# Post-Graduate Degree Programme
# (CBCS)
# in
# ZOOLOGY
## (M.Sc. Programme)

## SEMESTER-III

# Parasitology and Immunology

# ZDSE(MJ)T-301

**Self-Learning Material**

**DIRECTORATE OF OPEN AND DISTANCE LEARNING**
**UNIVERSITY OF KALYANI**
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# Director's Message

Satisfying the varied needs of distance learners, overcoming the obstacle of distance and reaching the unreached students are the threefold functions catered by Open and Distance Learning (ODL) systems. The onus lies on writers, editors, production professionals and other personnel involved in the process to overcome the challenges inherent to curriculum design and production of relevant Self Learning Materials (SLMs). At the University of Kalyani a dedicated team under the able guidance of the Hon'ble Vice-Chancellorhas invested its best efforts, professionally and in keeping with the demands of Post Graduate CBCS Programmes in Distance Mode to devise a self-sufficient curriculum for each course offered by the Directorate of Open and Distance Learning (DODL), University of Kalyani.

Development of printed SLMs for students admitted to the DODL within a limited time to cater to the academic requirements of the Course as per standards set by Distance Education Bureau of the University Grants Commission, New Delhi, India under Open and Distance Mode UGC Regulations, 2020 had been our endeavour. We are happy to have achieved our goal.

Utmost care and precision have been ensured in the development of the SLMs, making them useful to the learners, besides avoiding errors as far as practicable. Further suggestions from the stakeholders in this would be welcome.

During the production-process of the SLMs, the team continuously received positive stimulations and feedback from Professor (Dr.) Amalendu Bhunia, Hon'ble Vice- Chancellor, University of Kalyani, who kindly accorded directions, encouragements and suggestions, offered constructive criticism to develop it within proper requirements. We gracefully, acknowledge his inspiration and guidance.

Sincere gratitude is due to the respective chairpersons as well as each and every member of PGBOS (DODL), University of Kalyani. Heartfelt thanks are also due to the Course Writers-faculty members at the DODL, subject-experts serving at University Post Graduate departments and also to the authors and academicians whose academic contributions have enriched the SLMs. We humbly acknowledge their valuable academic contributions. I would especially like to convey gratitude to all other University dignitaries and personnel involved either at the conceptual or operational level of the DODL of University of Kalyani.

Their persistent and coordinated efforts have resulted in the compilation of comprehensive, learner-friendly, flexible texts that meet the curriculum requirements of the Post Graduate Programme through Distance Mode.



All the Self Learning Materials are self-writing and collected from e-book, journals and websites.

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# Theory (Discipline Specific Elective – Major I) - [ZDSE(MJ)T-301]

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| | **Total counselling session 12hrs.** | | | |

# UNIT I & UNIT II

# Classification of Apicomplexa with examples

**Objective:** In this unit we will discuss about Classification of Apicomplexa with examples

## Introduction

The apicomplexa are a monophyletic group composed almost entirely of parasitic (ie, no free-living) species. Formerly the apicomplexa were part of a group called sporozoa and this name is still sometimes used. Recently there have been some suggestions to revert back to the name sporozoa. The parasite belonging to this group of protozoa don't possess any type of locomotion organelles, they show slight amoeboid change of form (body flexion). Electron microscopy revealed unique ultrastructural features among the various sporozoa which were subsequently used to redefine the groups. A defining characteristic of the apicomplexa is a group of organelles found at one end--called the apical end--of the organism. This 'apical complex' includes secretory organelles known as micronemes and rhoptries, polar rings composed of microtubules, and in some species a conoid which lies within the polar rings.

Apicomplexans have complex life cycles, and there is much variation among different apicomplexan groups. Both asexual and sexual reproduction are involved, although some apicomplexans skip one or the other stage. The basic life cycle may be said to start when an infective stage, or **sporozoite**, enters a host cell, and then divides repeatedly to form numerous **merozoites**. Some of the merozoites transform into sexually reproductive cells, or **gamonts**. Gamonts join together in pairs and form a **gamontocyst** (pictured above). Within the gamontocyst, the gamonts divide to form numerous **gametes**. Pairs of gametes then fuse to form **zygotes**, which give rise by meiosis to new sporozoites, and the cycle begins again. Apicomplexans are transmitted to new hosts in various ways; some, like the malaria parasite, are transmitted by infected mosquitos, while others may be transmitted in the feces of an infected host, or when a predator eats infected prey.

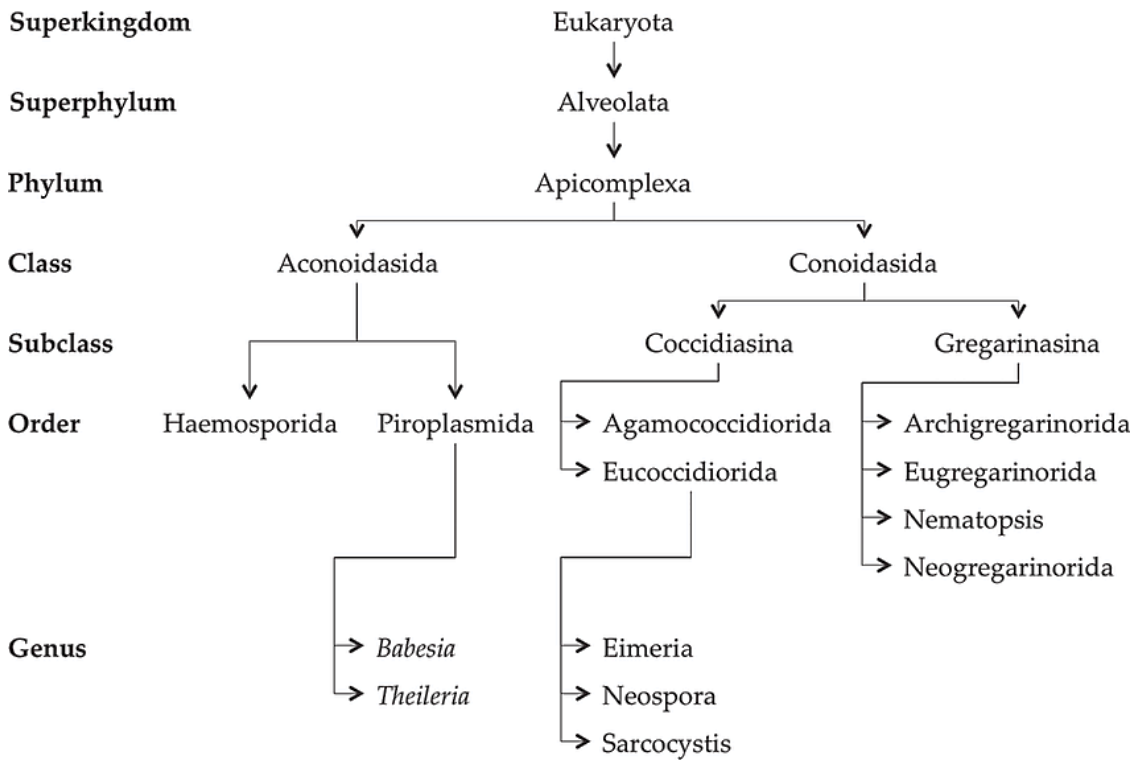
## What is Apicomplexa?

Previously called Protozoa, along with several other groups, the **Phylum Apicomplexa** is large and is further divided into 300 genera and over 60 families that consist of over 5000 species. The majority of species already identified are obligate intracellular parasites that infect a variety of animals (including human beings) causing a variety of diseases.
Because of the high diversity in this group, members have also been shown to exhibit a wide range of morphological shapes and characteristics depending on the genus and stage in their life cycle. This diversity also extends to the environment/habitat in which they are found in nature.

Common members of this group include:

- *Babesia*
- *Plasmodium*
- *Eimeria*
- *Isospora*
- *Haemoproteus*
- *Haemogregarina*

## Classification of Phylum Apicomplexa




**Superkingdom: Eukaryota** - Includes members with a membrane-bound nucleus

**Kingdom: Chromalveolata** (2005 classification) - A large group consisting of morphologically variable protists. The group is divided into four main groups that include Heterokontae, Alveolatae, Hacrobiae, and Rhizariae.

**Infrakingdom/superphylum: Alveolata** - Members of this group are single-celled eukaryotes that are widely distributed in different environments/habitats. They are characterized by a diverse means of locomotion as well as flattened vesicles known as alveoli. Apart from apicomplexans, other organisms classified in this group include ciliates and dinoflagellates.

The National Center for Biotechnology Information (NCBI; http://www.ncbi.nlm.nih.gov/) divides the phylum Apicomplexa into two classes: Aconoidasida and Conoidasida (Figure 1). The class Aconoidasida is divided into two orders: Haemosporida and Piroplasmida (containing the genera Babesia and Theileria), while the class Conoidasida is divided into two subclasses: Coccidiasina (containing the genera Eimeria, Neospora and Sarcocystis, that belong to order Eucoccidiorida) and Gregarinasina (Figure 1). It is estimated that subclass Coccidiasina separated from the class Aconoidasida ~705 million years ago.

## Phylum: Apicomplexa

### Characterictics

I. Obligate, intracellular protozoa
II. Distinct from other protozoa because they lack motor organelles (cilia and flagella), except for the male gametes during the sexual phase
III. Complicated life cycle, including sexual and asexual stages
IV. Sexual- Sporogony/ Sporogonic Cycle
V. Asexual- Schizogony/ Schizogonic Cycle
VI. They undergo schizogony/merogony (multiple fission/budding-production of merozoites), Gametogony (production of male and female gametes), and sporogony (sexual stage- production of sporozoites).
VII. The sporozoites have apical complex which is specific to sporozoans, this structure is used for attachment to the host.

The phylum is further divided into two classes that include:

- Aconoidasida
- Conoidasida

## Aconoidasida

Created in 1980, Aconoidasida is one of the two main classes of apicomplexan parasites.

It consists of members of the family Babesiidae and Theileriidae and has the following characteristics:

- Do not possess conoid in their apical complex
- Reproduce asexually through multiple fission
- Do not have specialized structures for locomotion
- Their oocysts do not have a cyst wall

## Conoidasida

The class Conoidasida was introduced by Levine in 1988. It's further divided into subclasses coccidia (mostly infect vertebrates) and gregarines (parasites of

invertebrates) which are characterized by a hollow conoid. They reproduce through sexual and asexual means and produce flagellated microgametes. Although some of the species have pseudopodia, they are mostly used for phagocytosis and not for movement.

## Distribution

As mentioned, the majority of already identified phylum Apicomplexa species are parasites of animals and human beings. For this reason, their distribution across the world is largely dependent on the species, environmental conditions (climate), and the host they infect.

Malaria parasites (*Plasmodium* species) are largely restricted to tropical and subtropical areas as well as in altitudes below 1,500m where they are responsible for high cases of malaria. On the other hand, *B. divergens*, a species of the Genus *Babesia*, is largely distributed in Europe with many cases (infections) being in human beings.

Depending on the type of host infected, Apicomplexa species are also found in different environments across the world. Moreover, the concentration of given species will vary from one environment to another.

In a study to investigate the distribution of coccidian parasites in aquatic and terrestrial environments in infected snakes, studies found a variation in infections between strictly terrestrial (52 percent) and semi-aquatic snakes (48 percent). This variation extends to different regions across the world.

## Characteristics

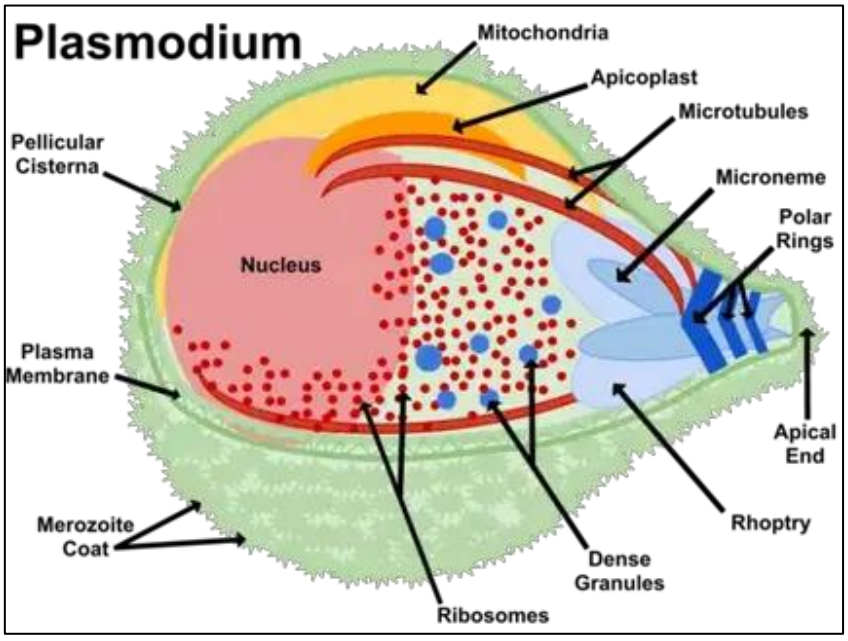



Fig: As a protist, the plasmodium is a eukaryote of the phylum Apicomplexa by Jfbranch14 / CC BY-SA (https://creativecommons.org/licenses/by-sa/3.0)

Apicomplexans are eukaryotic organisms and are therefore characterized by a complex structure as compared to the structure of prokaryotes. As eukaryotic organisms, they have membranous organelles (e.g. nucleus, endoplasmic reticulum, and mitochondria, etc).

They are also highly polarized cells that contain a given set of organelles that are only found in the phylum. They contain such secretory organelles as rhoptries and micronemes with given products that contribute to motility and cell invasion.

Flagella, used for motion, are commonly found in the gametes (male gametes). However, the number of flagella varies from one species to another. While male gametes in Plasmodium contain a single flagellum, Toxoplasma microgametes are bi-flagellated and thus contain two flagella. In both cases, the structure consists of 9 doublet microtubules and a central pair.

***** There are two types/categories of microtubules in apicomplexans. These include subpellicular microtubules (responsible for the shape and polarity of the apical complex) and spindle microtubules (involved in nuclear division during mitosis).

**Apical Complex**

The apical complex is the defining structure of apicomplexans. Located at the anterior end of adult obligate parasites, the apical complex consists of cytoskeleton structures and membrane-bound organelles.

The primary components of this structure include:

**The conoid** - The conoid is a cytoskeletal structure found in some of the apicomplexan parasites. It can be found in Toxoplasma and Eimeria species among others but is absent in such apicomplexans as Theileria and Plasmodium.

Essentially, the conoid is composed of counterclockwise spiraling filaments that form the cone-shaped structure at the apex of these apicomplexans. Based on molecular studies, filamentous subunits of the conoid have been shown to resemble microtubules. However, they are curled into a coil. They are about 250nm in diameter with two 400nm long microtubules located in the middle.

**Rhoptry** - Rhoptries are membrane-bound organelles (twin) with a pear-shaped appearance. They are part of the apical complex that secretes proteins at the apical tip.

Also described as secretory lysosomal granules, rhoptries (between 8 to12 per cell) also receive material from biosynthetic and endocytic cell pathways - Rhoptries are also associated with micronemes and dense granules in the parasite.

**Polar rings** - Found in all apicomplexans, polar rings serve as microtubule-organizing centers for the subpellicular microtubules.

## Probable questions:

1. Write down the characteristics of phylum Apicomplexa.
2. Write down the characteristics of class Aconoidasida with example.
3. Show a schematics diagram of Phylum apicomplexa.
4. What is conoid?
5. What is rhoptry?
6. What is polar ring?

# Suggested reading:


1. Cox, F. E. G. (1993). *Modern Parasitology*. 2nd ed. Blackwell Scientific Publications. Lea and Febiger, Philadelphia.
2. Martínez-Ocampo, F. (2018). Genomics of Apicomplexa. Farm Animals Diseases, Recent Omic Trends and New Strategies of Treatment. doi:10.5772/intechopen.72633

# UNIT III

# Origin of parasitic protozoa

**Objective:** In this unit we will discuss about origin of parasitic protozoa.

## Introduction

Protozoa are single celled organisms. They come in many different shapes and sizes ranging from an *Amoeba* which can change its shape to *Paramecium* with its fixed shape and complex structure. They live in a wide variety of moist habitats including fresh water, marine environments and the soil.

Some are parasitic, which means they live in other plants and animals including humans, where they cause disease. *Plasmodium,* for example, causes malaria. They are motile and can move by:

- Cilia - tiny hair like structures that cover the outside of the microbe. They beat in a regular continuous pattern like flexible oars.
- Flagella - long thread-like structures that extend from the cell surface. The flagella move in a whip-like motion that produces waves that propel the microbe around.
- Amoeboid movement - the organism moves by sending out pseudopodia, temporary protrusions that fill with cytoplasm that flows from the body of the cell.

## Origin of parasitic protozoa

Protists were a dominant form of life on Earth 1.5 billion years ago. While protozoans evolved early and have survived to the present day as unicellular organisms, they have undoubtedly undergone considerable evolutionary change. That many species must have become extinct as others appeared can be deduced from the limited fossil record of protozoans. Extinct fossil foraminiferan species, for example, number around 34,000, while there are only about 4,000 described living species.

