NATIONAL OPEN UNIVERSITY OF NIGERIA

FACULTY OF HEALTH SCIENCES

DEPARTMENT OF ENVIRONMENTAL HEALTH SCIENCES

COURSE CODE: EHS 303

COURSE TITLE: INTRODUCTION TOGENERAL PARASITOLOGY

COURSE GUIDE

EHS 303: INTRODUCTION TOGENERAL PARASITOLOGY

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COURSE GUIDE

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Introduction

Malaria is one of the most serious health problems facing the world today. The World Health Organization estimates that over 300 million new cases of malaria arise a year, with approximately two to three million deaths resulting from contraction. Malaria is endemic in tropical Africa, with an estimated 90% of the total malaria incidence and deaths occurring there, particularly amongst pregnant women and children. More specifically, malaria is causing various problems in Nigeria. Malaria is the only vector borne disease to be placed on World Health Organization's Disability Adjusted Life Years (DALYS) list. It is important to look at health problems like malaria that grossly affect the morbidity and mortality rates, as well as the economy of a developing country, such as Nigeria.

What you will learn in this course

In this course, you have the course units and a course guide. The course guide will tell you what the course is all about. It is general overview of the course materials you will be using and how to use those materials. It also helps you to allocate the appropriate time to each unit so that you can successfully complete the course within the stipulated time limit.

The course guide also helps you to know how to go about your Tutor-Marked Assignment which will form part of your overall assessment at the end of the course. Also, there will be regular tutorial classes that are related to this course, where you can interact with your facilitator and other students. Please, I encourage you to attend these tutorial classes.

Course Aims

The course aims to give you a understanding on human parasites

Course Objective

To achieve the aim set above, there are objectives. Each unit has a set of objectives presented at the beginning of the unit. These objectives will guide you on what to concentrate / focus on while studying the unit. Please read the objective before studying the unit and during your study to check your progress.

The Comprehensive Objectives of the Course are given below. By the end of the course/after going through this course, you should be able to:

- Define the term human parasite

Working through this course

To successfully complete this course, you are required to read each study unit, read the textbooks materials provided by the National Open University.

Reading the referenced materials can also be of great assistance.

Each unit has self-assessment exercises which you are advised to do and at certain periods during the course you will be required to submit your assignment for the purpose of assessment.

There will be a final examination at the end of the course. The course should take you about 17 weeks to complete.

This course guide will provide you with all the components of the course how to go about studying and hour you should allocate your time to each unit so as to finish on time and successfully.

The Course Materials

The main components of the course are:

- The Study Guide
- Study Units
- Reference / Further Readings
- Assignments
- Presentation Schedule

Study Unit

The study units in this course are given below:

MODULE 1: INTRODUCTION TO MAJOR HUMAN PARASITES OF PUBLIC HEALTH RELEVANCE

Unit 1: Phylogeny of parasites

Unit 2: Parasite physiology

Unit 3: Host parasite relationship

Unit 4: Evasive mechanism

MODULE 2: PARASITE LIFE CYCLE PATTERN

Unit 1: Host specificity

Unit 2: Parasite ecology

Unit 3: Infection infestation

Unit 4: Parasite pathology

MODULE 3: PATHOGENESIS AND SYMPTOMOLOGY OF PARASITIC DISEASES

Unit 1: Malaria

Unit 2: *Amoebiasis*

Unit 3: *Schistosomiasis*

Unit 4: *Onchocerciasis*

MODULE 4: STRATEGIES IN PARASITIC DISEASES CONTROL

Unit 1: Emerging and re-emerging parasitic control

Unit 2: Chemotherapy and chemoprophylaxis

Unit 3: Drug resistance in parasites

Unit 4: Prevention and control of parasite infections

There are activities related to the lecture in each unit which will help your progress and comprehension of the unit. You are required to work on these exercises which together with the TMAs will enable you to achieve the objectives of each unit.

Presentation Schedule

There is a time-table prepared for the early and timely completion and submissions of your TMAs as well as attending the tutorial classes. You are required to submit all your assignments by the stipulated time and date. Avoid falling behind the schedule time.

Assessment

There are three aspects to the assessment of this course.

The first one is the self-assessment exercises. The second is the tutor marked assignments and the third is the written examination or the examination to be taken at the end of the course.

Do the exercises or activities in the unit by applying the information and knowledge you acquired during the course. The tutor-marked assignments must be submitted to your facilitator for formal assessment in accordance with the deadlines stated in the presentation schedule and the assignment file.

The work submitted to your tutor for assessment will count for 30% of your total course work.

At the end of this course, you have to sit for a final or end of course examination of about a three hour duration which will count for 70% of your total course mark.

Tutor-Marked Assignment

This is the continuous assessment component of this course and it accounts for 30% of the total score. You will be given four (4) TMAs by your facilitator to answer. Three of which must be answered before you are allowed to sit for the end of course examination.

These answered assignments are to be returned to your facilitator.

You’re expected to complete the assignments by using the information and material in your readings references and study units.

Reading and researching into you references will give you a wider via point and give you a deeper understanding of the subject.

1. Make sure that each assignment reaches your facilitator on or before the deadline given in the presentation schedule and assignment file. If for any reason you are not able to complete your assignment, make sure you contact your facilitator before the assignment is due to discuss the possibility of an extension. Request for extension will not be granted after the due date unless there in exceptional circumstances.

2. Make sure you revise the whole course content before sitting or the examination. The self-assessment activities and TMAs will be useful for this purposes and if you have any comment please do before the examination. The end of course examination covers information from all parts of the course.

Course Marking Scheme

Assignment	Marks
Assignments 1 – 4	Four assignments, best three marks of the four count at 10% each–30% of coursemarks.
End of course examination	70% of overall course marks
Total	100% of course materials.

Facilitators/Tutors and Tutorials

Sixteen (16) hours are provided for tutorials for this course. You will be notified of the dates, times and location for these tutorial classes.

As soon as you are allocated a tutorial group, the name and phone number of your facilitator will be given to you.

These are the duties of your facilitator: He or she will mark and comment on your assignment. He will monitor your progress and provide any necessary assistance you need. He or she will mark your TMAs and return to you as soon as possible.

(You are expected to mail your tutored assignment to your facilitator at least two days before the schedule date).

Do not delay to contact your facilitator by telephone or e-mail for necessary assistance if you do not understand any part of the study in the course material. You have difficulty with the self assessment activities. You have a problem or question with an assignment or with the grading of the assignment.

It is important and necessary you acted the tutorial classes because this is the only chance to have face to face content with your facilitator and to ask questions which will be answered instantly. It is also period where you can say any problem encountered in the course of your study.

Summary

General parasitology is a course that introduces you to the scientific study of human parasites of public health relevance.

On completion of this course, you will have an understanding of basic knowledge on human parasite, the history of men and women who contributed to this field of study by their discoveries during their research works, the general characteristics. In addition you will be able to answer the following questions:

- Define the term phylogeny of parasites physiology
- State the host parasite relationship
- Explain the term Emerging and re-emerging diseases
- Define pathogenesis and symptomology of parasite disease.

The list of questions expected to be answer is not limited to the above list. Finally, you are expected to apply the knowledge you have acquired during this course to your practical life.

We wish you success in this course.

MODULE 1: INTRODUCTION TO MAJOR HUMAN PARASITES OF PUBLIC HEALTH RELEVANCE

Unit 1: Phylogeny of parasites

Unit 2: Parasites physiology

Unit 3: Host parasite relationship

Unit 4: Evasive mechanism

UNIT 1: PHYLOGENY OF PARASITES

CONTENTS

1.0 Introduction

The primary goal of comparing the phylogeny of a host and its parasite is to document the history of their association. For each host-parasite association we want to be able to decide if that association is due to descent or dispersal. But more than this, host-parasite assemblages offer exciting possibilities for the comparative study of rates of speciation and evolution in different organisms. The basis for such studies must be a phylogenetic analysis of the host-parasite association.

2.0 Objectives

At the end of this unit you should be able to:

- Know the primary goal of comparing the phylogeny of a host and its parasite

3.0 Main content

3.1 Comparing host and parasite Phylogenies

A phylogeny of a group of organisms has several components. The most basic is recency of common ancestry — two taxa are more closely related to each other than either is to a third taxon. This aspect of phylogeny can be depicted using a cladogram. The lengths of the branches of a cladogram have no meaning, only the groups are meaningful. By making the branch lengths proportional to the amount of evolutionary change we have an additive or metric tree (Fig. 1b). The sum of the lengths of the branches between any two taxa on an additive tree represents the amount of evolutionary change that has taken place since those two taxa diverged. A special case of the additive tree is the dendrogram, in which all the terminal taxa are equidistant from the root of the tree.

3. 2 Levels of Organization

When comparing host and parasite phylogenies the kinds of comparisons that are possible depends on the extent of our phylogenetic knowledge, which is mirrored in the kind of tree we use to represent that knowledge. The simplest comparison is between cladograms. Here we are asking whether or not hosts that are closely related harbor closely related parasites. However all tree building methods also provide some estimate of evolutionary change between taxa in the form of branch lengths. Hence we could also compare amounts of evolutionary change in the host and parasite clades by comparing lengths of the equivalent branches in the host and parasite phylogenies (that is, compare additive trees). This raises two problems. The first concerns identifying pairs of equivalent branches in the two trees.

The second problem, raised in a different context, is whether the characters used to estimate evolutionary change in the hosts and parasites are comparable. In morphological based studies this is likely to be a serious problem — for example, how does one rank changes in mammalian tooth enamel patterns with changes in setal formula in insects? Molecular data (especially nucleotide sequence. data) would seem to offer an attractive solution to this problem. In

principle nucleotide sites are comparable across taxa. This is not to say that the rates of change,

Positional and transition/transversion biases need be the same in the hosts and parasites, but we do at least have a common unit of measurement.

4.0 CONCLUSION

In this unit, you will be learning about comparing host and parasite phylogenies and its various kinds.

5.0 SUMMARY

The primary aim of this unit is to enlighten you on the comparison of host and parasites phylogenies.

6.0 TUTOR-MARKED ASSIGNMENT

- What is phylogeny of parasites
- Talk on the kinds of comparison

Solution

The primary goal of comparing the phylogeny of a host and its parasite is to document the history of their association. For each host-parasite association we want to be able to decide if that association is due to descent or dispersal

- Talk on the kinds of comparison

Solution

When comparing host and parasite phylogenies the kinds of comparisons that are possible depends on the extent of our phylogenetic knowledge, which is mirrored in the kind of tree we use to represent that knowledge. The simplest comparison is between cladograms. Here we are asking whether or not hosts that are closely related harbor closely related parasites. However all tree building methods also provide some estimate of evolutionary change between taxa in the form of branch lengths.

7.0 REFERENCE/FURTHER READING

Otunbanjo, O.A. (2008). Elements of Parasitology Second Edition. Panaf Publishing Inc. Abuja 196pp

Cribb, T. H., Bray, R. A., Olson P. D. and Littlewood D. T. J. (2003). Life cycle evolution in the Digenea: a new perspective from phylogeny. Advances in Parasitology 54, 197–254. [PubMed]

Drummond A., Pybus O. G. and Rambaut A. (2003). Inference of viral evolutionary rates from molecular sequences. Advances in Parasitology 54, 331–358. [PubMed]

Unit 2: PARASITE PHYSIOLOGY

1.0 Introduction

The localized sporulation shown by the hair-invading ringworm fungi on the surface of (in the ‘ectothrix’ type),or inside (the ‘endothrix’ type), the infected hair has also been advocated as a generic or infra-,generic criterion.

2.0 Objectives

At the end of this unit you will learn the two types of host parasites relationships.

3.0 Main content

3.1 Spatial relationship

The spatial relationship of the parasite to the host, that is, the localization of the pathogen to certain organs or tissues, has been used as a criterion for differentiating generic, specific, and intra-specific taxa. Some of the most

familiar examples of the use of these, what is called ‘ecological’, characteristics are provided by the Fungi Imperfection among which the genera *Phoma* and *Phyllostictu* are differentiated by the occurrence of the former on the stems and the latter on the leaves of the host plants. A similar, if not quite so clear-cut distinction is made between the allied pair of genera *Diplodina* (usually on stems, rarely on leaves) and *Aschochytu* (usually on leaves, less frequently on stems) in which the spores, produced as in Phoma and *PhylEosticta* in small flask-shaped structures (*pycnidia*) immersed in the host tissue, are %celled instead of 1-celled as in the first pair of genera, Another example at the generic level is provided by the *Dermatophytes* in which *Epidermophytm* is distinguished from *Microsporum* and *Trichophyton* by the inability to attack hair. On the other hand, the localized sporulation shown by the hair-invading ringworm fungi on the surface of (in the ‘ectothrix’ type), or inside (the ‘endothrix’ type), the infected hair has also been advocated as a generic or infra-generic criterion.

3.2 Physiological relationship

Although host specialization has been much more extensively employed as a taxonomic criterion than has localization of the parasite on the host it has rarely been used at a generic or supra-generic level, among the better known examples, the separation by Bergey *et al*., of plant pathogenic bacteria from similar coliform organisms as the tribe Erwiniae comprising the genera Erwinia (flagella *peritrichous*) and Phytomonas (flagella polar or absent) may be recalled. For specific and infra-specific taxa host specialization (or alleged specialization) has been exceedingly popular as any check-list of plant pathogenic fungi suggests. Up to 50 years or more of the specific epithets are found to be derived from host names. The reality of biological races is indisputable and their existence has for long tried the patience and exercised the ingenuity of both taxonomists and plant pathologists. It was among the rust fungi (*Uredinales*), a group of obligate plant parasites, that biological races were first recognized about 60 years ago, and since then the smuts and many other groups of fungi have been shown to exhibit the same phenomenon. Among the smuts, morphologically similar forms cause similar diseases in wheat and barley but the fungus from wheat will not infect barley and vice versa. This host specificity led to the erection of the two species, Ustilago *tritici*, the cause of loose smut of wheat, and the cause of loose smut of barley.

Sometimes small biometrical differences can be detected between such forms, but usually any morphological differences are too slight to have any practical taxonomic value and the taxonomic treatment is frequently modelled on that of the black stem rust, *Puccinia graminis*. This rust exhibits a series of races specialized for the parasitism of a range of cereals and grasses and it has been customary to treat these races as 'formae speciales' designated by Latin epithets (e.g. *Puccinia graminis*) to give names which have been used by many cereal pathologists and others as trinomials (e.g. *P . graminis tritici*). By using a series of carefully selected pure bred varieties of the host it is possible to demonstrate within one forma specialist the existence of large numbers of sub races (some 300 for *P . graminis tritici*) for the nomenclatural treatment of which the International Code of Botanical Nomenclature provides no guidance. A knowledge of such races and exchange of information on their geographical distribution is of great practical importance to plant pathologists who, therefore, agreed among themselves on an unofficial registration of these races at Prof, Stakman's laboratory at the University of Minnesota which acts as an international clearing house and allocates numbers by which the different races are distinguished.

4.0 Conclusion

On completion of this unit, you will learn in detail the host parasite relationship.

5.0 Summary

A knowledge and exchange of information on their geographical distribution is of great practical importance to plant pathologists who, therefore, agreed among themselves on an unofficial registration of these races at Prof, Stakman's laboratory at the University of Minnesota which acts as an international clearing house and allocates numbers by which the different races are distinguished.

6.0 Tutor Marked Assignment

- Write on the spatial relationship

Solution

The spatial relationship of the parasite to the host, that is, the localization of the

pathogen to certain organs or tissues, has been used as a criterion for differentiating generic, specific, and intra-specific taxa. Some of the most familiar examples of the use of these, what is called ‘ecological ’, characteristics are provided by the Fungi Imperfection among which the genera *Phoma* and *Phyllostictu* are differentiated by the occurrence of the former on the stems and the latter on the leaves of the host plants.

7.0 Reference/Further reading

Dennis. W.G. (1946). Notes on some parasite ascribed to Phoma and, related genera. Trans. B i .mycol. SOC. 11.

