region of the tegument, may be seen. These are unusual in that they do not have many cristae, reflecting the anaerobic metabolism of the organism. The tegumental nuclei are however not located in this outer layer, but are found within subtegumental cell bodies (**StC**), located beneath the circular (**Cm**) and longitudinal muscle (**Lm**) layers, embedded within the parenchymal tissues (**P**)

61

and mesenchymal musculature (**M**). These subtegumental cell bodies also contain other cellular elements such as golgi apparatus and lipid inclusion bodies (**L**) which are connected to the outer syncytium and areas of glycogen storage (**Gs**) by long protoplasmic extensions (**Pe**). The location of these important cellular elements away from the outer surface of the parasite, exposed to immunological attack by the parasites host, is an important adaptation to a parasitic lifecycle adopted by all of the parasitic platyhelminthes. The parenchymal tissues are similar to those of the trematodes and fill the spaces between the parasites internal organs (all cestodes and other platyhelminthes being acoelomate organisms). These tissues are a syncytial network formed by anastomosis of mesenchymal cells, with spaces filled with carbohydrate rich parenchymal fluid.

3.3　Larval Metacercaria

3.3.1　The Larval Cestodes

1) The Cestodarians

The cestodarians larvae, or **lycophore** are free swimming, being covered in cillia. They have a set of ten hooks at the extreme anterior of the body, thus differing from the larval eucestodes, which are equiped with 3 pairs of hooks. Anteriorly they are armed with penetration glands. The bodily form of these larvae bears a marked resemblance to the larvae of the trematodes, such as the miracidium in the digeneans, and the larval monogenean, the oncomiracidium.
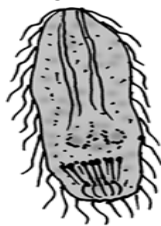
A lycophore

2) The Eucestodes (Tapeworms)
The eggs of Pseudophyllidean and Cyclophyllidean cestodes differ considerably. The egg of the pseudophyllidean tapeworm closely resembles that of the trematodes, having a thin shell wall, and an operculum, which on hatching opens to reseales the free swimming larvae. This illustrates the close relationship

between the two major groups of platyhelminth parasites. In contrast, the egg of the cyclophyllideans tapeworms is very different, having a very thick, resistant egg shell, with no operculum.
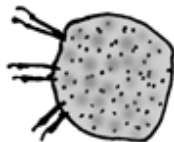

The pseudophyllidean and cyclophyllidean ova

The larvae emerging from these eggs also differ. The pseudophyllidean egg hatches to release a free swimming larvae called a coracidium. This has an outer layer of ciliated epidermal cells with which it swimms through the water before being ingested by the parasites first intermediate host. This is often a copepod. Inside the copepod the ciliated epidermis is shed, to release a larvae that initially resembles that of the newly hatched cyclophyllideans. This has 6 hooks, arranged in pairs, and is a common feature throughout the eucestodes. On the basis of the presence of these hooks, present in both the eucestodes and cestodarians, many authors believe that the cestodes originally evolved from an ancestor common to the extant monogeneans.


Coracidium larva

The larval cyclophyllidean, as with the pseudophyllidean, is equiped with 3 pairs of hooks. Both groups use these hooks to penetrate the gut wall of its intermediate host after being ingested, before developing into the other larval forms described below in more detail.


Larva of cyclophyllidean

3.4   Metacestodes

A number of different larval forms of cestodes (metacestodes) are seen, these include the following;
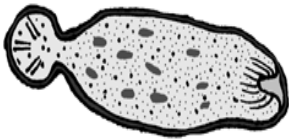
A Procercoid of **Pseudophyllidean** (e.g *D. latum*)

A larval form of **Pseudophyllidean** cestodes, (e.g. *D. latum*, *Ligula intestinalis*). Here two forms of the procercoid are shown. Firstly an immature procercoid, and secondly a mature infective procercoid. In the lifecycle of these parasites there are two intermediate hosts. The procercoid being found in the first of these (usually a small crustacean e.g. *Cyclops*).In appearance these larvae have solid bodies with the remains of the embryonic hooks from the onchosphere larvae at the posterior of the parasite.


A Plerocercoid of **Pseudophyllidean** (e.g *D. latum*)
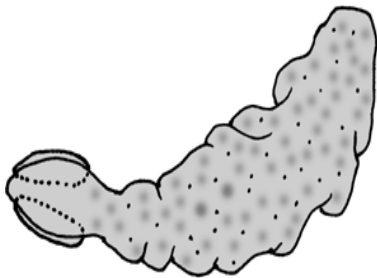
A larval form of **Cyclophyllidean** Cestodes, (e.g. *Hymenolepis* sp.)

A larval form of **Pseudophyllidean** and other Cestodes, (e.g. *D. latum, Ligula intestinalis*). In the lifecycle of these parasites there are two intermediate hosts (see the cestode life cycle page). The plerocercoid being found in the second of these (usually a fish or amphibian). In appearance, these are elongated larvae with solid bodies which are much larger than the preceding procercoid larvae. In these stages the embryonic hooks are absent. The plerocercoids of some Pseudophyllideans already show the start of the development of the sexual organs (e.g. *Schistocephalus solidus, Ligula intestinalis*), whilst those of *Schistocephalus solidus* are also already divided into proglottids).
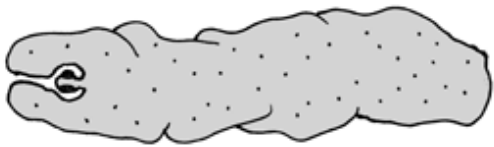
A larval form of **Cyclophyllidean** Cestodes, (e.g. *Hymenolepis* sp.). This larval form is usually found in species where the intermediate host is an invertebrate, usually an insect.
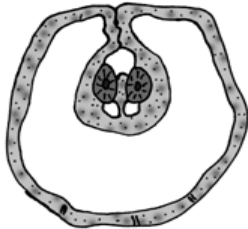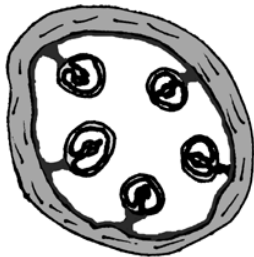
A larval form of **Cyclophyllidean** Cestodes, (e.g. *Taenia solium*)

A larval form of **Cyclophyllidean** Cestodes, (e.g. *Mesocestoides* sp.).

A larval form of **Cyclophyllidean** Cestodes, (e.g. *Taenia taeniaeformis*).

A larval form of **Cyclophyllidean** Cestodes, (e.g. *Taenia multiceps*).

A larval form of **Cyclophyllidean** Cestodes, (e.g. *Echinococcus granulosus*). - Hydiatid cyst

4.0   CONCLUSION

In this unit, you learnt how to describe the body plan of adult and larval cestodes.

5.0 SUMMARY

The body plan of adult cestode is divided into scolex, neck and strobila. The scolices of the order Caryophyllidea (parasites of freshwater fish) have no special attachment organs while the Pseudophyllidea have weakly muscular grooves which are armed with bothria. The Cyclophyllidea have four acetabulate suckers. In addition to these are glandular areas, protrusible suckers and rostellum depending on the species of the cyclophillidean. The strobila is made up of proglottids containing the male and the female reproductive organs. The larvae of cestodes vary with species with some being ciliated and as such are free swimming. Some however, have operculum with thin shell wall. Others have thick shell wall with 6 hooks.

6.0 TUTOR-MARKED ASSIGNMENT

1. Differentiate between the larvae of psedophyllidean and cyclophyllidean
2. Give a concise description of the various larval forms of cestodes

7.0 REFERENCE/FURTHER READINGS

Ukoli, F.M.A. (1990). Introduction of Parasitology in Tropical Africa. John Wiley and Sons Ltd., Chichester.

UNIT 2   TAPEWORMS AND EXAMPLES

CONTENTS

1.0   INTRODUCTION

Cestodes or tapeworms are the most specialised of the Platyhelminthe parasites. All cestodes have at least one, and sometimes more than one, secondary or intermediate host as well as their primary host. While the intermediate hosts are often invertebrates of some sort, the primary host is normally a vertebrate. However, in some cases both hosts are vertebrates, as in the common Beef

Tapeworm (*Taenia saginatus*), and in a few species there may be only a single host. A number of tapeworms include mankind in their life cycles but infection is not normally a serious health problem and can be cured. There are more than 1,000 species of tapeworms known to science, and nearly every species of vertebrate is liable to infection from at least one species of tapeworm.

2.0   OBJECTIVES

At the end of this unit, student should be able
To describe the morphology and life cycle of a named cestode.
To explain the epidemiology and control of these parasites

3.0   MAIN CONTENT

3.1   *Diphyllobothrium latum* (the Broad Fish Tapeworm)

Diphyllobothrium latum is the fish tapeworm of man. It has a fairly cosmopolitan distribution, but is particularly common in the Baltic region, Russia and the Great Lakes region of the U.S.A.

3.1.1   Morphology of the adult Tapeworm

The adult parasites are typically between 2 and 12 m in length by up to 2 cm in width, but may grow even longer in some cases. The anterior organ of attachment is a bothria, a pair of shallow, elongated muscular grooves, typical of tapeworms of the order Pseudophyllidea. The body is divided into proglottids, as is the case of all pseudophyllidean tapeworms. These proglottids are broader than they are long, except at the terminal end, where they are approximately square in shape. Internally the proglottids are typical of pseudophyllidean tapeworms, with numerous testes and vitellaria arranged on the lateral margins of the proglottid, with a central bilobed ovary. An important difference between this parasite and the other tapeworms of man is that the uterus open to the exterior (cyclophyllidean tapeworms have closed uteruses). Eggs are therefore actively deposited by the parasite, in contrast to the disintegration of the proglottids seen in the other human tapeworms.
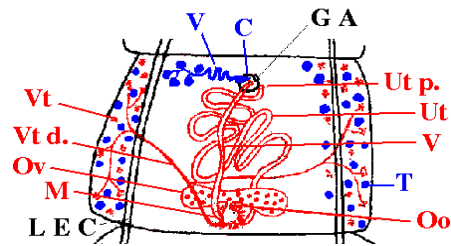
The Female Reproductive System   The Male Reproductive System of *D. latum*

Key - Ov - Ovary (bilobed in *D. latum*.) ;**Oo** - Ootype (where the egg is formed); **Ut** - Uterus (In the pseudophyllideans this opens to the outside, via the uterine pore); **Ut p.** - Uterine pore (**not** present in the cyclophyllideans); **V** - Vagina (a long straight tube); **Vt** - Vitelline glands (secreting substances that make up the egg yolk and shell);**Vt d.** - The Vitelline duct (connecting the vitelline gland, which are diffuse and are situated lateraly in *D. latum* ;**M** - The Mehlis gland (A cluster of unicellular shell glands, absent in some species) **T** - Testes (dorso-lateral in *D. latum* ;**V** - Vas deferens; **C** - Cirrus (a protrusible muscular organ, opening anterior to the vagina in a common genital atrium); **G A** - Genital Atrium (a cup shaped sinus, where the cirrus and vagina have common openings); **L E C** - The Lateral Excretory Canal

3.1.2   Life cycle and Transmission

Diphylobothrium latum has a typical Pseudophyllidean tapeworm lifecycle. In addition to the adult parasites in the definitive host, (i.e. man), there are two intermediate hosts containing larval stages. Eggs are passed from man in the faeces and hatch in water to release a small motile embryonic parasite, the **Coracidium**. This is internally similar to the hexacanth larvae of the Cyclophyllidean tapeworms, being equipped with 6 hooks, but this hexacanth larva is covered in a ciliated embryophore. The coracidium is a free swimming stage, but cannot survive long. For further development, it must be ingested by the first intermediate host, a copepod. On ingestion the embryonic larvae penetrates the arthropods gut wall, entering the haemocoel to develop into the first larval stage, the procercoid, measuring 50µm in length. This larva, (as well as the next larval stage, the plerocercoid described above) is very different from the cyclophyllidean parasite larvae in that they have elongated and solid bodies. In addition, the procercoid bears the embryonic hooklets on a posterior bulb like

rounded growth, the cercomer. To continue the lifecycle, the copepod must be ingested by the next intermediate host, a fish. The procercoid penetrates the gut wall of the fish, and develops into the next larval stage, the plerocercoid (**sparganum**), measuring 4 - 5 mm in length, in the viscera or musculature of the fish. These plerocercoids have again elongated solid bodied parasites, but differ from the procercoids in the absence of the cercomer and hooklets, and at the anterior end having a developed attachment organ, the bothridium, similar to the adult parasite. A number of different species of fish may act as intermediate hosts for the plerocercoids of *D. latum*, but the highest densities of plerocercoids are found in carnivorous fish such as the pike. These high parasite loads are because, in addition to infection by ingestion of the copepod plus procercoid, if another infected fish is eaten the plerocercoids within the body tissues of this predated fish are released in the intestine of the carnivorous fish. These then migrate through the intestinal wall, to invade the new host, which is then acting as a Paratenic host for these secondary plerocercoids. The plerocercoids are, in addition, very long lived, and may achieve very high parasite densities. Man is infected by ingestion of raw or undercooked fish, the plerocercoids emerging in the intestine to grow into the adult parasite. In addition to man a number of other fish eating mammals may also be infected, including cats, dogs, pigs, bears. Therefore *D. latum* in addition to being a parasite of man, also causes zoonotic infection. In man multiple infections may occur, sometimes of very high numbers (up to 143 worms have been reported from a single individual). In these cases the parasites length is considerably reduced.
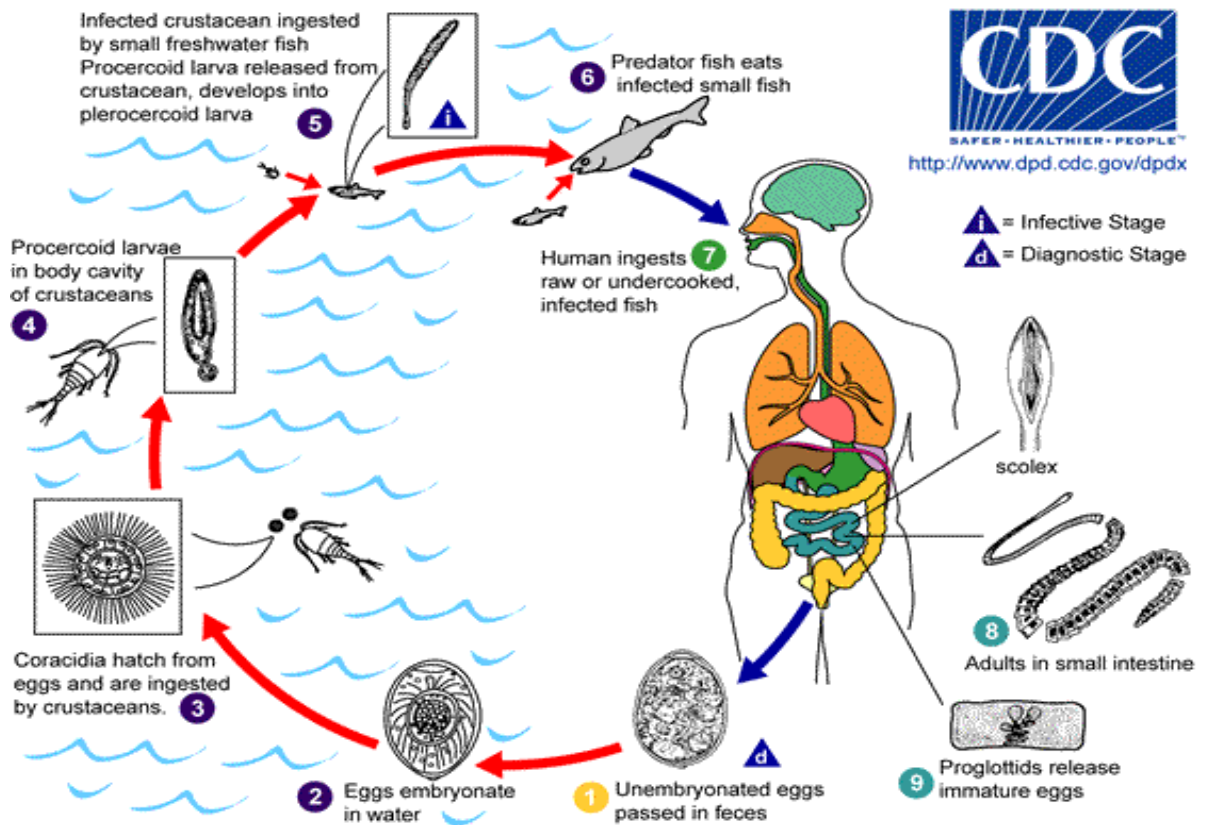
Life cycle of *D. latum*
http://www.dpd.cdc.gov/dpdx

3.1.3 Pathology of Infection

Infection, as is often the case with adult tapeworms, presents a variable range of pathology, but again is not commonly the cause of serious disease in man. Symptoms, when they occur, include a variety of non-specific abdominal signs, including abdominal pain and loss of weight, and are often very similar to the symptoms displayed during infection with adult *Taenia*. However, *D. latum* differs from *Taenia* in absorbing much more vitamin B12, (between ten and fifty times more) than other tapeworms. Infection may therefore result in a macrocytic hypochromic anaemia in some cases, vitamin B12 having an important role in formation of blood cells. This feature of the disease is much more common in the Baltic region, particularly in Finland. This tapeworm derived anaemia may be due to host derived genetic factors. It is also more commonly seen when the tapeworm is situated higher in the intestine.