Only a small number of protozoans, most of which are amoebae, have left fossil remains. The calcareous shells of the foraminiferans and calcium-secreting coccolithophores (a group of algae), for example, produced substantial geologic strata in the chalk formed during the Cretaceous Period (145.5 million to 65.5 million years ago) and the well-developed foram-limestones of the Paleozoic Era (542 million to 251 million years ago), Early Cretaceous Epoch (145.5 million to 99.6 million years ago), and Cenozoic Era (65.5 million years ago to the present). The fossil-forming radiolarians date to late Precambrian times, and the testate lobose amoeba *Melanocyrillium* dates to the late Precambrian geologic record of the Grand Canyon in northwestern Arizona, U.S. The testate amoeba *Nebela* is found in deposits from the Cretaceous Period.

- **Foraminifera**

The most abundant and important fossil protozoans are the foraminiferans. This entirely marine group is extremely important as stratigraphic markers in oil exploration. Because species have appeared and then become extinct frequently during geologic history and because they have fairly wide geographic distribution, particularly planktonic species, their value is in showing distinct phases in geologic history and, with specific species, in typifying particular beds of rock or strata. Foraminiferans are also important in the reconstruction of paleoceanographic circulation patterns.

The poor fossil record of protozoans has hampered attempts at unraveling the complexities of their evolution. Modern biochemical and electron microscopy techniques, however, are providing evidence for new affinities between groups and are elucidating possible evolutionary pathways. Comparisons of flagellar structures, mitochondria, and nuclear and plastid characteristics in conjunction with ribosomal RNA (ribonucleic acid) sequences are revealing the relationships of various taxa.

The ancestral eukaryote organism is thought to have been an amoeboid creature that relied on anaerobic or microaerophilic metabolism (microaerophilic organisms survive on only very small amounts of oxygen). The evolution of mitochondria (the centres of aerobic respiration in the cell) as organelles from endosymbiotic bacteria and the establishment of oxidative pathways allowed a more efficient cellular energy balance, which led the way to the evolution of an enormously diverse array of eukaryotic organisms. Some of the early amoeboid eukaryotes developed flagella to enhance their food-gathering abilities and to provide a more efficient mode of propulsion. The flagellates gradually evolved different ways of life, and their structures became modified accordingly. As phagotrophs that ingested bacteria for food, they in some cases came to establish symbiotic associations with photosynthetic species, and ultimately the endosymbionts became plastids within the cell. Some of the flagellates came to depend entirely on photosynthesis and to abandon heterotrophy completely, though many still retain both heterotrophic and autotrophic nutrition as mixotrophs. (Some present-day mixotrophs, however, may be only secondarily mixotrophic, having reestablished heterotrophy in conjunction with photosynthesis.)

- **Parasitic protozoa**

A considerable number of protozoans became parasitic, a mode of life that evolved independently among the protozoans many times. Ciliates and amoebae became symbionts in the intestinal tracts of both vertebrates and invertebrates as a result of surviving the digestive enzymes of the predator. (Most present-day parasites among these protists are intestinal parasites.) Once inside the intestine of the host, they multiplied and gradually, through mutation and selection, came to rely on the resistant

cyst as a means of survival and dispersal, losing the ability to survive in a free-living feeding form.

The process of parasitism probably arose in several independent cases. The trypanosomes, for example, evolved from free-living forms, adapting to life in the alimentary canal of primitive invertebrates during late Precambrian times (570 million years ago). They evolved with their hosts, becoming symbionts in a wide variety of invertebrates, including annelids, nematodes, and mollusks. It was in the insects, however, that they underwent their most extensive evolutionary explosion into two groups. At this stage they were transmitted from insect to insect by resistant cysts passed in the feces and ingested by subsequent hosts. When insects developed the habit of sucking vertebrate blood, which is believed to have occurred about 40 million years ago, the protozoan symbionts that lived in the gut entered the blood of vertebrates, probably as feces left by the insect were rubbed into the wound. The blood provided a rich environment for the flagellates and thus evolved the two-host life cycles seen today in the *Leishmania* and *Trypanosoma* groups.

The apicomplexans, which also inhabit the blood of vertebrates at some stage in their life cycle, probably evolved from a basal primitive stock seen today as the gregarines, which are parasites of invertebrates. They gave rise to a group of parasitic organisms of which the coccidia, with a one-host life cycle, are primitive survivors. At first these protozoans lived in the gut of their vertebrate host, but they gradually began invading host tissues and eventually became adapted to spending part of their life cycle in the bloodstream. There they were taken up by blood-feeding insects, and an insect vector host became incorporated into the life cycle. Associated modifications in the reproductive pattern, as seen in *Plasmodium*, which belongs to the Haemosporina, also occurred. This series of events appears to have happened at least twice in the evolution of apicomplexan life cycles.

## Probable questions:

1. Write down the characteristics of parasitic protozoa.
2. Describe the origin of parasitic protozoa.
3. Describe the origin of foraminiferans.
4. Write down the locomotary structure of protozoa?

## Suggested reading:


1. Protozoa: Written by Johanna E.M. Laybourn-Parry, Julia M. Diaz: https://www.britannica.com/science/Paramecium
2. Cox, F. E. G. (1993). *Modern Parasitology*. 2nd ed. Blackwell Scientific Publications. Lea and Febiger, Philadelphia.
3. Martínez-Ocampo, F. (2018). Genomics of Apicomplexa. Farm Animals Diseases, Recent Omic Trends and New Strategies of Treatment. doi:10.5772/intechopen.72633

# UNIT IV

# Some general consideration of protozoan parasites: Population & Communities

**Objective:** In this unit we will discuss about general consideration of Population & Communities of protozoan parasites.

## Introduction

## General Concepts

## Protozoa

Protozoa are one-celled animals found worldwide in most habitats. Most species are free living, but all higher animals are infected with one or more species of protozoa. Infections range from asymptomatic to life threatening, depending on the species and strain of the parasite and the resistance of the host.

### Structure

Protozoa are microscopic unicellular eukaryotes that have a relatively complex internal structure and carry out complex metabolic activities. Some protozoa have structures for propulsion or other types of movement.

### Classification

On the basis of light and electron microscopic morphology, the protozoa are currently classified into six phyla. Most species causing human disease are members of the phyla Sacromastigophora and Apicomplexa.

### Life Cycle Stages

The stages of parasitic protozoa that actively feed and multiply are frequently called trophozoites; in some protozoa, other terms are used for these stages. Cysts are stages with a protective membrane or thickened wall. Protozoan cysts that must survive outside the host usually have more resistant walls than cysts that form in tissues.

### Reproduction

Binary fission, the most common form of reproduction, is asexual; multiple asexual division occurs in some forms. Both sexual and asexual reproduction occur in the Apicomplexa.

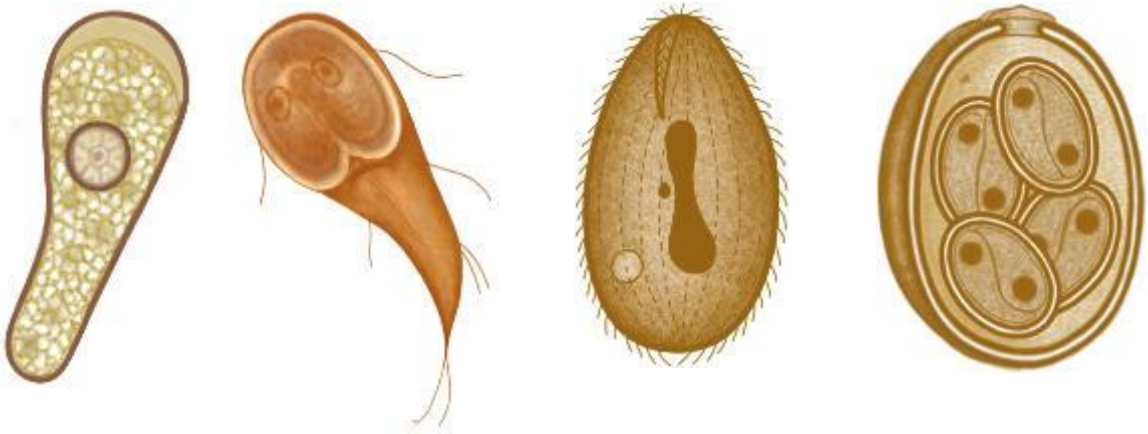
### Nutrition

All parasitic protozoa require preformed organic substances—that is, nutrition is holozoic as in higher animals.

## Biodiversity

Four main groups of protozoa are recognized on the basis of their locomotion using specialized subcellular and cytoskeletal features:

amoebae flagellates ciliates sporozoa



> **Amoebae** use pseudopodia (singular: pseudopodium) to creep or crawl over solid substrates. Pseudopodia (or 'false feet') are temporary thread-like or balloon-like extensions of the cell membrane into which the protoplasm streams. Similar amoeboid motion has been observed in cells of many life-forms, especially phagocytic cells (e.g. human macrophages).

> **Flagellates** use elongate flagella (singular: flagellum) which undulate to propel the cell through liquid environments. Flagella are 'whip-like' extensions of the cell membrane with an inner core of microtubules arranged in a specific 2+9 configuration (2 single central microtubules surrounded by 9 peripheral doublets). This configuration is conserved throughout eukaryotic biology, many organisms produce flagellated cells (e.g. human spermatozoa).

> **Ciliates** use numerous small cilia (singular: cilium) which undulate in waves allowing cells to swim in fluids. Cilia are 'hair-like' extensions of the cell membrane similar in construction to flagella but with interconnecting basal elements facilitating synchronous movement. Ciliated cells are found in specialized tissues and organs in many other higher life-forms (e.g. human bronchial epithelial cells).

> **Sporozoa** ('spore-formers') were originally recognized not on the basis of their locomotion, but because they all formed non-motile spores as transmission

stages. Recent studies, however, have shown that many pre-spore stages move using tiny undulating ridges or waves in the cell membrane imparting a forward gliding motion, but the actual mechanisms involved are not yet known.

- **Colonies and aggregates**

Protozoans are motile; nearly all possess flagella, cilia, or pseudopodia that allow them to navigate their aqueous habitats. However, this commonality does not represent a unique trait among protozoans; for example, organisms that are clearly not protozoans also produce flagella at various stages in their life cycles (e.g., most brown algae). Protozoans are also strictly non-multicellular and exist as either solitary cells or cell colonies. Nevertheless, some colonial organisms (e.g., *Dictyostelium discoideum*, supergroup Amoebozoa) exhibit high levels of cell specialization that border on multicellularity.

An important feature of colonies is the material that holds the component zooids together. In spheroid colonies, a matrix is secreted during development of the colony. Spheroid colonies of a few radiolarians (for example, *Collozoum*), unusually large examples, may measure 4–6 cm (1.6–2.4 in.) in width. In arboroid colonies, a branching pattern is produced by a branching stalk or by attachment of loricae to one another. Dispersal of such colonial species sometimes involves migratory stages. In certain spheroid colonies, reproductive and somatic zooids are differentiated. More often, members of a colony are similar. In addition to colonial types, certain protozoa may form aggregates by not separating promptly after fission. Palmella stages, analogous to spheroid colonies, are aggregates of nonflagellated organisms.

## Intestinal microbiota

The intestinal bacterial microbiota is a complex community of bacteria which is comprised of at least several hundred species. These organisms form a symbiotic relationship that influences human physiology and disease progression. Epidemiological studies have shown that the composition of the intestinal bacterial microbiota can correlate with the development of, or resistance to, obesity, malnutrition, and allergic disease and may also influence cognitive function and development. The intestinal microbiota is not limited to prokaryotes, with archaea and eukaryotes potentially contributing to clinical variation.

Microbiota compositions can vary significantly from one person to the next, even within healthy individuals or twins in the same household. Several studies have noted that the bacterial microbiota may influence the virulence of individual pathogens and potentially add variability to the outcomes of parasitic protozoan infections. For example, coculture with *Escherichia coli* strains can augment or attenuate the virulence of *Entamoeba histolytica* (35, 36). Recently reported studies highlight the impact of the microbiota on infections with enteric protozoa and on infection with extraintestinal *Plasmodium* parasites.

# Mucosal parasites and microbiota interactions in human populations

Mucosal infection with the enteric protozoa *Entamoeba*, *Giardia*, *Cryptosporidium*, and *Blastocystis* can be asymptomatic or cause diarrhea, abdominal pain, and/or weight loss. The infecting parasites reside in the intestinal mucosa and therefore are surrounded by the mucosa-associated microbiota. It has been proposed that the dynamic interplay that occurs between the protozoan parasite, host microbiota, and host immune system shapes the clinical outcome of enteric infections.

Infection with the gut parasite *Entamoeba* was significantly correlated with fecal microbiome composition and diversity. *Entamoeba* species infection was predicted by the composition of an individual's gut microbiota with 79% accuracy in a study of the farming and fishing populations in southwest Cameroon. One of the most important taxa in predicting an infection with *Entamoeba* was *Prevotellaceae*. In a separate independent study focused on the *E. histolytica*-associated diarrhea that is common in Bangladeshi infants, levels of *Prevotella copri*, a member of the *Prevotellaceae*, were found to be elevated in patients with diarrheagenic *E. histolytica* infections. The Cameroonian study was focused on infected adults who were not experiencing symptomatic amebiasis; therefore, it is interesting that both *P. copri* and *Prevotella stercorea* were significantly downregulated in infected individuals. Both studies suggest that microbiota composition may play a significant role during an *E. histolytica* infection. These studies also highlight the potential influence of inflammation driven by the gut microbiome in altering parasite infection outcomes. Elevated levels of *P. copri* have been associated with severe inflammation and an increased risk of autoimmune disease and colitis, suggesting that the organism is proinflammatory

*Cryptosporidium*, *Giardia*, *Blastocystis,* and *Trichomonas* infections may also be influenced by the gut microbiota. A retrospective study of volunteers who were originally enrolled in *Cryptosporidium* infectivity studiesexamined the relationship between the relative abundances of several bacterial taxa commonly found in adults prior to or within 48 h of infection and infection outcomes. The patients that were protected from infection had a greater abundance of *Proteobacteria* and lower *Bacteriodetes* and *Verrucomicrobia* levels than infected subjects. There was a higher ratio of *Firmicutes* to *Bacteriodetes* in uninfected subjects than in infected subjects. Seven specific taxa had differences of at least 2.5-fold between the two groups. Specifically, uninfected subjects had increased relative abundances of the indole-producing bacteria *Escherichia coli* CFT073 and *Bacillus* spp., as well as *Clostridium* spp. In contrast, infected subjects had increased relative abundances of *Bacteroides fragilis*, *Bacteroides pyogenes*, and *Prevotella bryantii*, as well as *Akkermansia muciniphila*. Presently, the mechanism by which increased indole production may protect from *Cryptosporidium* is unknown. Indole may directly adversely affect the parasite or perhaps alter host tissues to enhance the innate response by increasing epithelial integrity and/or stimulating anti-inflammatory pathways.

A study of intestinal parasite infection in individuals in southern Côte d'Ivoire utilizing PCR-temporal temperature gel electrophoresis (TTGE) and quantitative PCR demonstrated that TTGE profiles clustered into four significantly different groups, i.e., groups that are positive for *Giardia duodenalis*, positive for *Entamoeba* spp. and *Blastocystis hominis*, negative for protozoa, and positive for all three parasites. Quantitative PCR of selected bacterial species in these four groups showed that there was a significant increase in the relative abundance of *Bifidobacterium* in *G. duodenalis*-positive patients. This study suggested that the tested intestinal protozoans can induce significant changes in the microbiome which result in substantially different bacterial communities.

The relative abundances of *Faecalibacterium prausnitzii* and *E. coli* have been used as a marker of the inflammatory bowel disease (IBD)-induced dysbiosis associated with increased *E. coli* levels. Application of this tool to samples from a patient cohort in Côte d'Ivoire suggested that the Côte d'Ivoire and Cameroonian study results were in agreement and that an increase in microbiome diversity occurs in asymptomatic *Entamoeba* species infections. The Côte d'Ivoire results also suggest that this observation may be extended and that an increase in microbiome diversity also occurred during *Blastocystis hominis* infections. It is controversial, however, whether *Blastocystis* can cause diarrhea. Part of the reason for this controversy might be due to the tremendous genetic diversity within *Blastocystis* spp. *Blastocystis hominis* consists of at least seven morphologically identical but genetically distinct organisms. The gut microbiome which *Blastocystis* encounters upon infecting a human host may also influence clinical outcomes. Audebert et al. compared the microbiomes of *Blastocystis*-colonized and *Blastocystis*-free patients in a case-control study design that controlled for environmental and clinical risk factors, such as seasonal variation. The authors also reported a higher bacterial diversity in the fecal microbiota of *Blastocystis*-colonized patients, with a higher abundance of *Clostridia* as well as a lower abundance of *Enterobacteriaceae*. These results suggested that *Blastocystis* colonization may be associated with expansion of members of the intestinal microbiota generally associated with a healthy gut microbiota, rather than with expansion of bacteria associated with gut dysbiosis.