Legendre P. and Fortin M.-J. (1989). Spatial pattern and ecological analysis. Vegetation 80, 107–138

Levy P. S. and Lemeshow S. (2008). Sampling of Populations: Methods and Applications. Wiley-Blackwell, Oxford

Unit 3: Evasive mechanism

1.0 Introduction

Most living species are parasitic (Windsor 1988). Hosts, in turn, deploy their immune system to prevent infections or keep the parasites in check. The immune system is one of an organism's most complex systems and shows many signs of co-evolution with parasites. It is well tuned to its task, as otherwise long-lived multi-cellular organisms would probably not be able to survive and outpace their short-lived and numerous parasites. Nevertheless, some of the major questions in the field of evolutionary ecology have been to understand why immune responses are not always maximally efficient; in other words, why immune responses vary among host species and vary with many other factors, such as environment, stage of the host's life cycle or infection by different parasite types.

2.0 Objectives

At the end of this unit, you will be able to know the classification of evasive mechanism, and also you will get to know the definition of the term Pathogenesis.

3.0 Main content

3.1 Pathogenesis

Pathogenesis is a process based on physiological, biochemical or molecular mechanisms, and which leads to harmful effects for the host, for example, to the depletion of its resources, tissue destruction, detrimental changes in behavior, reduced fecundity, premature death and so forth. A general evolutionary ecological definition of virulence is considered to be a reduction in host fitness as a consequence of the infection by a parasite. 'Virulence' in a more narrow but commonly used sense refers to an increase in host mortality. Hence, pathogenesis (as used here) is the major process by which virulence is generated. Therefore, pathogenesis is the mechanism responsible for generating the virulence effects that are of interest for evolutionary ecologists. The respective mechanisms can be very different, however. The biomedical, veterinary or parasitological literature lumps them altogether under the term 'pathogenesis'. Unfortunately, in the discussions on the evolution of virulence, the relationship of pathogenesis to virulence has generally been treated as a black box (Weiss, 2002).

(a) Pathogenesis and virulence

The evolutionary theory of virulence rests on trade-offs in the life history of the parasite. Trade-offs describe the situation where an investment of an organism's resources into one fitness component (e.g. to increase survival) goes at the expense of another fitness component (e.g. the same investment reduces transmission success). The trade-off-based approach has generated a range of successful predictions that match how virulence, generally defined as the loss of host fitness resulting from an interaction with a parasite, varies with different ecological conditions (Frank, 1996). Because evolutionary theory is rather general, there are some obvious limitations. For example, the life cycle of parasites that need to kill their host (as is true for most parasitoids) will not match models describing transient microbial infections. Similarly, parasites that have recently invaded a host population may not yet have been subject to co-

evolution as assumed by theory (Weiss, 2002). Further factors can also lead to a mismatch between prediction and observation, such as the effect of environmental conditions and host status (e.g. nutrition, temperature; Ferguson & Read 2002; Beck *et al*., 2004; Bedhomme *et al*., 2004), or plasticity in the response (Taylor *et al*., 2006).

(b) Immune evasion and tolerance

Clearance of the parasite from the host is a threshold process—the parasite is either cleared or not. To the extent that this capacity is affected by parasite immune evasion, immune evasion also has threshold properties. By contrast, a gradual control of the infection by the host is often thought to be the cause of long-lasting, chronic infections, where the pathogen is kept at a level below damage. This issue is related to the concept of 'tolerance', which has been primarily developed in plant pathology (Clarke, 1984; Kover & Schaal, 2002). Tolerance is considered to be the ability of the host to reduce or buffer the effect of an infection on host fitness. It implies no or, more likely, a negative relationship between the levels of infection (e.g. as measured by infection intensity) and of host fitness (e.g. loss of body mass or fecundity)

4.0 Conclusion

Virulence in a more narrow but commonly used sense refers to an increase in host mortality. Hence, pathogenesis (as used here) is the major process by which virulence is generated.

5.0 Summary

Therefore, pathogenesis is the mechanism responsible for generating the virulence effects that are of interest for evolutionary ecologists. The respective mechanisms can be very different, however. The biomedical, veterinary or parasitological literature lumps them altogether under the term 'pathogenesis'. Unfortunately, in the discussions on the evolution of virulence, the relationship of pathogenesis to virulence has generally been treated as a black box

6.0 Tutor-marked Assignment

- Discuss on the term pathogenesis

Solution

Pathogenesis is a process based on physiological, biochemical or molecular mechanisms, and which leads to harmful effects for the host, for example, to the depletion of its resources, tissue destruction, detrimental changes in behaviour, reduced fecundity, premature death and so forth. A general evolutionary ecological definition of virulence is considered to be a reduction in host fitness as a consequence of the infection by a parasite. 'Virulence' in a more narrow but commonly used sense refers to an increase in host mortality. Hence, pathogenesis (as used here) is the major process by which virulence is generated. Therefore, pathogenesis is the mechanism responsible for generating the virulence effects that are of interest for evolutionary ecologists. The respective mechanisms can be very different, however. The biomedical, veterinary or parasitological literature lumps them altogether under the term 'pathogenesis'. Unfortunately, in the discussions on the evolution of virulence, the relationship of pathogenesis to virulence has generally been treated as a black box.

7.0 References/Further reading

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UNIT 4: PARASITES PHYSIOLOGY

CONTENTS

1.0 Introduction

Parasite physiology

Parasite physiology is the scientific discipline concerned with the study of the mechanisms and processes that enable parasites to grow and reproduce. A host in which parasites reproduce sexually is known as the *definitive, final* or *primary* host. In *intermediate* hosts, parasites either do not reproduce or do so asexually, but the parasite always develops to a new stage in this type of host. In some cases a parasite will infect a host, but not undergo any development, these hosts are known as paratenicor transport hosts.

The paratenic host can be useful in raising the chance that the parasite will be transmitted to the definitive host. For example, the cat lungworm (*Aelurostrongylus abstrusus*) uses a slug or snail as an intermediate host; the first stage larva enters the mollusk and develops to the third stage larva, which is infectious to the definitive host—the cat. If a mouse eats the slug, the third stage larva will enter the mouse's tissues, but will not undergo any development.

2.0 Objectives

At the end of this unit, you will get to know:

- How parasite reproduce
- How do parasite invade the body
- Types of plant parasite

3.0 Main Content

3.1 Overview of the term parasite

Parasitic life cycles occur in a variety of forms, all involving the exploitation of one or more hosts. Those that must infect more than one host species to complete their life cycles are said to have *complex or indirect* life cycles, while those that infect a single species have *direct* life cycles.

If a parasite has to infect a given host in order to complete its life cycle, then it is said to be an obligate parasite of that host; sometimes, infection is *facultative*—the parasite can survive and complete its life cycle without infecting that particular host species. Parasites sometimes infect hosts in which they cannot complete their life cycles; these are *accidental* hosts.

A host in which parasites reproduce sexually is known as the *definitive*, *final* or *primary* host. In *intermediate* hosts, parasites either do not reproduce or do so asexually, but the parasite always develops to a new stage in this type of host. In some cases a parasite will infect a host, but not undergo any development, these hosts are known as paratenicor transport hosts. The paratenic host can be useful in raising the chance that the parasite will be transmitted to the definitive host. For example, the cat lungworm (*Aelurostrongylus abstrusus*) uses a slug or snail as an intermediate host; the first stage larva enters the mollusk and develops to the third stage larva, which

is infectious to the definitive host—the cat. If a mouse eats the slug, the third stage larva will enter the mouse's tissues, but will not undergo any development.

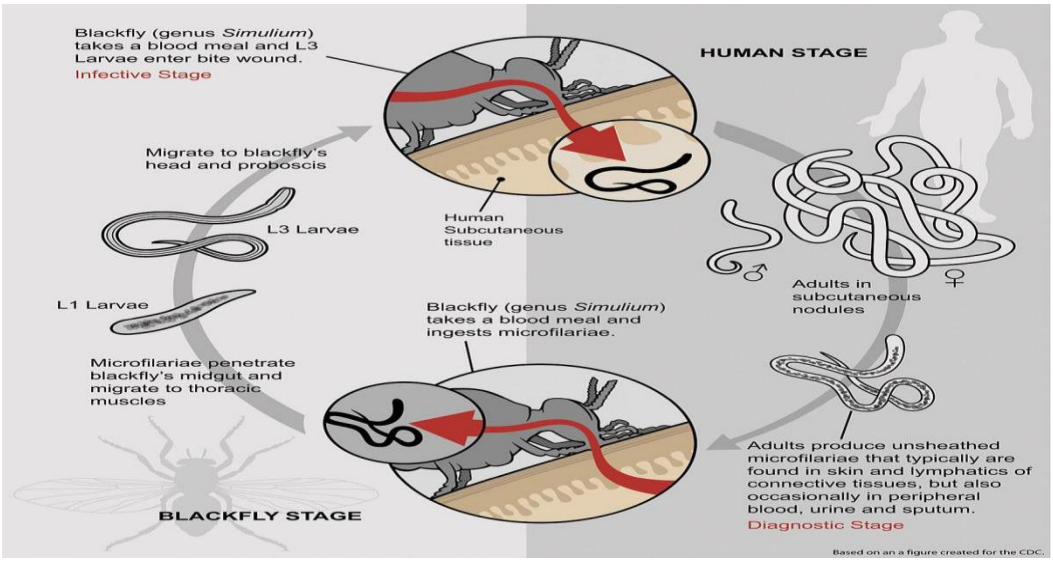

Fig. 1: Parasitic life cycle.***Source:*** *www.researchgate.com*

What is a parasite?

A parasite is an organism that lives in another organism, called the host, and often harms it. It depends on its host for survival.

Without a host, a parasite cannot live, grow and multiply. For this reason, it rarely kills the host, but it can spread diseases, and some of these can be fatal.

Parasites, unlike predators, are usually much smaller than their host and they reproduce at a faster rate.

Fast facts on parasites

- Parasites live on or in other organisms and thrive to the detriment of their host.

- Many different parasites can affect humans, and they can pass on diseases such as malaria and trichomoniasis.

- Ensuring that food is fully cooked, using insect repellant, and following good hand hygiene rules can reduce the risk of getting parasites.

Parasites range from microscopic in size to over 30 meters in length.

A parasite is an organism that lives within or on a host. The host is another organism.The parasite uses the host's resources to fuel its life cycle. It uses the host's resources to maintain itself.

Parasites vary widely. Around 70 percent are not visible to the human eye, such as the malarial parasite, but some worm parasites can reach over 30 meters in length.Parasites are not a disease, but they can spread diseases. Different parasites have different effects.

Endoparasite

These live inside the host. They include heartworm, tapeworm, and flatworms. An intercellular parasite lives in the spaces within the host's body, within the host's cells. They include bacteria and viruses.Endoparasites rely on a third organism, known as the vector, or carrier. The vector transmits the endoparasite to the host. The mosquito is a vector for many parasites, including the protozoan known as Plasmodium, which causes malaria.

Epiparasite

These feed on other parasites in a relationship known as hyperparasitism. A flea lives on a dog, but the flea may have a protozoan in its digestive tract. The protozoan is the hyper-parasite.

Types of parasites

There are three main types of parasites.

Protozoa: Examples include the single-celled organism known as Plasmodium. A protozoa can only multiply, or divide, within the host.

Helminths: These are worm parasites. Schistosomiasis is caused by a helminth. Other examples include roundworm, pinworm, trichina spiralis, tapeworm, and fluke.

Ectoparasites: These live on, rather than in their hosts. They include lice and fleas.

Symptoms

There are many types of parasite, and symptoms can vary widely. Sometimes these may resemble the symptoms of other conditions, such as a hormone deficiency, pneumonia, or food poisoning.

Some parasite-related problems, such as giardiasis and amebic dysentery, can cause abdominal pain.

Symptoms that might occur include:

- skin bumps or rashes
- weight loss, increased appetite, or both
- abdominal pain, diarrhea, and vomiting
- sleeping problems
- anemia
- aches and pains
- allergies
- weakness and general feeling unwell
- fever

However, parasites can pass on a wide variety of conditions, so symptoms are hard to predict.

Often there are no symptoms, or symptoms appear long after infection, but the parasite can still be transmitted to another person, who may develop symptoms.

Human parasites

Many types of parasites can affect humans. Here are some examples of parasites and the diseases they can cause.

Acanthamoebiasis

This tiny ameba can affect the eye, the skin, and the brain. It exists all over the world in water and soil. Individuals can become infected if they clean contact lenses with tap water.

Babesiosis

This disease that comes from parasites that are spread by ticks. It affects the red blood cells. The risk is highest in summer in the Northeast and upper Midwest of the United States.

Balantidiasis

This is passed on by *Balatidium coli*, a single-cell parasite that usually infects pigs but can, in rare cases, cause intestinal infection in humans. It can be spread through direct contact with pigs or by drinking contaminated water, usually in tropical regions.

Blastocystosis

This affects the intestines. The blastocystis enters humans through the fecal-oral route. A person can get it by eating food or drink contaminated with human or animal feces where the parasite is present.

Coccidiosis

This affects the intestines. Coccidia is passed on through the fecal-oral route. It is found around the world. It can also affect dogs and cats, but these are different kinds. Dogs, cats, and humans cannot normally infect each other.

Amoebiasis

This is caused by the parasite *Entamoeba histolytica*. It affects the intestines. It is more likely in tropical regions and in areas with high population density and poor sanitation. It is transmitted through the fecal-oral route.

Giardiasis

Giardia, or "beaver fever" affects the lumen of the small intestine. If humans ingest food or water contaminated with faeces, dormant cysts may infect the body.

Isosporiasis or cystosporiasis

This disease is caused by the *Cystoisospora belli*, previously known as *Isospora belli*. It affects the epithelial cells of the small intestine. It exists worldwide and is both treatable and preventable. It is passed on through the fecal-oral route.

Leishmaniasis

This is a disease that is passed on by parasites of the Leishmania family. It can affect the skin, the viscera, or the mucous membranes of the nose, mouth, and throat. It can be fatal. The parasite is transmitted by types of sandflies.

Primary amoebic meningoencephalitis (PAM)

This is passed on through a free-living ameba known as *Naegleria fowleri*. It affects the brain and the nervous system, and it is nearly always fatal within 1 to 18 days. It is transmitted through breathing in contaminated soil, swimming pools, and contaminated water, but not from drinking water.

Malaria

Different types of plasmodium affect the red blood cells. It exists in tropical regions and is transmitted by the Anopheles mosquito.

Rhinosporidiosis

This is caused by *Rhinosporidium seeberi*. It mainly affects the mucous of the nose, conjunctiva, and urethra. It is more common in India and Sri Lanka but can occur elsewhere. Polyps result in nasal masses that need to be removed through surgery. Bathing in common ponds can expose the nasal mucous to the parasite.

Toxoplasmosis

This is a parasitic pneumonia caused by the parasite *Toxoplasma gondii*. It affects the liver, heart, eyes and brain. It occurs worldwide. People can become infected after ingesting raw or undercooked pork, lamb, goat, or milk, or though contact with food or soil that is contaminated with cat feces.

A person with a healthy immune system will not usually have symptoms, but it can pose a risk during pregnancy and for those with a weakened immune system.

Trichomoniasis

Also known as "trich" this is a sexually transmitted infection (STI) caused by the parasite*Trichomonas vaginalis*. It affects the female urogenital tract. It can exist in males, but usually without symptoms.

Trypanosomiasis (Sleeping sickness)

This is passed on when the tsetse fly transmits a parasite of the Trypanosoma family. It affects the central nervous system, blood, and lymph. It leads to changes in sleep behaviour, among other symptoms, and it is considered fatal without treatment. It can cross the placenta and infect a foetus during pregnancy.

Chagas disease

This affects the blood, muscle, nerves, heart, esophagus and colon. It is transmitted through an insect bite. Over 300,000 people in the U.S. have the parasite that can lead to this disease.

Worms

Worms, or helminth organisms, can affect humans and animals.

Anisakiasis: This is caused by worms that can invade the intestines or the stomach wall. The worms are passed on through contaminated fresh or undercooked fish and squid.

Roundworms can be passed on by raccoons.

Roundworm: *Ascariasis*, or a roundworm infection, does not usually cause symptoms, but the worm may be visible in feces. It enters the body through consuming contaminated food or drink.

Raccoon roundworm: *Baylisascaris* is passed on through raccoon stools. It can affect the brain, lungs, liver, and intestines. It occurs in North America. People are advised not to keep raccoons as pets for this reason.

Clonorchiasis: Also known as Chinese liver fluke disease, this affects the gall bladder. Humans can become infected after ingesting raw or poorly processed or preserved freshwater fish.

Dioctophyme renalis infection: The giant kidney worm can move through the wall of the stomach to the liver and eventually the kidney. Humans can become infected after eating the eggs of the parasite in raw or undercooked freshwater fish.