3.1.4 Epidemiology and Control

Infection occurs by consuming raw or undercooked fish harbouring sparganum. Therefore to avoid infection in man, fish should be properly cooked, killing the infective plerocercoids.

3.2 *Dipylidium caninum* - (the Dog Tapeworm)

This Tapeworm is primarily a parasite of the Dog and the Cat. However man, and in particular children, may also be infected.

3.2.1 Lifecycle

Similar to *Taenia saginata,* the proglottids of this tapeworm are actively motile, and are able to crawl out of the anus of the definitive host as well as being passed in the faeces. The eggs of this species of tapeworm are contained in egg-capsules, each containing up to twenty eggs. These eggs are ingested by the parasite's intermediate host, in this case an invertebrate arthropod such as fleas (only the larval flea can be infected) or the dog louse *Trichodectes canis*. The onchosphere larvae is released in the arthropods gut and penetrates through the gut wall, developing into a cysticercoid, similar to the hymenolepid larval tapeworms. Infection of the Definitive host, whether dog, cat or man, occurs on ingestion of the larval parasite, either when the intermediate host is ingested, or ingestion of the crushed bodies of these hosts. For example, if the dog licks the face of the child just after it has bitten a flea or louse. On ingestion the cysticercoid larvae develops into the adult parasite in the small intestine in about twenty days.

3.2.2 Morphology

Larvae - The larvae are roughly pear-shaped, and follow the typical cysticercoid body pattern.

Adults - These are relatively short tapeworms, measuring between 15 and 17cm in length and consisting of up to 170 proglottids. These are elongated in form, the gravid proglottids, measuring approximately 12 x 3m and packed full of egg-capsules, having the appearance of grains of rice. The scolex by which the parasite attaches to the wall of the small intestine has four large acetabulate suckers, a

retractile rostellum and six rows of 30 to 150 rose-thorn shaped hooks. The eggs which are typical Cyclophyllidean tapeworm eggs, are round in shape and measure up to 60μm, and are held within egg-capsules.
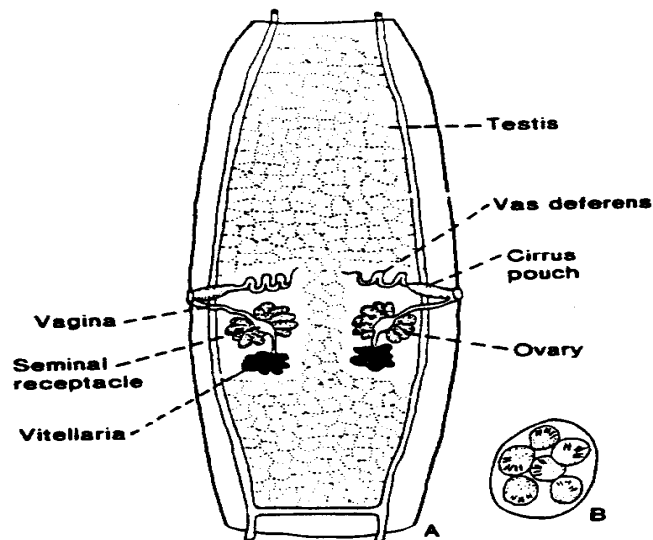
Dipylidium caninum. (A) Mature Proglottid; (B) Egg Packet Containing Eggs.

3.2.3 Pathology of Infection.

The infection appears to be asymptomatic and generally non-pathogenic, although there may be some degree of mild pruritis, or itching, around the perineum due to the presence of emerging proglottids.

3.3 Tapeworms of the Genus *Hymenolepis*

There are a number of species in this genus, two of which are common parasites of man.

H. nana - The Dwarf Tapeworm

This tapeworm is relatively small, growing up to 4cm in length, the size of the parasite being inversely proportional to the number of worms present in the

infection. Infections, which are more commonly seen in children in warmer climates, are characterised by the presence of numerous parasites (both cysticercoid larvae and adults) in the small intestine. Infection is by ingestion of soil contaminated with faeces containing eggs and may give rise to abdominal discomfort.

H. diminuta - The Rat Tapeworm

This tapeworm is much longer than *H. nana*, growing up to 60cm or more in length. This is primarily a parasite of the rat, humans only being infected by accidental ingestion of the insect intermediate host. This species is of more importance as a research model for the study of the biochemistry, physiology, chemotherapy and immunology of tapeworm infections. In addition there are a number of species found in animals, including;

H. carioca - A common non-pathogenic parasite of fowl in the USA.
H. microstoma - A parasite of rodents.
H. lanceolata - A pathogenic parasite of ducks, geese and other anseriform domestic fowl.
H. coronula- A parasite of anseriform domestic fowl.
H. cantaniana - A parasite of chickens and other galliform domestic fowl.

3.3.1 Morphology

Apart from their relative sizes, these two parasites of man are very similar, *H. nana* being up to 4cm in size, the strobila consisting of up to 200 proglottids, whilst *H. diminuta* grows up to 60cm or more in length and the strobila consists of up to 1000 proglottids. These proglottids are trapezoidal in shape, and are approximately four times as wide as they are long. Each proglottid contains three round testes, a bi-lobed ovary, a compact vitelline gland and a large uterus opening to a lateral genital pore (as does the cirrus). The scolex in both parasites have four suckers and a retractile rostellum which in *H. nana* is equipped with 20 - 30 hooks (the rostellum is unarmed in *H. diminuta*). Finally the eggs of the two species both have the characteristic thickened walls of all cestode eggs, but may easily be differentiated. Those of the yellowish brown *H. diminuta* eggs are much rounder than colourless *H. nana* eggs and are larger with 60 - 80µm in diameter. In *H. nana* the eggs are oval in shape, measuring ~ 40 by 50µm and contains an

oncosphere equipped with 3 pairs of embryonic hooks (i.e. a "hexacanth" larvae) and long wavy filaments (absent in *H. diminuta*) which lie in the space between the larvae and the egg shell wall.

The two species infecting man have rather different lifecycles which will be considered separately here.

3.3.1.1 *H. nana*

This parasite has rats and mice as well as man as the definitive host, and differs from *H. diminuta* and almost all other tapeworm in that an intermediate host is not required, although fleas and beetles may be used. The embryonated eggs are passed in the faeces where they contaminate soil. If the eggs are ingested by the definitive host the oncosphere is activated and breaks out of the egg and penetrates the gut villus. Here it develops as a cysticoid larvae in about 4 days before rupturing into the gut lumen. Once ruptured, the scolex attaches to the gut mucosa and the parasite develops into the adult tapeworm after about 15 to 20 days. If the insect intermediate hosts are utilised the lifecycle is similar to that of *H. diminuta* below. In heavy infections eggs liberated by adult worms in the intestine may hatch here rather than passing out of the body, to give autoinfection.
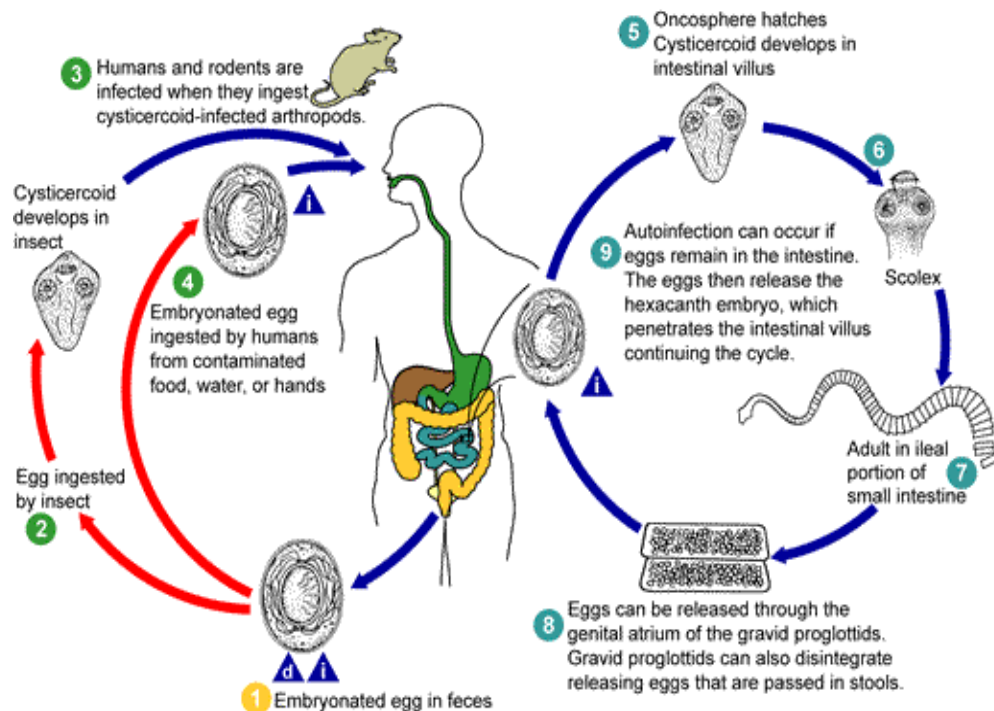
Life cycle of *H. nana*
http://www.dpd.cdc.gov/dpdx

3.3.1.2   *H. diminuta*

This parasite as in most tapeworms does require an intermediate host. Embryonated eggs pass out of the body of the definitive host in the faeces and are ingested by the insect intermediate hosts. Many insects may act as intermediate hosts for this parasite, the most common being fleas and beetles such as the flour beetle. When ingested by the intermediate host the oncosphere larvae become activated, break out of the egg shell and penetrate into the insect's body cavity where they develop into a cysticercoid larvae. For completion of the lifecycle, the infected intermediate host must be eaten by the definitive host. On ingestion, the cysticercoid larva becomes activated, the scolex becomes attached to the gut mucosal wall, and the parasite develops into the adult tapeworm.

An interesting feature of *Hymenolepis* tapeworms is that they undergo a diurnal migration within the gut, which is associated with the feeding patterns of the host. From about 4pm to 4am few parasites are seen in the lower part of the small intestine, whilst from about 4am to 4pm many parasites are seen in the upper part of the small intestine. This was first observed in *H. diminuta* and subsequently in other species, and is indicative of a nocturnal feeding pattern by the parasite.

3.3.2 Pathology of Infection.

These parasites are not very pathogenic, usually with asymptomatic infections. In man infected with *H. nana* there may be a slight irritation of the gut mucosa and slight abdominal pain, and with very heavy infections (>2000 worms) there may also be some diarrhoea. In the bird species there may be enteritis and intestinal obstruction with some species.

3.0 CONCLUSION

In this unit, you learnt about the morphology, life cycle, pathology and epidemiology of the broad fish tapeworm, dog tapeworm and tapeworm of man of the genus *Hymenolepis*

5.0 SUMMARY

D. latum 'a psedophyllidean' infects fish which in turn infects man when fed on raw or undercooked fish. Rats and mice as well as man are the definitive hosts of *H. nana*, and therefore difers from *H. diminuta* and almost all other tapeworm in that an intermediate host is not required, although fleas and beetles may be used. *Dipylidium caninum* are dog tapeworms that can as well infect human expecially children.

6.0 TUTOR-MARKED ASSIGNMENT

1. Explain the transmission cycle of *D. latum*

2.   What are morphological differences between the mature proglottids of *Dipylidium caninum* and *Hymenolepis* spp?

7.0   REFERENCE/FURTHER READINGS

Bonsdorff (1977). Diphyllobothriasis in man, New York, Academic Press, Inc.

UNIT 3   TAPEWORMS OF MAN AND OTHER HUMAN'S CESTODES

1.0   INTRODUCTION

Two species from the genus *Taenia* are common parasites of man, these being *Taenia solium* (the Pork tapeworm) and *Taenia saginata* (the Beef tapeworm). *Taenia saginata* has a cosmopolitan distribution, with estimates of approximately 50 million cases of infection world-wide annually. As with *T. saginata* and *T.*

solium this parasite has a cosmopolitan distribution, with estimates of approximately 50 million cases of infection world-wide annually. However the incidence of infection may vary considerably, and may be influenced by a number of factors such as religious inhibitions on eating pork, as in many Islamic countries, or in other countries by high degrees of sanitation, limiting exposure of the intermediate hosts to human faeces. This parasite has pigs as the main intermediate host, but man may also act as an intermediate host for this parasite as well as being infected with the adult tapeworms. This aspect of the parasites lifecycle has important implications for the pathology associated with infection with this parasite.

Echinococcus granulosus is one of the three species of *Echinococcus* that is generally accepted as parasites of man. It is the causative agent of Hydatid disease in man and many other mammals. It occurs in Europe and Artic region of North America.

3.0   MAIN CONTENTS

3.1   *Taenia* spp

3.1.1   Life cycle of *Taenia* spp

This parasite has cattle or related animals as its main intermediate hosts, although other animals such as camels, llamas and some antelopes may also occasionally be infected. The larval form in these animals is a cysticercus in the muscles and heart. These are infected by ingestion of the eggs of the tapeworm, shed from the faeces of the carnivorous definitive host, in this case man. Once ingested the eggs hatch to release the hexacanth larvae, which migrate through the intestinal wall to reach the blood or lymphatic systems, from where it is carried to the tissues, particularly the heart and other muscles to develop into the cysticercus. Man is infected by ingestion of undercooked or raw meat, the bladder wall of the cysticercus being digested in the intestine to release the scolex of the parasite. This attaches to the intestinal wall and grows into the mature adult tapeworm.
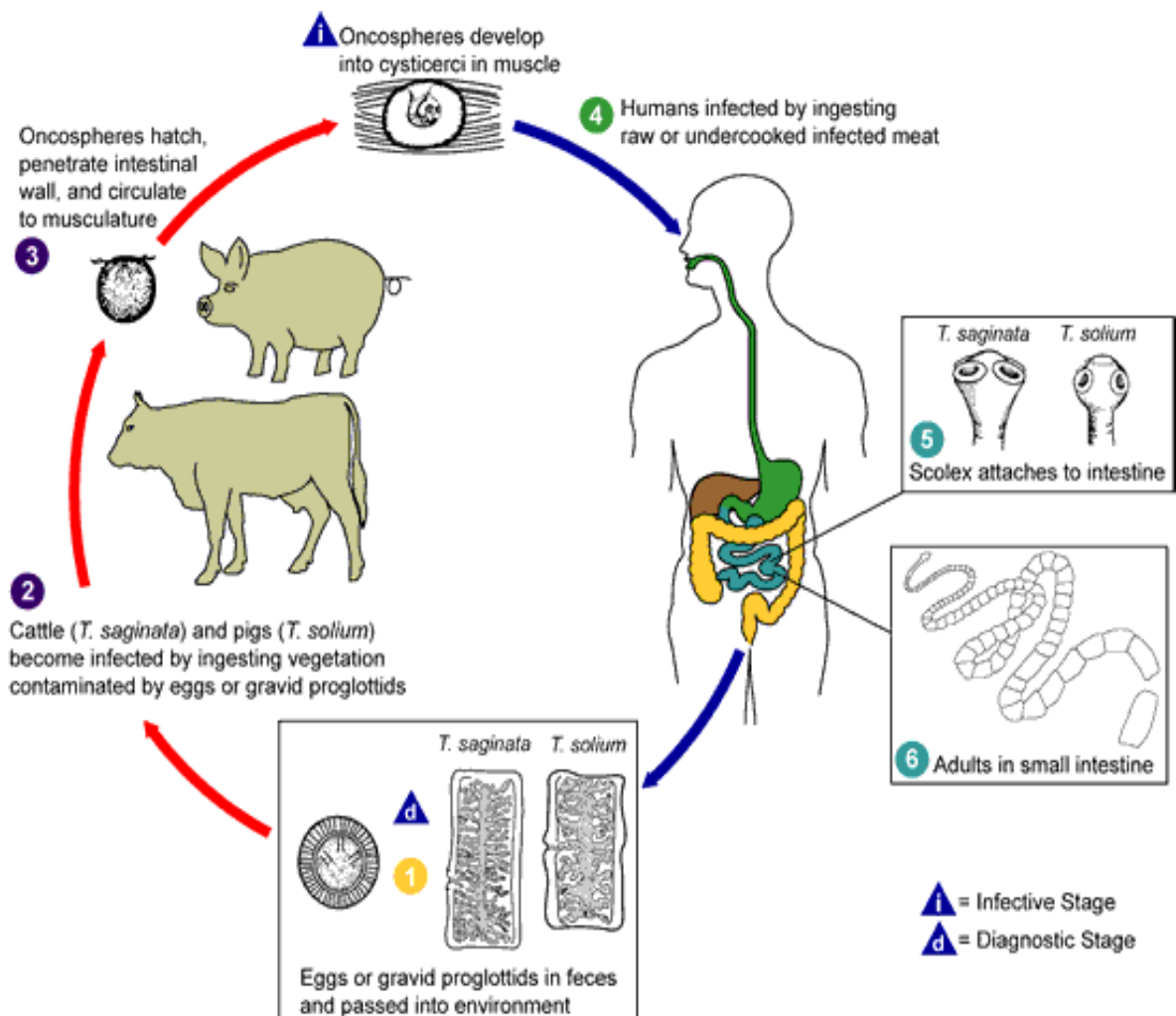
Life cycle of *T. saginata* and *T. solium*

3.1.2 Morphology

3.1.2.1 *Taenia saginata*

Larvae - These cysticerci are approximately 7.5-10mm wide by 4-6mm in length.