## Plasmodium and gut microbiota

Approximately 60% of the world's population is at risk of infection with *Plasmodium*. However, the distribution of clinical malaria is highly heterogeneous. In studies in Kenya and Senegal, the number of clinical episodes of disease ranged from 0 to 40 per child over a 5-year period in the same community. Clinical variation has been attributed to genetic differences. For example, heterozygous carriers of the hemoglobin variant HbS, associated with sickle cell disease, are healthy and are protected from severe forms of malaria, including cerebral malaria. Variation in exposure and variance in immune response are also implicated. However, these factors may not completely explain such a

large clinical variation. The intestinal bacterial microbiota might represent an environmental factor that may contribute to this variability.

In a recent study, stool samples were collected from a cohort of Malian children and adults just before the *P. falciparum* transmission season. The compositions of gut bacterial communities in these individuals were determined and compared to the risks of acquiring *P. falciparum* infection and febrile malaria. A significant association was found between microbiota composition and the prospective risk of *P. falciparum* infection. The intestinal microbiota of subjects who did not become infected had a significantly higher proportion of *Bifidobacterium* and *Streptococcus* species than subjects who became infected with *P. falciparum*. However, no relationship was observed between microbiota composition and the risk of developing febrile malaria once *P. falciparum* infection was established. The authors note that this is possibly due to a lack of statistical power. The preliminary finding of an association between gut microbiota composition and *P. falciparum* infection risk suggests that alteration of the composition of the intestinal microbiota may decrease the risk of *P. falciparum* infection in areas where malaria is endemic and may potentially augment partially effective malaria vaccines.

Gut bacteria might influence extraintestinal disease via many pathways, such as by alteration of adaptive immunity and augmentation of the magnitudes of T cells and B cell-mediated responses and perhaps by enhancement of innate immune pathways via trained immunity. Mechanisms underlying these extraintestinal effects are poorly understood. Metabolic products, such as short-chain fatty acids, or host-derived factors, such as damage-associated molecular pattern molecules induced by the microbiota, might be partially responsible for these effects. The metabolite pools present in animal models with differential, microbiota-dependent susceptibility to *Plasmodium* infection varied significantly in one study, with decreases in nucleotides, amino acids, and the substrates involved in the biosynthesis of these compounds in resistant mice, along with more-robust T and B cell responses. The gut microbiota has also been shown to have a systemic influence on serum metabolites in both animal models and humans. Blood-stage parasites have been shown to be highly susceptible to metabolic dysregulation induced by antimalarials and might also be influenced by changes induced by the microbiota. Therefore, the intestinal microbiota may influence the clinical outcome of a *Plasmodium* infection via alteration of the metabolome and modulation of innate or adaptive immunity.

## Probable questions:

1. Describe the colonization pattern of Plasmodium.
2. Describe the colonization pattern of gut bacteria.
3. Describe the colonization pattern of *E. coli*.
4. Describe the colonization pattern of mucosal parasite.
5. State important characteristics of bacterial colonization.

## Suggested reading:


1. Noble, E. R. and Noble G. A. (1989). Parasitology. The biology of animal Parasites. 6th ed. Lea and Febiger, Philadelphia.
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4. Smyth, J. D. (1994). Animal Parasitology. 3rd ed. Cambridge University Press.
5. Burgess, S.L., Gilchrist, C.A., Lynn, T.C., Petri, W.A., Jr. (2017). Parasitic protozoa and interactions with the host intestinal microbiota. Infect Immun 85:e00101-17. 10.1128/IAI.00101-17.
6. ASM Journals:Infection and Immunity, Vol. 85, No. 8; Parasitic Protozoa and Interactions with the Host Intestinal Microbiota; Stacey L. Burgess, Carol,A. Gilchrist, Tucker C. Lynn, William A. Petri Jr.
7. https://www.ncbi.nlm.nih.gov/books/NBK8325/

# UNIT V
# Some general consideration of protozoan parasites: Ecological niche

**Objective:** In this unit we will discuss about general consideration of ecological niche of protozoan parasites.

## INTRODUCTION

An **ecological niche** refers to the interrelationship of a species with all the biotic and abiotic factors affecting it.

A niche may be influenced by biotic and abiotic factors of an ecosystem. However, the niche of a species in a particular ecosystem will help set the features of its environment as these features will be crucial to its survival.

**Fig-The flightless dung beetle occupies an ecological niche: exploiting animal droppings as a food source.**

## DEFINITION

In ecology, a niche is the match of a species to a specific environmental condition.

It describes how an organism or population responds to the distribution of resources and competitors and how it in turn alters those same factors (for example, limiting access to resources by other organisms, acting as a food source for predators and a consumer of prey).

The type and number of variables comprising the dimensions of an environmental niche vary from one species to another and the relative importance of particular environmental variables for a species may vary according to the geographic and biotic contexts.

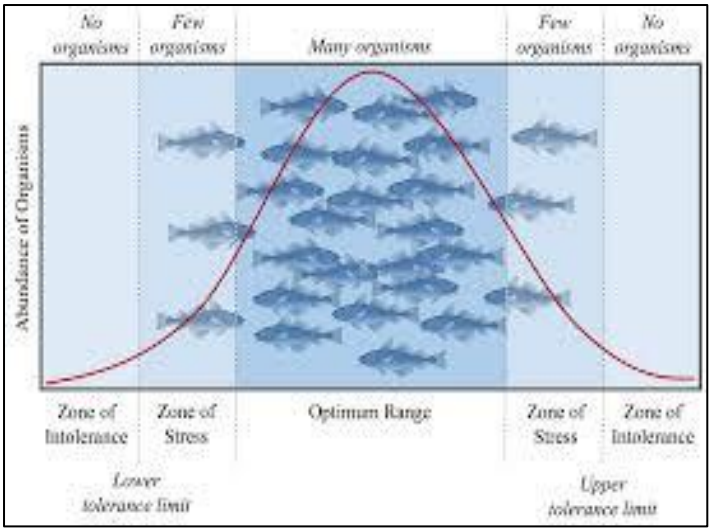



**Fig-Ecological Niche**

## TYPES OF ECOLOGICAL NICHE ACCORDING TO ODUM

## Habitat or Spatial Niche

- This pertains to the physical space inhabited by an organism. For example, consider an invertebrate community (millipedes) underneath a fallen log in a forest.
- In the dynamic view of the entire forest, some logs can be very old while some are new; some are untouched while some are recently disturbed. This variability also determines the distribution of the millipede species in that particular habitat.
- Here, the habitat under consideration is the forest floor of maple-oak vegetation. One species of millipede is predominant at the centre of logs, while another is at the superficial wood of logs.
- Likewise, different species of millipedes are found under the log, beneath the bark, beneath the litter, etc.

## Trophic Niche

- In the case of trophic niche, two species occupy the same habitat but have different functional roles or trophic positions.
- This is because of their variations in food habits. Example – Aquatic birds *Corixa* and *Notonecta* live in the same pond but inhabit different trophic

niches. *Notonecta* is an active predator that swims its way through to feed on other animals. Whereas *Corixa* feeds mostly on decaying vegetation.

- Another classic example would be Darwin's finches or Galapagos finches that showed adaptive radiation. The beak shape and size of these passerine birds are adapted as per their food sources.

**Fig-Darwin's finches**

## Multifactor or Hypervolume Niche

- This concept is based on the various environmental factors to which organisms of a population are uniquely adapted.
- Hutchinson (1957) emphasised his view of the n-dimensional hypervolume as a metaphor for the niche. Here, n is the number of environmental factors (temperature, humidity, etc.) for a given organism in a particular space or hypervolume.
- Since there are a large number of environmental factors, it is represented as n-dimensional hypervolume.

## TYPES OF ECOLOGICAL NICHE ACCORDING TO HUTCHINSON

## Fundamental Niche

- A particular organism living in their niche without disturbing or competing with other species.
- There is no interference of other species.
- It is larger than realised niche but it is a theoretical niche.

## Realised Niche

- When two organisms live in a same niche in competition.
- There is an interference of other species.
- It is smaller than fundamental niche but it is an actual or exact niche from where an organism belongs to.

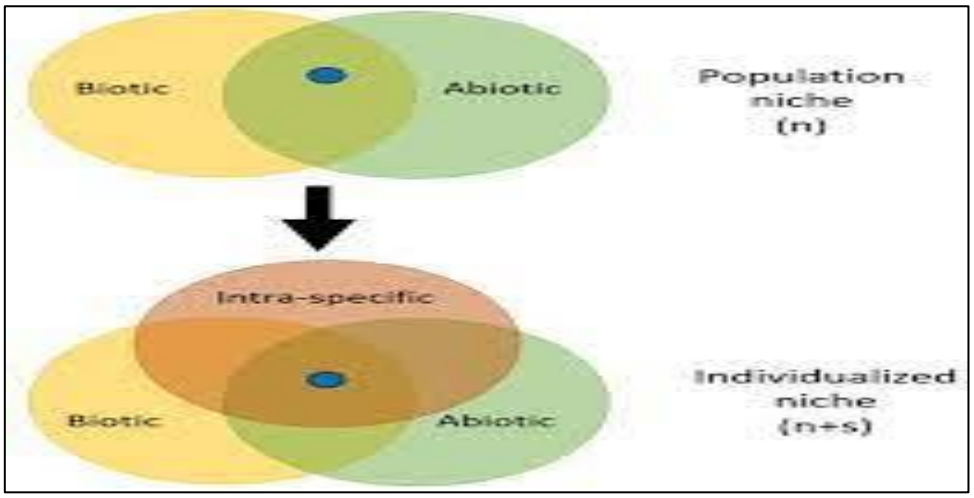



**Fig- Ecological niche according to Hutchinson**

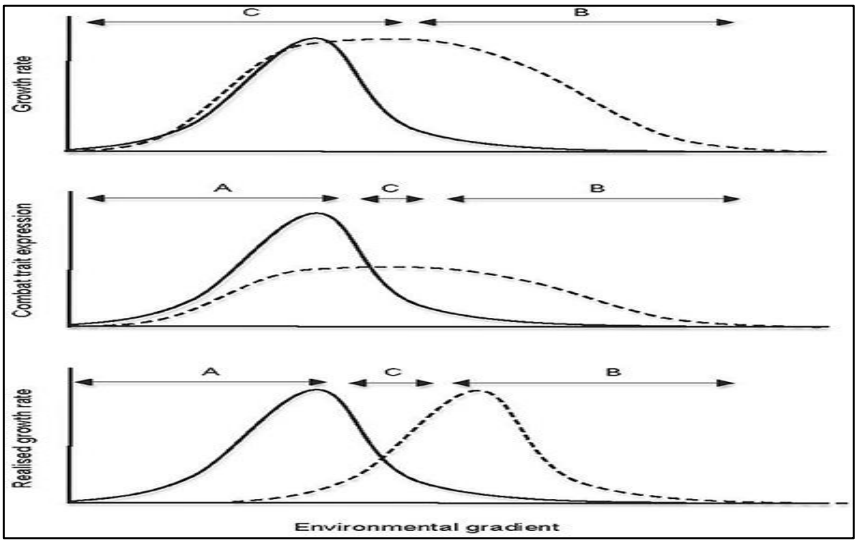



**Fig-Plotting the realised niche by estimating variation**

## Contemporary niche theory

- Contemporary niche theory (also called "classic niche theory" in some contexts) is a framework that was originally designed to reconcile different definitions of niches and to help explain the underlying processes that affect Lotka-Volterra relationships within an ecosystem. The framework centres around "consumer-resource models" which largely split a given ecosystem into resources (e.g. sunlight or available water in soil) and consumers (e.g. any living thing, including plants and animals), and attempts to define the scope of possible relationships that could exist between the two groups.

- In contemporary niche theory, the "impact niche" is defined as the combination of effects that a given consumer has on both a). the resources that it uses, and b). the other consumers in the ecosystem. Therefore, the impact niche is equivalent to the Eltonian niche since both concepts are defined by the impact of a given species on its environment.

- The range of environmental conditions where a species can successfully survive and reproduce (i.e. the Hutchinsonian definition of a realized niche) is also encompassed under contemporary niche theory, termed the "requirement niche". The requirement niche is bounded by both the availability of resources as well as the effects of coexisting consumers (e.g. competitors and predators).

# Coexistence under contemporary niche theory

Contemporary niche theory provides three requirements that must be met in order for two species (consumers) to coexist:

1. The requirement niches of both consumers must overlap.
2. Each consumer must outcompete the other for the resource that it needs most. For example, if two plants (P1 and P2) are competing for nitrogen and phosphorus in a given ecosystem, they will only coexist if they are limited by different resources (P1 is limited by nitrogen and P2 is limited by phosphorus, perhaps) and each species must outcompete the other species to get that resource (P1 needs to be better at obtaining nitrogen and P2 needs to be better at obtaining phosphorus). Intuitively, this makes sense from an inverse perspective: If both consumers are limited by the same resource, one of the species will ultimately be the better competitor, and only that species will survive. Furthermore, if P1 was outcompeted for the nitrogen (the resource it needed most) it would not survive. Likewise, if P2 was outcompeted for phosphorus, it would not survive.
3. The availability of the limiting resources (nitrogen and phosphorus in the above example) in the environment are equivalent.

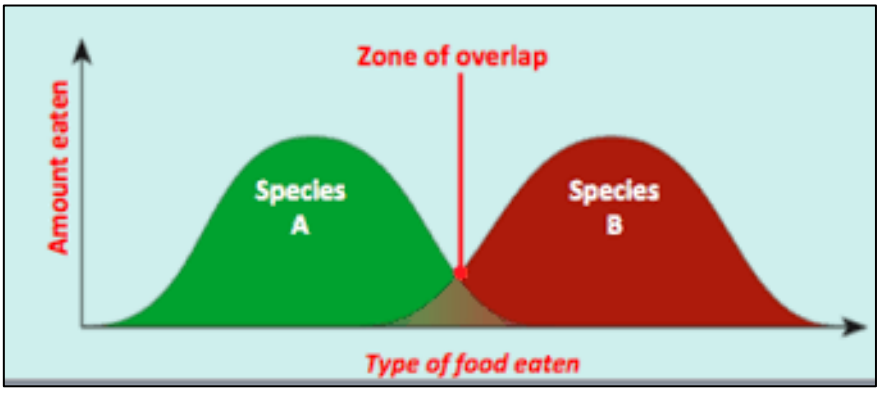
## Niche differentiation

- In ecology, niche differentiation (also known as niche segregation, niche separation and niche partitioning) refers to the process by which competing species use the environment differently in a way that helps them to coexist. The competitive exclusion principle states that if two species with identical niches (ecological roles) compete, then one will inevitably drive the other to extinction. This rule also states that two species cannot occupy the same exact niche in a habitat and coexist together, at least in a stable manner. When two species differentiate their niches, they tend to compete less strongly, and are thus more likely to coexist. Species can differentiate their niches in many ways, such as by consuming different foods, or using different areas of the environment.

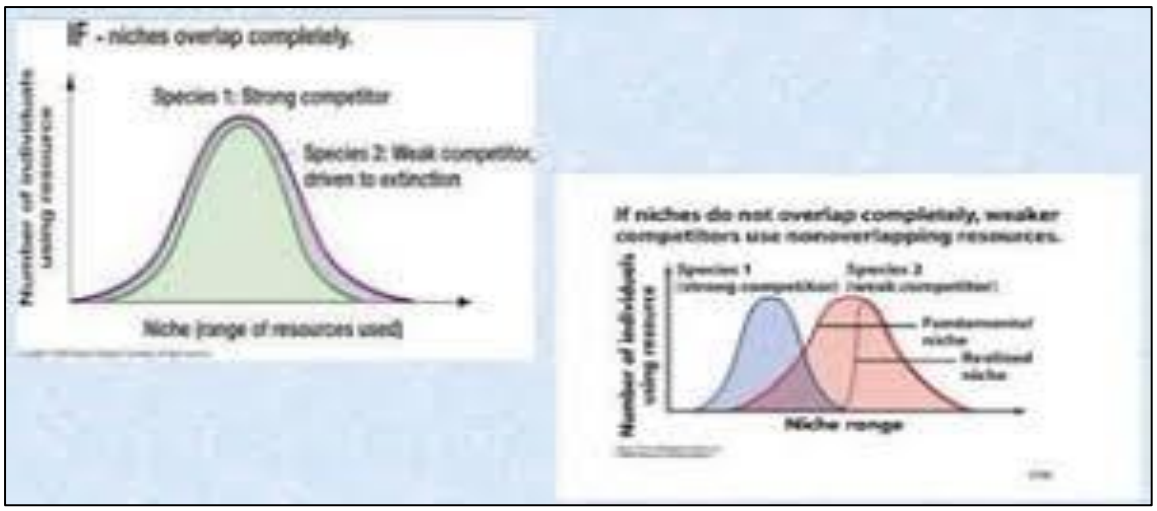
As an example of niche partitioning, several anole lizards in the Caribbean islands share common diets—mainly insects. They avoid competition by occupying different physical locations. Although these lizards might occupy different locations, some species can be found inhabiting the same range, with up to 15 in certain areas. For example, some live on the ground while others are arboreal. Species who live in different areas compete less for food and other resources, which minimizes competition between species. However, species who live in similar areas typically compete with each other.