Diphyllobothriasis tapeworm: This affects the intestines and blood. Humans can become infected after eating raw fish that live wholly or partly in fresh water. Prevalence has increased in some parts of the developed world, possibly due to the growing popularity of sushi, salted fillets, ceviche, and other raw-fish dishes.

Guinea worm: This affects subcutaneous tissues and muscle and causes blisters and ulcers. The worm may be visible in the blister. As the worms are shed or removed, they enter the soil or water, and are passed on from there.

Hookworms can cause intestinal disease.

Hookworm: These can cause intestinal disease. They lay their eggs in soil and the larvae can penetrate the skin of humans. Early symptoms include itching and a rash. They are most common in damp places with poor sanitation.

Hymenolepiasis: Humans can become infectedby ingesting material contaminated by rodents, cockroaches, mealworms, and flour beetles.

Echinococcosis tapeworm: Cystic echinococcosis can lead to cysts in the liver and lungs, and alveolar echinococcosis can cause a tumor in the liver. Humans can be infected after eating foods contaminated by the feces of an infected animal, or from direct contact with an animal.

Enterobiasis pinworm: A pinworm, or threadworm, *Enterobius vermicularis* can live in the colonand rectum of humans. The worm lays eggs around the anus while a person sleeps, leading to itching. It spreads through the oral-fecal route.

Fasciolosis liver fluke: This affects the gall bladder and liver. It is common in countries where cattle or sheep are reared, but rare in the U.S. It can affect the liver and the bile ducts and it causes gastrointestinal symptoms. It passes from one mammal to another through snails. A person may get it from eating watercress, for example.

Fasciolopsiasis intestinal fluke: This affects the intestines. It can also transmitted when consuming contaminated water plants or water.

Gnathostomiasis: This causes swellings under the skin, and occasionally affects the liver, the eyes, and the nervous system. It is rare, but it can be fatal. It occurs in Southeast Asia. It is transmitted by eating freshwater fish, pigs, snails, frogs, and chicken.

Loa loa filariasis: Also known as loaisis, this is caused by the *Loa loa* worm, or African eye worm. It causes itchy swellings on the body. It occurs mainly in Central and West Africa and is transmitted through deerfly bites.

Mansonellosis: This is passed on through the bites of midges or blackflies. It affects the layers under the surface of the skin, but it can enter the blood. It can lead to *angioedema*, swellings, skin rash, fever, and joint problems. It is present in Africa and Central America.

River blindness: Caused by a worm known as *Onchocerca volvulus*, this affects the eyes, skin, and other body tissues. It is found near fast flowing water. It is transmitted through the bite of a blackfly. It occurs in South America, but 90 percent of cases are in Africa.

Lung fluke: Also known as *paragonimiasis*, this affects the lungs, causing symptoms similar to those of tuberculosis (TB). However, it can reach the central nervous system, leading to meningitis. It is transmitted when eating undercooked or raw freshwater crabs, crayfishes, and other crustaceans. It is most common in parts of Asia.

Schistosomiasis, bilharzia, or snail fever: There are different types of schistosomiasis. They can affect the skin and internal organs. It results from exposure to fresh water that has snails in it that are infected with the blood fluke, or trematode worm. The worms are not found in the U.S. but they are common worldwide.

Sparganosis: Humans can become infected if they eat foods tainted with dog or cat feces that contains the larvae of a tapeworm of the Spirometra family. It can lead to a migrating abscess under the skin. It is rare.

Strongyloidiasis: This can lead to severe and possibly fatal immunodeficiency. The parasite penetrates through the skin and affects the lungs, skin, and intestines. It is passed on through direct contact with contaminated soil. It most occurs in tropical and subtropical regions.

Different types of tapeworm can affect the intestines, the liver, or the lungs.

Beef and pork tapeworms: Taeniasis is caused by tapeworms of the taenia family. They affect the intestines. They are passed on by eating undercooked beef or pork.

Toxocariasis: A roundworm transmits this infection from animals to humans. It affects the eyes, brain, and liver. It is caused by accidentally swallowing the eggs of the parasite, for example, when young children play with soil. Nearly 14 percent of people in the U.S. have antibodies, suggesting that millions have been exposed. Most never have symptoms.

Trichinosis: This is caused by the roundworm of the Trichinella family. Infection can lead to intestinal symptoms, fever, and muscle aches. It is passed on by eating undercooked meat.

Whipworm: Also known as trichuriasis, whipworms live in the large intestine. Eggs are passed in feces. It is common all over the world. Humans can become infected when ingesting the eggs, for example on unwashed fruit or vegetables.

Elephantiasis lymphatic filariasis: This is transmitted through mosquito bites. The adult worms live in the lymph system. Infection can lead to *lyphedema* and elephantiasis, in which swelling can cause disfigurement and disability. In the Americas, it is passed on by the *Culex quinquefasciatus*mosquito.

Ringworm is sometimes mistaken for a worm, but it is not a worm. It is a fungal infection.

Ectoparasites

These are parasites that live on the outside of the body, such as fleas.

Bed bugs are **ectoparasites**: They live on the outside of the body.

Bedbug: These can affect the skin and vision. They are found all over the world. Sharing clothing and bedding can spread infection. They may be present in newly rented accommodation and hotel rooms.

Body lice: These are common worldwide. Infection can spread through sexual activity, skin-to-skin contact, and sharing bedding or clothing.

Crab lice: These affect the pubic area and eyelashes. They are common all over the world and spread through sexual activity, skin-to-skin contact, and sharing bedding or clothing.

Demodex: These affect the eyebrow and eyelashes. They are common all over the world and can spread through prolonged skin contact.

Scabies: This affects the skin. It is common all over the world and can spread through sexual activity, skin-to-skin contact, and sharing bedding or clothing.

Screwworm: This is transmitted by a fly, and it affects skin and wounds. It is found in Central America and North Africa.

Head lice: These live on the scalp and affect the hair follicles. They are common all over the world and spread through head-to-head contact. A reaction to their saliva causes itching.

Parasites come in many shapes and sizes and can lead to a wide variety of symptoms and health issues. Some parasites are treatable and others are not.

Prevention

To increase your chance of avoiding parasites:

- find out which kind are prevalent in your area or in locations you may travel
- take precautions, for example, using insect repellant in places where mosquitoes are common
- be careful to eat only well-cooked fish and meat
- when traveling, drink only water from bottles with a sealed top
- take care when bathing in fresh-water lakes or rivers

If you have any symptoms, see a doctor.

4.0 Conclusion

The paratenic host can be useful in raising the chance that the parasite will be transmitted to the definitive host. For example, the cat lungworm (*Aelurostrongylus abstrusus*) uses a slug or snail as an intermediate host; the first stage larva enters the mollusk and develops to the third stage larva, which is infectious to the definitive host—the cat. If a mouse eats the slug, the third stage larva will enter the mouse's tissues, but will not undergo any development.

5.0 Summary

Parasite physiology is the scientific discipline concerned with the study of the mechanisms and processes that enable parasites to grow and reproduce.

A host in which parasites reproduce sexually is known as the *definitive*, *final* or *primary* host. In *intermediate* hosts, parasites either do not reproduce or do so asexually, but the parasite always develops to a new stage in this type of host. In some cases a parasite will infect a host, but not undergo any development, these hosts are known as paratenicor transport hosts.

6.0 Tutor-Marked Assignment

- Briefly write on what you know on the physiology of parasites.

Solutions

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Wilson, Edward O. *(2014).* The Meaning of Human Existence. *W. W. Norton & Company. p. 112.* ISBN 978-0-87140-480-0. *Parasites, in a phrase, are predators that eat prey in units of less than one. Tolerable parasites are those that have evolved to ensure their own survival and reproduction but at the same time with minimum pain and cost to the host*

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MODULE 2: PARASITE LIFE CYCLE PATTERN

Unit 1: Host specificity

1.0 Introduction

A small percentage of females *Anopheles* mosquitoes mate before they emerge and may arrive at the host already fertile, allowing them to construct a gallery and produce offspring without another male. This is assumed to occur either with a male that entered a natal gallery, or a sibling. In some parasitic species, e.g., *Dendroctonus micans* (Kugelann) and *Dendroctonus punctatus* LeConte, females attack by themselves, so mating occurs pre-emergence, or at least pre-attack (Grégoire, 1988; Furniss, 1995), or possibly both, as there is evidence that multiple mating can occur in *D. micans*. Exceptions to females being the pioneering sex among monogamous species occur in some genera, such as the ambrosia beetle genus *Gnathotrichus*, in which the male initiates attack and is joined by one female. This may indicate that monogamy is a derived state in these genera. In polygamous species, the male initiates gallery construction in the form of a nuptial chamber. Females will attempt to join the male, who may resist entrance, i.e., in polygamous species the male controls mate selection. Subsequent females encounter increasing resistance by the male. In some cases, a late-arriving female may enter a gallery by excavating her own entrance, i.e., thus circumventing male mate selection.

Some polygamous species include *pseudogynous* females, i.e., females that require mating, but produce offspring *parthenogenetically* without the use of male gametes. In some *scolytine* beetles, notably a few genera in the bark beetle tribe *Dryocoetini* and all species of the ambrosia beetle tribe Xyleborini, sex determination is by haplo-diploidy, with unmated diploid females producing haploid dwarf males with which they may later mate. Sib mating and fungal symbiosis are closely associated with this evolutionary path.

2.0 Objectives

At the end of this unit you will get to know how female mosquitoes mate. Female mosquitoes attack by themselves, so mating occurs pre-emergence, or at least pre-attack

3.0 Main content

3.1 Definition of term

A small percentage of females mate before they emerge, and may arrive at the host already fertile, allowing them to construct a gallery and produce offspring without another male. This is assumed to occur either with a male that entered a natal gallery, or a sibling. In some parasitic species, e.g., *Dendroctonus micans* (Kugelann) and *Dendroctonus punctatus* LeConte, females attack by themselves, so mating occurs pre-emergence, or at least pre-attack (Grégoire, 1988; Furniss, 1995), or possibly both, as there is evidence that multiple mating can occur in *D. micans*. Exceptions to females being the pioneering sex among monogamous species occur in some genera, such as the ambrosia beetle genus *Gnathotrichus*, in which the male initiates attack and is joined by one female. This may indicate that monogamy is a derived state in these genera. In polygamous species, the male initiates gallery construction in the form of a nuptial chamber. Females will attempt to join the male, who may resist entrance, i.e., in polygamous species the male controls mate selection. Subsequent females encounter increasing resistance by the male. In some cases, a late-arriving female may enter a gallery by excavating her own entrance, i.e., thus circumventing male mate selection.

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because host specificity is primarily determined by the ability of the ambrosia fungus to thrive in novel hosts. Consequently, ambrosia beetles are easily transported in dunnage or wood products, and many, e.g., *Xyleborinus saxeseni* (Ratzeburg), now have an almost worldwide distribution.

Host Specificity

Antricola and certain species of *Ornithodoros*). For example, during his graduate student years, Sonenshine colonized the bat tick, *Ornithodoros kelleyi*, in the laboratory. To feed the ticks, he had to maintain a colony of bats collected from limestone caves or attics of old buildings. No other hosts would do. Similarly, the cattle ticks *Boophilus microplus* and *B. annulatus* feed solely on cattle and, when available, on white-tailed deer. Other species exhibit limited host specificity (e.g., *D. variabilis*). Larvae and nymphs of *D. variabilis* feed on a wide range of small mammals (e.g., white-footed mice and meadow voles), but never on carnivores, ungulates, humans, or other large mammals.

4.0 Conclusion

Although the range of confirmed hosts is astounding, these opportunistic ticks have preferred hosts (e.g., mice for the immatures; deer, sheep, and other mammals for the adults). Host specificity is also strongly influenced by ecological adaptations, so that ticks adapted to a particular habitat in a given region of the world will encounter only vertebrates adapted to the same habitat.

5.0 Summary

As tick–host associations evolved, ticks gradually developed the ability to facilitate long-term feeding by evading or suppressing host homeostatic systems. For example, *I. scapularis* saliva contains pharmacologically active compounds that suppress edema and inflammation in their hosts while enhancing vasodilation. This leads to greater blood flow into the wound site without the pain and intense itching sensation so characteristic of the bites of mosquitoes or biting flies. These adaptations are most effective for the hosts encountered most frequently by each tick species, so-called preferred hosts, but less effective for uncommon hosts.

6.0 Tutor-marked Assignment

- How does a tick-host association evolve?

Solution

Ticks gradually developed the ability to facilitate long-term feeding by evading or suppressing host homeostatic systems. For example, *I. scapularis* saliva contains pharmacologically active compounds that suppress edema and inflammation in their hosts while enhancing vasodilation. This leads to greater blood flow into the wound site without the pain and intense itching sensation so characteristic of the bites of mosquitoes or biting flies. These adaptations are most effective for the hosts encountered most frequently by each tick species, so-called preferred hosts, but less effective for uncommon hosts.

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UNIT 2: PARASITE INFECTION INFESTATION

1.0 Introduction

2.0 Objectives

3.0 Main content

- **3.1 Parasite infection**
- **3.2 parasite infestation**

4.0 Conclusion

5.0 Summary

6.0 Tutor marked assignment

7.0 Reference/further reading

1.0 Introduction

Parasitic infection is a big problem in tropical and subtropical regions of the world. Malaria is one of the deadliest parasitic diseases. Parasitic infections can also occur in the United States. Common parasitic infections found in the United States include:

- Trichomoniasis
- Giardiasis
- Cryptosporidiosis
- Toxoplasmosis

2.0 Objectives

At the end of this unit, you will learn the differences between parasitic infection and parasitic infestation.

3.0 Main content

3.1 parasite infection

Parasitic infection is a big problem in tropical and subtropical regions of the world. Malaria is one of the deadliest parasitic diseases. Parasitic infections can also occur in the United States. Common parasitic infections found in the United States include:

- Trichomoniasis
- Giardiasis
- Cryptosporidiosis
- Toxoplasmosis

The symptoms of parasitic infections vary depending on the organism. For example:

- Trichomoniasis is a sexually transmitted infection caused by a parasite that often produces no symptoms. In some cases, it may cause itching, redness, irritation, and an unusual discharge in your genital area.
- Giardiasis may cause diarrhea, gas, upset stomach, greasy stools, and dehydration.

- Cryptosporidiosis may cause stomach cramps, stomach pain, nausea, vomiting, dehydration, weight loss, and fever.
- Toxoplasmosis may cause flu-like symptoms, including swollen lymph nodes and muscle aches or pains that can last for over a month.

Parasitic infections can be caused by three types of organisms:

- protozoa
- helminths
- ectoparasites

Protozoa are single-celled organisms that can live and multiply inside your body. Some infections caused by protozoa include giardiasis. This is a serious infection that you can contract from drinking water infected with *Giardia* protozoa.

Helminths are multi-celled organisms that can live in or outside of your body. They're more commonly known as worms. They include flatworms, tapeworms, thorny-headed worms,and roundworms.

Ecto-parasites are multi-celled organisms that live on or feed off your skin. They include some insects and arachnids, such as mosquitoes, fleas, ticks, and mites.

Parasitic infections can be spread in a number of ways. For example, protozoa and helminths can be spread through contaminated water, food, waste, soil, and blood. Some can be passed through sexual contact. Some parasites are spread by insects that act as a vector, or carrier, of the disease. For example, malaria is caused by parasitic protozoa that are transmitted by mosquitoes when they feed on humans.

3.2 Parasite Infestation

Amebiasis

Among various amebas, the *Entamoeba histolytica* is the one that invades tissues in man. As long as it remains in the lumen of the colon (luminal phase), it causes no problems. When it invades the bowel wall (invasive phase) it causes a diarrhoic syndrome and may spread to the liver where it forms amebic abscesses which are usually solitary. The patient experiences pain and

tenderness in that region and general symptoms with fever. The diagnosis of abscess is made with imaging techniques. The diagnosis of amebiasis is made by serological tests for amebic antibodies. The treatment is by drugs and rarely surgical. The first association of ameba with liver abscess was described by Loesch in St. Petersburg, Russia, in 1875.The figure shows clusters of ameba trophozoites in the tissue, large, up to 60 microns in diameter. This is the active mobile form. They have one small eccentric nucleus and cytoplasmic vacuoles sometimes containing red cells. The cystic form with round shape and multiple nuclei does not occur in the tissues.