Adults - The adult tapeworms have an average length of about 5 meters, consisting of approximately 1000 proglottids, but may grow up to 17 metres in length occasionally, and are therefore longer than the adult forms of *Taenia*

solium. The mature proglottids have approximately double the number of testes that *T. saginata* has and are larger. The gravid proglottids are also larger, measuring approximately 20mm long by 6mm wide with a uterus with more lateral branches than *T. solium*. These gravid proglottids when detached from the strobila may be very active, not only crawling away from the faeces when passed, but often actively emerging from the anus to deposit eggs from the ruptured uterus around the perianal region. The scolex in this tapeworm may also be differentiated from *T. solium* as it is slightly larger, at approximately 2mm in diameter and is unarmed, without any hooks, although the 4 acetabular suckers are still present.

3.1.2.2   *Taenia solium*

Larvae - These small cysticerci (refered to as *Cysticercus cellulosae*) are approximately 6- 18mm wide by 4 - 6mm in length when found in the muscles or subcutaneous tissues (the normal sites for the larva of this parasite). The cysticerci may however be found in other tissues such as those of the central nervous system where they may grow much larger, up to several centimetres in diameter.

Adults - The adult tapeworms have an average length of about 3 meters, but may grow up to 8 metres in length occasionally, and follow the typical morphology of cestode tapeworms. The strobila consists of between 800 and 1000 proglottids. The mature proglottids having trilobed ovaries with a small central lobe in addition to the two lateral lobes and only approximately half the number of testes that *T. saginata* has. The gravid proglottids, measuring approximately 12mm long by 6mm wide, have a uterus with between 8 to 12 lateral branches, less than *T. saginata*. The scolex in this tapeworm may also be differentiated from *T. saginata* as it is equipped with a low rostellum with a double crown of approximately 30 hooks.
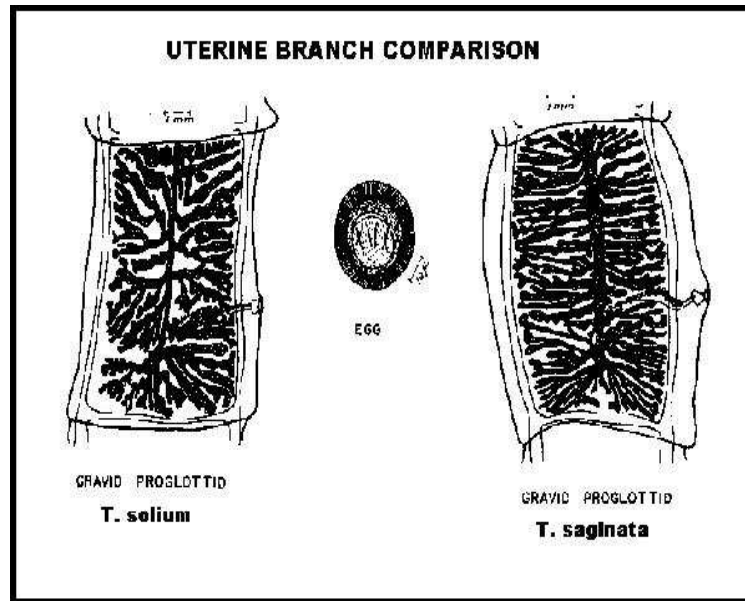
Figure showing the distinctions between the proglottid of *T. solium* and *T. saginata*

Major Differences between *T. saginata* and *T. solium*

Features	*T. saginata*	*T. solium*
Size	3-7m(sometimes Upto 25m) long	2-3m(sometimes up to 10m) long
No. of proglottids	1000-2000 (sheds 3-10 daily)	800-1000 (sheds 8-10 daily)
No. of eggs per proglottid	100000	40000
Scolex	Cuboidal, up to 2.0mm In diameter	Spheroid, about 1.0mm in diameter
Rostellum	Absent	Present, armed with two circlets of 22-32 hooks
No. of testes	800-1200	300-500
Shape of ovary	Bilobed	Trilobed
Gravid uterus	15-25 lateral branches on each side	7-13 lateral branches on each side
Vaginal sphincter	Present	Absent
Gravid proglottid	When detached, active and creep out through anus and craw about individually	When detached, passive

3.1.3   Pathology of Infection.

3.1.3.1 *T. saginata*

Larvae - Unlike *T. solium*, *T. saginata* does not utilise man as an intermediate host, and therefore pathology due to the larval form is not a feature in human disease. In cattle the cysticercus, refered to as *Cysticercus bovis* (named before the parasite life cycle had been determined, and the connection between the two forms had been established) is completely asymptomatic.

Adults - The pathology of infection with adult *T. saginata* is highly variable. Often infections are completely asymptomatic, but in other cases some degree of pathology may be seen, most seriously intestinal blockage. In some cases vitamin deficiency may be the result of excessive absorption of nutrients by the parasite, although this aspect of tapeworm pathology is more a feature of infection with the fish tapeworm *D. latum*. In addition infection may be accompanied by a broad range of non-specific symptoms, including more commonly, (if seen at all), abdominal pain, digestive disturbances, excessive appetite *or* loss of appetite, weakness and weight loss.

3.1.3.2   *T. solium*

Larvae - Infection with the larval form of *T. solium Cysticercus cellulosae*, (called "Cysticercosis") may have severe consequences, the annual world-wide mortality due to cysticercosis having been estimated at approximately 50 000 cases. In man the cysticerci mainly develop in the subcutaneous tissues, but infections in both the Central Nervous System (C.N.S.) and ocular tissues are also very common. Infection of the C.N.S. may cause severe pain, paralysis, optical and/or psychic disturbances and epileptic convulsions, mainly due to mechanical pressure as the larvae develop. Later there may be loss of consciousness and even death. infections involving the eye may give rise to discomfort, and can cause detachment of the retina.

Adults - Usually only a single adult specimen is present, which may cause a slight degree of mucosal inflammation. The actual effects on the host may vary considerably, often there are few symptoms, but in some cases a variety of non-specific symptoms such as constipation, epigastric pain and diarrhoea, are present.

Very rarely there may be perforation of the intestinal wall, with subsequent peritonitis. However, more seriously, as detailed above, the presence of adult worms carries the risk of autoinfection due to reverse-peristalsis resulting in cysticercosis, it being estimated that approximately 25% of cases of *Cysticercus cellulosae* infections in man being acquired by this route.

3.1.4 Diagnosis of Infection by *Taenia* spp

- Demonstration of scolex and proglottids in the faeces. However, scolex are rarely excreted in faeces
- The eggs of *T. saginata* and *T. solium* are similar. However, most laboratory diagnosis is through the observation of *Taenia* spp eggs in faecal sample.
- Examination of gravid uterus shows 15-25 lateral branches in *T. Saginata* and 7-13 lateral branches (counted from the main stem) in *T. Solium* when short chains of 5-8 proglottids passed out in faeces are pressed through glass slides.
- The scolex of *T. saginata* is easily distinguished from that of *T. solium* in that it has only 4 suckers but no hooks.
- Radiological examination of the intestinal tract may reveal tapeworm infection.

3.1.5 Epidemiology and control

The prevalence of *Taenia* infection is on the increase due to the following factors;
1. Intensification of animal production
2. Development of meat industries in several developing countries
3. Consumption of undercooked beef and pork by tourist visiting highly endemic areas
4. Consumption of semi-cooked meat in manufactured food products like hamburgers, etc.
5. Accelerating urbanisation with decreased efficiency of sewage systems
6. Sewage farming

In view of the above listed epidemiological factors that favour transmission, the following measures can be taken to reduce prevalence;
1. Proper meat inspection services before usage in meat industries. Diseased meat should be condemned and destroyed
2. Lightly infected beef with cysticerci can be rendered safe for consumption by freezing at -10°C for at least 10 days

3. Cooking of meat well before eating
4. High standards of sanitation will reduce transmission
5. Immunization against bivine cysticercosis.

3.2   Other *Taenia* cestodes

3.2.1 Infection by Adult tapeworms

Taenia taeniformis - This parasite has a cosmopolitan distribution, the adult parasites are normally found in cats and related carnivores, but it has been reported from an Argentinean child. The adult tapeworms are about 60cm long, and are unusual in that they lack a neck. The scolex is large and equipped with two rows of hooks, whilst the posterior gravid proglottids have a characteristic bell shape. The larvae, which is found in wild rodents, is a strobilocercus, a development of a cysticercus where the scolex has evaginated, but is still attached to the bladder of the cysticercus by a short segmented strobila.

Taenia bremneri (Syn. ***T. confusa***) - reported from man in Africa, Japan and the United State of America. This parasite may be a synonym of *T. saginata.*

Taenia africanus - reported a few times in East Africa. This tapeworm has broad segments and an unarmed scolex with a small apical sucker.

3.2.2 Infection by Larvae (Metacestode Infections)

Taenia multiceps - The adult tapeworms of this species are found in dogs and related canids. The larva is a fluid containing cyst 5cm or more in diameter, containing several hundred protoscolices, and is called a coenurus. It is normally found in the brain or spinal cord of sheep and goats where it is an important pathogen. In these animals, it causes a condition known as 'gid' or 'staggers' as the coenurus develops along with an associated destruction of nervous tissue. The larval form may rarely infect man, where it causes a condition called coenurus cerebralis, on accidental ingestion of tapeworm eggs from the faeces of dogs.

Taenia serialis - A similar parasite to *T. multiceps*, the coenurus larvae, measuring 4cm in diameter or larger, is usually found in the subcutaneous and intramuscular tissues of lagomorphs. The adult tapeworms are found in dogs and foxes with a cosmopolitan distribution. They measure about 70 cm in length and have a scolex

with two rows of about 30 hooks. The larvae have been reported very rarely in man.

Taenia glomerulatus - The larvae normally infect rodents, but the coenurus larvae have also been reported as rarely infecting man in Africa.

3.3   *Echinococcus* spp

Three species of *Echinococcus* have been generally accepted as parasites of man

3.3.1   *Echinococcus granulosus*

The causative agent of Hydatid disease in man and many other mammals. The dog acts as the definitive host for this species. A number of sub-species of this parasite have also been described, the most universally accepted being *E. g. granulosus* (thought to be the original species found in Europe, although now more widespread) and *E. g. canadensis* (the indigenous species of the Arctic region of North America, for more details see below). In addition there is considerable strain variation within this parasite, with differing preferences for intermediate hosts. For example in Ireland a strain exists whose larvae only infect the horse, man being resistant to infection.

3.3.1.1   Life cycle

Dogs and other canids are parasitised by the adult tapeworm. When shed by the tapeworm, the gravid proglottids disintegrate in the dogs intestine, and eggs which are passed in the faeces, are highly resistant, being able to survive freezing and drying on the ground for up to a year. Many mammals apart from man may act as intermediate hosts, in particular sheep and horses. The situation is highly complex as at least 9 sub-species have been identified, all with different host specificity;

E.g. granulosus - Adult form in most canids apart from the red fox, hydatids in sheep, pigs, cattle, man and many wild ruminants.

E.g. equinus - Adults in canids, hydatids in horses and other Equidae, but probably not man.

E.g. canadensis - Adults in canids, hydatids in caribou, reindeer and man.

E.g. borealis - Adults in canids, hydatids in many cervids and man.

In addition, in parts of Kenya there is a strain or sub-species that is particularly adapted to transmission between man and domesticated dogs.

The egg enters the host by ingestion, either from contaminated grass (as is the case in infections of herbivorous ruminants), or in the case of man, by contamination, (for example by the dog licking face after it has been cleaning itself) or other examples of bad hygiene, followed by transfer to the mouth. The egg then hatches in the intestine, penetrates the gut wall, and travels via the lymphatic or blood system throughout the body, from where they lodge within the body tissues. The cysts may develop anywhere within its intermediate hosts body, but as the circulatory blood stream passes from the mesenteric blood vessels to the liver, it is in the liver that the majority of the cysts (in about 65% of cases) are found. Next in frequency of infection are the lungs (about 20%), brain (1%), peritoneal cavity (8%), kidneys (3%) and bone marrow or other organs. Development of the cysts to produce infective protoscolices takes approximately 1 to 2 years. On the death of the intermediate host, either directly by predation on the part of the dog, or by the scavenging of the dead cadaver, (the protosocialises are also highly resistant, being able to survive in carrion for several weeks), the cyst is ingested along with the offal. The cyst wall is then digested, liberating the protoscolices which quickly evaginate, penetrating deeply into the crypts of Lieberkuhn, and developing to adult worms in approximately 7 to 9 weeks. Due to the presence of many protoscolices in each hydatid cyst, dogs may be infected with many *E. granulosus* (tapeworms).
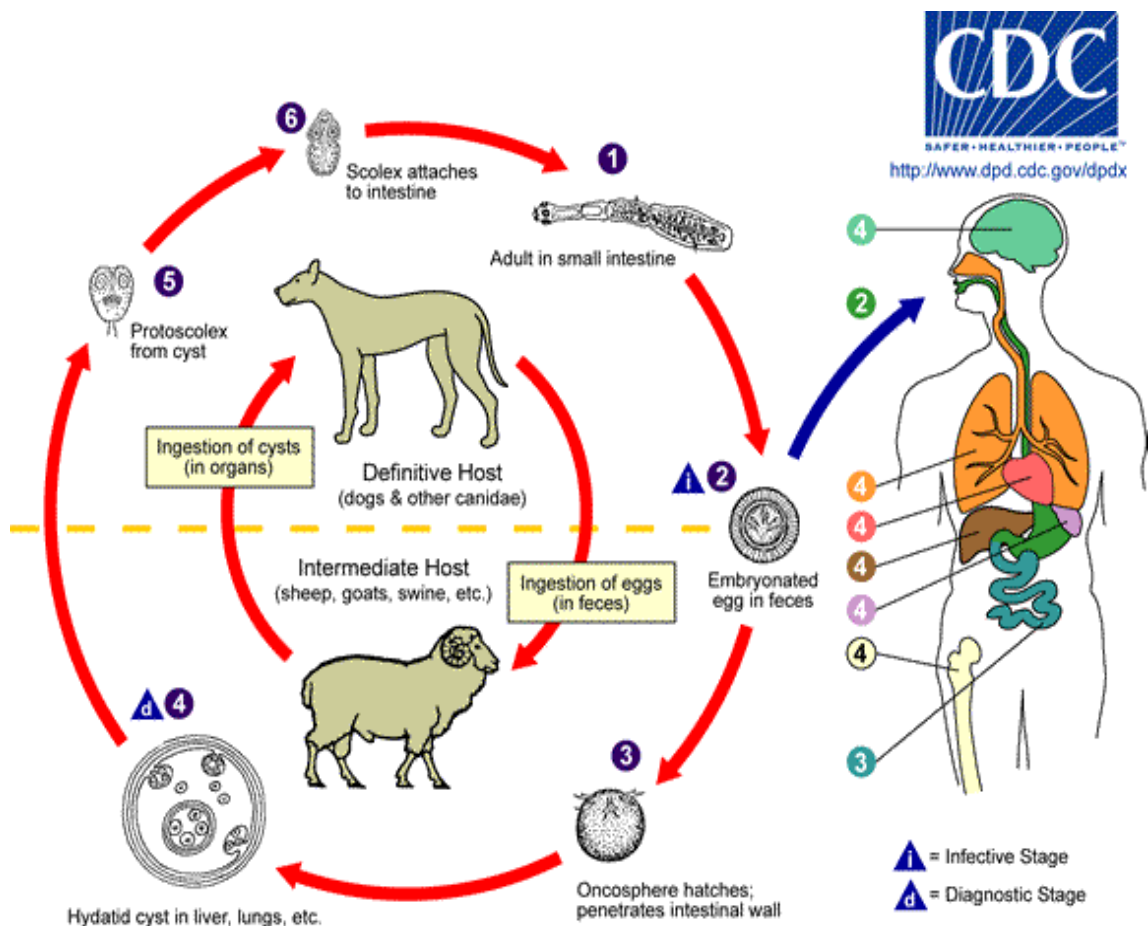
Life cycle of *E. granulosus*
http://www.dpd.cdc.gov/dpdx

3.3.1.2   Morphology

Larvae - These Metacestodes (called 'Hydatids') are large, roughly spherical, fluid filled hollow bladders, containing numerous protoscolices (forming the so-called hydatid sand), brood capsules, and daughter cysts which are identical in form to their parent cyst. The cyst wall itself consists of an outer laminated hyaline wall, supporting the whole cyst. Beneath this there is a nucleated germinal layer, studded with developing brood capsules, which may eventually break off to float freely in the fluid filled cyst. The protoscolices are formed within the brood capsules, which may rupture to give the free protoscolices in the hydatid fluid.