## Niche Overlap

- Niche overlap described the sharing of niche space by two or more species.
- It is the index of similarity between the resource utilization of two species.




**Figure(A)**




**Figure(B)**

**Fig(A) and (B)-Niche Overlap**

## Niche Width

Niche width describes the range of niche dimension occupied by a species as well as other factors such as temperature and air on water pressure level as well as interspecific and intraspecific interaction**.**

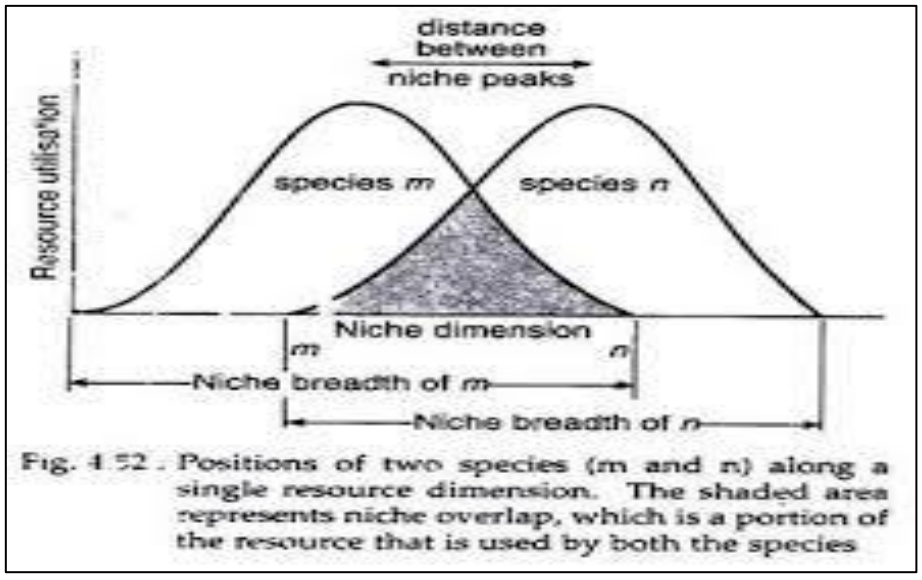



Fig. 4.52. Positions of two species (m and n) along a single resource dimension. The shaded area represents niche overlap, which is a portion of the resource that is used by both the species

**Fig-Position of species along a single resource dimension.**

## Resource Partitioning

- Resource partitioning is the phenomenon where two or more species divides out resources like food, space, resting sites etc. to coexist. For example, some lizard species appear to coexist because they consume insects of differing sizes.
- Alternatively, species can coexist on the same resources if each species is limited by different resources, or differently able to capture resources.
- Different types of phytoplankton can coexist when different species are differently limited by nitrogen, phosphorus, silicon, and light. In the Galapagos Islands, finches with small beaks are more able to consume small seeds, and finches with large beaks are more able to consume large seeds.
- If a species' density declines, then the food it most depends on will become more abundant (since there are so few individuals to consume it). As a result, the remaining individuals will experience less competition for food.
- Although "resource" generally refers to food, species can partition other non-consumable objects, such as parts of the habitat. For example, warblers are thought to coexist because they nest in different parts of trees. Species can also partition habitat in a way that gives them access to different types of resources. As stated in the introduction, anole lizards appear to coexist because each uses different parts of the forests as perch locations. This likely gives them access to different species of insects.

## The Ecological niche in Aquatic ecosystem

- Ecological niches are found in all types of ecosystems. At the level of concept or definition, there is no distinction between aquatic niches and terrestrial niches. Even so, aquatic environments are distinctive in that some (but not all) of the niche axes most likely to be important differ from those of terrestrial environments. Important dimensions of the ecological niche for aquatic organisms include temperature, dissolved oxygen, habitat structure, predation, and plant nutrients.
- The range of temperature for aquatic ecosystems is much narrower than the range for terrestrial ecosystems because liquid water has a minimum temperature of 0 °C. Thermal thresholds are weak for phytoplankton and zooplankton, as shown by the distribution of species across wide ranges of latitude and elevation. Thermal thresholds are more important for large invertebrates and especially for fishes. For example, the family Salmonidae (salmon and trout) contains many species that are intolerant of waters exceeding 15–20 °C. Similarly, perennially cool waters, such as montane or subarctic lakes, cannot sustain populations of many kinds of warmwater fishes, such as most species of the sunfish family (Centrarchidae).

- Water holds only approximately 10 mg/L of oxygen at low temperatures and 6–7 mg/L at high temperatures. Thus, respiration can make water anoxic if it is not offset by photosynthetically produced oxygen or by contact of water with the atmosphere. High rates of respiration in water that is in contact with sediment, for example, can remove all oxygen from water in a matter of a few days.
- Some aquatic environments are much more subject to oxygen depletion than others. Mountain streams of high gradient with low amounts of respiration are on one end of the spectrum, in that they have very little potential for oxygen depletion, whereas fertile lakes or wetlands, where there is much respiration and less efficient gas exchange with the atmosphere, show a much higher probability of oxygen depletion.
- Aquatic environments show an exceptional range of potential for producing autotroph biomass. Waters that have very low concentrations of nitrogen and phosphorus have production potential that falls well below that of any soil-based environment where moisture is present. In contrast, aquatic ecosystems with high concentrations of phosphorus and nitrogen have production potential for autotrophs that may be 1000 times higher than the least productive waters. Competition for nutrients is paramount in unproductive waters, which contain species having very high affinity for phosphorus and nitrogen. The most productive aquatic environments also support specialized taxa, but with different kinds of adaptations. Balance of nutrients may also be an important determinant of niche. For example, the nitrogen-fixing blue-green algae (heterocystous cyanobacteria) are, unlike other algae, capable of converting gaseous N2 to ammonia that can be used in making amino acids. When inorganic nitrogen is in short supply, these organisms are capable of continuing growth when other autotrophs cannot.

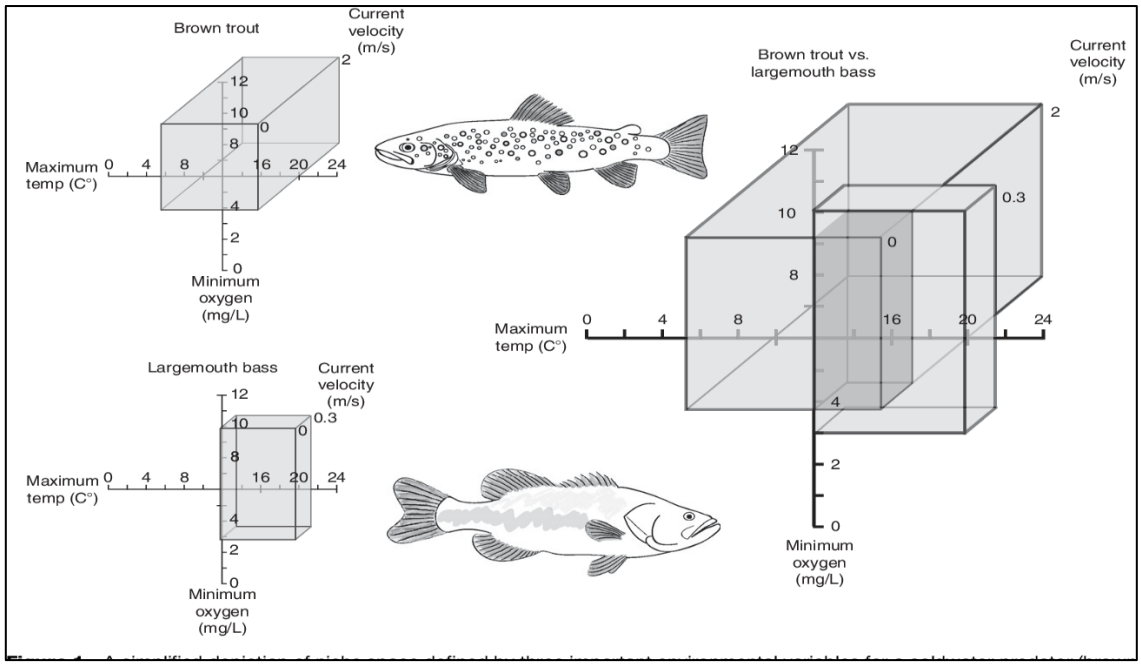



**Fig-Ecological niche in aquatic ecosystem**

# GAUSE PRINCIPLE

The Gause Principle, also known as the "Competitive Exclusion Principle", is a key concept in ecology related to ecological niches. Formulated by the Russian ecologist G.F. Gause in the 1930s, the principle states that two species that compete for the exact same resources cannot stably coexist. If two species have identical niches, one will outcompete the other, leading to the exclusion or extinction of the less competitive species.

## Key Points of the Gause Principle:

- Stable Coexistence through Niche Differentiation: Species that occupy different niches can coexist more stably. This differentiation can occur through behavioural, morphological, or physiological adaptations. For example, plants might have different root depths to access water and nutrients at different soil levels, reducing competition.
- **Competitive Exclusion:** According to the Gause Principle, one species will inevitably have a slight advantage over the other. This could be due to faster reproduction, more efficient resource utilization, or better adaptation to environmental conditions. Over time, this advantage allows the dominant species to outcompete and exclude the other species from the niche.

## Examples of the Gause Principle in Action:

**Paramecium Experiment:** Gause's original experiments with Paramecium species (single-celled organisms) demonstrated the principle. When two species, Paramecium aurelia and Paramecium caudatum, were cultured together with limited resources, P. aurelia consistently outcompeted P. caudatum, leading to the latter's decline.

## Importance of the Gause Principle:

**Understanding Biodiversity:** The principle helps explain the mechanisms that maintain species diversity in ecosystems. By recognizing how species avoid direct competition, ecologists can better understand patterns of biodiversity and species distribution.

**Conservation and Management:** Knowledge of competitive exclusion can inform conservation strategies. For instance, introducing new species into an ecosystem without considering their potential to outcompete native species can lead to unintended consequences, including the decline of native species.

**Predicting Ecological Changes**: The principle can be used to predict how ecosystems might change in response to environmental changes, such as habitat destruction, climate change, or the introduction of invasive species. Understanding niche dynamics helps

predict which species might be at risk of exclusion and how community composition might shift.

## IMPORTANCE OF ECOLOGICAL NICHE

The concept of an ecological niche is crucial in understanding how species interact with their environment and with each other. An ecological niche encompasses the role a species plays in its ecosystem, including its habitat, the resources it uses, and its interactions with other organisms. Here are several reasons why ecological niches are important:

**1. Biodiversity and Species Coexistence:** The concept of the niche helps explain how multiple species can coexist in the same environment. By occupying different niches, species can reduce competition for resources. This specialization allows for greater biodiversity within ecosystems.

**2. Resource Allocation and Utilization:** Understanding the niche of a species helps in identifying how resources are used within an ecosystem. This can inform conservation efforts and resource management, ensuring that critical resources are maintained for various species.

**3. Ecosystem Stability and Functioning:** Each species' niche contributes to the overall functioning of the ecosystem. For example, pollinators, decomposers, and predators each play specific roles that help maintain the balance and health of ecosystems. The loss or change of a species' niche can lead to cascading effects on ecosystem stability.

**4.Adaptive Evolution and Speciation:** Niches drive evolutionary processes by promoting adaptations that allow species to exploit different resources or environments. This can lead to speciation, where populations evolve into distinct species adapted to specific niches.

**5.Invasive Species and Ecosystem Impact:** Understanding ecological niches can help predict the impact of invasive species, which can disrupt existing niches and outcompete native species. This knowledge is essential for managing and mitigating the effects of invasive species on ecosystems.

**6.Climate Change and Habitat Alteration:** As environmental conditions change due to climate change, the niches of many species will shift. Understanding current niches helps predict how species distributions might change, which is critical for developing conservation strategies and managing biodiversity in the face of climate change.

**7. Human Impact and Conservation:** Human activities often alter habitats and resources, impacting the niches of many species. By understanding ecological niches, conservationists can design better strategies to protect species and restore ecosystems, ensuring that the necessary conditions for species' survival are maintained.

## Examples of Niche Importance:

- **Keystone Species:** Species like sea otters have a niche that involves preying on sea urchins. By controlling urchin populations, they maintain the health of kelp forest ecosystems. Loss of sea otters can lead to overpopulation of urchins and destruction of kelp forests.
- **Pollinators:** Bees occupy a niche that involves pollinating plants. Their role is crucial for the reproduction of many flowering plants, including crops. Declines in bee populations can lead to reduced plant reproduction and impacts on food production.
- **Decomposers:** Fungi and bacteria occupy niches as decomposers, breaking down dead organic matter and recycling nutrients back into the ecosystem. Without these decomposers, ecosystems would accumulate dead matter, and nutrient cycling would be disrupted.
- In summary, ecological niches are fundamental for understanding the dynamics of ecosystems, the interactions among species, and the processes that drive biodiversity and ecosystem functioning.

## Probable questions:

1. Define ecological niche. Explain the phenomenon with reference to protozoa.
2. Discuss the types of ecological niche.
3. What is niche overlap and niche width? Discuss with examples.
4. State the importance of ecological niche?
5. How Gause's principle is related with ecological niche?
6. What is the ecological role of parasites?

## Suggested reading: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# UNIT VI & UNIT VII

# Some general consideration of protozoan parasites: Temperature and climate

**Objective:** In this unit we will discuss about general consideration of temperature and climate of protozoan parasites.

## TEMPERATURE

Climate factors are often ignored in a study of parasite unless they are directly related to the control of disease of economic importance. Such as the various plants and animal diseases transmitted by insects.

The temperature at which a parasite grows is a major significance. Many species of protozoa and helminths (e.g., *Hymenolepis nana*) have been maintained experimentally at abnormally high temperatures and they have consequently changed their morphology as well as their physiologic relations to a marked degree.