Giardiasis

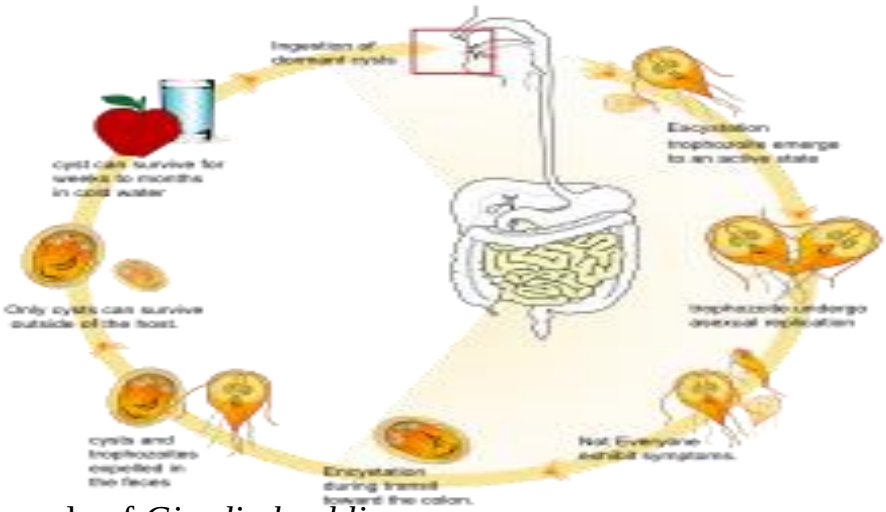

Fig 3 LIfe cycle of *Giardia lamblia*

Source: https://en.wikipedia.org/wiki/Giardia_lamblia

This organism causes a diarrhoic syndrome. It rarely affects the liver where it can cause cholecystitis, cholangitis and granulomatous hepatitis. The diagnosis is made with light microscopy in the stools. This flagellate protozoan represents a historical curiosity. It was first seen by the inventor of the microscope, Leeuwenhoek, in 1881, in his own stools. Its pathological significance, however, was recognized tow hundred years later by Lambl of Prague in 1859. The organism in its tropho stage is flat, rounded at one end and pointed at the other end. It has towed paired nuclei and 4 pairs of flagella. The encysted form is smaller without flagella and four nuclei.

Cryptosporidiosis

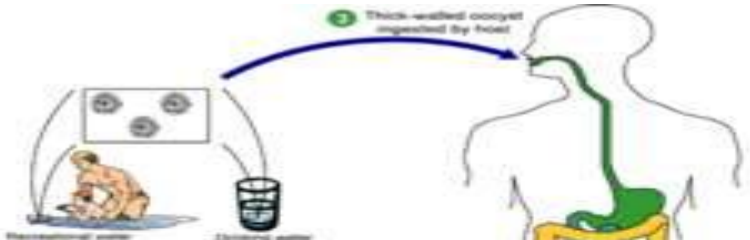

Fig 4 Life cycle of Cryptosporidium

Source: https://en.wikipedia.org/wiki

Cryptosporidium is a parasite of domestic and wild animals. In normal humans it causes no damage but in immunocompromised individuals it can cause a protracted diarrhoic syndrome and cholecystitis and cholangitis in the liver. The diagnosis is made by light microscopy. This organism is acid-fast positive.

Malaria

Fig 5 Malaria vector

www.greendaily.com/*Anopheles Mosquito* Images

There are four species of Plasmodium that cause disease in humans: *P. falciparum, P.malariae, P. vivax, P. ovale*. They have a sexual life cycle in the mosquito (sporogony) and an asexuals cycle in humans (schizogeny)They are transmitted by the female of the Anopheles mosquito. With their bites they inject sporozoites of these plasmodia into the blood stream. The organisms concentrate and proliferate in the liver where they form paranuclear masses(schizontes) visible even with a light microscope. Individual organisms (merozoites) are released from these masses and go to infect and destroy red cells. Schizontes of P.vivax and ovale may stay in hepatocyte for long time (hypnozoites) and cause relapses at distance of months and years; schizontes of *P.falciparum and malariae* do not have a long hepatic phase and develop into schizontes and merozoites in the red cells, capable to reinfect red cells but not hepatocytes. Gametocytes which develop from merozoites are sucked by mosquitoes to infect other individuals. There are, in summary two phases: extraerythrocytic and erythrocytic. Infected red cells will stick to endothelial cells of capillaries causing sequestration of red cells and disfunction of the microcirculation (anoxia) in various organs and fever. *P.vivax*and *ovale* cause benign tertian malaria; P. falciparum causes malignant tertian malaria; *P. malariae* causes quartan malaria. The malarial attacks consist of chills and fever recurring at 48 hour intervals for tertian and at 72 hour intervals for quartan type. Each attack lasts for-12 hours and coincide with the rupture of erythrocytes and release of a new pousse' of organisms.

Toxoplasmosis

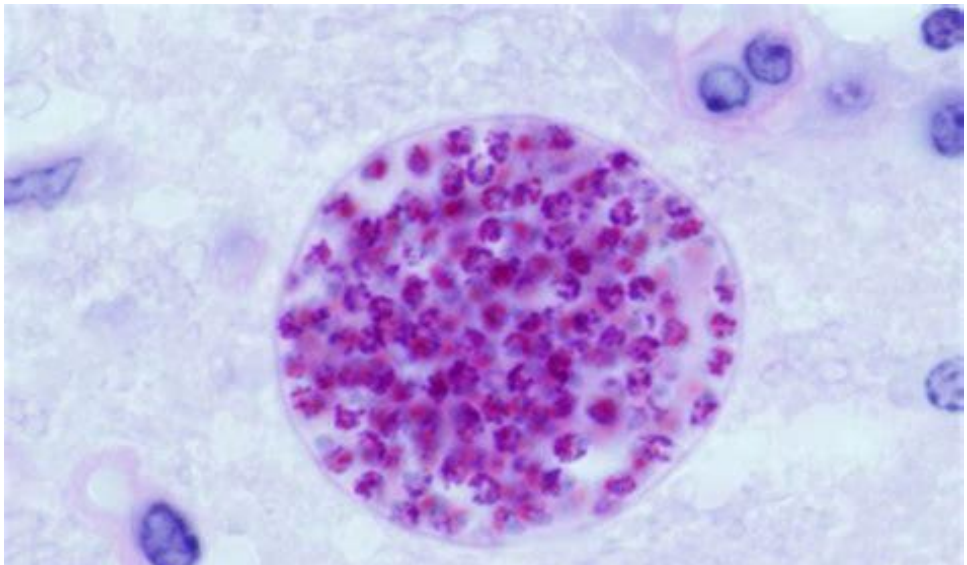

Fig 6: *Toxoplasma gondii*

Source: *www.health.zone/Toxoplasma Gondii Images*

It is due to Toxoplasma gondii, so-called because it was found in a rat by the name gondi in North Africa. It is acquired from raw meat and cat feces. It affects immunosuppressed individuals. It is a major intercurrent infection in cardiac and liver transplants. Fetuses are infected from the placenta and develop severe brain damage. Fluorescent antibody test for IgG and IgM are the best for diagnosis. It is readily seen in H&E sections. It is treatable.

Schistosomiasis

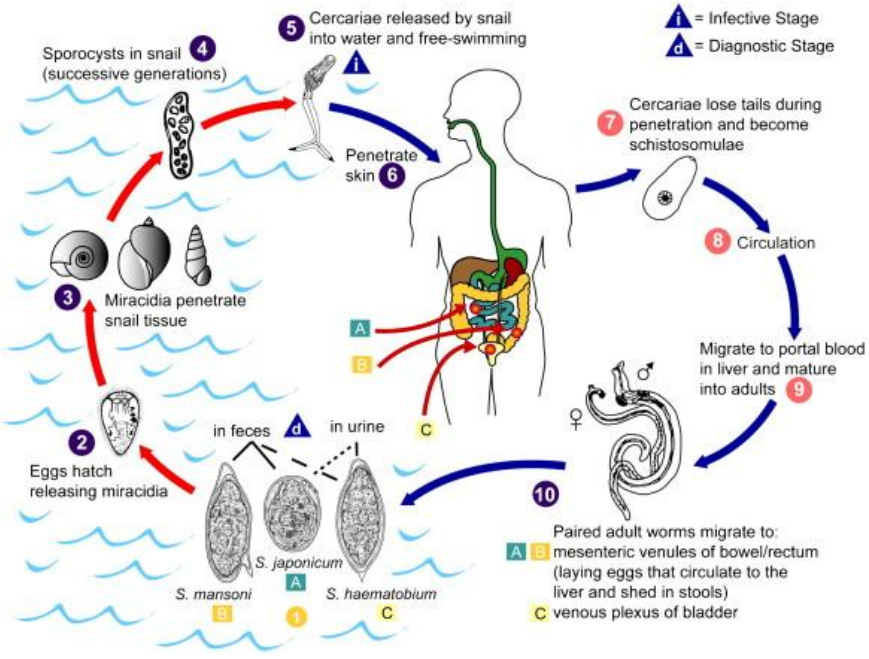

Fig 7: Life cycle of Schistosomiasis.
Source: www.researchgate.net

Schistosoma are trematodes (flat worms). The species that cause infestation in men are: *S. mansoni, S. japonicum , S. intercalatum, S. mekongi* which parasitize the intestinal venules and capable of spreading to the liver and *Schistosoma hematobium* which resides in the vesical venules and damage the urinary bladder and ureters. The first of these parasites (hematobium) was discovered in Egypt in 1852 by a German pathologist, Bilharz.

Clonorchiasis

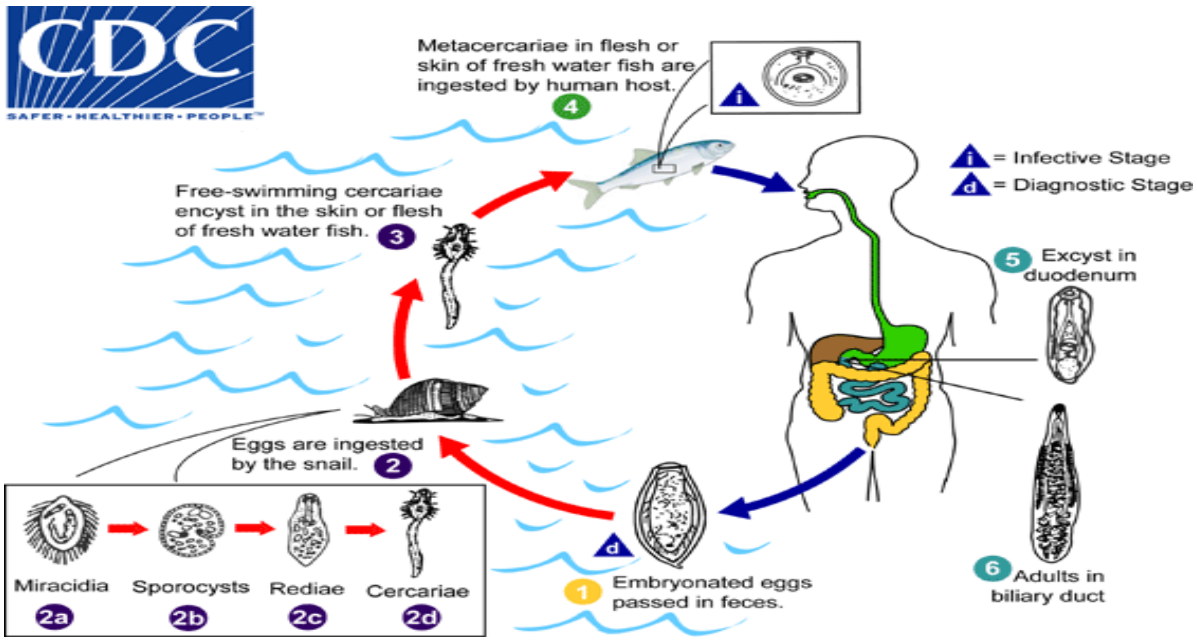

Fig 8: Life cycle of Clonorchiasis
Source: https://www.cdc.gov/dpdx/index.html

Clonorchis (opistorchis) sinensis is a flat fluke, 10-25 mm in length and 3-5 mm in width, reddish, transparent that resides in the major intra-hepatic bile ducts. It is found mainly in the Far East. It is acquired by eating row or poorly cooked fish which carry the eggs of this parasite in their scales. The infection causes dilatation of intrahepatic bile ducts resembling cysts and may cause purulent cholangitis with multiple liver abscesses due to *E.coli*. The presence of the fluke induces glandular hyperplasia of the bile ducts with increased mucus secretion. The glandular hyperplasia may be prominent and adenomatous. Cholangicarcinoma is associated with this infestation. Other tow similar trematodes are found in Poland and Siberia I) and in Thailand (*Opistorchis viverrini).* They cause a disease similar to *O.snensis* and are associated with cholangiocarcinoma.

Toxocariasis.

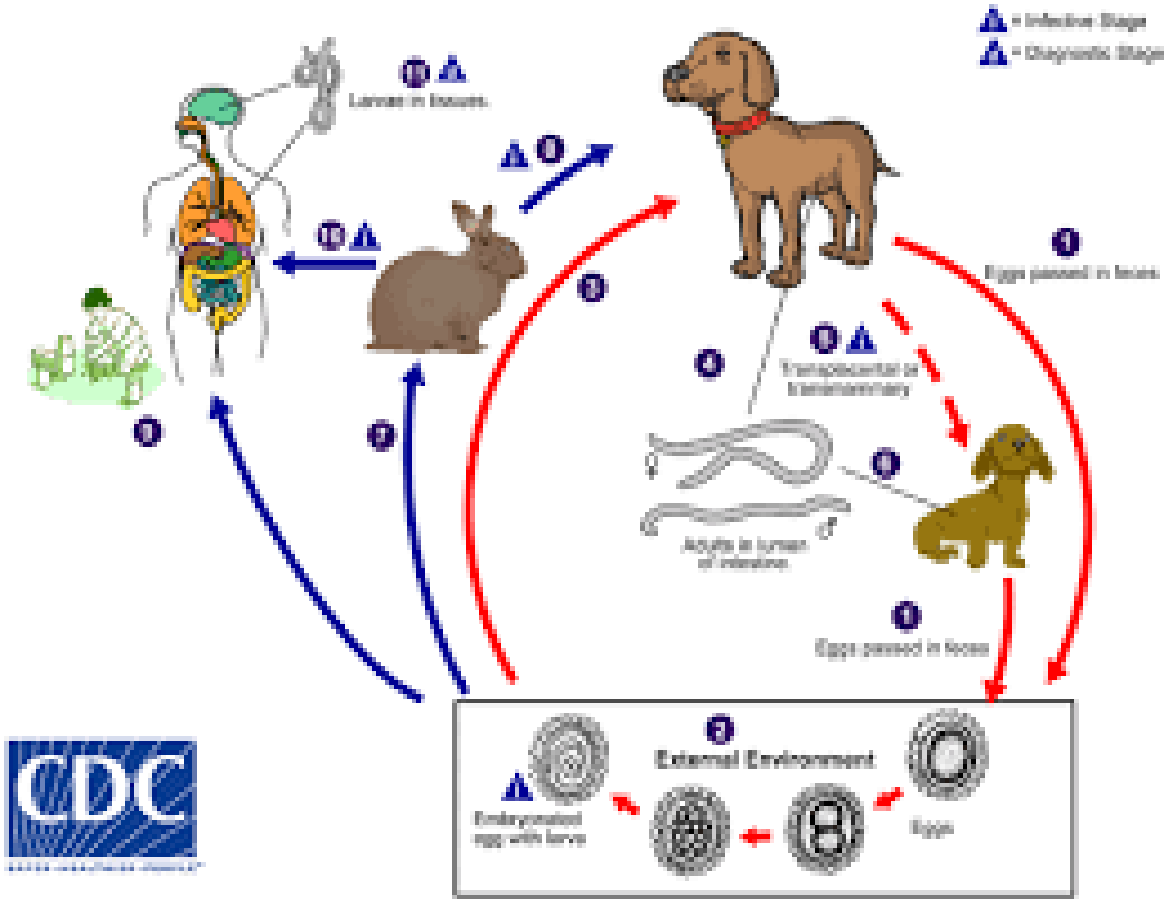

Fig 9: Life cycle of Toxocariasis.
Source: https://www.cdc.gov/parasites/toxocariasis/biology.html

Toxocara canis and *T.catis* are intestinal ascarids of dogs and cats. In humans, their ingested larvae migrate to the liver where they form granulomas in the portal tracts. The disease is more frequent in children being more in contact with dogs. The liver is the most affected organ. Here the granulomas can be seen as small-size whitish spots under the capsule. Invasion of other organs may also occur. The granulomas are mostly composed of eosinophil with some histiocytes at their periphery.