They vary considerably in size depending on where in the body they form, which may be almost any organ of the body. Those found in the liver (the most common

organ affected) may be approximately 20cm in diameter, but those found in the peritoneal cavity may sometimes be very much larger, containing several litres of fluid. For example one case has been reported of a cyst 50cm in diameter, containing 16 litre of fluid.

Adults - The adult parasites in the dog represent one of the smallest of the tapeworms. They measure between 3 and 9mm in length, and usually consist of only 3 proglottids, an immature, a mature, and a gravid proglottid. The scolex is globular in shape, and has a prominent rostellum, armed with a double row of between 30 and 36 hooks. The eggs are very similar to those of the genus *Taenia*, and measure between 30 and 40µm in diameter

3.3.1.3   Pathology of Infection.

Larvae - In domesticated animals clinical signs appear to be uncommon, whilst in man they will vary in their seriousness depending on where in the body the hydatid develops, and how large it grows. Sometimes, the infection is asymptomatic, the only evidence of infection being the presence of calcified cysts on autopsy after death due to an unrelated cause. The major pathology is due to the size of the cyst, giving rise to pressure related injury. A complication may arise if the cyst is ruptured, possibly due to blows to the body, muscular strain, or during operations. In this case the contents of the hydatid is released into the body's circulatory system, and the liberated protoscolices may give rise to numerous secondary cysts throughout the body. In addition the hydatid cyst fluid is highly allergenic and cyst rupture may result in anaphylactic shock and rapid death.

Adults - The adult tapeworm is usually non-pathogenic to its canine hosts, although sometimes in very heavy infections there may be some inflammation of the intestinal wall.

3.3.2   *Echinococcus multilocularis*

It is the causative agent of highly pathogenic Alveolar Hydatid disease in man and other mammals. The fox is the most important definitive host, although dogs, and occasionally cats, may also be infected with the adult parasite. Again there appears to be a number of sub-species of this organism, *E. m. multilocularis* in Europe and *E. m. sibiricencis* in North America.

This is very similar to that of *E. granulosus*, but with more adaptations for colder climates. For example the eggs are highly resistant to cold temperatures, being able to survive at -20° for more than 2 weeks. In addition the pre patent period in the definitive host is much shorter, usually between 4 to 5 weeks.

3.3.2.1　Morphology

Larvae - The larval *E. multilocularis* is very different from that of *E. granulosus*. In this case the 'cyst' grows invasively by external budding, forming a diffuse growth through the infected organ, replacing that organ's tissues. The growth itself, (it cannot truly be called a cyst as there is no real cyst wall), is composed of numerous cavities containing a gelatinous matrix within which protoscolices and numerous brood capsules are produced, and which in its behaviour, most closely resembles a malignant neoplasm. In contrast to *E. granulosus* this growth is also very rapid, infective protoscolesces being present after only 2 to 3 months, as compared to the 1 to 2 years in the related metacestode.

Adults - The adult parasite is very similar to *E. granulosus*, being slightly smaller, with a maximum length of approximately 4mm, and consisting of 4 to 5 proglottids.

3.3.2.2　Pathology of Infection

Larvae - The multilocular cyst is highly pathogenic due to its fast growth rate and invasive nature, in extreme cases completely replacing liver tissue. As the cyst lacks the tough laminated layer seen in *E. granulosus*, and by its nature grows by budding, metastases of growth may also be seen, colonising other body organs. Due to this aspect of the parasite, it may also be transferred by transplantation. This parasite must be considered one of the most pathogenic of the parasitic helminths.

Adults - As with *E. granulolsus* the adult tapeworm is usually non-pathogenic to its canine hosts.

- **CONCLUSION**

At the end of this unit, you learnt the morphology, pathology, life cycles and control of tapeworms.

5.0   SUMMARY

The two major tape worms infecting man are *Taenia saginata* (beef tape worms) and *T. solium* (pork tape worm). Infections by these tapeworms often occur following the consumption of raw or undercooked beef and pork. Others *Taenia* spp which have man as accidental host are *T. taeniformis, T. bremneri, T. multiceps, T. serialis* and *T. glomerulatus.*

Dog and other canids are the definitive hosts of *Echinococcus granulosus* with the hydatid cyst of the parasite causing the pathological effects seen in man. Proper cooking of beef and pork could prevent infection due to *T. saginata* and *T. solium* while good sanitary condition can as well prevent infection by *E. granulosus.*

6.0   TUTOR-MARKED ASSIGNMENT

1. Briefly discuss the epidemiology and control of human tapeworms

2. Based on the knowledge of transmission of *Echinococcus granulosus,* highlight the control measures of the parasite

7.0   REFERENCES/FURTHER READINGS

Ash, L. R. and Orihel, T.C. (1987). Parasites: a Guide to Laboratory Procedures and Identification, 1st ed. American Society of Clinical Pathologists Press, Chicago. 328 pp.

Baumeister, S., Pohlmeyer, K., Kuschfeldt, S., Stoye, M. (1997). Prevalence of Echinococcus multilocularis and other metacestodes and cestodes in the muskrat (Ondatra zibethicus LINK 1795) in Lower Saxony. Dtsch Tierarztl Wochenschr. 104(10):448-52.

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MODULE 4   **NEMATODES**

Unit 1 General features and life cycles of nematodes
Unit 2 Soil transmitted helminths
Unit 3 Blood and Tissue nematodes
Unit 4 Air-borne nematodes

UNIT 1   GENERAL FEATURES AND LIFE CYCLES OF NEMATODES

CONTENTS

1.0   Introduction
2.0   Objectives
3.0   Main contents
       3.1   General features
       3.2   The Basic Life cycle of the Major Groups of Nematodes

 4.0   Conclusion
 5.0   Summary
 6.0   Tutor-Marked Assignment
 7.0   References/Further Readings

1.0   INTRODUCTION

Nematode infections in humans include ascariasis, trichuriasis, hookworm, enterobiasis, strongyloidiasis, filariasis, and trichinosis, among others. The phylum Nematoda, also known as the roundworms, is the second largest phylum in the animal kingdom, encompassing up to 500,000 species. Members of Nematoda are elongated, with bilaterally symmetric bodies that contain an intestinal system and a large body cavity. Many roundworm species are free living in nature. Recent data have demonstrated that approximately 60 species of roundworms parasitize humans. Intestinal roundworm infections constitute the largest group of helminthic diseases in humans. According to a 2005 report by the World Health Organization (WHO), approximately 0.807-1.221 billion humans have ascariasis, 604-795 million have trichuriasis, and 576-740 million have hookworm infections worldwide.

2.0   OBJECTIVES

At the end of this unit, students should be able to:

1. List the various examples of nematodes with thier common names
2. Describe the general morphological features of nematodes
3. Explain the life cycles of the major groups of nematodes

3.0 MAIN CONTENTS

3.1 General features

Nematodes are cylindrical rather than flattened; hence the common name roundworm. The body wall is composed of an outer cuticle that has a noncellular, chemically complex structure, a thin hypodermis, and musculature. The cuticle in some species has longitudinal ridges called alae. The bursa, a flap-like extension of the cuticle on the posterior end of some species of male nematodes, is used to grasp the female during copulation.

The cellular hypodermis bulges into the body cavity or pseudocoelom to form four longitudinal cords; a dorsal, a ventral, and two lateral cords which may be seen on the surface as lateral lines. Nuclei of the hypodermis are located in the region of the cords. The somatic musculature lying beneath the hypodermis is a single layer of smooth muscle cells. When viewed in cross-section, this layer can be seen to be separated into four zones by the hypodermal cords. The musculature is innervated by extensions of muscle cells to nerve trunks running anteriorly and posteriorly from ganglion cells that ring the midportion of the esophagus.

The space between the muscle layer and viscera is the pseudocoelom, which lacks a mesothelium lining. This cavity contains fluid and two to six fixed cells (celomocytes) which are usually associated with the longitudinal cords. The function of these cells is unknown.

The **alimentary canal** of roundworms is complete, with both mouth and anus. The mouth is surrounded by lips bearing sensory papillae (bristles). The oesophagus, a conspicuous feature of nematodes, is a muscular structure that pumps food into the intestine; it differs in shape in different species.

The intestine is a tubular structure composed of a single layer of columnar cells possessing prominent microvilli on their luminal surface.

The excretory system of some nematodes consists of an excretory gland and a pore located ventrally in the mid-esophageal region. In other nematodes this structure is drawn into extensions that give rise to the more complex tubular excretory system, which is usually H-shaped, with two anterior limbs and two posterior limbs located in the lateral cords. The gland cells and tubes are thought to serve as absorptive bodies, collecting wastes from the pseudocoelom, and to function in osmoregulation.

Nematodes are usually bisexual. Males are usually smaller than females, have a curved posterior end, and possess (in some species) copulatory structures, such as
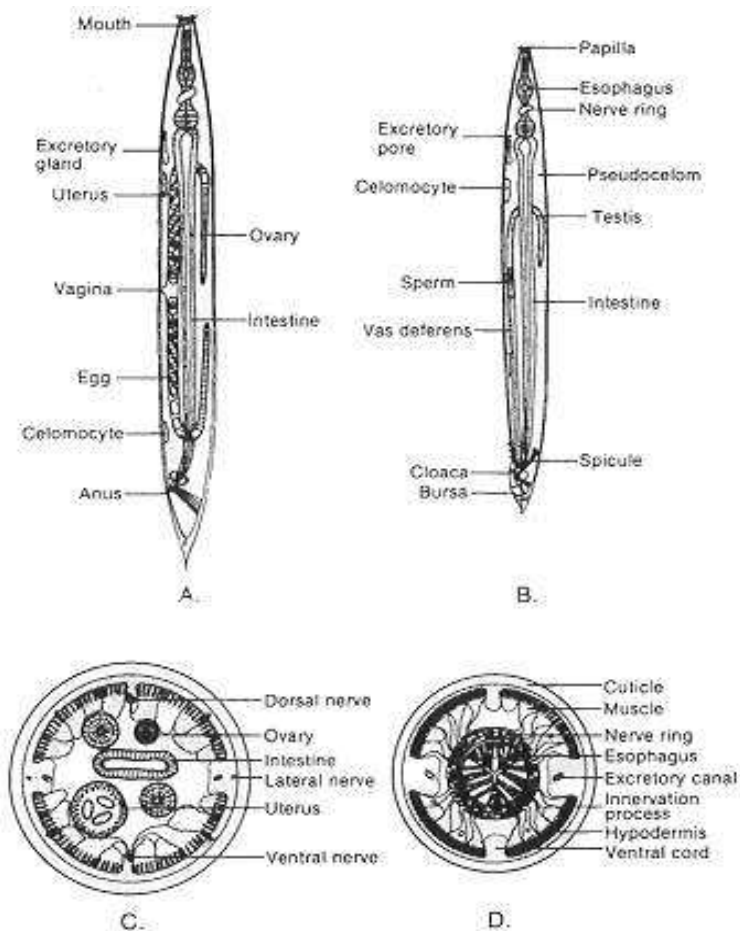
Structure of nematodes. (A) Female. (B) Male. Transverse sections through the mid region of the female worm (C) and through the esophageal region (D).

spicules (usually two), a bursa, or both. The **males** have one or (in a few cases) two testes, which lie at the free end of a convoluted or recurved tube leading into a seminal vesicle and eventually into the cloaca.

The **female** system is tubular also, and usually is made up of reflexed ovaries. Each ovary is continuous, with an oviduct and tubular uterus. The uteri join to form the vagina, which in turn opens to the exterior through the vulva.

Copulation between a female and a male nematode is necessary for fertilization except in the genus *Strongyloides,* in which parthenogenetic development occurs (i.e., the development of an unfertilized egg into a new individual). Some evidence indicates that sex attractants (pheromones) play a role in heterosexual mating. During copulation, sperm is transferred into the vulva of the female. The sperm enters the ovum and a fertilization membrane is secreted by the zygote. This membrane gradually thickens to form the chitinous shell. A second membrane, below the shell, makes the egg impervious to essentially all substances except carbon dioxide and oxygen. In some species, a third proteinaceous membrane is secreted as the egg passes down the uterus by the uterine wall and is deposited outside the shell. Most nematodes that are parasitic in humans lay eggs that, when voided, contain either an uncleaved zygote, a group of blastomeres, or a completely formed larva. Some nematodes, such as the filariae and *Trichinella spiralis*, produce larvae that are deposited in host.

3.2   THE BASIC LIFE CYCLE OF THE MAJOR GROUPS OF NEMATODES

The life cycles of the parasitic species vary considerably, as would be expected from such a large and diverse group. There are however a number of common features. Firstly, the parasite undergoes a series of moults through larval stages (designated **L1** to the adult **L5** form). Secondly, in most (but not all) nematodes it is the **L3** larvae that is the infective form, important exceptions to this being the Ascarids, such as *Ascaris lumbricoides* and the pinworms, where it is either the **L1** larvae, or eggs containing **L1** or **L2** larvae that are infective. Thirdly the **L3** form onwards in all species undergoes a migration within the body of the definitive host as it matures into the adult parasite, usually via the bloodstream or lymphatic system to the heart, lungs, trachea, and then to the intestine. Finally, in most cases

the parasite leaves the definitive host as thin walled eggs in the faeces, important exceptions being the viviparous filarial worms (where **L1** larvae infect intermediate hosts, usually in the blood meals of biting arthropods), *Strongyloides stercoralis*, (where the **L1** larvae are found in the faeces), and the viviparous *Trichinela spiralis*, where the larvae do not leave the body as such, but develop to the **L3** stage which then encysts in the muscles, infection being by ingestion of undercooked contaminated meat. Infection of the definitive host may be by a variety of routes, such as the oral route, where eggs are accidentally ingested, also many filarial worms are infective via the bite of flies, as previously decribed, and the **L3** larvae of many nematodes such as the hookworms and other related nematodes are directly invasive.

In terms of complexity, the simplest life cycles are those of the pinworms, where adults living in the colon mate and lay eggs which pass out in the faeces, infection being either by the oral route with eggs, or perianally, where eggs hatch around the anus and **L1** larvae migrate back through the anus.

The most diverse is probably that of *S. stercoralis*, where there are a number of alternative lifecycles which it may undergo, either as a completely free living soil nematode, or as the standard infective **L3** larvae with tissue migration to the intestine, or even occasionally full completion of the life cycle within the intestine, and finally in immunocompromised hosts a life-threatening disseminated infection can occur, with parasites found throughout the body.

4.0 CONCLUSION

In this unit, you learnt the general structures of nematodes and the basic life cycles of the major groups.

5.0 SUMMARY

Nematodes are roundworms with pseudocoelom (lacking a mesothelium lining). The alimentary canal is complete having mouth and anus. The intestine is a tubular structure composed of a single layer of columnar cells possessing prominent microvilli on their luminal surface. The excretory system of some nematodes consists of an excretory gland and a pore with complex tubular excretory system, which is usually H-shaped. Nematodes are usually bisexual. Males are usually smaller than females. Copulation between a female and a male nematode is necessary for fertilization except in the genus *Strongyloides,* in which

parthenogenetic development occurs (i.e., the development of an unfertilized egg into a new individual). Parasite undergoes a series of moults through larval stages with L3 larva mostly being the infective stage. Infection of the definitive host may be by accidental ingestion of eggs, bite of flies and skin penetration by the infective L3 larval form as in the case of hookworms.