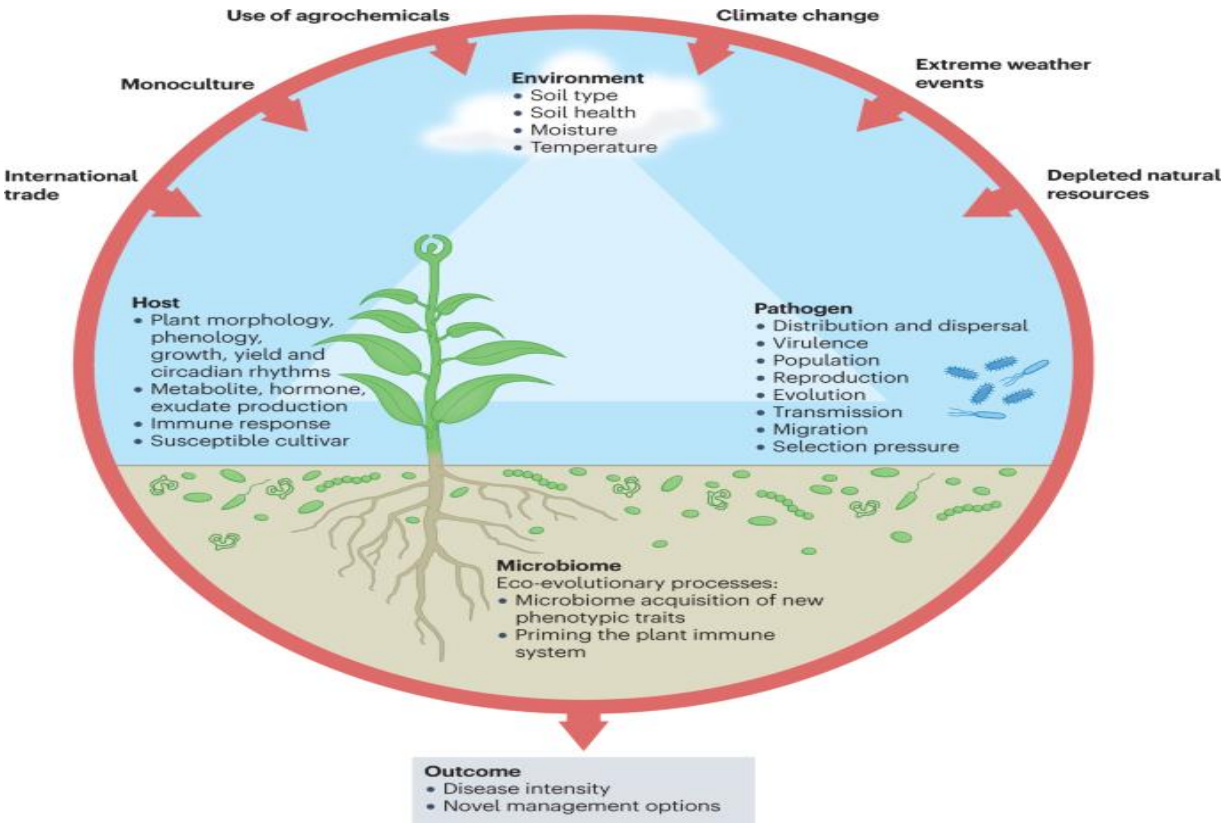


**Fig- Climate change impact on plant pathogen**

## There are many protozoan parasites which are affected by temperature and climate. These are described below:

- ***Trypanosoma rotatorium*** is highly polymorphic parasite in the blood of frogs. Studies have been carried out to determine how temperature affects the density of the flagellates in peripheral blood and to determine in internal temperatures of bullfrogs in their natural environment. The peripheral parasitaemia of the parasite *Rana catesbeina* was found to be markedly affected by temperature. Over the long-term high temperatures are always coincident with high peripheral parasitaemia and vice versa over the short term. Increase in temperature bring about a corresponding increase in parasite level and vice versa. It is proposed that the control of peripheral parasitaemia is due to the change in the level of metabolic activity of the host.
- Temperature affects both the reaction of host salamander, *Triturus viridescens,* to the parasite. *Trypanosoma diemyctyli,* and the response of parasite to the host. Infection of *T. diemyctyli* occurs in adult salamanders, and a critical point of change in the nature of infections from a pathogen to a nonpathogen is at about 20˚ C. The infection is pathogenic at lower temperatures only, and at higher temperatures the metabolic rate of the host is great enough to reduce the numbers of trypanosomes probably through the production of antibodies.
- ***Giardia*** is currently well established in harsh northern climates, where cooler, wetter conditions favour survival and transmission of cysts. It is possible that warming temperature will decrease environmental survival of cysts.
- ***Plasmodium*** is also depended on temperature. The optimum temperature for the development of the exogenous cycle of Plasmodium vivax was 28°C, the lowest temperature at which the cycle was completed in the shortest time.
- **Helminths** sometimes go through more than a 60˚C. Change in temperature from host to host during the normal course of their lifecycle.
- ***Schistocephalus*** suggests that two enzyme systems may be present, each of which responds differently to temperature changes. One enzyme system may control somatic growth in Plerocercoid larvae, with a peak efficiency near 23˚C, while the other system may control malnutrition in adult worms with a peak efficiency near 40˚C.
- ***Haemonchus contortus*** become progressively later. The reverse is true during the second half of the year. A low humidity accompanied by either low or high temperature. The largest number of climbing larvae are to be found during rainy season. Free larvae of this nematode's third stage larvae are the infective stage for grazing animals that become infected by eating the larvae with grass. The main factors concerned with the vertical migration of the larvae up the leaves are temperatures and humidity. Most of the larvae are in the grass blades during early morning and evening. The time of the morning maximum become progressively earlier while

passing from winter to summer and the time of evening maximum and other livestock nematodes can develop experimentally in cultivation over temperature range of about 10° to 35° C, or more. But the optimum is between 20° and 30° C. Larval mortality is high above 40° C, but some larvae may survive for short periods at 50° C, or more.

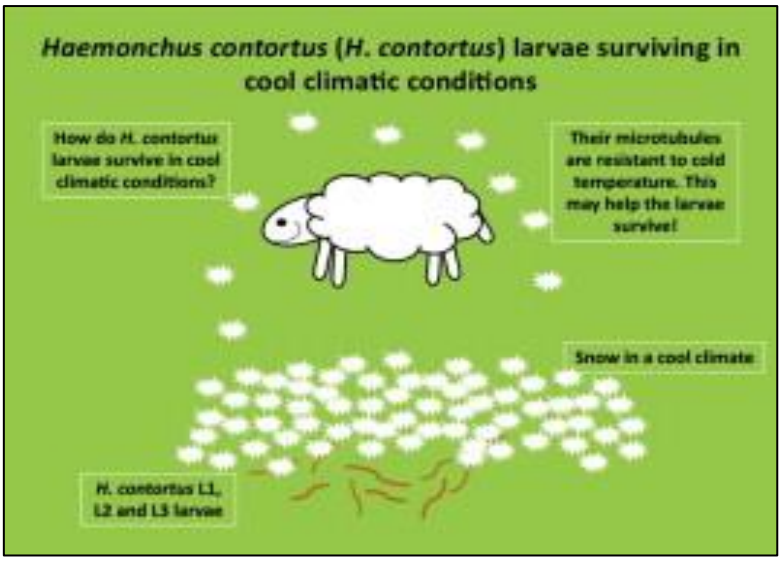


**Fig-*Haemonchus contortus* in climate condition**

1. Plerocercoid larvae in the stomach of *Gymnodactylus* (lizard) migrate well if the host is kept at room temperature but at 37°C migration does not take place. Relatively few experiments have dealt with the effort of high temperatures on developing parasites. But adverse effects on *Hymenolepis didiminuta* are greatest when exposure occurs during the sensitive period of maximum larval growth and development. Major indicators of sensitivity to high temperature (38.5° to 40° C for 24 hours) are failure of scolex-withdrawal and inhibition of infectivity for mammalian host. Such exposures to high temperature have little or no effect when applied at other time during development.

- ***Urceolaria*** has shown that the population changes in the parasite are not influenced by change in the host population. But that the changes are largely initiated by fluctuations in the surrounding populations of bacteria which fluctuations in turn are influenced by the rainfall. Temperature was found to be a secondary factor only. These ciliates tolerate marked chemical changes in the water in which they live and they feed chiefly on bacteria that are on or near the freshwater triclad turbellarian flatworms that serves as hosts.

Many parasites are said to be not only more abundant in the tropics but less adapted to colder climates. Such conclusions often result from considering each facet of information separately rather than studying and appreciating the full complex of ecologic and epidemiology implications.

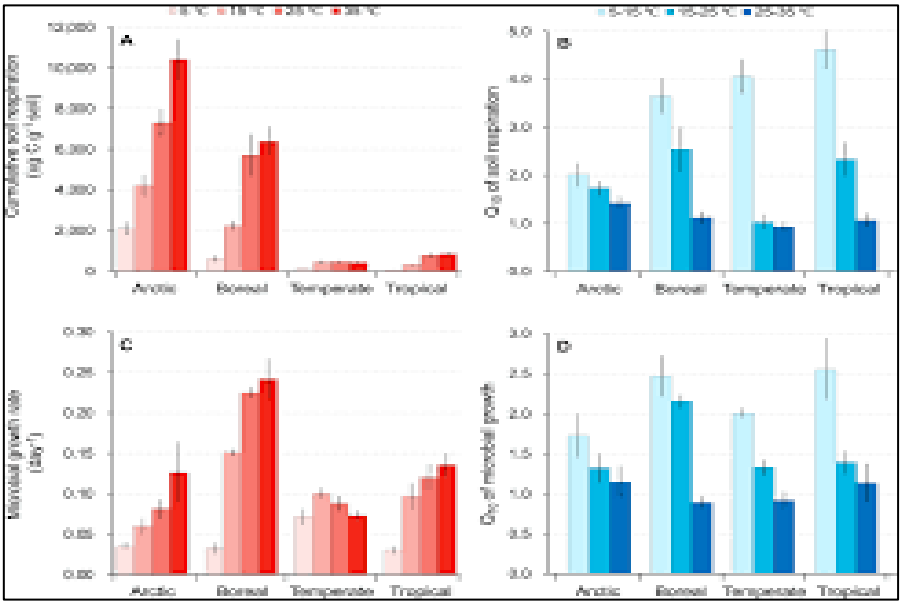


**Fig-**Temperature **sensitivity of soil: microbial biodiversity**

A number of protozoan parasites have been preserved by freezing and after thawing were found to be in good condition and able to grow and reproduce in a normal manner.

## Probable questions:

1. How does temperature influence the existence of parasites?
2. Explain how climate governs the distribution of parasites?
3. Comment on photoperiodism in protozoa?
4. "The distribution of Parasites and their hosts is directly or indirectly governed in large measure by climate"- Explain.
5. Describe the necessity of specific environmental conditions for the growth and development of parasites.
6. Describe the climate and seasonal variation of protozoan parasites?

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# UNIT VIII
# Some general consideration of protozoan parasites: Mutualistic intestinal Protozoa

**Objective:** In this unit we will discuss about general consideration of Mutualistic intestinal of protozoan parasites

## INTRODUCTION:

The term mutualism can be simply defined as a relationship in which both species are mutually benefited.

- It is present in the species or between the two different species.

**EXAMPLE**

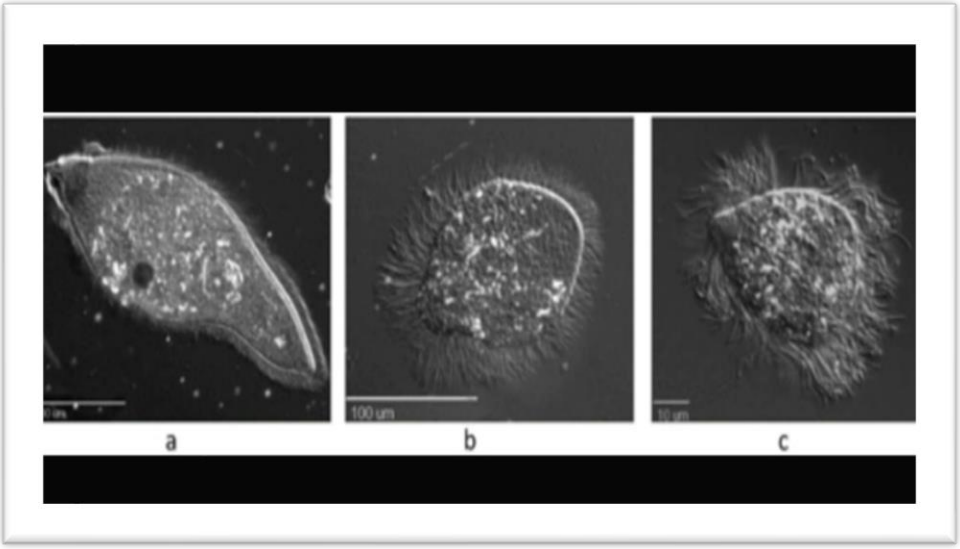
1. Mutualistic relationship between termites and their intestinal protozoan fauna.
2. Most of the protozoa in the rumen are ciliates Species belonging to the phylum Ciliophora.

## INSECTS

- A classic example of mutualism is the cooperation between termites and their intestinal protozoa. They live in the insect's gut specially in hind gut.
- Intestinal flagellated protozoans and termites exhibit obligative mutualism, a strict interdependency.
- *Cryptocercus*, rely on flagellated eukaryotic symbionts in the hind gut to digest their wood diet.
- Flagellates undergo encystment cycles tightly coordinated with the molting cycle of their host.
- The trophozoite stage is passed directly from parents to offspring via hindgut fluid.

The protozoa in nymphs of zootermopsis constitute one seventh to one-third of the total weight of host and parasites.

- Most of the larger flagellates in termites belong to the order Hypermastigida.
- The three species of flagellate protozoa found in the hindgut of the Formosan subterranean termites.




*a)* ***Pseudotrichonymphagrassii***

*b)* ***Holomastigotoideshartmanni***

*c)* ***Spirotrichonymphaleidyi***

## TRANSMISSION

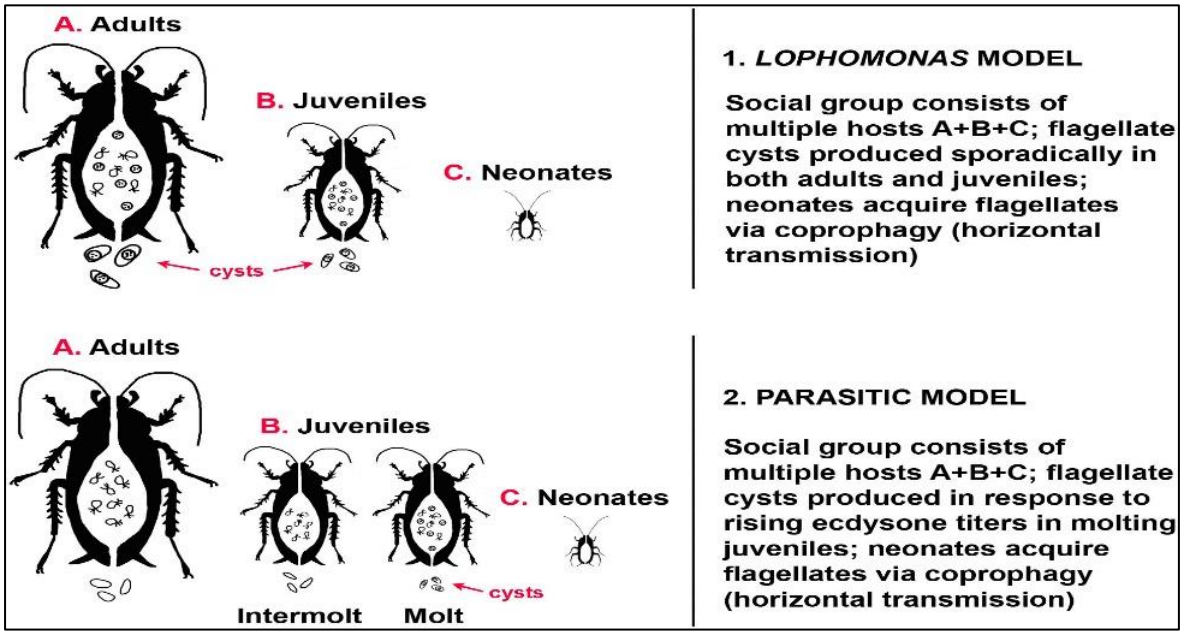



**FIG: MODEL OF TRANSMISSION DYNAMICS IN COCKROACH**

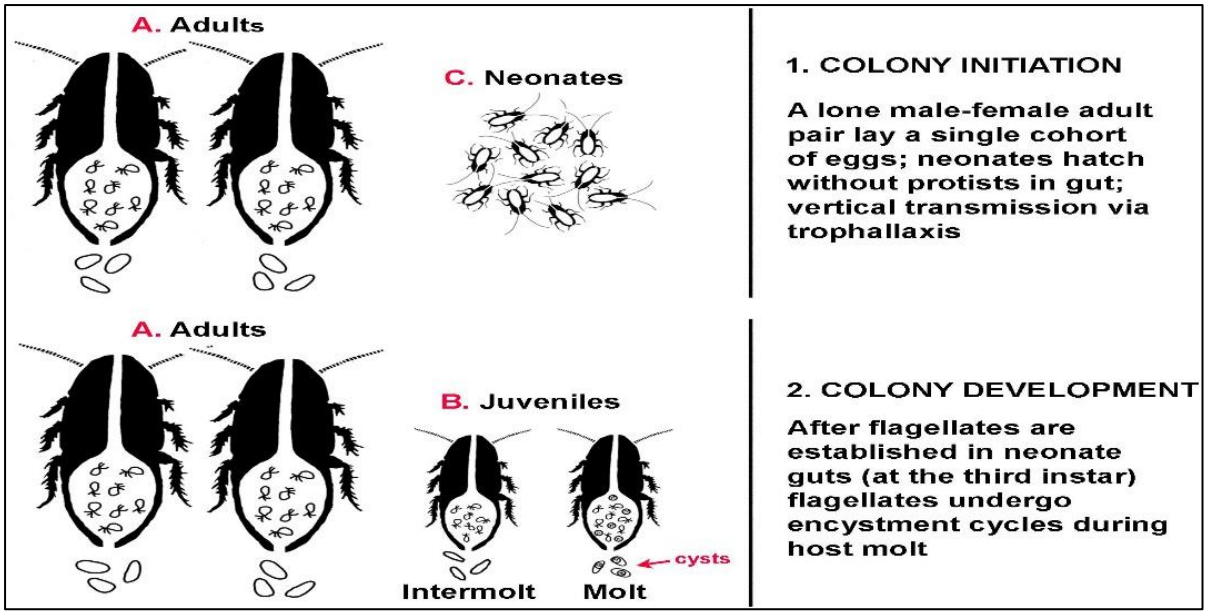



**FIG: TRANSMISSION DYNAMICS DURING COLONY ONTOGENY IN SUB SOCIAL WOOD FEEDING COCKROACH**

## TRANSMISSION

The mechanism by which they are transmitted is important because it influences the extent to which symbiont fitness interests are aligned with those of the host. There are two basic strategies-

- ✓ **Horizontal transmission-** It occurs across positions in space and is assumed to be the basal condition.
- ✓ **Vertical transmission-** Which takes place across generation in time, from parent to offspring. Vertical transmission that favors mutualistic relationship becomes consistently associated with a host.

## TRANSMISSION FROM PARASITISM TO MUTUALISM:

They comprise a fluid spectrum of interaction from mutually beneficial to neutral to exploitative. Although mutualistic relationship such as the one between *Cryptocercus* and its flagellates can arise in several ways from other interactions along the spectrum mutualism originate from an antagonistic parasitic relationship. The most commonly cited catalyst driving the change from parasitism to mutualism is the switch from horizontal to vertical transmission. Parasites become totally dependent on host pushing the flagellates to evolve metabolically in a way more favorable to the host.