Capillariasis

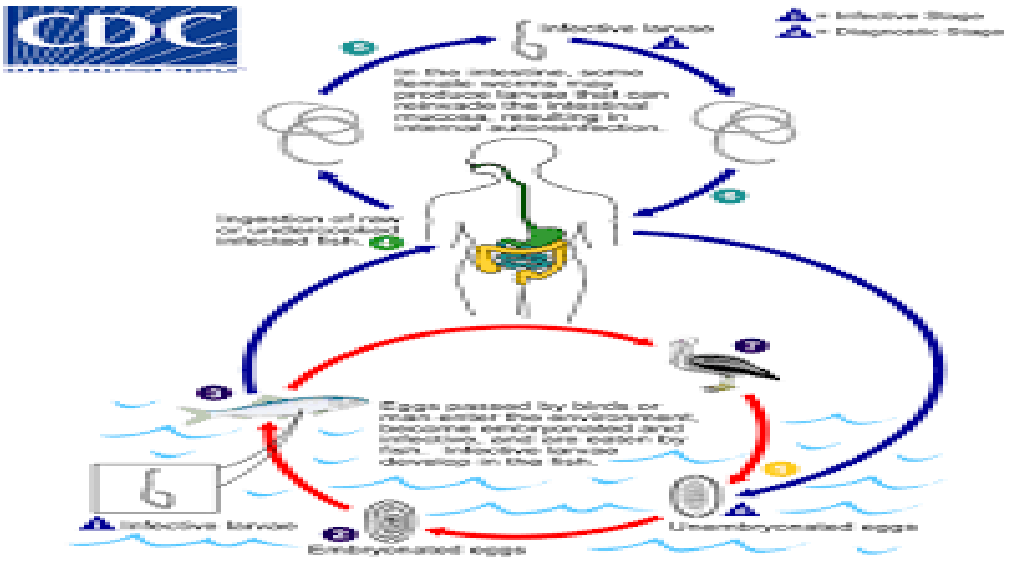

Fig 10: Life cycle of Capillariasis

Source: https://www.cdc.gov/parasites/capillaria/biology_c_philippinensis.html

It is due infestation with *Capillaria hepatica*, a common worm parasite of rats, and les commonly of squirrels, maskrat, hare beaver, chimpanzee and other monkeys. Few cases have been reported in man. The worm liver in the liver where it deposits its eggs. The eggs cause necrosis and fibrosis in the li and are not released in the feces. The infestation is contracted by injesting the infected liver. The worm and and eggs resemble Trichuris. The eggs have a birefringent. There is no treatment.

4.0 Conclusion

Parasitic infections can be spread in a number of ways. For example, protozoa and helminths can be spread through contaminated water, food, waste, soil, and blood. Some can be passed through sexual contact. Some parasites are spread by insects that act as a vector, or carrier, of the disease. For example, malaria is caused by parasitic protozoa that are transmitted by mosquitos when they feed on humans.

5.0 Summary

The infestation is contracted by injesting the infected liver. The worm and eggs resemble Trichuris. The eggs have a birefringent. There is no treatment.

6.0 Tutor-marked Assignment

Define the following terms:

- Toxoplasmosis
- Malaria

Solution

Toxoplasmosis
It is due to *Toxoplasma gondii*, so-called because it was found in a rat by the name gondi in North Africa. It is acquired from raw meat and cat feces. It affects immune suppressed individuals. It is a major intercurrent infection in cardiac and liver transplants. Fetuses are infected from the placenta and develop severe brain damage. Fluorescent antibody test for IgG and IgM are the best for diagnosis. It is readily seen in H&E sections. It is treatable.

Malaria
There are four species of Plasmodium that cause disease in humans: *P. falciparum, P.malariae, P. vivax, P. ovale.* They have a sexual life cycle in the mosquito (sporogony) and an asexuals cycle in humans (schizogeny)They are transmitted by the female of the Anopheles mosquito. With their bites they inject sporozoites of these plasmodia into the blood stream. The organisms concentrate and proliferate in the liver where they form paranuclear masses(schizontes) visible even with a light microscope. Individual organisms (merozoites) are released from these masses and go to infect and destroy red cells.

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Unit 3: PARASITE ECOLOGY

1.0 Introduction

2.0 Objectives

3.0 Main content

3.1 Definition of term

4.0 Conclusion

5.0 Summary

6.0 Tutor marked assignment

7.0 Reference/further reading

1.0 Introduction

Parasites possess a remarkable range of traits that have important consequences for their ecology and distribution. These traits, including various life histories and behaviors, have evolved as strategies to locate available hosts, survive and disperse among hosts, reproduce, and persist. Some parasites are highly specific to a single host species, while others are generalists and can use many species as hosts. Parasites can have complex life cycles, in which the adult parasite occupies a single host (often a vertebrate) and immature stages require one or multiple invertebrates, or they can have simple life cycles with direct transmission within one host species. Parasites can also have a profound range of effects on hosts and the surrounding communities, which can vary by geographic location and local environmental conditions. Every aspect of parasite biology and ecology affects the abundance, habitat distribution, and biogeography of parasite species.

Scientists at SERC take a multidisciplinary approach to parasite ecology research across a diverse range of ecosystems, from trees and plants in forests to invertebrates and fish in coastal ecosystems. Our approach combines biodiversity surveys and experiments with molecular and evolutionary ecology to gain a broad understanding of host-parasite interactions across spatial, temporal, and taxonomic scales.

2.0 Objectives

At the end of this unit you will get to know that Parasites possess a remarkable range of traits that have important consequences for their ecology and distribution. These traits, including various life histories and behaviors, have evolved as strategies to locate available hosts, survive and disperse among hosts, reproduce, and persist

3.0 Main content

3.1 Definition of term

Although parasites have traditionally been defined by a combination of conceptual and taxonomic features, I use an entirely conceptual definition here. I consider a parasite to be any small organism (including viruses) that lives in close association with a host organism and for which it seems reasonable to assume that the host carries some cost. These costs may be clearly visible, in the form of reduced fecundity or survival, but may in some cases be subtle. For example, reduced sexual attractiveness (leading to reduced mating success) or reduced competitive ability may not be very visible. I devote an entire chapter to discussing the fitness costs caused by parasites. This conceptual definition of a parasite includes members of various taxa, such as viruses, bacteria, fungi, and protozoa, but also includes functional categories (not taxonomically defined), such as pathogens and helminths. In contrast to typical predators, parasites do not always kill their hosts, and if they do, it may take a considerable amount of time, during which the parasite may be transmitted to other hosts, and the host remains in the community competing with other organisms for space, food, and mating partners.

In the literature on Cladocera and more specifically on *Daphnia*, parasites are often distinguished from epibionts. Whereas the former are usually endoparasites, i.e., located within the body of the host, the latter are located on the body surface and may therefore be labeled as ectoparasites. In the main part of this book, I concentrate on endoparasites and exclude epibionts. However, this is not to say that epibionts are not parasites or are not important. In fact, I believe that most epibionts fulfill the definition of parasites used here, because they are often closely associated with their hosts and cause harm to their hosts. This harm may not be directly visible, but there are certainly

increased costs for swimming, which may have consequences for other fitness components, such as fecundity, survival, competition, and mate finding (Threlkeld et al. 1993). It has also been suggested that epibiontic filter feeders compete with their hosts for food (Kankaala and Eloranta 1987). On the other hand, it has been suggested that under certain conditions, high loads of algal epibionts may provide additional food for the host and thus result in a net benefit (Barea-Arco et al. 2001). However, this form of a food supplementation is certainly not the typical effect of epibionts.

I do not include epibionts in this book, because I feel that there is less need to discuss the epidemiology of this functional group than for endoparasites. However, I will refer to them whenever it might further our understanding of*Daphnia*–parasite interactions.

HOST PARASITE INTERACTION

Parasites may be directly or indirectly involved in the ecology and evolution of a broad range of phenomena: host population dynamics and extinctions, maintenance of genetic diversity, sexual selection, evolution of genetic systems, and evolution of sexual recombination, to name just a few. Certainly, parasites possess features that make them very attractive as explanatory factors in the evolution and ecology of their hosts. These features include their high abundance in nearly every ecosystem, their typically narrow host range (compared with typical predators), their adverse effects on their hosts (e.g., reduced fecundity and survival), and density dependence during horizontal transmission (Anderson 1979, 1993; Anderson and May 1978; May and Anderson 1979; Price 1980).

On the other hand, hosts are the environment for the parasites and thus define their niche. Most parasites are not viable outside of their hosts for extended periods (not considering resting stages) and therefore—from the parasite's point of view—parasite and host form an inseparable biological unit. Thus, parasite ecology is closely linked to the ecology of its hosts, and the parasite's natural history is best seen in the light of its host's biology. In this book, I focus largely on members of the genus *Daphnia* as hosts. Whenever possible, I include information on other Cladocerans.

4.0 Conclusion

Scientists at SERC take a multidisciplinary approach to parasite ecology research across a diverse range of ecosystems, from trees and plants in forests to invertebrates and fish in coastal ecosystems. Our approach combines biodiversity surveys and experiments with molecular and evolutionary ecology to gain a broad understanding of host-parasite interactions across spatial, temporal, and taxonomic scales.

5.0 Summary

Parasites can also have a profound range of effects on hosts and the surrounding communities, which can vary by geographic location and local environmental conditions. Every aspect of parasite biology and ecology affects the abundance, habitat distribution, and biogeography of parasite species.

6.0 Tutor-Marked Assignment

- Define the term parasite

Solution

Parasite can bedefined as the combination of conceptual and taxonomic features. A parasite is consider to be any small organism (including viruses) that lives in close association with a host organism and for which it seems reasonable to assume that the host carries some cost. These costs may be clearly visible, in the form of reduced fecundity or survival, but may in some cases be subtle. For example, reduced sexual attractiveness (leading to reduced mating success) or reduced competitive ability may not be very visible.

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Unit 4: PARASITE PATHOLOGY

1.0 Introduction

PARASITE PATHOLOGY

Parasite pathology deals with the study of parasitic diseases transmissions, their signs and symptoms, causes, and their chemotherapy.

A parasitic disease, also known as parasitosis, is an infectious disease caused or transmitted by a parasite. Many parasites do not cause diseases. Parasitic diseases can affect practically all living organisms, including plants and mammals. The study of parasitic diseases is called parasitology.

Some parasites like *Toxoplasma gondii* and *Plasmodium* spp. can cause disease directly, but other organisms can cause disease by the toxins that they produce.

Although organisms such as bacteria function as parasites, the usage of the term "parasitic disease" is usually more restricted. The three main types of organisms causing these conditions are protozoa(causing protozoan infection), helminths (helminthiasis), and ectoparasites. Protozoa and helminths

are usually endoparasites (usually living inside the body of the host), while ectoparasites usually live on the surface of the host. Protozoa are single-celled, microscopic organisms that belong to the kingdom Protista. Helminths on the other hand are macroscopic, multicellular organisms that belong to the kingdom Animalia. Occasionally the definition of "parasitic disease" is restricted to diseases due to endoparasites.

2.0 Objectives

At the end of this unit, you will get to know what is meant by parasite pathology.

3.0 Main content

3.1 Sign and symptoms

Symptoms of parasites may not always be obvious. However, such symptoms may mimic anemia or a hormone deficiency.[5] Some of the symptoms caused by several worm infestations can include itchingaffecting the anus or the vaginal area, abdominal pain, weight loss, increased appetite, bowel obstructions, diarrhea, and vomiting eventually leading to dehydration, sleeping problems, worms present in the vomit or stools, anemia, aching muscles or joints, general malaise, allergies, fatigue, nervousness. Symptoms may also be confused with pneumonia or food poisoning.[6]

The effects caused by parasitic diseases range from mild discomfort to death. The nematode parasites *Necator americanus* and *Ancylostoma duodenale* cause human hookworm infection, which leads to anaemia, protein malnutrition and, in severely malnourished people, shortness of breath and weakness. This infection affects approximately 740 million people in the developing countries, including children and adults, of the tropics specifically in poor rural areas located in sub-Saharan Africa, Latin America, South-East Asia and China. Chronic hookworm in children leads to impaired physical and intellectual development, school performance and attendance are reduced. Pregnant women affected by a hookworm infection can also develop aneamia, which results in negative outcomes both for the mother and the infant. Some of them are: low birth weight, impaired milk production, as well as increased risk of death for the mother and the baby.

3.2 Causes

Mammals can get parasites from contaminated food or water, bug bites, or sexual contact. Ingestion of contaminated water can produce Giardia infections.

Parasites normally enter the body through the skin or mouth. Close contact with pets can lead to parasite infestation as dogs and cats are host to many parasites.

Other risks that can lead people to acquire parasites are walking with barefeet, inadequate disposal of feces, lack of hygiene, and close contact with someone carrying specific parasites, and eating undercooked foods, unwashed fruits and vegetables or foods from contaminated regions.

Parasites can also be transferred to their host by the bite of an insect vector, i.e. mosquito, bed bug, fleas.

3.3 Treatment

Parasitic infections can usually be treated with antiparasitic drugs.

Albendazole and mebendazole have been the treatments administered to entire populations to control hookworm infection. However, it is a costly option and both children and adults become reinfected within a few months after deparasitation occurs raising concerns because the treatment has to repeatedly be administered and drug resistance may occur.

Another medication administered to kill worm infections has been pyrantel pamoate. For some parasitic diseases, there is no treatment and, in the case of serious symptoms, medication intended to kill the parasite is administered, whereas, in other cases, symptom relief options are used. Recent researches have also proposed the use of viruses to treat infections caused by protozoa.

4.0 Conclusion

The three main types of organisms causing these conditions are protozoa(causing protozoan infection), helminths (helminthiasis), and ectoparasites. Protozoa and helminths are usually endoparasites (usually living inside the body of the host), while ectoparasites usually live on the surface of the host. Protozoa are single-celled, microscopic organisms that belong to the kingdom Protista. Helminths on the other hand are macroscopic,

multicellular organisms that belong to the kingdom Animalia. Occasionally the definition of "parasitic disease" is restricted to diseases due to endoparasites.

5.0 Summary

Parasitic diseases can affect practically all living organisms, including plants and mammals. The study of parasitic diseases is called parasite pathology.

Some parasites like *Toxoplasma gondii* and *Plasmodium* spp. can cause disease directly, but other organisms can cause disease by the toxins that they produce.

6.0 Tutor-Marked Assignment

- What do you understand by the term Parasite pathology?

- State the route of infection in parasite

Solution

-What do you understand by the term Parasite pathology?

Parasite pathology deals with the study of parasitic diseases transmissions, their signs and symptoms, causes, and their chemotherapy.

A parasitic disease, also known as parasitosis, is an infectious disease caused or transmitted by a parasite. Many parasites do not cause diseases. Parasitic diseases can affect practically all living organisms, including plants and mammals. The study of parasitic diseases is called parasitology.

- State the route of infection in parasite

Some parasites like *Toxoplasma gondii* and *Plasmodium* spp. can cause disease directly, but other organisms can cause disease by the toxins that they produce.

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MODULE 3: PATHOGENESIS AND SYMPTONOLOGY OF PARASITIC DISEASES

Unit 1: Malaria

Unit 2: *Amoebiasis*

Unit 3: *Schistosomiasis*

Unit 4: *Onchocerciasis*

UNIT 1: MALARIA

1.0 Introduction

2.0 Objectives

3.0 Main content

3.1 Major causes of malaria infection

4.0 Conclusion

5.0 Summary

6.0 Tutor marked assignment

7.0 Reference/further reading

1.0 Introduction

About 3.3 billion people – half the world's population – are at risk of malaria due to the main causative parasite, *Plasmodium falciparum*. Over 200 million cases of malaria occur each year, 90% of which occur in Africa, and 655 000 people died from the disease in 2010, making it one of the world's most important health problems. Increasing resistance of the parasite to currently available drugs has created an urgent need to discover new treatments.

However, this requires an improved understanding of the pathogenesis of malaria.