6.0   TUTOR-MARKED ASSIGNMENT

1. With the aid of well labeled diagrams describe the general features of roundworms.
2. Highlight the basic life cycles of nematode

7.0   REFERENCE/FURTHER READINGS

Ukoli, F.M.A. (1990). Introduction of Parasitology in Tropical Africa. John Wiley and Sons Ltd., Chichester.

UNIT 2   **SOIL TRANSMITTED HELMINTHS**

CONTENTS

- **INTRODUCTION**

Soil-transmitted helminth (STH) infection is highly endemic in tropical and subtropical areas of sub-Saharan Africa, Asia and Latin America, where up to 2 billion people have active infections. STH infection has remained largely

neglected by the global health community because the people most affected are among the most impoverished and because the infection causes chronic ill health with insidious clinical presentations, rather than severe acute illness or high mortality. However, it is now recognized that STH infection causes significant morbidity worldwide with 39 million disability adjusted life years (DALYs) lost each year - more than those lost to malaria (36 million yearly) and approaching those lost to tuberculosis (47 million yearly). Hookworm infection alone causes the loss of 22 million DALYs.

2.0 OBJECTIVES

At the end of this unit, the students should be able to

1. Give examples of soil transmitted helminths(STHs)
2. Describe their life cycles with emphasis on the route of infection
3. Describe the diagnostic features of the parasites

3.0 MAIN CONTENTS

3.1 *Ascaris lumbricoides* (large roundworm of man)

Infection with this roundworm is extremely common, with estimates of the annual incidence of infection being greater than 1500 million cases, or around one quarter of the worlds population. In addition to the species in man, *Ascaris lumbricoides*, a morphologically indistinguishable species *Ascaris suum* is found in the pig. Other related genera include *Parascaris* in equines, and *Toxascaris* in a variety of domesticated animals.

3.1.1 Morphology

The adult *Ascaris lumbricoides* are large white, or pinkish-white, cylindrical roundworms, slightly narrower at the head. The more slender males measure between 10 to 30cm long and have a curved tail with two spicules, but no copulatory bursa. The females are very similar, being slightly larger at between 20 to 35cm long, a vulva approximately a third of the length of the body down from the head, and have a blunt tail. They are both characterised by having a smooth, finely striated, cuticle, and a mouth, which is characteristic of all of the Ascarids

(e.g. *Toxocara*), having three lips each equipped with small papillae. Internally they follow the generalised body plan of all nematodes, and have a cylindrical oesophagus opening into a flattened ribbon like intestine.

The eggs consist of a thick transparent inner shell which is covered in a thick, warty, albuminous coat.

Eggs unfertilized (left) fertilized (right) of *A. lumbricoides*.

3.1.2   Life cycle

These parasites have a direct life cycle, with no intermediate hosts. The adult parasite lives in the lumen of the small intestine of man, usually only feeding on the semi-digested contents of the gut, although there is some evidence that they can bite the intestinal mucous membrane and feed on blood and tissue fluids. The female parasite is highly prolific, laying an estimated 2 million eggs daily. In the intestine, these only contain an unembryonated mass of cells, differentiation occurring outside the host. This requires a temperature less than 30°C, moisture and oxygen, before the development of the young L1 larvae after approximately 14 days. Eggs containing the L2 larvae take another week to develop, before they are infective to man, and may remain viable in the soil for many years if conditions are optimal. Infection occurs on ingestion of raw food, such as fruit or vegetables, that is contaminated with these infective eggs. The eggs then hatch in the small intestine, to release the L2 rhabditiform larvae (measuring approximately 250 by 15µm in size. These do not simply grow into the adult forms in the intestine, but must then undergo a migration through the body of their host. These L2 larvae penetrate the intestinal wall, entering the portal blood stream, and then migrate to the liver, then heart, then after between 1 to 7 days, the lungs. Here they moult twice on the way to form the L4 larvae, (measuring approximately 1.5mm

long), then burrow out of the blood vessels, entering the bronchioles. From here they migrate up through the air passages of the lungs, to the trachea. They then enter the throat and are swallowed, finally ending up in the small intestine where they mature and mate, to complete their life cycle.

3.1.3 Pathology of Infection.

The majority of infections (~85%) *appear* to be asymptomatic, in that there is no gross pathology seen. However the presence of these parasites appears to be associated with the same general failure to thrive in their hosts seen with many of these intestinal nematodes. In terms of more easily identified pathology, this may be divided into three areas;

Pathology Associated with the Ingestion and Migration of Larvae

Severe symptoms of *Ascaris* infection may be associated with the migrating larvae, particularly in the lungs. If large numbers of these larvae are migrating through the lungs simultaneously this may give rise to a severe haemorrhagic pneumonia. More commonly, as is the case with most infections, the haemorrhages are smaller in scale, but still may lead to breathing difficulties, pneumonia and/or fever. A complication here is that many of the parasites proteins are highly allergenic. Due to this the presence of the migrating larvae in the lungs is often associated with allergic hypersensitivity reactions such as asthmatic attacks, pulmonary infiltration and urticaria and oedema of the lips.

Pathology Associated with Adult Parasites in the Intestine

The most common symptoms of infection are due to the adult parasite, and consist of rather generalised digestive disorders, such as a vague abdominal discomfort, nausea, colic (e.t.c.). These symptoms are dependent to some extent on the parasite's burden of the host, which in severe cases may consist of many hundreds or even thousands of parasites, although these are extreme cases. In the case of these heavy infections the presence of many of these large parasites may contribute to malnutrition in the host, especially if the hosts (often children) are undernourished. A more serious, and potentially fatal, condition may arise in these more heavy infections, where the mass of worms may block the intestine and need to be surgically removed. This may also occur sometimes on treatment for other

intestinal nematodes such as hookworms, where the curative drug dose for these parasites irritates the ascarids.

Pathology due to "Wandering" Adults outside of the Intestine

Adult parasites often leave the small intestine to enter other organs, (sometimes in response to anti-helminthic drugs used to treat other intestinal nematode infections), where they may cause various types of pathology, sometimes with severe consequences. For example adult *Ascaris* worms may migrate to the bile duct, which may then become blocked causing jaundice and a general interference in fat metabolism. Adult parasites may also migrate to the appendix, or through the intestinal wall, both conditions which may cause a fatal peritonitis as they may well carry intestinal bacteria to these sites. They may, alarmingly, sometimes migrate forward through the intestinal tract, to be either vomited up or emerging through the nose. More seriously, if they enter the trachea they may cause suffocation.

3.1.4 Diagnosis

Definitive diagnosis is by demonstration of the characteristic eggs in faecal samples or by identifying adult worms passed out spontaneously by the host.

3.1.5 Epidemiology and Control

Infection occurs through ingestion of parasites' eggs in food. The eggs are highly resistant to adverse environmental conditions. This with other factors highlighted below are often associated with transmission of infection;
- Lack or inadequate waste disposal facilities
- Improper washing of hands before eating
- Improper washing of fruits and vegetables before consumptions
- Unkept rooms and dwelling places that harbour mechanical carriers of parasites, etc.

Provision of good waste disposal system and good personal hygiene will help to control infections.

3.2 The human hookworms

The hookworms belong to the Order Strongylida, a very large order, and of great interest as it contains many important pathogens of man and domesticated animals. This order is further subdivided into three Superfamilies, the Strongyloidea (the hookworms in man), and two related groups, the Superfamily 'Trichostrongyloidea', intestinal nematodes which are of veterinary importance in many domesticated animals (e.g. *Haemonchus contortus* in cattle and *Nippostrongylus brasiliensis* in rodents) and members of the Superfamily 'Metastrongyloidea' (the lungworms, in domesticated animals).

In man there are two species capable of causing intestinal infections, *Ancylostoma duodenale* native to parts of Southern Europe, North Africa and Northern Asia parts of Western South America, and *Necator americanus* in Central and Southern Africa, Southern Asia, Australia and the Pacific Islands. These are very important human pathogens. It has been estimated that there are 1200 million cases of hookworm infection in man annually, of which about 100 million of which are symptomatic infections with accompanying anaemia. In addition, the larvae of several species of hookworms infecting domesticated animals may penetrate human skin, causing pathology even though they do not develop to the adult parasites in man.

3.2.1 Morphology

The adult parasites are small cylindrical worms, 0.5-1.5mm long (*Ancylostoma duodenale* being slightly larger than *Necator americanus*). The posterior end of the male worm is equipped with a characteristic copulatory bursa, used to hold the female nematode in place during mating. The females themselves have a vulva situated near the center of the body, slightly anterior in *Necator* and slightly posterior in *Ancylostoma*. The anterior end of the parasites are formed into a buccal

Scanning electron micrograph of the mouth capsule of *Ancylostoma duodenale* (left), note the presence of four "teeth," two on each side and *Necator americanus* (right)

capsule, absent in members of the other Strongylida superfamilies, by which the different genera and species within the group may be differentiated. For example members of the genus *Necator* have capsules equipped with cutting plates on the ventral margins, and within the capsule itself small dorsal teeth. In contrast members of the genus *Ancylostoma* have pairs of teeth on the ventral margin of the capsule. The number of teeth will vary between different species of *Ancylostoma* , but is usually between one and four pairs.
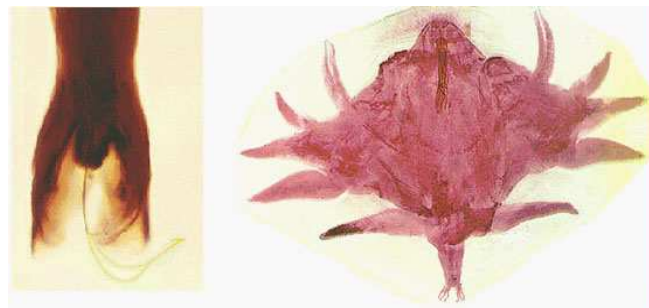
Left picture: Copulatory bursa and spines of *N. americanus* (a side view); Right picture: copulatory bursa of *A. duodenale* (a top view)

The eggs are bluntly rounded, thin shelled, and are almost indistinguishable between the different species, measuring approximately 60 by 40 µm, the eggs of *Ancylostoma* being slightly larger than those of *Necator*.

The Morphological Differences between Two species of Hookworms

| Features	*A. duodenale*	*N. americanus*
Size	Larger	Smaller
Shape	single curve, looks like C	double curves, looks like S
Mouth	2 pairs of ventral teeth	1pair of ventral cutting plates
Copulatory bursa	circle in shape	oval in shape
Copulatory spicule	1pair with separate endings	1pair of which unite to form a terminal hooklet
Caudal spine	Present	No
Vulva position	post-equatorial	pre-equatorial

3.2.2 Life cycle

The life cycles of all the hookworms are very similar. The eggs are passed in the faeces, once exposed to air they mature rapidly if conditions are right, with both moisture and warmth essential for development. When matured, they hatch to liberate a rhabditiform (i.e. having an oesophagus where a thick anterior region is connected via a neckline region with a posterior bulb) L1 larvae after a few days.

These larval nematodes feed on bacteria and organic material in the soil, where they live and grow for about two days before undergoing the first moult. After about five days more growth they moult again, to produce a much more slender L3 larvae. The L3 larvae has a much shorter oesophagus, is a non-feeding form, and is the infective form of the parasite. Infection takes place by penetration of the skin, for example when walking with bare feet over contaminated damp soil, followed by entry into the circulatory system. Here they are carried to the heart, and then lungs. Once in the lungs, they are too large to pass through the capillary bed there. Instead they become trapped, and the burrow through the capillary epithelium, entering the air spaces. They then migrate up through bronchi and trachae, and are then swallowed. Once swallowed, they pass into the intestine and bury themselves between the intestinal villi. Here they moult to form the L4 larvae, equipped with a buccal capsule allowing adherence to the gut wall. After

about thirteen days post-infection they moult for the final time, producing immature adult worms. These mature over three to four weeks (i.e. five to six weeks after infection), then mate and commence egg laying to complete the life cycle. These parasites show a very high fecundity, female *Necator americanus* producing up to 10,000 eggs daily, while female *Ancylostoma duodenale* produces up to 20,000 eggs daily.
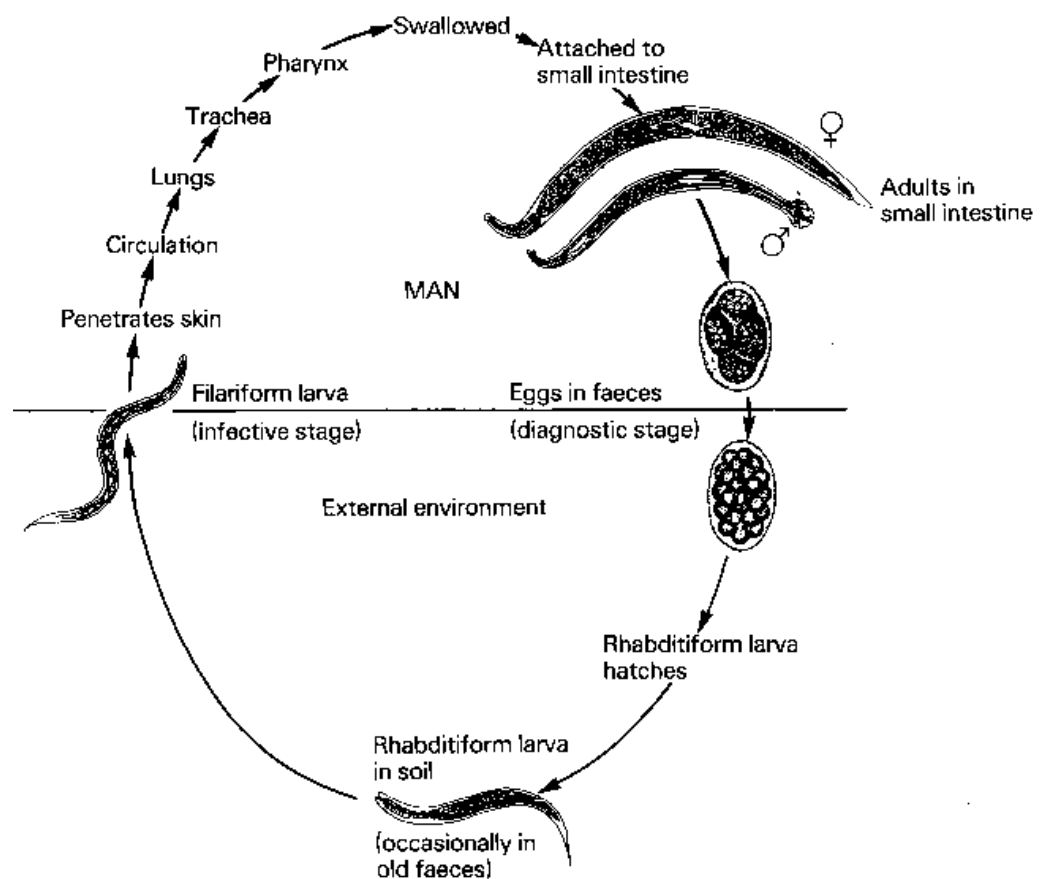

Life cycle of Human Hookworms

3.2.3 Pathology of Infection.

The Pathology associated with hookworm infections may be divided roughly into two areas. Firstly the pathology associated with the presence of the adult parasite in the intestine, and secondly the pathology associated with the penetration of, and migration of the larval worms within the skin.

The adult hookworms attach themselves to the intestinal wall using their buccal capsules. Their preferred site of infestation is in the upper layer of the small intestine, but in very heavy
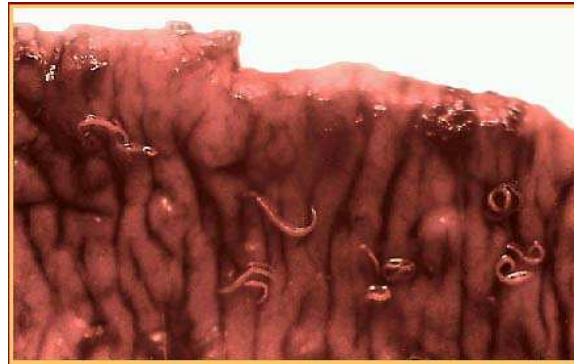

Adults in intestinal mucosa

infections (where many thousands of worms may be present) the parasites may spread down as far as the lower ileum. Once attached to the intestinal wall, the hookworm mouthparts penetrate blood vessels, and the parasites obtain nutrition by sucking blood. A single *Necator americanus* will take approximately 30 µl of blood daily, while the larger *Ancylostoma duodenale* will take up to 260 µl. The gross pathology of the disease is very dependent on the intensity of infection. Light infections appear asymptomatic, but in heavy infections, the continuous loss of blood leads to a chronic anaemia, with down to 2gm of haemoglobin per 100ml of blood in extreme cases. Experiments carried out in the 1930's showed that in dogs infected with 500 *Ancylostoma caninum* a similar species to the human parasite, nearly a pint of blood a day was lost. This leads to permenant loss of iron and many blood proteins as well as blood cells. This in turn has consequences for further production of erythrocytes, which have been shown to contain less haemoglobin, as well as being reduced in size and smaller in numbers. This form of anaemia may be directly fatal, but more often, it induces more non-specific symptoms, the most noticeable being the severe retardation in growth and development, both physical and mental, in infected children, and a general weakness and lassitude, often wrongly interpreted as "laziness".