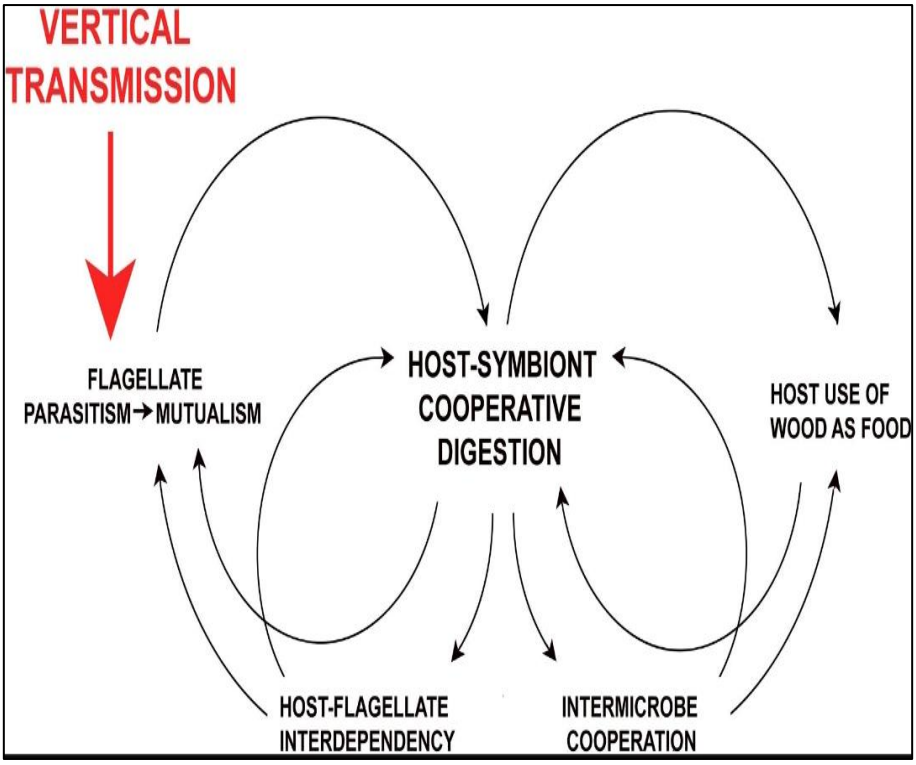



Fig: Transmission from parasitism to mutualism

## ROLE

**Cockroach**- Gut flagellates must utilize host resources to meet their needs like nutrients and energy. In cockroaches coprophagy has benefits that may outweigh any cost of mutualism.

The presence of protozoa resident in the gut may play a role in shaping the way that the host body responds to infections.

Cockroach feces offer fragmented moistened and softened fare and are enriched with lipids, carbohydrates, amino acids, unsaturated fatty acid, sterols.

Feces additionally serve as inoculum in the horizontal transmission of beneficial gut bacteria.

A parasitic flagellate of cockroaches that insinuates its cyst into this ingestible fecal bonanza has a high probability of finding a new host.

Protozoa help the cockroach by releasing unique enzymes and digestive substances. This makes it fit enough to digest almost anything ranging from nylon to plastic as well as both edible and inedible food.

- The protists get a safe home in the termite's gut and a continual supply of ingested wood. The termite is unable to digest cellulose by itself so it uses the protists in its gut to do so.

- Termites also known as wood ants eat wood to meet their nutritional demand. Wood contains 40-50% cellulose. These protozoa reside in hind gut of termites and secrete an enzyme Cellulase.
- Cellulase enzyme is capable to hydrolyze the beta-linkages of cellulose and form short chain fatty acid. Thus, in return termites provide its gut and protection and food that is Cellulose.
- These protozoans would die outside the termite and termite also cannot survive without them.

Few members that do feed possess in their intestines presumably elaborate enzymes that digest wood. The first nymphal instar acquires its protozoa by proctodeal feeding at the anus of an infected termite.

During each molting period protozoa are temporarily lost to be regained as originally acquired. Only the adult, asexual, nondividing forms ordinarily occur in termites. During molting period, they are directly influenced by the rise in base of the gut fluids.

Encystment of the protozoa does not take place.

Termites can be defaunated by incubating them at high temperature for 24 hours or by submitting them to two atmospheres of oxygen, after the protozoa are dead the insect will continue eating wood but they will starve to death because they cannot digest their food. If they are soon refaunated they will survive.

- Termites depend upon the protozoan in their gut or digestive tract to digest the complex sugars in wood into simple molecules that they can use for food.
- The termite cells use these acids like acetic acid as nourishment just like our cells do.
- The termite protozoa also produce gases during this breakdown process.
- Methane gas is a major product and termites are large source of methane in our atmosphere.

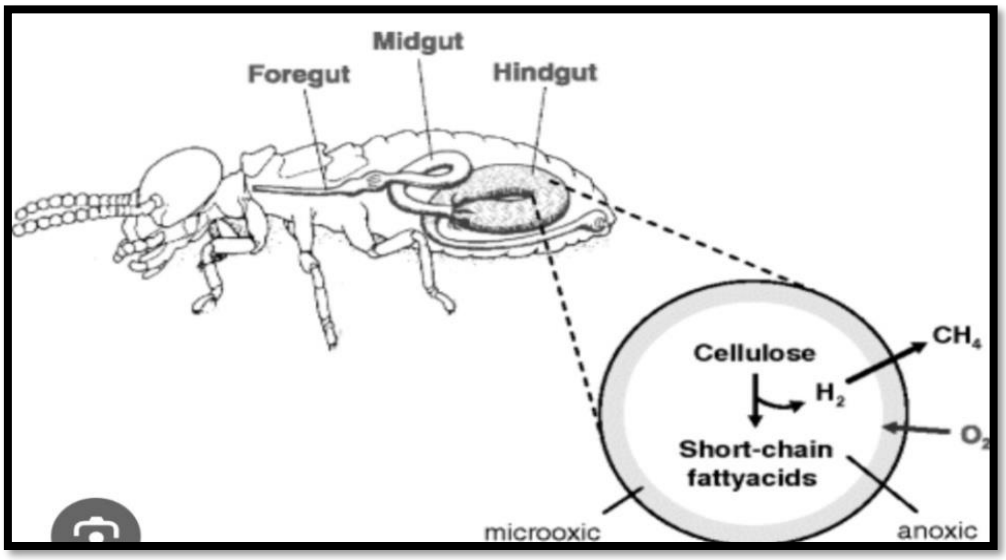


Fig: Role of protozoa in gut of termite

## RUMINANTS:

Ruminants are special populations and common herbivores such as cattle and sheep have an important organ that can digest fibrous plant resources. The microbial system in the rumen is very complex, and it is mainly composed of protozoa. Among rumen protozoa ciliates are the largest most abundant and most important. It is a single cell eukaryotic organism.

Protozoa helps to ferment feed into the rumen so that organisms can provide nutrients to maintain the physiological activities of the host.

Rumen protozoa can swallow rumen bacteria reduce the rate of starch digestion and protect animals from acidosis.

Some rumen protozoa can swallow fiber substances and digest cellulose by producing related carbohydrate enzyme.

In sheep and goat the gymnostome ciliates occur in numbers between 160,000 and 200,000 per milliliter of rumen content.

*Diplodinium*per milliliter of rumen contents in cows fed timothy hay and concentrate with a range between 800-30000.

Protozoa helps in utilization of anaerobic intracellular protozoa for synthesis of essential building materials high energy phosphate for the ability of the protozoa to survive.

Starvation for several days followed by the administration of a copper sulfate solution. Cellulose and hemicellulose continue to digest at same rate.

Absorb soluble carbohydrates of the host stomach and convert the carbohydrate into starch, helping to keep sugars from being immediately fermented.

It may store large amount of glycogen.

About 66gm of protein are supplied the host in the form of ciliates each day.

The exact contribution would fluctuate from day to day as numbers and kinds of protozoa vary.

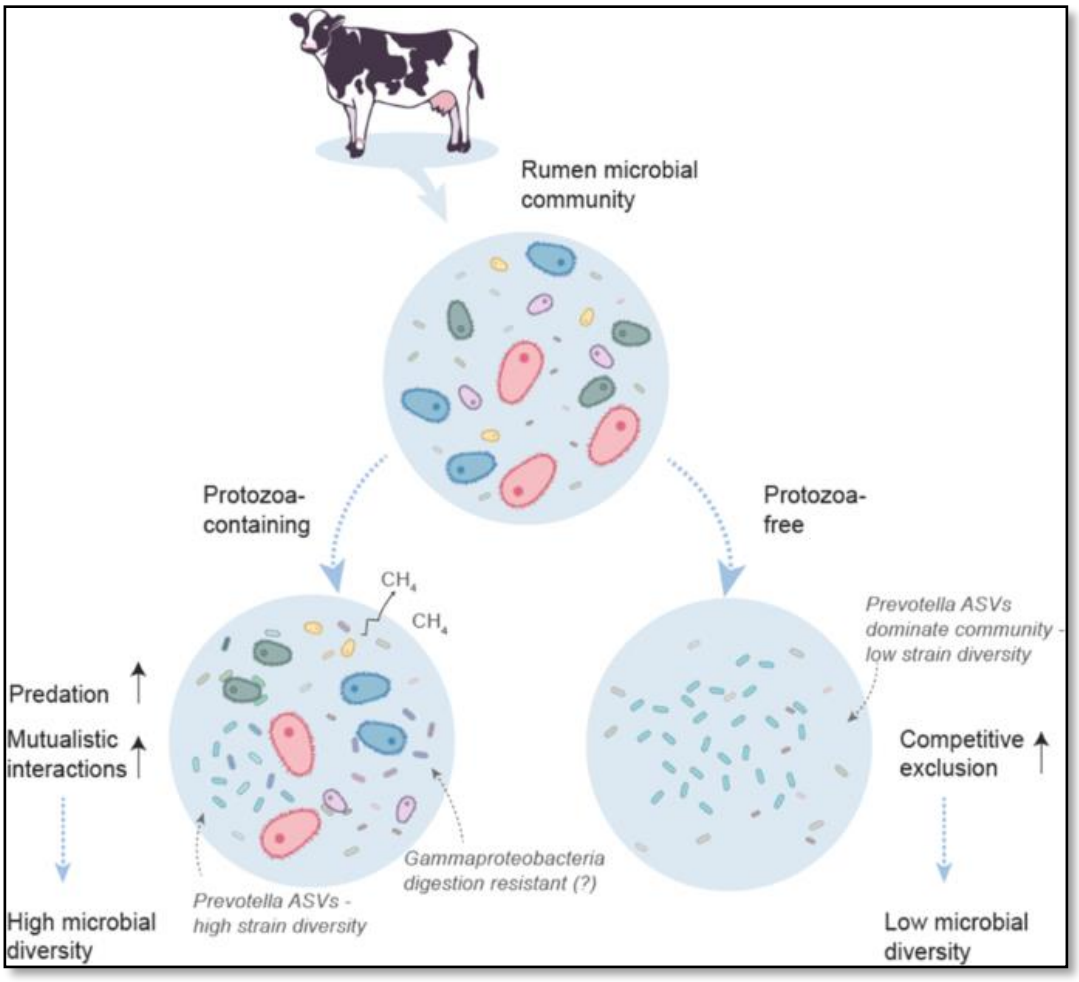



Fig: Mutualistic Intestinal Protozoa In Cattle

## Probable questions:

1. Write a note on mutualistic intestinal protozoa.
2. State the role of Intestinal Protozoa in synthesizing cellulose.
3. “A classical example of mutualism is the cooperation between termites and their intestinal protozoa fauna”- Justify.
4. Mention two major roles of protozoans in the rumen.
5. Describe about Transmission from Parasitism to mutualism.

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# UNIT IX
# Arthropods as blood suckers

**Objective:** In this unit we will discuss about Arthropods as blood suckers

## Introduction:

Hematophagous arthropods (ticks and insects) transmit various life-threatening diseases resulting in over one million human deaths annually. Exploiting vertebrates for blood demanded extensive sensory and behavioral adaptations that are apparent across the evolutionary range of vector species, from primitive ticks to advanced dipterans.

## Free-Living Bloodsuckers:

Free-living bloodsuckers includes mosquitoes, black flies, biting midges, sandflies, horse flies, and deer flies among the "lower" Diptera and tsetse and many Muscidae among the Cyclorrhapha. They are bound to the host mainly as a source of food, and they are characterized by high mobility. Like black flies, mosquitoes and horse flies are capable of migrating actively or being carried by the wind several dozen kilometres away from the breeding site. However, their flight range is normally restricted to 1-2 km or even to hundreds of meters; sandflies are restricted to an even smaller area. Host-seeking behaviour is provided for by well-developed organs of sight and also by orientation to $CO_2$ and other chemicals given off by the host.

A potentially broad polyphagy is typical of most species. Blood of the vast majority of species of vertebrate animals probably fulfills the nutritional requirements of these hematophages and varies only quantitatively in its nutritional value, so that the influence of feeding on different species of mammals or birds by mosquitoes is mainly restricted to their fecundity. Host specificity, if present, is usually ecological in nature. Therefore, unrelated groups of vertebrates occupying the same habitats, possessing similar circadian rhythms, and characterized by other bionomic similarities may be hosts for one species of bloodsucker. Preferential feeding by mosquitoes, black flies, and midges on mammals or birds is often observed; by sand flies of the genus *Sergentomyia* on reptiles and of the genus *Phlebotomus* on mammals. In *Aedes aegypti* host selection between mammals and birds has been shown to be genetically fixed. Several species of *Anopheles* feed predominantly on man, which makes them particularly dangerous malaria vectors.

Though Diptera possess the capability of feeding at almost any site on the host body, various spatial preferences have been observed. Most Tabanidae feeding on ungulates prefer the limbs, abdomen, head, and neck, but larger forms (*Tabanus bovinus*, *T. sudeticus*) more frequently feed on the back. Significant factors in the choice of feeding site among different species of horse flies are the size of their feeding apparatus, the

height and density of host pelage, thickness of skin, and depth of capillaries. Selectivity with respect to blood meal site has also been noted in mosquitoes and other biting flies, and in some cases, it is directed at decreasing the efficiency of defensive host behaviour.

Difficulties in host location and normal completion of a blood meal have resulted in the phenomenon of gonotrophic rhythm ("harmony" in Russian) in most free-living bloodsuckers. This refers to the coordination between blood digestion and ovarian development, which allows consumption in a single blood meal of the minimal quantity of food sufficient to ensure the development of one egg batch. Gonotrophic rhythm was first discovered in mosquitoes, and later in black flies, biting midges, sand flies and horse flies. However, it is lacking in Cyclorrhaphous tsetse and stable flies (*Stomoxys*). Disturbances in gonotrophic rhythm may appear in the form of gonotrophic dissociation in some species of overwintering mosquitoes, autogenic development of eggs (oviposition without adult feeding and utilizing food reserves acquired in the larval stage), and abundant feeding on sugar in the imaginal stage.

The amount of blood ingested is normally not more than 1.5-2 times the weight of an unfed individual. Decrease in the amount of ingested blood results in a lower number of eggs produced. The minimal amount of blood required for oviposition may be highly variable. It is 0.4 to 0.6 of the body mass in all studied species of *Culex* and not less than 1 in *Anopheles maculipennis*.

Egg production from one gonotrophic cycle is moderate and varies from 40-60 in sand flies, 100-500 in mosquitoes, 900-1000 in black flies and 300-600 in horse flies. Fecundity may be as high as several thousand eggs over repeated gonotrophic cyIes. Thus, in females of *Anopheles maculipennis*, 13 successive gonotrophic cycles may result in 2000-3500 eggs. Longevity of the adult female varies from several days to several weeks, and rarely to several months, as in the case of hibernating mosquitoes.

Among the families of Diptera that include hematophages, most of those in the Nematocera and the Tabanidae (aside from autogeny), as well as the Glossinidae, consist entirely of obligatory bloodsuckers. The Ceratopogonidae, in addition to bloodsuckers, include nectarophages and entomophages. Among Rhagionidae bloodsuckers are represented by only a few genera. Muscidae include facultative bloodsuckers, mucophages, and coprophages.

## Nest-Burrow Bloodsuckers:

Nest-burrow bloodsuckers (most Argasidae, many Gamasina, some Siphonaptera, Cimicidae, some Reduviidae) are, apart from food, bound to the host by a common habitat, i.e. the nest or burrow to which their complete life cycle is confined. Analysis of species composition of bloodsucking arthropods from burrows of the great gerbil (*Rhombomys opimus*), little souslik (*Citellus pygmaeus*), voles, and other rodents, as well as from the nests of birds, has shown that there are constant complexes of bloodsuckers, which are bound by trophic relationship not only with their principal hosts but also with secondary

or accidental vertebrate hosts and invertebrate nidicoles.