Malarial infection begins when a person is bitten by an infected female anopheles mosquito and Plasmodium spp (species) parasites in the form of sporozoites are injected into the bloodstream. The sporozoites travel to the liver, multiplying asexually over the next 7–10 days. During this time there are no symptoms. The parasites, now in the form of merozoites, emerge from the liver cells in vesicles and travel through the heart to the capillaries of the lungs. The vesicles eventually disintegrate, releasing the merozoites to enter the bloodstream where they invade and multiply in erythrocytes. When the cells burst, the parasites invade more erythrocytes. Clinical symptoms, including fever, occur in synchrony with the rupture of infected erythrocytes and the release of erythrocyte and parasite debris, including malarial pigment (hemozoin) and glycophosphatidylinositol, the putative 'malaria toxin

2.0 Objectives

On completion of this unit, you will get to know the first stage of malaria infection and how it gradually evolves.

3.0 Main content

3.1 Major cause of malaria infection

Vitamin A and Malaria

Malarial infection is accompanied by reductions in serum vitamin A concentrations from ≥120 mmol/l to ≤70 μmol/l (<20 μg/dl), a level usually taken to indicate deficiency in children.

In rodent models of malaria there is an inverse correlation between parasitemia and host vitamin A. Reduced serum vitamin A levels are also found consistently in children with malaria. Such observations have led to the suggestion that *P. falciparum* uses vitamin A from the host for its metabolism; in fact, *P. falciparum* selectively absorbs vitamin A from host tissues. This selective uptake of vitamin A was shown by Mizuno *et al*. in a study in which a standard isolate of the parasite was cultured with H-labeled vitamin A at concentrations of the vitamin normally present in human serum. The H-labeled vitamin A accumulated in the parasites in a parasitemia-dependent manner. Radioactivity

levels detected in the parasites, found mostly in the cytoplasm, also increased with parasite maturation from the ring to the late *trophozoite* stage.

Vitamin A supplementation appears to have a protective effect against the disease. For instance, a randomized double-blind, placebo-controlled trial of vitamin A supplementation on morbidity prevention in children in a malaria-endemic area of Papua New Guinea, showed that supplementation every three months for 13 months led to a 30% reduction in the number of confirmed *P. falciparum* febrile episodes, compared to the placebo group, and a 68% decrease in parasite density. A more recent randomized controlled trial on the effect of a single dose of 200 000 IU of vitamin A with daily zinc supplementation for six months on children in Burkina Faso resulted in a significant 30% reduction in slide-confirmed malaria fevers. One mechanism suggested for the parasite density-lowering effect is an up regulation of the phagocytotic receptor CD36 and down regulation of cytokines such as TNF-alpha by binding of 9-cis-retinoic acid (RA) to the *peroxisome proliferator*-activated receptor gamma (PPARgamma) or Retinoid-X-Receptor (RXR). However, while vitamin A supplementation reduces the incidence of uncomplicated malaria by about one-third, it does not appear to reduce the risk of deaths that can be specifically attributed to malaria. The therapeutic potential of vitamin A is also complicated by the fact that retinol (ROL) antagonizes the antimalarial effect of artemisinin. Reversal of the effect of a major drug treatment for malaria suggests that vitamin A may in some way contribute to the disease.

4.0 Conclusion

A more recent randomized controlled trial on the effect of a single dose of 200 000 IU of vitamin A with daily zinc supplementation for six months on children in Burkina Faso resulted in a significant 30% reduction in slide-confirmed malaria fevers. One mechanism suggested for the parasite density-lowering effect is an up regulation of the phagocytotic receptor CD36 and down regulation of cytokines such as TNF-alpha by binding of 9-cis-retinoic acid (RA) to the *peroxisome proliferator*-activated receptor gamma (PPARgamma) or Retinoid-X-Receptor (RXR).

5.0 Summary

Vitamin A supplementation reduces the incidence of uncomplicated malaria by about one-third, it does not appear to reduce the risk of deaths that can be specifically attributed to malaria. The therapeutic potential of vitamin A is also complicated by the fact that retinol (ROL) antagonizes the anti-malarial effect of artemisinin.

6.0 Tutor-marked Assignment

- What is the function of Vitamin A supplement?

Solution

Vitamin A supplementation reduces the incidence of uncomplicated malaria by about one-third, it does not appear to reduce the risk of deaths that can be specifically attributed to malaria. The therapeutic potential of vitamin A is also complicated by the fact that retinol (ROL) antagonizes the anti-malarial effect of artemisinin.

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UNIT 2: AMOEBIASIS

1.0 Introduction

Amoebiasis is a common infection of the human gastro-intestinal tract. Amoebiasis is more closely related to poor sanitation and socioeconomic status than to climate. It has worldwide distribution. It is a major health problem in China, South East and West Asia and Latin America, especially Mexico.

Amoebiasis is a disease caused by the parasite *Entamoeba histolytica*. Only about 10% to 20% of people who are infected with *E. histolytica* become sick from the infection.

2.0 Objectives

On completion of this course, we will get to know what is meant by amoebiasis, its causes, preventions and controls.

3.0 Main contents

3.1 Symptoms

The clinical spectrum ranges from asymptomatic infection, diarrhoea and dysentery to fulminant colitis and peritonitis as well as extra-intestinal *amoebiasis*.

Acute amoebiasis can present as diarrhoea or dysentery with frequent, small and often bloody stools.

Chronic amoebiasis can present with gastrointestinal symptoms plus fatigue, weight loss and occasional fever.

Extra-intestinal amoebiasis can occur if the parasite spreads to other organs, most commonly the liver where it causes amoebic liver abscess. Amoebic liver abscess presents with fever and right upper quadrant abdominal pain.

Other organs can also be involved, including pleuropulmonary, cardiac, cerebral, renal, genitourinary, peritoneal, and cutaneous sites. In developed countries, amebiasis primarily affects migrants from and travellers to endemic regions, men who have sex with men, and immunosuppressed or institutionalized individuals.

3.2 Causes

Amoebiasis is caused by parasite Entamoeba histolytica. Several protozoan species in the genus *Entamoeba* colonize humans, but not all of them are

associated with disease. It exists in two forms- Vegetative (trophozoite) and cystic forms (cyst). *Trophozoites* multiply and encyst in the colon. The cysts are excreted in stool and are infective to humans. Cysts remain viable and infective for several days in faeces, water, sewage and soil in the presence of moisture and low temperature.

Transmission occurs via:

- Faecal–oral route, either directly by person-to-person contact or indirectly by eating or drinking faecally contaminated food or water.
- Sexual transmission by oral-rectal contact is also recognized especially among male homosexuals.
- Vectors such as flies, cockroaches and rodents can also transmit the infection.

The incubation period for *E histolytica* infection is commonly 2-4 weeks but may range from a few days to years.

The use of night soil for agricultural purposes favours the spread of the disease. Epidemic/ outbreaks (occurrence of more cases of a disease than would be expected in a community or region during a given time period) are usually associated with sewage seepage into the water supply.

3.3 Diagnosis

Entamoeba histolytica must be differentiated from other intestinal protozoa. Microscopic identification of cysts and *trophozoites* in the stool is the common method for diagnosing *E. histolytica*. Differentiation is based on morphologic characteristics of the cysts and trophozoites.

In addition, *E. histolytica* trophozoites can also be identified in aspirates or biopsy samples obtained during colonoscopy or surgery.

3.4 Management

For symptomatic intestinal infection and extra intestinal disease, treatment with antiamoebic drugs should be taken with consultation of a physician. Asymptomatic patients infected with *E. histolytica* should also be treated with antiamoebic drugs, because they can infect others and because 4%–10% develop disease within a year if left untreated.

Liver aspiration- Liver aspiration is indicated only if abscesses are large (> 12 cm), abscess rupture is imminent, medical therapy has failed, or abscesses are present in the left lobe.

3.5 Prevention

Amoebiasis can be prevented and controlled both by non-specific and specific measures.

Non-specific measures are concerned with-

1. Improved water supply– The cysts are not killed by chlorine in amount used for water disinfection. Water filtration and boiling are more effective than chemical treatment of water against amoebiasis.
2. Sanitation–Safe disposal of human excreta coupled with the sanitary practice of washing hands after defecation and always before handling and consuming food.
3. Food safety– Uncooked fruits and vegetables should be washed thoroughly with safe water, peel fruits, and boil vegetables prior to eating. Measures should also include the protection of food and drink from flies and cockroaches and the control of these insects. Carriers, who pass cysts and are involved in handling food, whether at home, at street stalls, or in catering establishments, should be actively detected and treated since they are major transmitters of amoebiasis.
4. Health education of the public as well as health personnel at all levels about sanitation and food hygiene-Elementary hygienic practices should be propagated and constantly reinforced in schools, health care units, and the home through periodic campaigns using the mass media.
5. General social and economic development-The implementation of individual and community preventive measures (e.g., washing of hands, proper excreta disposal) should be an essential part of these activities.

4.0 Conclusion

Amoebiasis is a common infection of the human gastro-intestinal tract. Amoebiasis is more closely related to poor sanitation and socioeconomic status than to climate. It has worldwide distribution. It is a major health problem in China, South East and West Asia and Latin America, especially Mexico.

5.0 Summary

Amoebiasis can be prevented and controlled both by non-specific and specific measures.

6.0 Tutor-marked Assignment

- Define Schistosomiasis and state the symptoms

Solution

The clinical spectrum ranges from asymptomatic infection, diarrhoea and dysentery to fulminant colitis and peritonitis as well as extra-intestinal *amoebiasis*. Acute amoebiasis can present as diarrhoea or dysentery with frequent, small and often bloody stools.

Chronic amoebiasis can present with gastrointestinal symptoms plus fatigue, weight loss and occasional fever.

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UNIT 3: SCHISTOSOMIASIS

1.0 Introduction

Schistosomiasis is a disease that is caused by parasites (genus *Schistosoma*) that enter humans by attaching to the skin, penetrating it, and then migrating through the venous system to the portal veins where the parasites produce eggs and eventually, the symptoms of acute or chronic disease (for example, fever, abdominal discomfort, blood in stools). Health officials consider the disease to be a worm infection, or helminthiasis.

2.0 Objectives

At the end of this unit, you will get to know what is meant by Schistosomiasis, signs, symptoms and treatment of Schistosomiasis.

3.0 Main content

Signs, symptoms and treatment of Schistosomiasis

What are the symptoms and signs of schistosomiasis?

Although a few patients may have minor skin irritation when the cercariae enter the skin, most people do not develop symptoms until the eggs develop (about one to two months after initial skin penetration). Then, fever, chills, cough, and muscle aches can begin within one to two months of infection. However, most people have no symptoms at this early phase of infection. Unfortunately, a few patients develop acute schistosomiasis (Katayama fever) during this one- to two-month period, and their symptoms resemble those for serum sickness and are as follows:

- Fever
- Abdominal pain (liver/spleen area)
- Bloody diarrhea or blood in the stools
- Cough
- Malaise
- Headache
- Rash
- Body aches

The majority of people who develop chronic schistosomiasis have symptoms develop months or years after the initial exposure to the parasites. The following is a list of most symptoms associated with chronic schistosomiasis. Patients usually have a few of these symptoms.

- Abdominal pain
- Abdominal swelling (ascites)
- Bloody diarrhea or blood in the stools
- Blood in the urine and painful urination
- Shortness of breath and coughing
- Weakness
- Chest pain and palpitations
- Seizures
- Paralysis
- Mental status changes
- Lesions on the vulva or the perianal area

What is the treatment for schistosomiasis?

Currently, the drug used in most people is praziquantel (Biltricide); however, it only is effective against adult worms and does not affect eggs or immature worms. Treatment with this drug is simple and its dose is based on the patient's weight with two doses given on one day. However, the drug causes rapid disintegration of the worm which, in turn, allows the human immune system to attack the parasite. This immune response can cause localized reactions, which may increase the patient's symptoms. Corticosteroids are often used to reduce the symptoms of this reaction. Unfortunately, this response limits the use of praziquantel. Praziquantel and oxaminquine or artemether are used by some clinicians early in infections, or to treat individuals infected with both malaria and schistosomes, respectively.

Ocular schistosomiasis should not be treated with this praziquantel. Other organs with heavy parasite infections may not function well and require supportive care until the hyperimmune response abates after drug administration. Other drugs (oxamniquine, metrifonate, artemisinins, and trioxolanes) have been used in some patients but have limited effectiveness. New drugs are in development. Infectious disease specialists, ophthalmologists, and surgeons may treat someone with a schistosomiasis infection.

Surgical care may include removal of tumor masses, ligation of esophageal varices, shunt surgeries, and granuloma removal.

4.0 Conclusion

Although a few patients may have minor skin irritation when the cercariae enter the skin, most people do not develop symptoms until the eggs develop (about one to two months after initial skin penetration). Then, fever, chills, cough, and muscle aches can begin within one to two months of infection. However, most people have no symptoms at this early phase of infection. Unfortunately, a few patients develop acute schistosomiasis (Katayama fever) during this one- to two-month period, and their symptoms resemble those for serum sickness.

5.0 Summary

Treatment with this drug is simple and its dose is based on the patient's weight with two doses given on one day. However, the drug causes rapid disintegration of the worm which, in turn, allows the human immune system to attack the parasite. This immune response can cause localized reactions, which may

increase the patient's symptoms. Corticosteroids are often used to reduce the symptoms of this reaction. Unfortunately, this response limits the use of praziquantel. Praziquantel and oxaminquine or artemether are used by some clinicians early in infections, or to treat individuals infected with both malaria and schistosomes, respectively.

6.0 Tutor-Marked Assignment

- List the signs, symptoms and treatment of Shistosomiasis.

Solution

Although a few patients may have minor skin irritation when the cercariae enter the skin, most people do not develop symptoms until the eggs develop (about one to two months after initial skin penetration). Then, fever, chills, cough, and muscle aches can begin within one to two months of infection. However, most people have no symptoms at this early phase of infection. Unfortunately, a few patients develop acute schistosomiasis (Katayama fever) during this one- to two-month period, and their symptoms resemble those for serum sickness and are as follows:

- Fever
- Abdominal pain (liver/spleen area)
- Bloody diarrhea or blood in the stools
- Cough
- Malaise
- Headache
- Rash
- Body aches

The majority of people who develop chronic schistosomiasis have symptoms develop months or years after the initial exposure to the parasites. The following is a list of most symptoms associated with chronic schistosomiasis. Patients usually have a few of these symptoms.

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UNIT 4: ONCHOCERCHIASIS

1.0 Introduction

Simulium blackflies are the obligate intermediate hosts of *O. volvulus*. The flies breed in fast-flowing rivers, and infective-stage larvae (L3) are released from infected blackflies during a blood meal. L3 larvae undergo two molts to become adult worms, which can be detected in subcutaneous collagenous nodules. Females release approximately 1,000 microfilariae (L1) per day over a 9- to 14-year period, and the cycle is continued after uptake by blackflies during a blood meal.

2.0 Objectives

At the end of this unit, you will get to know what is meant by Onchocerhiasis, Signs, symptoms and treatment of onchocerciasis.

3.0 Main content

Signs, symptoms and treatment of onchocerciasis

There are different stages of onchocerciasis. In earlier stages, you may not have any symptoms. It can take up to a year for symptoms to appear and the infection to become apparent.

Once the infection becomes severe, symptoms may include:

- skin rashes
- extreme itching
- bumps under the skin
- loss of skin elasticity, which can make skin appear thin and brittle
- itching of the eyes
- changes to skin pigmentation
- enlarged groin
- cataracts
- light sensitivity
- loss of vision

In rare cases, you may also have swollen lymph glands.

The death of microfilariae induces intense inflammatory reactions, provoking most of skin and eye symptoms and clinical manifestations. Therefore, infected people develop rashes, severe itching and various skin lesions. The skin wastes away and loses elasticity, causing hanging groin. Sometimes the pigmentation layer of the skin is also affected, particularly in the lower legs known as leopard skin. Nodules also develop under the skin over several years.

Some infected people develop eye lesions which can lead to visual impairment and sometimes permanent blindness. Onchocerciasis is the world's second leading infectious cause of blindness. In the 1970s, blindness affected up to 50% of adults in some regions in West Africa, and people abandoned the fertile river valleys in fear of contracting the disease. Poverty and famine increased.

Economic losses were estimated at US$30 million, and onchocerciasis posed an obstacle to socioeconomic development.