3.2.4 Diagnosis
Identify characteristic eggs in feacal samples. Note the eggs of *N. americanus* and *A. duodenale* are morphologically identical.
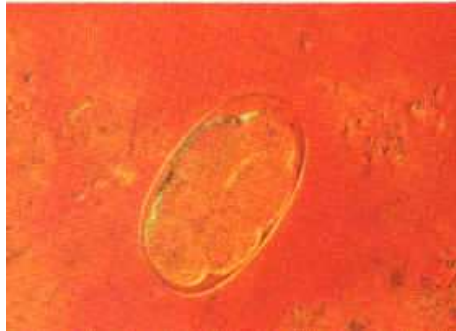
Egg of Hookworm

3.2.5 Epidemiology and Control
The factors of epidemiological importance include;

- Poor sanitation through contamination of soil through direct defaecation on the ground.
- Skin exposure to infections e.g. by walking about bare-footed
- Favourable environmental conditions that enhance eggs and larval development.
- Loose, humus soil with reasonable drainage and aeration
- Even distribution of rainfall throughout the year.

Control is by improvement in the standard of sanitation, raising the nutritional status of the population especially in relation to iron content, and mass treatment with suitable worm expeller (vermifuge)

3.3 *Trichuris trichiura* (human whipworm)

The first written record of *Trichuris trichiura* was made by Morgani, an Italian scientist, who identified the presence of the parasite in a case of worms residing in the colon in 1740. Exact Morphological description and figures were first recorded in 1761 by Roedere, a German physicist. Soon after morphology and visual representation of the worms, *Trichuris trichiura* was given taxonomy (during the 18th century).

This is the third most common round worm of humans. It is distributed worldwide, with infections more frequent in areas with tropical weather and poor

sanitation practices, and among children. It is estimated that 800 million people are infected worldwide. The southern United States is endemic for trichuriasis.

3.3.1 Morphology

Adult worms are usually 3–5 cm long, with females being larger than males as is typical of nematodes. The thin, clear majority of the body (the anterior, whip-like end) is the oesophagus, and it is the end that the worm threads into the mucosa of the colon. The widened, pinkish gray region of the body is the posterior, and it is the end that contains the parasite's intestines and reproductive organs.Trichuris trichiura has characteristic football-shaped eggs, which are about 50-54µm long and contain polar plugs (also known as refractile prominences) at each end.
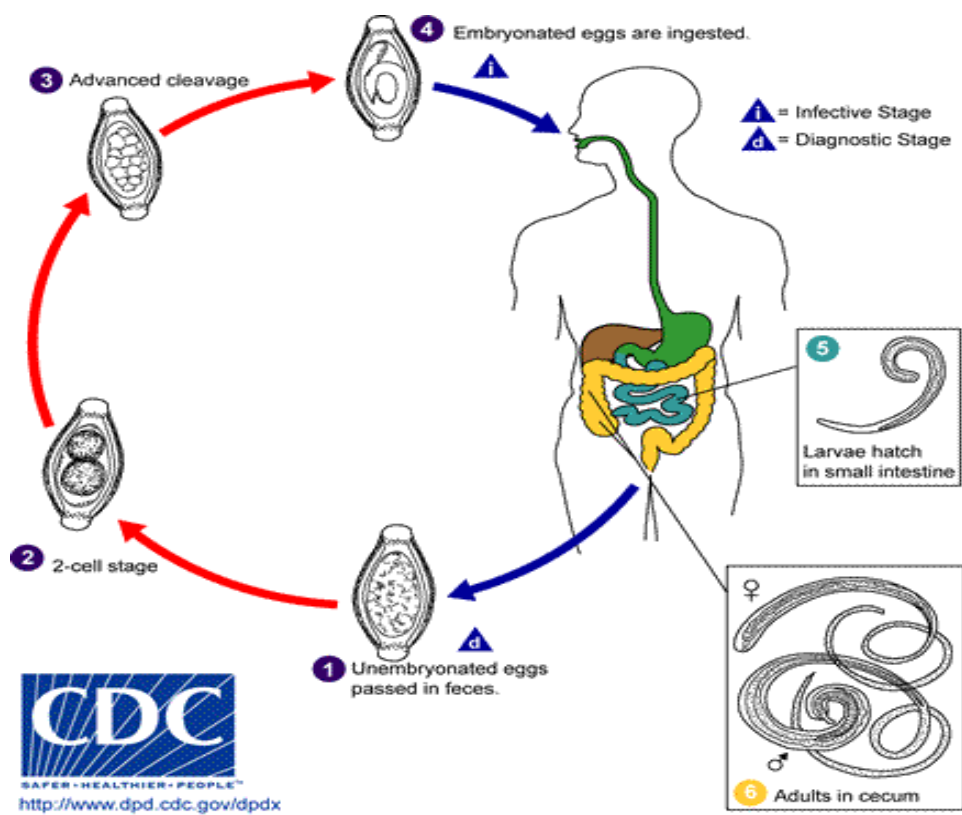

Egg and adult of *Trichuris trichiura*

3.3.2 Life cycle and transmission

Humans can become infected with the parasite due to ingestion of infective eggs by mouth contact with hands or food contaminated with egg-carrying soil. However, there have also been rare reported cases of transmission of *Trichuris trichiura* by sexual contact. Some major outbreaks have been traced to contaminated vegetables (due to presumed soil contamination).

Unembryonated eggs (unsegmented) are passed in the faeces of a previous host to the soil. In the soil, these eggs develop into a 2-cell stage (segmented egg) and then into an advanced cleavage stage. Once at this stage, the eggs embryonate and then become infective, a process that occurs in about 15 to 30 days). Next, the

infective eggs are ingested by way of soil-contaminated hands or food and hatch inside the small intestine, releasing larvae into the gastrointestinal tract. These larvae burrow into a villus and develop into adults (over 2–3 days). They then migrate into the cecum and ascending colon where they thread their anterior portion (whip-like end) into the tissue mucosa and reside permanently for their year-long life span. About 60 to 70 days after infection, female adults begin to release unembryonated eggs (oviposit) into the cecum at a rate of 3,000 to 20,000 eggs per day, linking the life cycle to the start.

Life cycle of *Trichuris trichiura*

3.3.3 Signs and symptoms

Light infestations are frequently asymptomatic (have no symptoms). Heavier infestations, especially in small children, can present gastrointestinal problems including abdominal pain and distention, bloody or mucous-filled diarrhoea, and

tenesmus (feeling of incomplete defecation, generally accompanied by involuntary straining). While damage may be done to the GI tissue and appendicitis may be brought on (by damage and oedema of the adjacent lumen) if there are large numbers of worms or larvae present, it has been suggested that the embedding of the worms into the ileo-cecal region may also make the host susceptible to bacterial infection. Severe infection may also present with rectal prolapse, although this is typically seen only in heavy infections of small children. High numbers of embedded worms in the rectum cause oedema, which causes the rectal prolapse. The prolapsed, inflamed and oedematous rectal tissue may even show visible worms.

Growth retardation, weight loss, nutritional deficiencies, and anaemia (due to long-standing blood loss) are also characteristic of infection, and these symptoms are more prevalent and severe in children.

3.3.4 Diagnosis

A stool ova and parasites examination reveals the presence of typical whipworm eggs. Typically, the Kato-Katz thick-smear technique is used for the identification of the *Trichuris trichiura* eggs in the stool sample.

Although colonoscopy is not typically used for diagnosis, but there have been reported cases in which colonoscopy has revealed adult worms. Colonoscopy can directly diagnose trichuriasis by identification of the threadlike form of worms with an attenuated, whip-like end. Colonoscopy has been shown to be a useful diagnostic tool, especially in patients infected by only a few male worms and with no eggs presenting in the stool sample.

3.3.5 Epidemiology

Trichuris trichiura is the third most common nematode (roundworm) of humans. Infection of trichuris trichiura is most frequent in areas with tropical weather and poor sanitation practices. Trichuriasis occurs frequently in areas in which human feces is used as fertilizer or where defecation onto soil takes place. Trichuriasis infection prevalence is 50 to 80 percent in some regions of Asia (noted especially in China and Korea) and also occurs in rural areas of the southeastern United States. Infection is most prevalent among children, and in North America,

infection occurs frequently in immigrants from tropical or sub-tropical regions. It is estimated that 600-800 million people are infected worldwide with 3.2 billion individuals at risk.

3.3.6 Control and Prevention

Improved facilities for faeces disposal have decreased the incidence of whipworm. Handwashing before food handling, and avoiding ingestion of soil by thorough washing of food that may have been contaminated with egg-containing soil are other preventive measures. Mass Drug Administration (preventative chemotherapy) has had a positive effect on the disease burden of trichuriasis in East and West Africa, especially among children, who are at highest risk for infection. Improvement of Sewage and Sanitation systems, as well as improved facilities for faeces disposal have helped to limit defecation onto soil and contain potentially infectious faeces from bodily contact. A study in a Brazil Urban Centre demonstrated a significant reduction in prevalence and incidence of geohelminth infection, including trichuriasis, following implementation of a city-wide sanitation programme. A 33% reduction in prevalence of trichuriasis and a 26% reduction in incidence of trichuriasis was found in the study performed on 890 children ages 7–14 years old within 24 different sentinel areas chosen to represent the varied environmental conditions throughout the city of Salvador, Bahia, Brazil. Control of Soil Fertilizers has helped eliminate the potential for contact with human faecal matter in fertilizer in soil.

- **CONCLUSION**

  In this unit, student learnt the morphology, laboratory diagnosis, life cycle and control of various soil transmitted helminths.

- **SUMMARY**

  STH infection is caused by four major nematode species: *Ancylostoma duodenale* and *Necator americanus* (hookworms), *Ascaris lumbricoides* (roundworm) and *Trichuris trichiura* (whipworm). Infection is prevalent in areas with over-population and inadequate sanitation in tropical and sub-tropical countries, where the climate supports the survival of the parasite eggs or larvae in the warm and moist soil. After infective larvae enter the human body they develop into adult

worms and parasitize the gastrointestinal tract, sometimes for years. Some species of worms can produce up to 200,000 eggs per day. Eggs are excreted in the faeces and remain viable in the soil for several weeks or years depending on the species. It is common for a single individual, especially a child, to be infected with all three types of worms. Although STH infection rarely causes fatality, chronic infection with high worm burden can lead to serious health consequences. Infection is typically most intense and debilitating in school-age children, resulting in malnutrition, physical and intellectual growth retardation, and cognitive and educational deficits. *A. lumbricoides* may cause intestinal obstructions that require surgery, and *T. trichuria* may cause chronic colitis. Hookworm infection causes iron-deficiency anaemia because the worms feed on the intestinal wall causing tissue damage and blood loss. Hookworm infection is a leading cause of morbidity in children and pregnant women, and can have adverse results for the mother, the foetus and the neonate.

- **TUTOR-MARKED ASSIGNMENT**

1. State the morphological differences between the two hookworm species
2. What strategies you will implore for the control of soil transmitted nematodes?

7.0   REFERENCES/FURTHER READING

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UNIT 3   BLOOD AND TISSUE NEMATODES

CONTENTS

1.0   INTRODUCTION

Filariasis is caused by nematodes (roundworms) that inhabit the lymphatics and subcutaneous tissues. Eight main species infect humans. Three of these are responsible for most of the morbidity due to filariasis: *Wuchereria bancrofti* and *Brugia malayi* cause lymphatic filariasis, and *Onchocerca volvulus* causes onchocerciasis (river blindness). The other five species are *Loa loa*, *Mansonella perstans*, *M. streptocerca*, *M. ozzardi*, and *Brugia timori*. (The last species also cause lymphatic filariasis).

Among the agents of lymphatic filariasis, *Wuchereria bancrofti* is encountered in tropical areas worldwide; *Brugia malayi* is limited to Asia; and *Brugia timori* is restricted to some islands of Indonesia. The agent of river blindness, *Onchocerca volvulus*, occurs mainly in Africa, with additional foci in Latin America and the Middle East. Among the other species, *Loa loa* and *Mansonella streptocerca* are found in Africa; *Mansonella perstans* occurs in both Africa and South America; and *Mansonella ozzardi* occurs only in the American continent.

Another tissue invading parasite is *Trichinella spiralis* whose larval form is found in the muscular tissue of the host animal. *Trichinella spiralis* is in fact a complex of three closely related worm species. They are morphologically identical, but differ in their host specificity and their biochemical characteristics. *T. spiralis spiralis* occurs in moderate regions and infects mainly pigs. *T. spiralis nativa* occurs in the polar regions (polar bear, walrus). These parasites are resistant to freezing which is important for meat storage. *T. spiralis nelsoni* occurs in Africa and southern Europe with a reservoir in wild carnivores and wild pigs. *T. britovi* and *T. pseudospiralis* rarely cause infections. *T. pseudospiralis* can also infect some birds as well as mammals, unlike the other *Trichinella* species.

OBJECTIVES

At the end of this unit, the students should be able to

1. Give examples of blood and tissue invading parasites

2. Describe their life cycles and clinical features associated with their infections.

3. Describe the methods of diagnosis of their infections

3.0   MAIN CONTENTS

3.1   Filarial worms

3.1.1   *Wuchereria bancrofti*

Different species of the following genera of mosquitoes are vectors of *W. bancrofti* filariasis depending on geographical distribution. Among them are:

Culex (*C. annulirostris*, *C. bitaeniorhynchus*, *C. quinquefasciatus*, and *C. pipiens*); *Anopheles* (*A. arabinensis*, *A. bancroftii*, *A. farauti*, *A. funestus*, *A. gambiae*, *A. koliensis*, *A. melas*, *A. merus*, *A. punctulatus* and *A. wellcomei*); *Aedes* (*A. aegypti*, *A. aquasalis*, *A. bellator*, *A. cooki*, *A. darlingi*, *A. kochi*, *A. polynesiensis*, *A. pseudoscutellaris*, *A. rotumae*, *A. scapularis*, and *A. vigilax*); *Mansonia* (*M. pseudotitillans*, *M. uniformis*); *Coquillettidia* (*C. juxtamansonia*). During a blood meal, an infected mosquito introduces third-stage filarial larvae onto the skin of the human host, where they penetrate into the bite wound. They develop in adults that commonly reside in the lymphatics. The female worms measure 80 to 100 mm in length and 0.24 to 0.30 mm in diameter, while the males measure about 40 mm by .1 mm. Adults produce microfilariae measuring 244 to 296 μm by 7.5 to 10 μm, which are sheathed and have nocturnal periodicity, except the South Pacific microfilariae which have the absence of marked periodicity. The microfilariae migrate into the lymph and blood channels moving actively through lymph and blood. A mosquito ingests the microfilariae during a blood meal. After ingestion, the microfilariae loose their sheaths and some of them work their way through the wall of the proventriculus and cardiac portion of the mosquito's midgut and reach the thoracic muscles. There the microfilariae develop into first-stage larvae and subsequently into third-stage infective larvae. The third-stage infective larvae migrate through the haemocoel to the mosquito's prosbocis and can infect another human when the mosquito takes a blood meal.
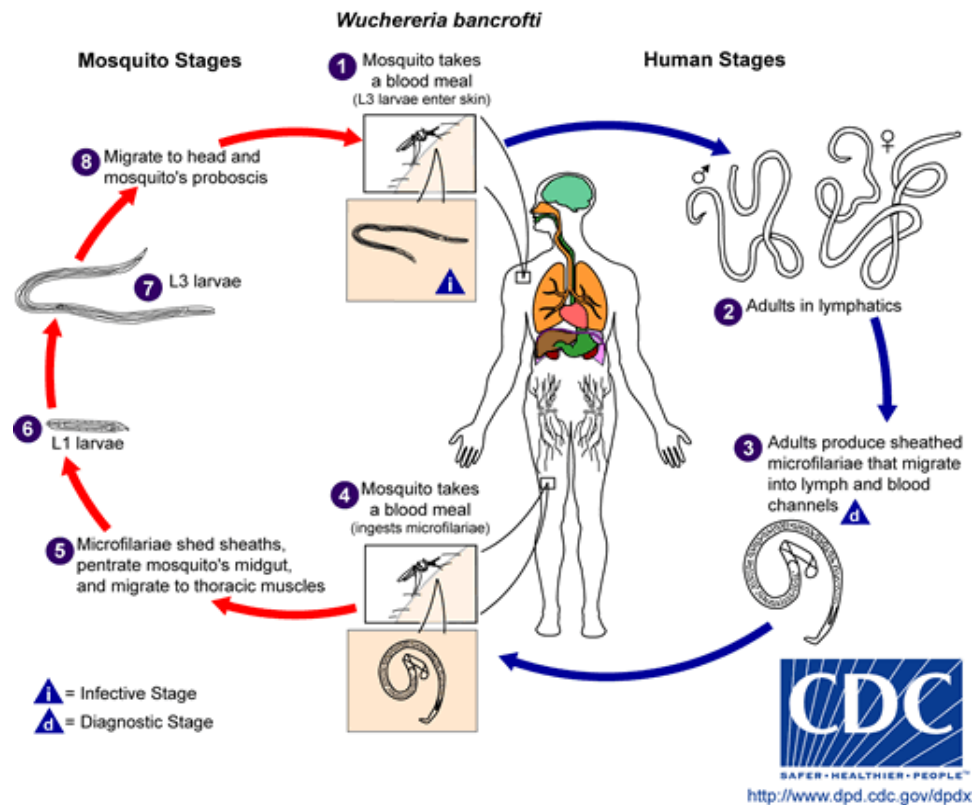
Life cycle of *W. bancrofti*