Host-parasite specificity of nest-burrow bloodsuckers is determined primarily by microclimate and by structure and degree of isolation of the burrows and nests from other vertebrate members of the ecosystem. For example, it was established in Siberian taiga that the shared fauna of gamasine mites in nests of voles of the genera *Clethrionomys* and *Microtus* was 56.7%, whereas for *Clethrionomys* and shrews of the genus Sorex it was 70%, despite the hosts in the latter case belonging to different orders. Most nest-burrow bloodsuckers are polyxenous to various degrees, which reflects the frequency of their contacts with different species of their potential hosts. Inhabitants of nests occupied for many years are oligoxenous because, like their hosts, they are ecologically isolated from other species of animals. Typical in this respect are nests of the bank swallow (*Riparia riparia*) with the fleas *Ceratophyllus styx* and *C. riparias* and the tick *Ixodes plumbeus*, and nests of the martin (*Delichon urbica*) with the fleas *C. hirundinis*, *C. delichoni*, and *C. jarreni*, the louse-fly *Craterina hirundinis*, and the bug *Oeciacus hirundinis*.

The amount of a single blood meal, the frequency of bloodsucking, and fecundity vary significantly in different taxonomic groups of this category of bloodsuckers. What they all have in common is a constant connection with the burrow or nest and only a short period of time spent on the body of the host, that is when feeding. The most heterogeneous in type of feeding are gamasine mites, among which even in the family Laelapidae, which is comprised largely of parasitic species, one can observe varying degrees of transition from predation or schizophagy to facultative hematophagy, as in the case of *Eulaelaps stabularis*, *Haemogamasus nidi*, *H. citelli*, and *H. ambulans*. Most Laelapidae are characterized by frequent blood meals (50-100 % of body weight), no gonotrophic rhythm, and low fecundity with rapid completion of life cycle. Many representatives of the family Dermanyssidae (*Ornithonyssus*, *Dermanyssus*, etc) are obligatory nest-burrow bloodsuckers. Typical of this family is the development of a gonotrophic rhythm and increase in weight by more than tenfold following a single blood meal. One oviposition series may include 3-20 eggs, and during a lifetime one individual may oviposit several times.

Among Siphonaptera a considerable number of species spend the longest part of their life cycle in nests or burrows ("nest fleas"). Thus, species of *Coptopsylla* are present on the host body only once, and no longer than 1-3 hr. No gonotrophic rhythm is observed in these fleas, and the mass of blood consumed at one time varies from 50 to 100 % of the weight of a hungry individual. Some species of "nest fleas," e.g. *Ceratophyllus garei* and *C. gallinae*, which are associated with birds, are capable of free movement from abandoned nests, whereas related parasites of martins (*C. farreni*, *C. hirundinis*, *C. delichoni*) occupy new nests of birds by passive dispersal on the hosts.

Despite the fact that they belong to different classes, similarities in parasitic relations are found between bloodsucking bugs of the families Reduviidae (Triatominae) and Cimicidae and soft ticks of the family Argasidae. These are obligatory bloodsuckers in all active stages of development, though in some species of soft ticks' hungry

individuals have been observed to consume newly fed ticks of their own species. Among Triatominae, cases of "cannibalism" (feeding on the blood meal taken by another individual) have been observed in nymphs of *Triatoma sanguisuga*, and entomophagy is known to occur in *T. rubrofasciata*. Feeding lasts several minutes (sometimes about 1-2 hr). The amount of food consumed during that time exceeds the weight of a hungry individual by tenfold or more. It provides nourishment for the molt to the next stage or for egg deposition (from several dozen to 200—300 eggs). Host specificity is in most cases poorly expressed. Thus, for example, many species of mammals, birds, and reptiles may be hosts of the genus *Ornithodoros*: in the laboratory the tick *O. papillipes* has been fed the blood of these different hosts without showing significant differences in its molting ability or fecundity. Species of soft ticks of the genus *Argas* that are found on birds have a higher food specialization and even in experimental conditions consume only a small amount of mammal blood. These bloodsuckers possess a remarkable capability for surviving long periods without food, e.g. several months in bugs and fleas, years in soft ticks. Some individuals of *Ornithodoros papillipes* have survived 10-11 years without nutrition.

## Temporary Ectoparasites with Rapid Feeding:

Rapid-feeding, temporary ectoparasites (some Siphonaptera and gamasine mites, the majority of Hippoboscidae, Nycteribiidae, and Streblidae) spend most of their life cycle in the fur or feathers of their hosts, only leaving to deposit eggs or puparia. They have a higher host specificity than nest-burrow bloodsuckers. Among fleas and Gamasina this type of host-parasite relationship is an expression of the evolutionary pathway of nest-burrow bloodsuckers in which the relationship with the host has been strengthened. Thus, "host fleas" (*Leptopsylla*, *Ceratophyllus*, *Xenopsylla*, and others) take frequent, small blood meals (with 1-3 hr intervals). Gonotrophic rhythm is lacking and egg development is timed to coincide with the stay on the body of the host. In species of the genus *Xenopsylla* a period on the host is followed by the development and oviposition of a considerable number of eggs during the following 28-48 hr. *Leptopsylla segnis* does not leave the host even for oviposition, so that all of the eggs are deposited in the fur and only later fall into the burrow. Average fecundity of host fleas varies from 4 to 19 eggs per day and during the whole period of reproduction from 200 to 300 eggs.

In Pupipara, temporary parasitism undoubtedly originated from ancestral forms of free-living bloodsuckers. Many Hippoboscidae that stay mainly in the feathers or fur of the host have preserved the ability to fly and have a more nearly fly-like appearance. Nycteribiidae and Streblidae stay permanently in the fur of bats, leaving only for deposition of puparia, and they are extraordinarily modified in appearance.

## Temporary Ectoparasites with Slow Feeding:

Slow-feeding, temporary ectoparasites (all hard ticks, larvae of chigger mites, many soft ticks, some gamasine mites of the family Macronyssidae, and some fleas) leave the host only to molt and to oviposit. Feeding lasts several days and is accompanied by intensive growth of the integument and visceral organs. This phenomenon of intermolt growth is rare among arthropods and is called neosomy. Such parasitism is typical of hard ticks, who's feeding generally lasts 3-5 days in adult females and 7 or more days in immature stages. The body weight of females increases by more than 100 times. At every developmental stage of the life cycle, ticks feed only once, and the blood meal is sufficient for the molt to occur to the next stage. Females deposit, depending on the species, from 1000-3000 (*Ixodes*, *Haemaphysalis*) to 10,000-20,000 eggs (*Hyalomma*, *Amblyomma*). Most ixodids are three-host species. In some species of *Hyalomma* and *Rhipicephalus*, after a blood meal the larvae remain on the host body and molt into nymphs that detach and leave the host after the blood meal (two-host species). In *Boophilus* species, *Hyalomma scupense*, etc. all molts occur on the host body (one-host species), and only the adults leave after the blood meal.

All developmental stages in most species of *Ixodes* and *Haemaphysalis* and often larvae and nymphs in other genera of ticks stay in the dwellings of their hosts. Accordingly, the host range of species in this ecological group is relatively narrow and they are oligoxenous to various degrees. In about 10% of ixodid species, the host is attacked by ticks waiting on the surface of the soil or on plants; these ticks usually feed on many different vertebrate species. Thus, hosts of *Ixodes persulcatus*, which is widespread throughout the taiga zone of the Palearctic, were found to include 200 species of mammals, 120 species of birds, and several species of reptiles.

A similar lack of host specificity is typical of many species of larval Trombiculinae, which also passively await. the host on the ground or on vegetation; some of these mites have been discovered on several dozen species of mammals and birds belonging to various orders.

In certain fleas (e.g. *Tunga* species) feeding on the host continuously for many days is accompanied, as in hard ticks, by neosomy and increase of fecundity to several thousand eggs. During feeding the semisedentary rabbit flea *Spilopsyllus cuniculi* frequently changes its attachment site, detaches from the host for oviposition, and then attacks new hosts. Sticktight fleas (*Echidnophaga*, *Vermipsilla*, *Dorcadia*) after attaching to a host do not leave it until the end of their life cycle. Eggs are laid in the fur and subsequently fall on the soil surface or burrow substratum. Host specificity of these fleas is much higher than that of most ticks. The final stage in this evolutionary trend among fleas is the intradermal parasitism of fleas ("jiggers") of the genus *Tunga*.

Slow feeding by ectoparasites stimulates different forms of cellular and humoral immunity in the host, which has been most carefully studied in hard ticks. Slow-feeding ectoparasites in tum have developed a number of adaptations to weaken or suppress immunologic responses. For example, mouthparts of some Acarina, when fastened in the

host skin, are contained in a tube or cone formed directly or indirectly by the action of salivary gland secretion, i.e. the cement sheath in hard ticks and the stylostome in chigger mites. This enables these ticks and mites to obtain liquid food from skin layers located much deeper than the distal ends of their mouthparts. This food is composed of the products of tissue lysis and an inflammatory infiltrate in the focus at the attachment site. Adaptation of ectoparasites to their hosts is reflected in localization of tick and flea attachment sites on certain parts of mammals and birds. Coordination of the drop-off rhythms of engorged ticks and the oviposition rhythms of stick-tight fleas with circadian rhythms has been demonstrated. In *Spilopsyllus cuniculi* and some other flea species, direct response of bloodsucking females to hormone levels in the blood of their rabbit hosts has been observed. The transition of attached female fleas from the resting state to intensive bloodsucking and egg development is stimulated by increased levels of corticosterone and other hormones.

## Permanent Ectoparasites:

Permanent ectoparasites (biting and sucking lice, various mites, bugs of the family Polyctenidae, and some louse flies of the family Hippoboscidae) are associated with the body of the host throughout their entire life cycle. They attach their eggs to hairs or feathers of the host. That these parasites are oligoxenous or monoxenous is due not only to the type of nutrition but also to environmental conditions on the host body. Features typical of this group are small and frequent feedings (on blood, lymph, secretions of oil and sweat glands, scales of epidermis, hair, or feathers), lack of gonotrophic rhythm, moderate or low fecundity, low individual longevity, inability to survive long periods without feeding, and a short life cycle. Infection of new hosts is mainly through contact, although cases of phoresy are known.

Among permanent ectoparasites, only sucking lice are known vectors of pathogens. Insects and mites that feed on epidermis or secretions of dermal glands have limited chance of contact with pathogens that cause transmissible diseases. Because all stages of lice may be completed on one host individual and transfer between individuals is irregular, only a few species are efficient vectors.

As a result of their epidemiological significance, the body louse and head louse (*Pediculus humanus corporis* and *P. h. capitis*) are among the most thoroughly studied permanent ectoparasites. Their development includes three nymphal instars that last 15-16 days; the longevity of the imago is 6-60 days. Off the host and unfed, they survive not more than 5-10 days. Nymphs take 3-8 blood meals per day and adult females 5-12. Feeding lasts several minutes, and during that time the amount of blood consumed by adult females constitutes 50-100% of the body weight of an unfed individual. Gonotrophic rhythm is lacking, and average daily production is 3-10 eggs; one female can lay 100-300 eggs during its lifetime. There is strict host specificity for humans, but under laboratory conditions lines of lice have been reared successfully on rabbits or with alternate feeding on man and guinea pig. It is possible to make *P. humanus* suck the blood

of many species of mammals and even pigeons under laboratory conditions. However, normal development on these atypical hosts is impossible. In some experiments even one blood meal on another host resulted in death of the insect.

Bugs of the family Polyctenidae have a mode of life similar to lice. These are parasites of bats that have repeated blood meals in every instar. Deprived of the opportunity to feed, nymphs die within 6 hr, imagoes in 30 hr, so that transfer to new hosts is possible only when there is direct contact.

**Probable questions:**

1. What do you mean by hematophagous arthropods?
2. Enumerate spatial preference of blood feeding by dipterans.
3. Explain gonotrophic rhythm.
4. What are the two mechanisms of blood feeding by temporary ectoparasites?
5. State the epidemiological significance of sucking louse.

**Suggested reading:**


1. Noble, E. R. and Noble G. A. (1989). Parasitology. The biology of animal Parasites. 6th ed. Lea and Febiger, Philadelphia.
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5. Smyth, J. D. (1994). Animal Parasitology. 3rd ed. Cambridge University Press.
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# UNIT-X

# Arthropods as disease transmitters

## Objectives:

In this section we will discuss on arthropods as disease transmitters.

## Introduction:

Arthropods are capable of serving as vectors, indicating that they play a major role in disease transmission. Arthropods that serve as vectors include mosquitoes, fleas, sand flies, lice, ticks, and mites. These arthropods are responsible for the transmission of numerous diseases. These types of vectors are considered to be hematophagous. These arthropod vectors are characterized as feeding on blood at some or all stages of their life cycles. The arthropods feed on the blood which typically allows parasites to enter the bloodstream of the host.

## Disease transmission:

The order Diptera is by far the most important with regard to disease vectors, primarily because of the family Culicidae (mosquitoes). Ticks (family Ixoxoidea) are the second most important disease vectors. Most vector-borne pathogens are zoonoses (pathogens of animals) and have a primary vertebrate reservoir host and a primary arthropod vector that maintains the transmission cycle in nature; this is usually via horizontal transmission but is sometimes facilitated by transovarial or vertical transmission. This primary cycle is usually inapparent in humans and domestic animals. On occasion, the pathogen may be introduced into the peridomestic or urban environment by secondary vectors or by the vertebrate host, which often establishes a secondary transmission cycle involving other vertebrate hosts and arthropod vectors. Humans and domestic animals are usually infected by bridge vectors from these secondary cycles and are often dead-end or incidental hosts, not contributing to the transmission cycle. Recent studies suggest, however, that humans may in fact play a previously unappreciated role as reservoir during the transmission of pathogens and parasites in what is typically referred to as anthropogenic transmission (e.g., visceral leishmaniasis caused by *Leishmania donovani* in India and Sudan). A basic vector-borne pathogen transmission cycle is shown in Fig. 147.1.

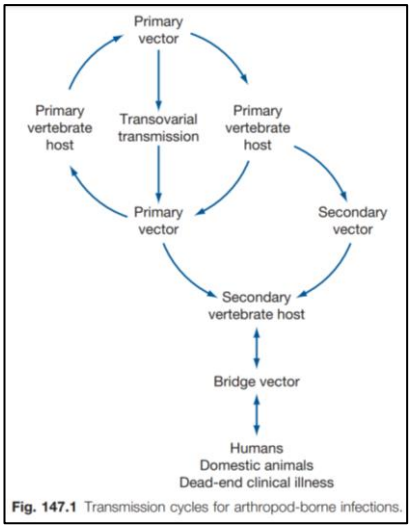

**Fig. 147.1** Transmission cycles for arthropod-borne infections.

An arthropod can transmit a pathogen from one person or animal to another in one of two basic ways.

**Mechanical Transmission**

Defined as transfer of a pathogen present on contaminated mouthparts or other body parts. There is no multiplication or developmental change of the pathogen on or in the insect. Examples include some enteroviruses, bacteria, and protozoa of human and veterinary importance. Insects, such as houseflies, can become contaminated with such pathogens while feeding on feces and transfer them to food. Parasites such as *Trypanosoma vivax* (of African origin) are transmitted to cattle in South America via the bite of tabanid flies.

**Biologic (or Horizontal) Transmission**

The second and most important type of disease transmission by arthropods is biologic. The pathogen must undergo development in the insect vector in order to complete its life cycle. There are four types of biologic transmission.

**Propagative**

The pathogen is ingested with a blood meal and undergoes simple multiplication within the vector. Examples are the arboviruses, which replicate extensively in the tissues of the arthropod and are transmitted to a new host in the saliva when another blood meal

is taken. The plague-causing bacteria *Yersinia pestis*, transmitted by the bite of infected fleas, also is in this category.

**Cyclopropagative**

The pathogen undergoes a developmental cycle (changes from one stage to another) and multiplication within the vector. Classic examples of cyclopropagative transmission include *Plasmodium*(malaria), in which a single ookinete, formed after gametogenesis, may give rise to thousands of sporozoites and *Leishmania* (leishmaniasis), in which infective metacyclic promastigotes develop after tissue amastigotes change to

promastigotes and multiply.

**Cyclodevelopmental**

The pathogen undergoes developmental changes from one stage to another but does not multiply. With the filariae, for example, a single microfilaria ingested by a mosquito can result in only one infective third instar (L3) larva. In most instances, however, the number of infective larvae is lower than the number of microfilariae ingested with the blood meal.

**Transovarial or Vertical**

Some viral and rickettsial disease agents are transmitted from the female parent arthropod through the eggs to the offspring. If the pathogen can infect the developing egg germ cells, it is termed transovarial transmission. With some arboviruses, only the ovarial sheath and oviduct are infected; the egg is infected as it passes down the oviduct and is inseminated. This is called vertical transmission. In either case, the newly hatched larval stages are infected with the pathogen, which is then transmitted to subsequent developmental stages of the arthropod (transstadial transmission). Finally, venereal transmission of certain viruses has also been documented. Male mosquitoes that become infected transovarially or vertically can transfer the infective virus to female mosquitoes in the seminal fluid during copulation. These latter types have some epidemiologic importance in the infection of humans and animals, and in the maintenance of the pathogen in nature.