Noddingsyndrome
Nodding syndrome is a neurological condition which affects children between the ages of 5 and 15 years causing progressive cognitive dysfunction, neurological deterioration, stunted growth and a characteristic nodding of the head. It has been reported in some onchocerciasis hyperendemic areas of United Republic of Tanzania, South Sudan and Uganda, without any proven causal relationship with *Onchocerca volvulus* parasite.

4.0 Conclusion

Nodding syndrome is a neurological condition which affects children between the ages of 5 and 15 years causing progressive cognitive dysfunction, neurological deterioration, stunted growth and a characteristic nodding of the head.

5.0 Summary

Onchocerciasis is the world's second leading infectious cause of blindness. In the 1970s, blindness affected up to 50% of adults in some regions in West Africa, and people abandoned the fertile river valleys in fear of contracting the disease. Poverty and famine increased. Economic losses were estimated at US$30 million, and onchocerciasis posed an obstacle to socioeconomic development.

6.0 Tutor-marked Assignment

- What is nodding syndrome

- List signs, symptoms and treatment of onchocerciasis

Solution

Nodding syndrome is a neurological condition which affects children between the ages of 5 and 15 years causing progressive cognitive dysfunction, neurological deterioration, stunted growth and a characteristic nodding of the head.

- List signs, symptoms and treatment of onchocerciasis

Once the infection becomes severe, symptoms may include:

- skin rashes
- extreme itching
- bumps under the skin
- loss of skin elasticity, which can make skin appear thin and brittle
- itching of the eyes
- changes to skin pigmentation
- enlarged groin
- cataracts
- light sensitivity
- loss of vision

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MODULE 4: STRATEGIES IN PARASITIC DISEASES CONTROL

Unit 1: Emerging and re-emerging parasitic control

Unit 2: Chemotherapy and chemoprophylaxis

Unit 3: Drug resistance in parasites

Unit 4: Prevention and control of parasite infections

Unit 1: Emerging and re-emerging parasitic control

1.0 Introduction

2.0 Objectives

3.0 Main content

3.1 Significance of Emerging and Re-emerging Parasitic Diseases

4.0 Conclusion

5.0 Summary

6.0 Tutor-Marked Assignment
7.0 Reference/Further Reading

1.0 Introduction

Emerging and re-emerging diseases are those diseases that have been newly appeared or that have existed in the past but are now rapidly increasing in frequency, geographical range, or both. For instance the United State Institute of Medicine has defined emerging infections as those whose incidences in humans have increased within the past two decades or threatens to increase in the near future. Emergence could be due to a new agent being widely distributed or the diagnosis of a pathogen that has been present and causing disease without detection. It could also be due to discovery that an existing pathogen is actually infectious. Emergence has also been used to describe the reappearance of a known infection after a decline in incidences.

2.0 Objectives

On completion of this unit, you will be learning more on what is meant by emerging and re-emerging diseases, significance and types.

3.0 Main content

3.1 Significance of Emerging and Re-emerging Parasitic Diseases

Emerging zoonotic diseases hamper both human and animal health and also cause economic loss. Emergence of drug resistant pathogens poses many challenges to governments of either channelling a number of resources for control the infections or lose the animals or humans.

TYPES OF EMERGING AND RE-EMERGING PARASITIC DISEASES

A good number of emerging and re-emerging diseases/parasites have been recognized and recorded in various parts of the world and are either helminth, protozoal/rickettsial or entomological/entomologically-borne in origin. In Nigeria, some members of these classes of parasites are also common. These include: lyme boreliosis, cryptosporidiosis, malaria and yellow fever. However, many other emerging parasites and disease conditions which are not on this list but are also tagged as emerging and re-emerging and occurring not necessarily

only in Nigeria or the entire West African region but in other regions of the world have been listed under the relevant subheadings namely helminth, protozoa/rickettsial and entomological/ vector borne emerging and re-emerging animal parasitic diseases.

4.0 Conclusion

Parasitic diseases can be broadly divided into two classes, namely animal health parasitic diseases which affect animals and human health parasitic diseases which affect humans. However, there is no clear distinction between them in the case of the zoonotic parasites which have the potential to infect both man and animals, thereby affecting the health of both classes. A good example of a zoonosis is echinoccocosis.

5.0 Summary

Emergence has also been used to describe the reappearance of a known infection after a decline in incidences. World health organization (WHO)/Food and agricultural organization (FAO)/ Office international desepizooties (OIE) joint consultation on emerging zoonotic diseases in May, 2004 defined an emerging zoonosis as a zoonosis that is newly recognized or newly evolved or that has occurred previously but shows an increase in incidence or expansion in geographical, host or vector range. Emerging infectious diseases are said to be dominated by zoonoses with majority of them originating from wildlife.

6.0 Tutor-marked Assignment

- Define emerging and re-emerging diseases.
- A good example of a zoonosis is?
- Types of emerging and re-emerging.

Solution

- Define emerging and re-emerging diseases.

Emerging infectious diseases (EID) are those diseases that have been newly appeared or that have existed in the past but are now rapidly increasing in frequency, geographical range, or both

- A good example of a zoonosis is?
  Echinoccocosis.
- Types of emerging and re-emerging

These include: lyme boreliosis, cryptosporidiosis, malaria and yellow fever

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Unit 2: Chemotherapy and chemoprophylaxis

1.0 Introduction

Chemotherapy is the use of any drug to treat any disease. But to most people, the word chemotherapy means drugs used for cancer treatment. It's often shortened to "chemo."

Chemoprevention (also **Chemoprophylaxis**) refers to the administration of a medication for the purpose of preventing disease or infection.[1] Antibiotics, for example, may be administered to patients with disorders of immune system function to prevent bacterial infections (particularly opportunistic infection).[2] Antibiotics may also be administered to healthy individuals to limit the spread of an epidemic, or to patients who have repeated infections (such as urinary tract infections) to prevent recurrence. It may also refer to the administration of heparin to prevent deep venous thrombosis in hospitalized patients.

In some cases, chemoprophylaxis is initiated to prevent the spread of an existing infection in an individual to a new organ system, as when intrathecal chemotherapy is administered in patients with malignancy to prevent further infection.

2.0 Objectives

At the end of this unit, you will know the differences between chemotherapy and chemoprophylaxis.

3.0 Main content

3.1 Definition of term

Chemotherapy is the use of any drug to treat any disease. But to most people, the word chemotherapy means drugs used for cancer treatment. It's often shortened to "chemo."

Surgery and radiation therapy remove, kill, or damage cancer cells in a certain area, but chemo can work throughout the whole body. This means chemo can kill cancer cells that have spread (metastasized) to parts of the body far away from the original (primary) tumor.

Goals of chemotherapy treatment

If your doctor has recommended chemotherapy to treat your cancer, it's important to understand the goals of treatment when making treatment decisions. There are three main goals for chemotherapy (chemo) in cancer treatment:

1. Cure
2. Control
3. Palliation

Cure

If possible, chemo is used to cure cancer, meaning that the cancer is destroyed – it goes away and doesn't come back.

Most doctors don't use the word "cure" except as a possibility or intention. So, when giving treatment that has a chance of curing a person's cancer, the doctor may describe it as treatment with *curative intent*.

There are no guarantees, and though cure may be the goal, it doesn't always work out that way. It often takes many years to know if a person's cancer is really cured.

Control

If cure is not possible, the goal may be to control the disease. Chemo is used to shrink tumors and/or stop the cancer from growing and spreading. This can help the person with cancer feel better and live longer.

In many cases, the cancer doesn't completely go away, but is controlled and managed as a chronic disease, much like heart disease or diabetes. In other cases, the cancer may even seem to have gone away for a while, but it's expected to come back. Then chemo can be given again.

Palliation

Chemo can also be used to ease symptoms caused by the cancer. This is called *palliative chemotherapy* or palliation.

When the cancer is at an advanced stage, meaning it's not under control and has spread from where it started to other parts of the body, the goal may be to

improve the quality of life or help the person feel better. For instance, chemo may be used to help shrink a tumor that's causing pain or pressure.

Chemoprevention (also **Chemoprophylaxis**) refers to the administration of a medication for the purpose of preventing disease or infection. Antibiotics, for example, may be administered to patients with disorders of immune system function to prevent bacterial infections (particularly opportunistic infection). Antibiotics may also be administered to healthy individuals to limit the spread of an epidemic, or to patients who have repeated infections (such as urinary tract infections) to prevent recurrence. It may also refer to the administration of heparin to prevent deep venous thrombosis in hospitalized patients.

In some cases, chemoprophylaxis is initiated to prevent the spread of an existing infection in an individual to a new organ system, as when intrathecal chemotherapy is administered in patients with malignancy to prevent further infection.

The use of chemoprophylaxis is limited primarily by two factors: risk and financial costs.

- All medications have the potential to cause side effects. In general, chemoprophylaxis should be initiated only when the benefits of treatment outweigh the risks.
- The cost associated with chemoprophylaxis may be prohibitive, particularly when the cost of treatment is high or the incidence of the target disease is low. Many forms of chemoprophylaxis are therefore not cost-effective.

Specific diseases

Using chemoprophylaxis as a treatment against early signs of tuberculosis has proven to be effective. In familial *adenomatous polyposis* physicians observed polyps regression with NSAIDs for anti-inflammatory therapy. Chemoprophylaxis is also used to treat several different varieties of meningococcal infections for close contact exposure to *Neisseria meningitidis.*

4.0 CONCLUSION

It's important to know that any treatment that's used to reduce symptoms or improve comfort is called *palliative care*. For example, anti-nausea treatments

or pain medicines are palliative, and can be used at all stages of treatment. It can be confusing when chemo is used as a palliative treatment, because it's most often used to try to cure or control the cancer. But when it's used with the goal of comfort, chemo becomes palliative care.

5.0 Summary

When the cancer is at an advanced stage, meaning it's not under control and has spread from where it started to other parts of the body, the goal may be to improve the quality of life or help the person feel better. For instance, chemo may be used to help shrink a tumor that's causing pain or pressure.

6.0 Tutor-Marked Assignment

Define the following terms

- Chemotherapy
- Chemoprophylaxis

Solution

-Chemotherapy is the use of any drug to treat any disease. But to most people, the word chemotherapy means drugs used for cancer treatment. It's often shortened to "chemo."

-Chemoprevention (also **Chemoprophylaxis**) refers to the administration of a medication for the purpose of preventing disease or infection.[1] Antibiotics, for example, may be administered to patients with disorders of immune system function to prevent bacterial infections (particularly opportunistic infection).[2] Antibiotics may also be administered to healthy individuals to limit the spread of an epidemic, or to patients who have repeated infections (such as urinary tract infections) to prevent recurrence. It may also refer to the administration of heparin to prevent deep venous thrombosis in hospitalized patients.

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Unit 3: Drug resistance in parasites

Unit 3: Drug resistance in parasites

1.0 Introduction

4.0 Objectives

3.0 Main content

3.3 Significance of Emerging and Re-emerging Parasitic Diseases

4.0 Conclusion

5.0 Summary

6.0 Tutor-Marked Assignment

7.0 Reference/Further Reading

1.0 Introduction

As with all other anti-infectives (antibiotics, anti-viral drugs, and anthelminthics), the limited arsenal of anti-protozoal drugs is being depleted by a combination of two factors: increasing drug resistance and the failure to replace old and often shamefully inadequate drugs, including those compromised by (cross)-resistance, through the development of new anti-parasitics. Both factors are equally to blame: a leaking bathtub may have plenty of water if the tap is left open; if not, it will soon be empty. Here, I will reflect on the factors that contribute to the drug resistance emergency that is unfolding around us, specifically resistance in protozoan parasites.

2.0 Objectives

At the end of this unit, you will learn how drugs

3.0 Main content

3.1 Drug resistance in parasite

Infections with protozoan pathogens will be with us for the foreseeable future. There is still no effective malaria vaccine despite enormous investment in money and effort over many years. *Leishmaniasis* is still spreading, including in Southern Europe. At least 100 million women worldwide suffer infection with the sexually transmitted *Trichomonas vaginalis*. Chagas Disease (American *trypanosomiasis*) affects communities from Texas to Argentina. Sleeping sickness (human African *trypanosomiasis*) remains a scourge in sub-Saharan Africa. Billions of people are infected with *Toxoplasma gondii*. Then, there are *Cryptosporidium* spp., *Entamoeba histolytica*, and *Giardia* spp. — but you get the idea.

In almost all cases, we rely on treatment (no vaccines) with a few old drugs that would never pass safety evaluation if entered into trials now. Despite the clear clinical need, there is in fact very little serious new drug development going on for these protozoan infections, and what little there is, is driven by private organisations including the Medicines for Malaria Venture (MMV), the Drugs for Neglected Diseases initiative (DNDi), the Wellcome Trust (in part through its support of the Drug Development Unit at the University of Dundee), the Bill and Melinda Gates Foundation [in part through the Consortium for Parasitic Drug Development (CPDD)], and the Institute for One World Health rather than governments or the private sector. These organisations do phenomenally good work with limited resources and have taken multiple new drugs or formulations into clinical use or trials. However, their budgets clearly fall far short of the

level of effort that is needed to tackle all these neglected protozoan diseases. The investment discrepancy with welfare diseases such as obesity, atherosclerosis, diabetes, and some forms of cancer does not need further elaboration.

Moreover, while malaria and the kinetoplastid diseases have a champion in MMV and DNDi, respectively, there is no such champion for trichomoniasis, cryptosporidiosis, *giardiasis*, toxoplasmosis, etc. And that is not mentioning important protozoan animal infections such as nagana (e.g. cattle; *Trypanosoma congolense*, *T. brucei*, and *T. vivax*), surra (e.g. camels, buffalo; *T. evansi*), babesiosis (cattle, dogs; *Babesia bovis*, *B. canis*), dourine (horses; *T. equiperdum*), theileriosis (cattle, sheep, goats; *Theileria annulata*, *Theileria parva*), and *leishmaniosis* (canines; *Leishmania* spp.), for which usually very few treatment options exist. For the veterinary applications, again, little drug development is taking place, as the problems are mostly those of tropical countries and poor farming communities and herdsmen, and this does not fit the necessarily profit-driven drug discovery model, nor does it translate into easy logistics of delivery and administration.
The inevitable consequence is the use, for decades, of the same old treatments, often inexpertly, predictably giving rise to the drug resistance that follows close behind. So far, so obvious; but is resistance so inevitable that we should just shrug and move on? After a century of chemotherapy, are there any lessons learned and steps that can be taken to minimise the impact of (early onset) drug resistance in protozoan disease?

Steps that can be taken to minimise the impact of (early onset) drug resistance in protozoan disease.

Actually, the best strategy is to avoid over-reliance on chemotherapy in the first place — anything that reduces transmission rates brings the disease burden down. Good sanitation reduces transmission of waterborne parasites such as *Giardia*, *Entamoeba*, and *Cryptosporidium*, just as condoms prevent trichomoniasis. Clearly, effective vaccines would be our best chance of actually eradicating specific diseases, but none are likely to be approved any time soon. While a commercial vaccine for canine leishmaniasis is now available, it appears to be more effective in preventing disease progression than infection rates, and one study concluded that a simple anti-flea collar on the dog prevented infection much more effectively. Similarly, insecticide-treated bed nets have saved many people from malaria, housing improvements, insecticide

and the screening of blood donors greatly reduced transmission of Chagas disease, and mathematical modelling shows that spraying cattle with insecticide is much more effective against tsetse-transmitted trypanosomiasis than chemotherapy, especially when combined with scent-baited tsetse traps or insecticide-treated tsetse 'targets' that can be as small as 0.06 m^2 to be effective. One form of tsetse control that has attracted much attention is the use of sterile insect technology (SIT), which eliminated the insects from the island of Zanzibar [8], but this is a very expensive option, and the release of large numbers of (sterile) tsetse risks a dramatic increase in transmission in the short term. Moreover, reinvasion by wild-type flies from adjourning areas would be impossible to prevent on much of the African continent. An example of an alternative to vector control is the use of trypanotolerant indigenous cattle, but these breeds usually have much lower food production value than imported ones, and can still carry and transmit the parasite, and may therefore perpetuate the problem by masking it. Indeed, there is a strong case to be made to also treat asymptomatic infections for two main reasons: continued transmission, and the risk that the infection will not remain asymptomatic. For *T. b. gambiense* sleeping sickness, asymptomatic carriers present a serious obstacle to eradication, however, even if they could be identified, the treatment of healthy carriers raises ethical issues, especially with the currently available drugs and their well-documented side effects.