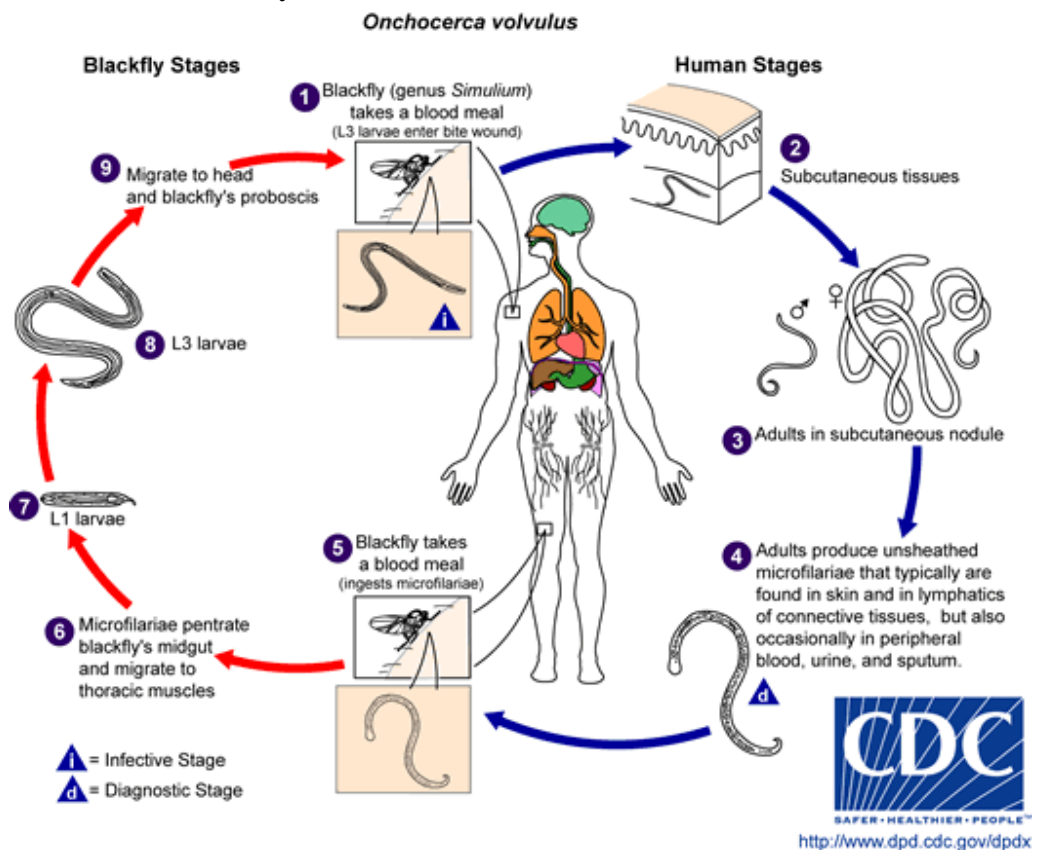

Microfilaria of *W. bancrofti*

3.1.2 *Onchocerca volvulus*

During a blood meal, an infected blackfly (genus *Simulium*) introduces third-stage filarial larvae onto the skin of the human host, where they penetrate into the bite wound. In subcutaneous tissues the larvae develop into adult filariae, which commonly reside in nodules in subcutaneous connective tissues Adults can live in

the nodules for approximately 15 years. Some nodules may contain numerous male and female worms. Females measure 33 to 50 cm in length and 270 to 400 µm in diameter, while males measure 19 to 42 mm by 130 to 210 µm. In the subcutaneous nodules, the female worms are capable of producing microfilariae for approximately 9 years. The microfilariae, measuring 220 to 360 µm by 5 to 9 µm and unsheathed, have a life span that may reach 2 years. They are occasionally found in peripheral blood, urine, and sputum but are typically found in the skin and in the lymphatics of connective tissues. A blackfly ingests the microfilariae during a blood meal. After ingestion, the microfilariae migrate from the blackfly's midgut through the haemocoel to the thoracic muscles. There the microfilariae develop into first-stage larvae and subsequently into third-stage infective larvae. The third-stage infective larvae migrate to the blackfly's proboscis and can infect another human when the fly takes a blood meal.

Life cycle of *O. Volvulus*

3.1.3 *Loa loa*

The vector for *Loa loa* filariasis are flies from two species of the genus *Chrysops*, *C. silacea* and *C. dimidiata*. During a blood meal, an infected fly (genus *Chrysops*, day-biting flies) introduces third-stage filarial larvae onto the skin of the human host, where they penetrate into the bite wound. The larvae develop into adults that commonly reside in subcutaneous tissue. The female worms measure 40 to 70 mm in length and 0.5 mm in diameter, while the males measure 30 to 34 mm in length and 0.35 to 0.43 mm in diameter. Adults produce microfilariae measuring 250 to 300 μm by 6 to 8 μm, which are sheathed and have diurnal periodicity. Microfilariae have been recovered from the spinal fluids, urine, and sputum. During the day they are found in peripheral blood, but during the noncirculation phase, they are found in the lungs. The fly ingests microfilariae during a blood meal. After ingestion, the microfilariae lose their sheaths and migrate from the fly's midgut through the haemocoel to the thoracic muscles of the arthropod. There the microfilariae develop into first-stage larvae and subsequently into third-stage infective larvae. The third-stage infective larvae migrate to the fly's proboscis and can infect another human when the fly takes a blood meal.
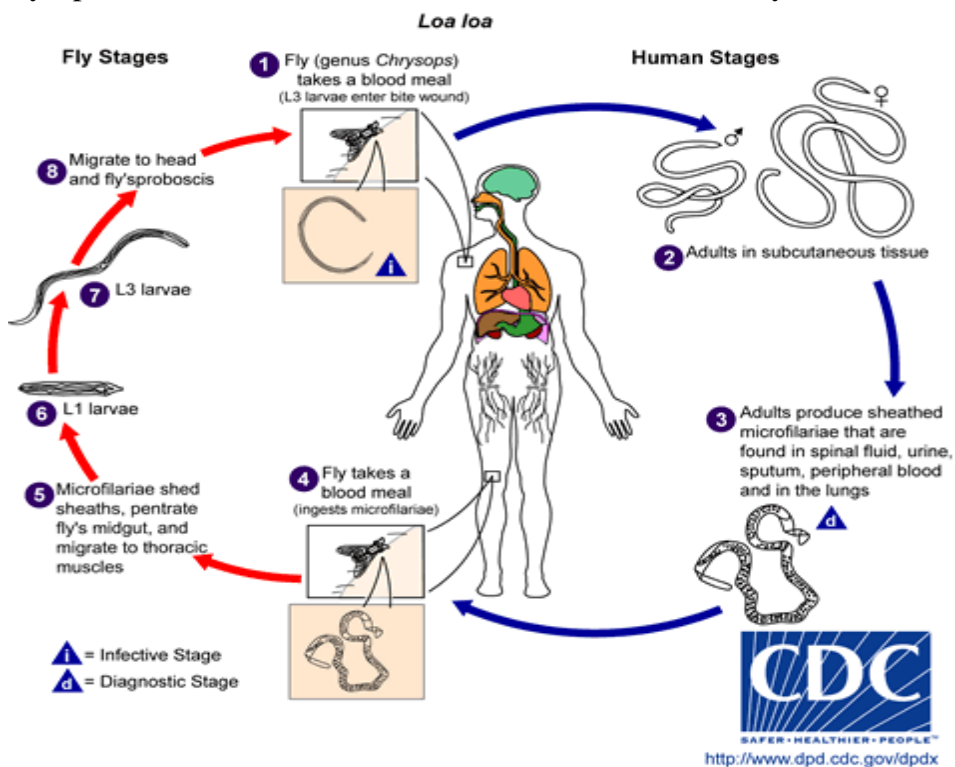

Life cycle *Loa loa*

3.1.4 *Brugia malayi*

The typical vector for *Brugia malayi* filariasis are mosquito species from the genera *Mansonia* and *Aedes*. During a blood meal, an infected mosquito introduces third-stage filarial larvae onto the skin of the human host, where they penetrate into the bite wound. They develop into adults that commonly reside in the lymphatics. The adult worms resemble those of *Wuchereria bancrofti* but are smaller. Female worms measure 43 to 55 mm in length by 130 to 170 μm in width, and males measure 13 to 23 mm in length by 70 to 80 μm in width. Adults produce microfilariae, measuring 177 to 230 μm in length and 5 to 7 μm in width, which are sheathed and have nocturnal periodicity. The microfilariae migrate into the lymph and enter the blood stream reaching the peripheral blood. A mosquito ingests the microfilariae during a blood meal. After ingestion, the microfilariae lose their sheaths and work their way through the wall of the proventriculus and cardiac portion of the midgut to reach the thoracic muscles. There the microfilariae develop into first-stage larvae and subsequently into third-stage larvae. The third-stage larvae migrate through the haemocoel to the mosquito's prosbocis and can infect another human when the mosquito takes a blood meal.
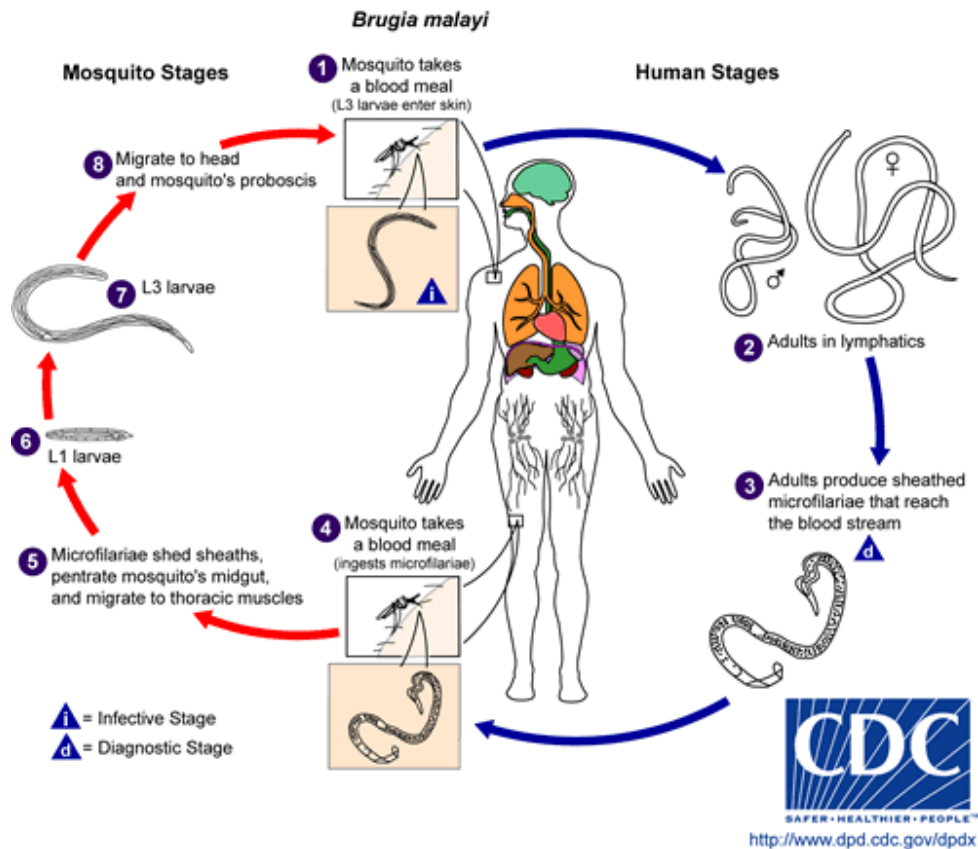
Life cycle of *Brugia malayi*

3.1.4.1   Clinical Features and Pathology

Lymphatic filariasis most often consists of asymptomatic microfilaremia. Some patients develop lymphatic dysfunction causing lymphedema and elephantiasis (frequently in the lower extremities) and, with *Wuchereria bancrofti*, hydrocele and scrotal elephantiasis. Episodes of febrile lymphangitis and lymphadenitis may occur. Persons who have newly arrived in disease-endemic areas can develop afebrile episodes of lymphangitis and lymphadenitis. An additional manifestation of filarial infection, mostly in Asia, is pulmonary tropical eosinophilia syndrome, with nocturnal cough and wheezing, fever, and eosinophilia. Onchocerciasis can cause pruritus, dermatitis, onchocercomata (subcutaneous nodules), and lymphadenopathies. The most serious manifestation consists of ocular lesions that can progress to blindness. Loiasis (*Loa loa*) is often asymptomatic. Episodic angioedema (Calabar swellings) and sub-conjunctival migration of an adult worm

can occur. Infections by *Mansonella perstans*, which is often asymptomatic, can be associated with angioedema, pruritus, fever, headaches, arthralgias, and neurologic manifestations. *Mansonella streptocerca* can cause skin manifestations including pruritus, papular eruptions and pigmentation changes. Eosinophilia is often prominent in filarial infections. *Mansonella ozzardi* can cause symptoms that include arthralgias, headaches, fever, pulmonary symptoms, adenopathy, hepatomegaly, and pruritus.

Elephantiasis caused by infection by *W. bancrofti*

3.1.4.2   Treatment and control

Ivermectin is effective in killing the larvae, but does not affect the adult worm. Preventive measures include vector control, treatment of infected individuals and avoidance of black fly.

3.1.4.3   Laboratory Diagnosis of filarial worms

Identification of microfilariae by microscopic examination is the most practical diagnostic procedure.

Microscopy

Examination of blood samples will allow identification of microfilariae of *Wuchereria bancrofti*, *Brugia malayi*, *Brugia timori*, *Loa loa*, *Mansonella*

perstans, and *M. ozzardi.* It is important to time the blood collection with the known periodicity of the microfilariae. The blood sample can be a thick smear, stained with Giemsa or haematoxylin and eosin. For increased sensitivity, concentration techniques can be used. These include centrifugation of the blood sample lyzed in 2% formalin (Knott's technique), or filtration through a Nucleopore® membrane. Examination of skin snips will identify microfilariae of *Onchocerca volvulus* and *Mansonella streptocerca.* Skin snips can be obtained using a corneal-scleral punch, or more simply a scalpel and needle. The sample must be allowed to incubate for 30 minutes to 2 hours in saline or culture medium, and then examined for microfilariae that would have migrated from the tissue to the liquid phase of the specimen.

Preparing Blood Smears for Microscopy Examination

If one uses venous blood, blood smears should be prepared as soon as possible after collection (delay can result in changes in parasite morphology and staining characteristics).

Thick smears

Thick smears consist of a thick layer of dehemoglobinized (lysed) red blood cells (RBCs). The blood elements (including parasites, if any) are more concentrated (app. 30×) than in an equal area of a thin smear. Thus, thick smears allow a more efficient detection of parasites (increased sensitivity). However, they do not permit an optimal review of parasite morphology. For example, they are often not adequate for species identification of filaria parasites: if the thick smear is positive for filaria parasites, the thin smear should be used for species identification.

Prepare at least 2 smears per patient!

1. Place a small drop of blood in the centre of the pre-cleaned, labeled slide.
2. Using the corner of another slide or an applicator stick, spread the drop in a circular pattern until it is the size of a dime (1.5 cm^2).
3. A thick smear of proper density is one which, if placed (wet) over newsprint, allows you to barely read the words.
4. Lay the slides flat and allow the smears to dry thoroughly (protect from dust and insects!). Insufficiently dried smears (and/or smears that are too thick) can detach

from the slides during staining. The risk is increased in smears made with anticoagulated blood. At room temperature, drying can take several hours; 30 minutes is the minimum; in the latter case, handle the smear very delicately during staining. You can accelerate the drying by using a fan or hair dryer (use cool setting). Protect thick smears from hot environments to prevent heat-fixing the smear.

5. Do not fix thick smears with methanol or heat. If there will be a delay in staining smears, dip the thick smear briefly in water to haemolyse the RBCs.

Thin smears

Thin smears consist of blood spread in a layer such that the thickness decreases progressively toward the feathered edge. In the feathered edge, the cells should be in a monolayer, not touching one another.

Prepare at least 2 smears per patient!

1. Place a small drop of blood on the pre-cleaned, labeled slide, near its frosted end.
2. Bring another slide at a 30-45° angle up to the drop, allowing the drop to spread along the contact line of the 2 slides.
3. Quickly push the upper (spreader) slide toward the unfrosted end of the lower slide.
4. Make sure that the smears have a good feathered edge. This is achieved by using the correct amount of blood and spreading technique.
5. Allow the thin smears to dry. (They dry much faster than the thick smears, and are less subject to detachment because they will be fixed.)
6. Fix the smears by dipping them in absolute methanol.

Special Procedures for Detecting Microfilariae

Blood microfilariae:

A. Capillary (fingerstick) blood
Since microfilariae concentrate in the peripheral capillaries, thick and thin smears prepared from fingerstick blood are recommended.
B. Anticoagulated (EDTA) venous blood (1 ml) should be concentrated by one of the following methods:

1. Centrifugation (Knott's technique)
   a. Prepare 2% formaldehyde (2 ml of 37% formaldehyde + 98 ml H_2O).
   b. Mix 9 ml of this 2% formaldehyde with 1 ml of patient's venous blood. Centrifuge at $500 \times g$ for 10 minutes; discard supernatant. Sediment is composed of WBCs and microfilariae (if present).
   c. Examine as temporary wet mounts.
   d. Prepare thick and thin smears; allow to dry; dip in absolute methanol before Giemsa staining to enhance staining of microfilariae.
2. Filtration
   a. Place Millipore® or Nucleopore® membrane filter (5 µm pore) in filter holder with syringe attachment.
   b. Mix 1 ml of venous blood (in EDTA) with 10 ml of 10% Teepol® 610 (Shell Co.); allow to stand for several minutes to allow lysis; transfer to a 10 ml Luer-Loc® syringe; attach the filter apparatus.
   c. Force the solution through the 5 µm pore filter, followed by several syringes of water to wash out the remaining blood, then 1 or 2 syringes full of air to clear excess fluid.
   d. Prepare a temporary wet mount by removing the filter and placing it on a glass slide, adding a drop of stain or dye and a coverslip.
   e. For permanent preparations, pass 2 to 3 ml of methanol through the filter while it is still in the holder; remove filter and dry it on a glass slide; then stain it with Giemsa stain, horizontally (so that the filter does not wash off the slide); coverslip filter before examining.

Diagnostic findings

- **Antigen detection** using an immunoassay for circulating filarial antigens constitutes a useful diagnostic approach, because microfilaremia can be low and variable. A rapid-format immunochromatographic test, applicable to *Wuchereria bancrofti* antigens, has been evaluated in the field. However, antibody detection is of limited value. Substantial antigenic cross reactivity exists between filaria and other helminths, and a positive serologic test does not distinguish between past and current infection.
- **Molecular diagnosis** using polymerase chain reaction is available for *W. bancrofti* and *B. malayi*.

▪ **Identification of adult worms** is possible from tissue samples collected during nodulectomies (onchocerciasis), or during subcutaneous biopsies or worm removal from the eye (loiasis).

3.2   *Trichinella spiralis*

3.2.1   Historical aspect

In 1835, a man died of tuberculosis in St Bartholomew's Hospital, London. Dr Paget, a first-year student, carried out the autopsy and observed fine hard white inclusions in the muscles. Similar inclusions had been observed by doctors from time to time in the past, but were attributed to commonplace muscle calcification, which quickly blunted the dissecting scalpel. Dr Paget inspected the lesions with a hand lens and quickly recognised their worm-like structure. The name "*Trichina spiralis*" was suggested. This name *Trichina* had already been given to a certain fly, however, and the name was later changed to "*Trichinella*". The discovery of the parasite was published by the famous biologist and palaeontologist Richard Owen, at that time assistant conservator of the museum of the Royal College of Surgeons. In 1859 Rudolph Virchow carried out transmission experiments in which infected human muscle was fed to a healthy dog. After only 3 to 4 days adult *Trichinella* worms were found in the dog's duodenum and jejunum.

3.2.2   Life cycle

More than 100 species of mammals are susceptible to the infection. Infections with *Trichinella spiralis* affect chiefly carnivores and omnivores, although infection of horses has also been described. People become infected with this nematode by eating raw or insufficiently cooked infected meat, often pork or wild boar. The larvae of *Trichinella spiralis* which are in the meat develop in a few days into adult worms (2-4 mm) in the wall of the small intestine. There they lay larvae (100 m m). These spread via the bloodstream to various muscles, including the heart, where they undergo encapsulation [*Trichinella pseudospiralis* does not form a capsule]. The larvae cannot continue to survive in the heart. The larvae are localised within the cells of the muscles, which is unique for a worm. After penetrating the muscle cell, a larva excretes a number of signal molecules and proteins, which convert the cell to what is called a nurse cell. In the cell the behaviour of the worm is rather similar to that of a virus. Many of its proteins are glycosylated and often carry an unusual sugar (tyvelose). These proteins are excreted from a special organ in the larva (the stichosome). Various muscle proteins such as actin and myosin change or disappear, nuclear division is

stimulated and mitochondria are damaged. Local angiogenesis is stimulated by excretion of a blood vessel growth factor and new blood vessels, originating from nearby venules, develop and form a network around the infected cell. The metabolism of the nurse cell and the parasite is essentially anaerobic. After 1 to 4 months the adult worms die. The larvae in the muscles sometimes survive for years and can remain viable for a long time even in rotting flesh. *Trichinella* is unique among worms in that all development stages take place in the same host. There is never a free stage outside the mammalian body.

3.2.3   Symptoms

Infection may be asymptomatic. In typical cases there is diarrhoea, abdominal pain, vomiting and fever a few days after eating infected meat. After 10 days the fever increases, the patient is very ill and debilitated, there are muscle pains and a typical peri-orbital oedema (differential diagnosis acute trypanosomiasis and nephrosis). This oedema is caused by invasion of the small muscles around the eye. There may be signs of myocarditis, encephalitis, urticaria and asthma. There is often very significant eosinophilia. The myositis causes an increase in the muscle enzymes (creatine phosphokinase, CK). After a few months the symptoms are reduced or disappear. Mild infections are self-limiting.

3.2.4   Diagnosis

Not many nematodes are found in muscle tissue. Occasionally a migrating third stage larva of *Ancylostoma, Toxocara* or *Gnathostoma* may be found (visceral larva migrans). *Dracuncula medinensis* may also be found in muscle tissue. Another, less common nematode which may be found here is *Haycocknema perplexum* (Tasmania).

3.2.5   Prevention

- Meat should be well boiled or roasted thoroughly.
- Importance of meat inspection. The diaphragm of a slaughtered animal is inspected (the piece of muscle is flattened between two glass slides and examined using transillumination). This technique (trichinoscopy) is not so good for *Trichinella pseudospiralis* because it is not surrounded by a capsule and is easily missed.
- Pig food (which may include infected rats) should be boiled for 30 minutes.

- To store pork for 10 days at -25° C is generally impractical in developing countries. In the West meat is sometimes irradiated with high doses of gamma rays, which will kill any larvae. *Trichinella spiralis nativa* is cold-hardy.

4.0   CONCLUSION

In this unit, students learnt the various life cycles and transmission patterns, clinical symptoms and preventions of blood and tissue nematodes.

5.0   SUMMARY

Filariasis is caused by nematodes that inhabit the lymphatics and subcutaneous tissues. Eight main species infect humans of which three of these are responsible for most of the morbidity due to filariasis: *Wuchereria bancrofti* and *Brugia malayi* cause lymphatic filariasis, and *Onchocerca volvulus* causes onchocerciasis (river blindness)

Infective larvae are transmitted by infected biting arthropods during a blood meal. The larvae migrate to the appropriate site of the host's body, where they develop into microfilariae-producing adults. The adults dwell in various human tissues where they can live for several years. The agents of lymphatic filariasis reside in lymphatic vessels and lymph nodes; *Onchocerca volvulus* in nodules in subcutaneous tissues; *Loa loa* in subcutaneous tissues, where it migrates actively; *Brugia malayi* in lymphatics, as with *Wuchereria bancrofti*; *Mansonella streptocerca* in the dermis and subcutaneous tissue; *Mansonella ozzardi* apparently in the subcutaneous tissues; and *M. perstans* in body cavities and the surrounding tissues. The female worms produce microfilariae which circulate in the blood, except for those of *Onchocerca volvulus* and *Mansonella streptocerca*, which are found in the skin, and *O. volvulus* which invade the eye. The microfilariae infect biting arthropods (mosquitoes for the agents of lymphatic filariasis; blackflies (*Simulium*) for *Onchocerca volvulus*; midges for *Mansonella perstans* and *M. streptocerca*; and both midges and blackflies for *Mansonella ozzardi*; and deerflies (*Chrysops*) for *Loa loa*). Inside the arthropod, the microfilariae develop in 1 to 2 weeks into infective filariform (third-stage) larvae. During a subsequent blood meal by the insect, the larvae infect the vertebrate host. They migrate to the appropriate site of the host's body, where they develop

into adults, a slow process than can require up to 18 months in the case of *Onchocerca*.

Infections with *Trichinella spiralis* affect chiefly carnivores and omnivores. People become infected by eating raw or insufficiently cooked infected meat, often pork or wild boar. The larvae of *Trichinella spiralis* which are in the meat develop in a few days into adult worms (2-4 mm) in the wall of the small intestine. There they lay larvae (100 mm). These spread via the bloodstream to various muscles, including the heart, where they undergo encapsulation. Vector control in case of filariasis and proper cooking of pork (*Trichinella spiralis*) are the control measures.

6.0   TUTOR-MARKED ASSIGNMENT

1.   Describe the life cycle of the parasite that causes elephantiasis

2.   How can you prevent infection by *Trichinella spiralis*?

7.0   REFERENCES/FURTHER READING

Duke (1972). Onchocerciasis. *Br. Med. Bull., 28*: 66-71

Neva and Ottessen (1978). Tropical (filarial) eosinophilia. *N. Engl. J. Med., 298*: 1129-1131

UNIT 4   AIR-BORNE NEMATODE

1.0   INTRODUCTION

The human pinworm *Enterobius vermicularis* is an ubiquitous parasite of man. It is estimated that over 200 million people are infected annually. It is more common in the temperate regions of Western Europe and North America, (it is being relatively rare in the tropics) and is found particularly in children. Samples of Caucasian children in the U.S.A. and Canada have shown incidences of infection of between 30% to 80%, with similar levels in Europe, and although these regions are the parasites strongholds, it may be found throughout the world, again often with high degrees of incidence. For example in parts of South America the incidence in school children may be as high as 60%. Interestingly non-Caucasians appear to be relatively resistant to infection with this nematode. As a species, and contrary to popular belief, *E. vermicularis* is entirely restricted to man, other animals harbouring related but distinct species that are non-infective to humans, but their fur may be contaminated by eggs from the human species if stroked by someone with eggs on their hands. In man anywhere where there are large number of children gathered together, (such as nurseries, play groups, orphanages etc.), especially if conditions are insanitary, are ready sources of infection, as one child may rapidly transmit the parasite to his or her fellows.

2.0 OBJECTIVES

At the end of the unit, students should be able to describe the morphology of the parasite, its life cycle, pathology, diagnosis and control.

3.0 MAIN CONTENTS

3.1 *Enterobius vermicularis* (the human pin-worm)

3.1.1 Morphology

These creamy white coloured nematodes are relatively small, with the female measuring only approximately 10mm by 0.4mm wide. The females have a cuticular expansion at their anterior ends, with a long pointed tail. The male parasites, which are much less numerous than the females, are much smaller, measuring only up to 5mm long, and have a curved tail, with a small bursa like expansion, and a single spicule. The head has a mouth with three small lips


Adult Pinworm

3.1.2 Life cycle

The adult parasites live predominantly in the caecum. The male and females mate, and the uteri of the females become filled with eggs. The gravid females (each containing up to 15000 eggs) then migrate down the digestive tract to the anus. From here they make regular nocturnal migrations out of the anus, to the perianal region, where air contact stimulates them to lay their eggs, before retreating back into the rectum. Eventually the female die, their bodies disintegrating to release many remaining eggs. These eggs, which are clear and measure about 55 by 30μm, then mature to the infectious stage (containing an L1 larvae) over 4 to 6

weeks. To infect the host, typically these eggs must then be ingested. The ingested eggs hatch in the duodenum.

The eggs themselves are sticky, and have a characteristic shape, shared with all members of the group Oxyridea, with an asymmetrical form, flattened on one side, (see below);
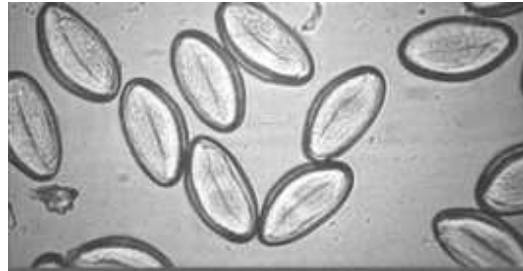
The ova of *Enterobius vermicularis*

The larvae then undergo a series of moults, as they migrate down the digestive tract. The adult worms then mature in the caecum, before copulating to complete the cycle (typically 6 weeks). Occasionally the eggs hatch in the perianal region itself, the resulting L1 larvae being fully infective, crawling back through the anus, then migrating up the intestine to the caecum (retroinfection).

3.1.3 Pathology of Infection.

The majority of infections with this nematode are asymptomatic, although in some cases the emerging females and the sticky masses of eggs that they lay may causes irritation of the perianal region, which in some cases may be severe. As the females emerge at night this may give rise to sleep disturbances, and scratching of the affected perianal area transfers eggs to the fingers and under the finger nails. This in turn aids the transmission of the eggs, both back to the original host (autoinfection), and to other hosts.

3.1.4 Diagnosis

Because eggs are rarely passed out with faeces, examination of faecal samples may not reveal them. This may account for negative results of enterobiasis in many of the surveys for helminth infections involving faecal samples in tropical

Africa. The most reliable diagnosis is by the cellophane tape swab. This involves the attachment of a piece of cellophane to the perianal region overnight. This is then examined for eggs under the microscope. Alternatively, the anus and perianal area can be examined under bright light at night, at which time adult worms can be seen glittering in the light.

3.1.5 Epidemiology and Control

The eggs of the parasite are air-borne, caught in clothing, household linen, curtain, carpets, etc. As such, infection is common in dry season than rainy season in the tropics. Maitenance of high standards of personal and domestic hygiene is therefore imperative for control and prevention.

4.0 CONCLUSION

At the end of this unit, student learnt the morphology, life cycle, pathology and control of pin-worm.

5.0 SUMMARY

Enterobius vermicularis is an air-borne parasitic infection common mostly in the temperate regions of the world. Adult female worms lay eggs in the perianal regions wnd infection occurs through direct ingestion of eggs containing the L1 larvae. Infection is usually asymptomatic but sometimes the sticky eggs could cause irritation of the perianal giving rise to scratching and sleep disturbance. Maitenance of high standards of personal and domestic hygiene is imperative for control and prevention.

6.0 TUTOR-MARKED ASSIGNMENT

Describe the morphology and life cycle of *Enterobius vermicularis*

- **REFERENCE/FURTHER READING**

Brown, H.W. and Neva, F.A. (1983). Basic clinical parasitology (5th edition). pp. 128-132