Vector control, repellents and barriers such as bed nets are all important in reducing transmission but more so for some protozoan diseases than others; *Toxoplasma* and *Giardia*, to name just two human parasites, are not transmitted by insect vectors, nor is dourine, which is caused by *T. equiperdum*. Moreover, full eradication by vector/transmission control is at best a long-term prospect, and meanwhile infected patients and animals must be effectively treated. Given that no new treatments will be introduced for most of these infections, we must make the best possible use of the current pharmacopoeia and not lose any to drug resistance. One good strategy is to go all-in with a full eradication programme of active case finding, mass treatment, diverse and aggressive vector control measures and the monitoring of treatment outcomes, with a second-line treatment option for the cases of drug failure – and see it through to the end. This does not allow for resistance to take hold and spread, picking off one valuable (near-irreplaceable) drug at the time. Of course, this requires a very high level of funding and organisation, and if not successfully implemented, the mass treatment programme can lead exactly to early-onset

resistance as was arguably the case with chloroquine and malaria. Such an approach is likely to work, but this ideal scenario is probably unrealistic given current realities such as the lack of political leadership, prioritisation and funding for the control of the diseases of the poor.

Is drug resistance really inevitable, or is it possible to develop drugs to which the parasites simply cannot develop resistance? It is worth exploring this question, especially at this time, when confidence in antibiotics is fast disappearing because of rapidly adapting pathogens. However, antibiotics are generally products produced by an organism to defend themselves against (other) bacteria, which means that bacteria have already been exposed to them for periods on an evolutionary scale, achieving an ecological balance. In other words, antibiotic resistance adaptations are as much part of the natural world as are the antibiotics themselves. But, is the situation any different for eukaryotic parasites and the anti-parasitic drugs, which are, with a few exceptions such as tetracycline for malaria, not natural compounds? Not fundamentally, no: protozoan parasites still adapt, and have become resistant to many standard treatments. Common adaptations include: target enzyme mutations reducing interactions with the drug (antifolates in malaria) reduced drug uptake (diminazene aceturate in *T. b. brucei* or antimonials in *L. donovani*); up-regulation of a metabolic bypass (methotrexate in *Leishmania*); failing to activate a prodrug (nifurtimox in *T. cruzi*); increased drug efflux (chloroquine in malaria); and even the failure to produce the target (amphotericin B in *Leishmania*). The genetic mechanisms of resistance include gene deletions, point mutations in targets or transporters (enabling or disabling), copy number variations, base pair insertions/deletions causing frame shifts in the target gene and the formation of chimeric genes through recombination.

Despite the plethora of mechanisms by which protozoa can acquire drug resistance, for some drugs this happens much faster than for others. In many cases, a single mutation or gene deletion is sufficient for a loss of sensitivity, sometimes followed by secondary mutations to give higher levels of resistance. Whereas a target protein is of course usually essential, active-site mutations that selectively lose affinity for an inhibitor, yet retain sufficient functionality, are certainly possible. In the case of a transporter mutation, the transporter is usually not essential, because of redundancy or its substrate is non-essential to the cell, and in such cases the mutation can be disabling to the transport of both the physiological substrate and the drug. In other cases, however, resistance does not come about so easily. The drug may not have a single protein target

that can be mutated but may bind instead to DNA as intercalator or minor groove binder **(**e.g. phenanthridines, diamidines), or e.g. to haem (chloroquine), or be more generally cytotoxic, with multiple targets (polypharmacology), as may be the case for heavy metal drugs (arsenicals, antimonials) and suramin , in which case selectivity depends mostly on selective entry into the parasite rather than into the host cells, through unique transporters or other uptake mechanisms.

In some such cases, resistance still occurs, through mutation or deletion of these transporters, but in the case of pentamidine, for instance, clinical resistance for sleeping sickness has not been reported despite being virtually the only drug used against early-stage *T. b. gambiense*sleeping sickness since the mid-1930s, including population-scale mass treatment campaigns, although various levels of resistance can be induced *in vitro*. The lack (of reports) of clinical resistance, in this case, can be traced to the fact that pentamidine is taken up by three different transport mechanisms, at least two of which, the aminopurine transporter AT1 and the High Affinity Pentamidine Transporter, HAPT1 (now identified as an aquaglyceroporin, TbAQP2), are highly efficient, allowing a fast accumulation of the drug; moreover, the drug action becomes irreversible after only a brief exposure, giving little scope for resistance to develop. Recently, AQP2-mutated *T. b. gambiense* strains have been isolated from sleeping sickness patients in the Democratic Republic of Congo and from South Sudan, who relapsed after melarsoprol treatment; these isolates were verified to be significantly resistant to both melarsoprol and pentamidine *in vitro*. However, it is of note that the relapses were after treatment with melarsoprol (which is also a substrate of both AT1 and AQP2), not pentamidine. As explained by Graf *et al*., the pharmacokinetics of pentamidine (but not of melarsoprol), especially the high peak plasma concentration and long clearance time, may prevent treatment failure even though the rate by which the drug enters the parasite is reduced dramatically, leading to a significant *in vitro* resistance phenotype. Similarly, there have been no reports of resistance to suramin, still in use for *T. b. rhodesiense* sleeping sickness after 100 years, and this is believed to be linked to the drug being internalised by endocytosis, which is uniquely fast in bloodstream trypanosomes, after binding to the invariant surface glycoprotein ISG75, of which there are multiple paralogues.

Interestingly, the known protozoan ‘drug transporters’ are mostly members of highly conserved gene families, and often involved in nutrient uptake or other household functions. For example, in *Leishmania* the miltefosine transporter is a

phospholipid translocase and methotrexate is taken up by a folate transporter, the *T. brucei* carrier for eflornithine is an amino acid transporter, while pentamidine and melarsoprol are taken up by an aminopurine transporter and an aquaglyceroporin. Thus, it is often hard to predict what might be a (potential) drug transporter in a protozoan: they are hidden in plain sight, masked by their innocuous roles. Conversely, creative use of the uniqueness of protozoan transporters from conserved families can be exploited for the selective targeting of therapeutic agents.

Thus, we see that polypharmacology, combined with an uptake mechanism that is not easily disabled by mutations in a single Open Reading Frame (ORF), can prolong a drug's lifespan to (most of) a century. This point can be illustrated with our work on curcumin and a series of analogues that display *in vitro* activity against *T. brucei* species, with an EC_{50} value of 53 nM for the most potent analogue, AS-HK014, compared with 2.7 µM for curcumin itself [38]. We were unable to induce any level of resistance against curcumin *in vitro*, although we easily induced a 50-fold level of resistance to the analogue, which brought it to the exact same level as curcumin, which we could not then surpass. Curcumin is believed to diffuse across the membrane and indeed act *on* the membrane [40], and we certainly were unable to measure any saturable uptake using ^{3}H-curcumin (unpublished data). The failure to induce curcumin resistance is consistent with the current model that attributes the many biological actions of curcumin to polypharmacology and a capacity to interact with membrane proteins. In contrast, the activity of analogue AS-HK014 was well defined (reaction with cellular thiols leading to depletion of trypanothione and glutathione), and resistance developed rapidly.

A more important example, of course, is the essential antimalarial drug artemisinin; it is estimated that widespread resistance to this drug, as is now spreading in South-East Asia, will result in well over 100 000 additional malaria deaths and US$385M/year in additional loss of economic activity. As artemisinin appears to act, when activated by haem, through highly promiscuous covalent binding to many cellular targets in the parasite, resistance through target mutations/gene deletion would appear impossible, and no transporter mutations have been found. Indeed, as noted by Wang *et al*., no *Plasmodium falciparum* strain has so far been found to be insensitive to artemisinin in a standard *in vitro* protocol. Yet treatment failure with artemisinin combination therapy (ACT) is undoubtedly real, and associated with mutations in a single gene, *kelch13* (K13), which is neither an artemisinin target nor a transporter.

However, drug resistance is not an all-or-nothing proposition, and the answer to the Sphinx's riddle is that the parasites remain sensitive but that the parasite clearance time is significantly increased in the adapted strains. The K13 mutations have the effect of shortening the duration of the trophozoite stage, during which the parasite is particularly susceptible to artemisinin, and lengthening the duration of the ring stage that is relatively insensitive due its low haem content. This adaptation of intra-erythrocyte development is yet another unimagined way by which parasites can develop clinical resistance, but the clinical resistance would probably not have taken hold if the ACT partner drugs had not suffered from resistance first. It is the function of these partner drugs to 'mop up' the parasites that survive the relatively short exposure to artemisinin, caused by the rapid clearance of the drug.

We thus see that combination chemotherapy as in ACT, or nifurtimox/eflornithine in late-stage sleeping sickness, can keep resistance effectively at bay, unless the parasite can either develop transmissible resistance to one of the component drugs separately (which is not true combination therapy) or if resistance to one of the drugs in the combination already exists in the pathogen population. In other words, it may be a really counterproductive idea to introduce a combination in order to try to 'salvage' a drug *after* resistance has been reported, because that may lead to the functional loss of both components in the combined formulation. This is not a trivial concern, as we truly cannot lose any of the main anti-protozoal drugs, and should feature in current discussions about the potential introduction of combinations against visceral leishmaniasis. Resistance against all the major anti-leishmanial drugs has been reported and, alarmingly, resistance against several combinations can be induced readily *in vitro*.

4.0 Conclusion

We see that it is extremely difficult to prevent the onset, or spread of drug resistance and that, therefore, the reality is that we are and will continue to lose life-saving treatments. While more can be done on the side of prevention, vector control, sanitation, etc., the only way to prevent a catastrophic inability to treat protozoan disease is by investing in new treatments. For that, genuine, long-term partners with very deep pockets are eagerly sought.

5.0 Summary

Infections with protozoan pathogens will be with us for the foreseeable future. There is still no effective malaria vaccine despite enormous investment in

money and effort over many years. *Leishmaniasis* is still spreading, including in Southern Europe. At least 100 million women worldwide suffer infection with the sexually transmitted *Trichomonas vaginalis*. Chagas Disease (American *trypanosomiasis*) affects communities from Texas to Argentina. Sleeping sickness (human African *trypanosomiasis*) remains a scourge in sub-Saharan Africa. Billions of people are infected with *Toxoplasma gondii*. Then, there are *Cryptosporidium* spp., *Entamoeba histolytica*, and *Giardia* spp. — but you get the idea.

6.0 Tutor-Marked Assignment

-Write on Steps that can be taken to minimise the impact of drug resistance in protozoan disease.

Solution

Actually, the best strategy is to avoid over-reliance on chemotherapy in the first place — anything that reduces transmission rates brings the disease burden down. Good sanitation reduces transmission of waterborne parasites such as *Giardia*, *Entamoeba*, and *Cryptosporidium*, just as condoms prevent trichomoniasis. Clearly, effective vaccines would be our best chance of actually eradicating specific diseases, but none are likely to be approved any time soon. While a commercial vaccine for canine leishmaniasis is now available, it appears to be more effective in preventing disease progression than infection rates, and one study concluded that a simple anti-flea collar on the dog prevented infection much more effectively.

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Unit 4: Prevention and control of parasite infections

1.0 Introduction

2.0 Objectives

3.0 Main content

3.1 What are the symptoms of parasitic infections?

3.2 What causes parasitic infections?
3.3 Who is at risk of parasitic infections?
3.4 How are parasitic infections diagnosed?
3.5 How are parasitic infections treated?
3.6 How can parasitic infections be prevented?

4.0 Conclusion

5.0 Summary

6.0 Tutor-Marked Assignment
7.0 Reference/Further Reading

Unit 4: Prevention and control of parasite infections

1.0 Introduction

What is a parasitic infection?

Parasites are organisms that live off other organisms, or hosts, to survive. Some parasites don't noticeably affect their hosts. Others grow, reproduce, or invade organ systems that make their hosts sick, resulting in a parasitic infection.

Parasitic infections are a big problem in tropical and subtropical regions of the world. Malaria is one of the deadliest parasitic diseases. Parasitic infections can also occur in the United States. Common parasitic infections found in the United States include:

- trichomoniasis
- giardiasis
- cryptosporidiosis
- toxoplasmosis

3.0 Main content

3.1 What are the symptoms of parasitic infections?

The symptoms of parasitic infections vary depending on the organism. For example:

- Trichomoniasis is a sexually transmitted infection caused by a parasite that often produces no symptoms. In some cases, it may cause itching, redness, irritation, and an unusual discharge in your genital area.
- Giardiasis may cause diarrhea, gas, upset stomach, greasy stools, and dehydration.

- Cryptosporidiosis may cause stomach cramps, stomach pain, nausea, vomiting, dehydration, weight loss, and fever.
- Toxoplasmosis may cause flu-like symptoms, including swollen lymph nodes and muscle aches or pains that can last for over a month.

3.2 What causes parasitic infections?

Parasitic infections can be caused by three types of organisms:

- protozoa
- helminths
- ectoparasites

Protozoa are single-celled organisms that can live and multiply inside your body. Some infections caused by protozoa include giardiasis. This is a serious infection that you can contract from drinking water infected with *Giardia* protozoa.

Helminths are multi-celled organisms that can live in or outside of your body. They're more commonly known as worms. They include flatworms, tapeworms, thorny-headed worms, and roundworms.

Ectoparasites are multicelled organisms that live on or feed off your skin. They include some insects and arachnids, such as mosquitos, fleas, ticks, and mites.

Parasitic infections can be spread in a number of ways. For example, protozoa and helminths can be spread through contaminated water, food, waste, soil, and blood. Some can be passed through sexual contact. Some parasites are spread by insects that act as a vector, or carrier, of the disease. For example, malaria is caused by parasitic protozoa that are transmitted by mosquitos when they feed on humans.

3.3 Who is at risk of parasitic infections?

Anyone can get a parasitic infection. But some people are at greater risk than others. You're more likely to contract a parasitic infection if you:

- have a compromised immune system or are already sick with another illness
- live or travel in tropical or subtropical regions of the world
- lack a clean supply of drinking water
- swim in lakes, rivers, or ponds where *Giardia* or other parasites are common
- work in childcare, work with soil regularly, or work in other contexts where you come into contact with feces on a consistent basis

Outdoor cats can come into contact with infected rodents and birds. This makes their owners more likely to contract toxoplasmosis, a type of protozoa. Toxoplasmosis can be very harmful for pregnant women and their developing babies. The infection is spread through cat feces. If you're pregnant, it's important to have someone else clean the litter box daily.

3.4 How are parasitic infections diagnosed?

Parasitic infections can be diagnosed in a number of ways. For example, your doctor might perform or order:

- A blood test
- A fecal exam: In such an exam, a sample of your stool will be collected and checked for parasites and their eggs.
- An endoscopy or colonoscopy: These tests may be ordered if the results of a stool exam are inconclusive. While you are sedated, your doctor will pass a thin flexible tube through your mouth or rectum and into your digestive system to examine your intestinal tract.

- X-rays, magnetic resonance imaging (MRI), or computerized axial tomography (CAT): These scans are used to check for signs of lesions or injury to your organs caused by parasites.

Your doctor may also order tests to check for bacteria or other things that can cause infections.

3.5 How are parasitic infections treated?

Your treatment plan will depend on your specific diagnosis. Typically, your doctor will prescribe medications. For example, they may prescribe medications to treat trichomoniasis, giardiasis, or cryptosporidiosis. They probably won't prescribe medications for toxoplasmosis if you're not pregnant and otherwise healthy, unless you have a severe and prolonged infection.

Your doctor may also recommend other treatments to relieve your symptoms. For example, many parasitic infections can cause diarrhea, which often leads to dehydration. Your doctor will likely encourage you to drink plenty of fluids to replenish those you lose.

3.6 How can parasitic infections be prevented?

There are several steps you can take to lower your risk of contracting a parasitic infection:

- Practice safe sex, using a condom.
- Wash your hands regularly, especially after handling uncooked food or feces.
- Cook food to its recommended internal temperature.
- Drink clean water, including bottled water when you're traveling.
- Avoid swallowing water from lakes, streams, or ponds.
- Avoid cat litter and feces when you're pregnant.

If you suspect you have a parasitic infection, make an appointment with your doctor. They can help diagnose the cause of your symptoms and recommend a treatment plan. By getting early treatment, you can help stop the spread of infection to other people.

4.0 Conclusion

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6.0 Tutor-Marked Assignment

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