**Extrinsic Incubation Period**

In all types of biologic transmission, a period of time is required for the pathogen to develop (or multiply, depending on the case) within the arthropod vector and be able to be transmitted. For a number of parasites, including filarial worms, *Plasmodium*, and *Leishmania*, it requires change from a non-infective to an infective stage that can be successfully transmitted during blood feeding by the vector. This period between infection of the arthropod vector and transmission is called the extrinsic incubation period (EIP). The EIP may vary between 3 and 14 days, depending on the pathogen, the vector, and various environmental factors, including temperature.

**Entomologic Inoculation Rate**

The entomologic inoculation rate (EIR) defines the number of infected bites an individual may receive during a period of time, be it a day, a month, or a year. EIR is important to define risk of exposure to vector-borne disease, along with several other biotic and abiotic factors.

**Barriers to Pathogen Development**

Generally, transmission requires an ability of the pathogen to escape barriers in the vector that are generally associated with vector competence. For example, viruses must overcome the midgut infection barrier and the midgut escape barrier to reach the hemolymph (blood), be disseminated, and ultimately invade the salivary glands (transmission barrier). For *Plasmodium*, escape from the peritrophic matrix that

envelops the blood meal, invasion of midgut cells and ability to survive the mosquito's innate immune responses, and invasion of the salivary glands by the infectious sporozoites are limiting steps for parasite development and transmission (Fig. 147.2).

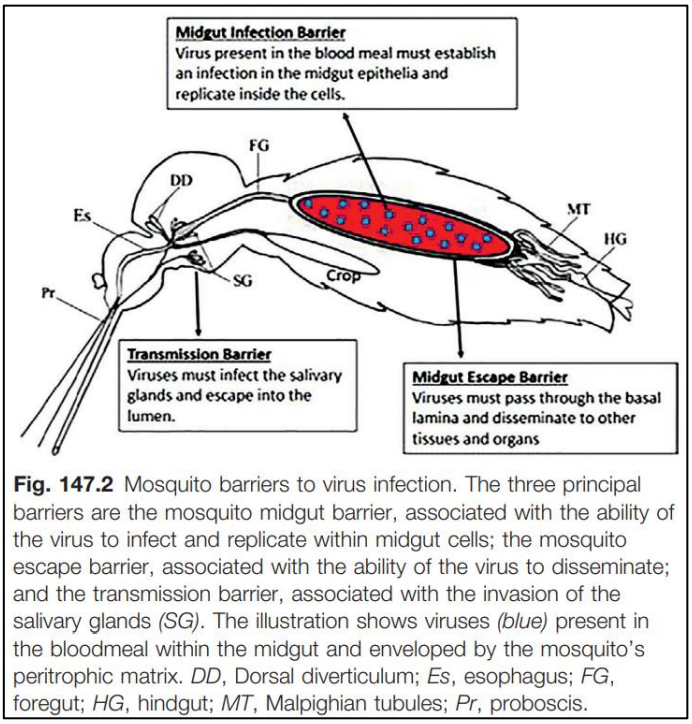


**Fig. 147.2** Mosquito barriers to virus infection. The three principal barriers are the mosquito midgut barrier, associated with the ability of the virus to infect and replicate within midgut cells; the mosquito escape barrier, associated with the ability of the virus to disseminate; and the transmission barrier, associated with the invasion of the salivary glands *(SG)*. The illustration shows viruses *(blue)* present in the bloodmeal within the midgut and enveloped by the mosquito's peritrophic matrix. *DD*, Dorsal diverticulum; *Es*, esophagus; *FG*, foregut; *HG*, hindgut; *MT*, Malpighian tubules; *Pr*, proboscis.

Several such barriers have also been identified for other vector–pathogen pairs. It is important to realize that, in many instances, a pathogen and a vector co-evolved so that the vector may indeed transmit the pathogen. Hence, not all arthropods may be able to transmit a given pathogen.

## • Factors Influencing Transmission

The ability of insects to transmit an agent depends on the interaction of complex intrinsic and extrinsic factors. Successful mechanical transmission depends on the degree of contact insects have with humans and on the arthropod feeding behavior. For example, the domestic housefly has been incriminated as a mechanical vector of various intestinal pathogens, primarily because this insect breeds in large numbers, lives in intimate contact with humans, and has the habit of feeding on feces and food. Tabanidae flies (horseflies) are efficient mechanical vectors of viruses and protozoa because of frequent interrupted blood feeding. Certain flies can mechanically transmit the bacteria that cause yaws and other tropical diseases from open sores.

The ability to transmit a pathogen biologically varies greatly among species of arthropods and even among geographic strains or isolated populations within a species. There can be variation in the susceptibility to becoming infected and subsequently to transmitting a pathogen. Thus, for example, within a single mosquito species, it is common to find geographic strains that are good and poor vectors. Because vector

competence (susceptibility to infection, growth of the pathogen, and transmission) is genetically controlled, it may be expected to change as a result of selective pressures on either the pathogen or the arthropod over time.

In addition to innate susceptibility to infection, the overall vectorial capacity is influenced by other biologic and behavioral characteristics of the arthropod. The degree of contact the species has with humans is influenced by the blood meal host preference; the intrinsic blood-feeding and resting behavior of the arthropod; and the population density of the vector, animal, and human hosts. Longevity, resting behavior, flight behavior, and oviposition (breeding) behaviors are important intrinsic factors that are influenced by extrinsic environmental factors, such as temperature, humidity, wind, and rainfall.

Other extrinsic factors may influence whether an individual insect becomes infected with a pathogen. For example, it has been shown that mosquitoes ingesting blood containing both microfilariae and Rift Valley fever virus have a higher viral infection rate because disseminated virus infection is facilitated by microfilariae escaping from the midgut into the hemocoel. Finally, infection of the arthropod and subsequent transmission is influenced by the strain of pathogen. This is especially important with the arboviruses, where certain strains or subtypes of virus have greater infectivity and more rapid replication in the vectors.

Because arthropods are cold-blooded, transmission of diseases in temperate regions is seasonal, usually only occurring during warm months. Cessation of transmission in these regions is usually determined by temperature and day length. In the tropics and subtropics, transmission generally occurs year-round. In these areas, increased seasonal transmission is most frequently correlated with the rainy season.

Finally, it has been shown that vector saliva plays important roles during transmission due to its anti-hemostatic, anti-inflammatory, and immunomodulatory activities. Recently it has also been shown that bacteria present in the vector gut and deposited on the skin during the bite elicit immune responses that can be associated with certain clinical outcomes.

**Probable questions:**

1. Describe with a flow diagram transmission cycle of an arthropod vector.
2. What are the different types of transmission of pathogen in arthropod vectors?
3. What do you mean by extrinsic incubation period and entomologic inoculation rate.
4. State the factors responsible for controlling transmission of pathogens in arthropod vectors.

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# Unit-XI
# Parasite-host specificity with reference to protozoan parasites

## Objectives:

In this section we will discuss on parasite-host specificity with reference to protozoan parasites.

## Introduction:

Host specificity was defined as the degree to which a parasite or symbiont is restricted in its range of hosts (Simpson and Weiner, 1989). It is a simple concept encompassing evolutionary history of the parasite's associations with different host lineages (Page, 2003). It is an important attribute of the parasite's life-history, as it predicts the likelihood that a parasite will successfully invade a new area, or adjusts to new hosts following its introduction to a new ecogeographical area.

## Parasites and Hosts:

In heteroxenous life cycles a distinction is first made between the final host and intermediate host. Sexual reproduction takes place in the final (definitive) host. Some confusion in terminology can occasionally arise with this definition; for plasmodia, for example, the more important host from an anthropocentric viewpoint is the human being. However, fertilization takes place in the mosquito, and hence the insect must be regarded as the definitive host. Another part of the life cycle of parasites takes place in the intermediate host, where significant developmental processes or asexual reproduction occur. This is the distinguishing factor between intermediate hosts and pure transmission agents (vectors), which transmit pathogens mechanically (e.g., through the stylets of bloodsucking insects). Several intermediate hosts may be exploited in succession during a life cycle, and these are known as first or second intermediate hosts. In some life cycles, a host individual plays the roles of the final and intermediate hosts simultaneously. In many cases, transmission from intermediate to definitive hosts occurs by predation of the former by the latter. The larval stages of some parasites can also be transmitted from smaller to larger intermediate hosts through the food chain, without any significant morphological changes occurring. Such hosts, known as paratenic hosts, accumulate the larvae, and their insertion in the life cycle facilitates transmission of these larvae to the definitive host.

At each stage of their life cycle, many parasites are optimally adapted to a particular host species, which may represent their main host in fat, that is, the one they have coevolved with for a long time. On this host, growth and reproduction are optimal and

the parasite enjoys a long-life cycle. By contrast, living conditions are worse for the parasite in alternative host in fat that nonetheless allow the survival of the parasite, with the result that these hosts play a less significant role in the perpetuation of the life cycle. These alternative hosts, however, may serve as reservoir hosts in fat and be of major epidemiological importance–when control measures have been used on the main host, for example, chemotherapy on farm animals, the parasite cycle in wild reservoir hosts cannot be eradicated and a reinfection may take place via these hosts. By contrast, parasite development stages can sometimes occur in a wrong host or dead-end host, where no further transmission can take place (e.g., *Toxoplasma gondii* in humans).

One indispensable basis for the establishment of a host–parasite relationship is the susceptibility of the host. Susceptibility is essentially determined by the behavioral, physiological, and morphological characteristics of the host, and also by the host's innate and adaptive immune responses. Within a population, therefore, the host genotype often determines the individual degree of susceptibility–certain hosts may thus be predisposed for infection. Acquired characteristics, such as physical condition or age, can also affect an individual host's susceptibility to parasites.

Host resistance to a parasitic infection can depend on the immune responses of the host. This becomes clear when a host only becomes susceptible when elements of the immune system are disabled. For example, *Aotus* monkeys–used as experimental animals in malaria vaccine research–can only be reliably infected after splenectomy. However, resistance can also be defined by biochemical factors. *T. brucei*, for instance, is killed by a protein in human serum, which is associated with high-density lipoproteins. Immunity is the term used when a past infection leaves behind protective immune responses. In the case of parasitic infections, an existing infection often provides immunity to further infections. A concomitant immunity (premunition) like this permits already-established parasites to survive, but leads to the elimination of new infective stages trying to infect the host. This situation can cause parasite density to be downregulated to a tolerable level for the host. Hosts with defective immune systems are often more susceptible to parasites; these hosts may consequently be colonized by opportunistic pathogens, which are present only in low densities or not at all in immunocompetent individuals. Such opportunistic parasitic infections are common in AIDS patients – and in many cases, these infections are the direct cause of death. Examples of this are the frequent occurrence of *Toxoplasma gondii*, *Cryptosporidium parvum*, and *Leishmania* species in AIDS patients and other immunocompromised persons.

Parasites can specialize in varying degrees in the way they exploit their hosts. The degree of specialization is expressed in the host specificity, which combines the number of host species that can be used at any stage of the life cycle, and the relative prevalence and intensity of infection by the parasite on these hosts. For instance, parasites that can infect only one host species or infect a few host species but achieve high prevalence and intensity on only one of these species have a high degree of host specificity ("narrow host specificity"). Parasites with wide host specificity can colonize a wide range of hosts successfully, and often achieve high prevalence or intensity of infection on many of these

hosts. For example, certain stages of *Trypanosoma cruzi* and *Toxoplasma gondii* exploit almost all mammals as hosts and invade almost all types of the hosts' nucleated cells. Relying on the host range combined with information on prevalence and intensity of infection as a measure of specialization can be, however, misleading. Let us consider two related parasite species, A and B, each using four host species and achieving almost equal prevalence and intensity in all their hosts. However, the hosts of parasite A belong to distantly related families, whereas those of parasite B all belong to the same genus. Therefore, we can easily argue that parasite B displays higher host specificity than A, since its hosts are restricted to a narrower phylogenetic spectrum. Host specificity is the outcome of colonization of new hosts and adaptation to these hosts over evolutionary time, and the more host-specific parasites are those that cannot make the large jump necessary to colonize animal species not closely related to their main host. In this context, several parasites have made the "jump" from wild or domestic animals to humans; diseases caused by parasites transmitted between vertebrates and humans under natural conditions are referred to as zoonoses.

The establishment of parasites in a susceptible host result in an infection. In strict context, this term applies only if an increase in the number of parasites occurs by replication of the original parasite within the host, as in the case of protozoa. The term formerly used for these groups is "infestation." The period during which diagnostically relevant parasite stages appear, such as plasmodia in the blood, is known as patency. The period from infection to patency is called prepatency or the prepatent period, while the period until the onset of the first symptoms is known as the incubation period. For helminth parasites, prepatency corresponds to the period from initial infection of the host to the onset of egg production, when eggs start appearing in the feces or urine of the host; patency then corresponds to the adult life of the worm, from the onset of egg production to its death. In accordance with an international agreement, the infection and the resulting disease are known by the name of the parasite with the suffix -osis, for example, toxoplasmosis. However, form any diseases the suffix-iasis is in wide use.

The term used when an infection with the same pathogen occurs after a parasitosis has healed is reinfection. An infection contracted in addition to an existing parasitosis and caused by the same species of parasite is known as superinfection. Simultaneous infections with multiple pathogen species are known as mixed infections. Both superinfections and mixed infections have consequences for host welfare, as the overall harmful effect on the host may depend on additive or synergistic effects between the different infections. If a host infects itself with stages that originate from its own infection, it is called autoinfection.

The harmful effect which the host suffers from parasites may have different causes and manifestations, and is measured in different ways by different groups of researchers. As a measure of the impairment caused by parasites, evolutionary biologists use the reduction in the host's genetic fitness attributable to infection. This decrease in host fitness is referred to as the virulence of the parasite, and is quantified as the relative difference between the reproductive capacity of the infected host compared to what

reproduction it could achieve without the infection. In the assessment of medical importance, the parameters morbidity (incidence of disease) and mortality (incidence of death) are used. A quantification is determined by calculating the disability-adjusted life years (DALYs); this is a WHO index into which up to 140 individual parameters flow for the assessment of a disease. Finally, the harmful effect of parasitic infections in livestock is calculated by determining the loss of productivity (e.g., milk yield in cows and wool production in sheep) and the cost of infection control.

At a physiological level, parasitic infections usually have a pathogenic impact or effect; this is generally described as pathogenicity and the defined molecular factors that are important in this context are called pathogenicity factors. The amoebapore protein produced by *Entamoeba histolytica* is a pathogenicity factor, since it plays a crucial role during tissue invasion. The term used for the quantitative expression of pathogenicity is virulence (Latin virulentus=full of poison), which was originally a means of assessing a pathogen's degree of aggressiveness. Unfortunately, the same term is used by evolutionary biologists to refer to the parasite's effect on host genetic fitness. From a physiological perspective, the host is therefore not only harmed by food deprivation – the destruction of cells or tissues through the action of toxic metabolic products and immune responses that harm the host's own tissue (immunopathology) also cause damage. It used to be thought that phylogenetically ancient parasite–host relationships should be characterized by a relatively low pathogenicity, since parasites which only minimally harm their hosts may persist longer over the course of evolution. However, following both theoretical work and experimental studies with fast-evolving pathogens, it is now accepted that the pathogenicity (or virulence in its evolutionary sense) of parasites can increase over the course of evolution. Indeed, under a range of circumstances, natural selection can favor aggressive exploitation of the host, resulting in high parasite replication and transmission rates, at the expense of long-term host survival.

## Mucosal parasites and microbiota interactions in human Populations

Mucosal infection with the enteric protozoa *Entamoeba*, *Giardia*, *Cryptosporidium*, and *Blastocystis* can be asymptomatic or cause diarrhoea, abdominal pain, and/or weight loss. The infecting parasites reside in the intestinal mucosa and therefore are surrounded by the mucosa-associated microbiota. It has been proposed that the dynamic interplay that occurs between the protozoan parasite, host microbiota, and host immune system shapes the clinical outcome of enteric infections.

Infection with the gut parasite *Entamoeba* was significantly correlated with fecal microbiome composition and diversity. *Entamoeba* species infection was predicted by the composition of an individual's gut microbiota with 79% accuracy in a study of the farming and fishing populations in southwest Cameroon. One of the most important taxa in predicting an infection with *Entamoeba* was *Prevotellaceae*. In a separate independent

study focused on the *E. histolytica*-associated diarrhea that is common in Bangladeshi infants, levels of *Prevotella copri*, a member of the Prevotellaceae, were found to be elevated in patients with diarrheagenic *E. histolytica* infections. The Cameroonian study was focused on infected adults who were not experiencing symptomatic amebiasis; therefore, it is interesting that both *P. copri* and *Prevotella stercorea* were significantly downregulated in infected individuals. Both studies suggest that microbiota composition may play a significant role during an *E. histolytica* infection. These studies also highlight the potential influence of inflammation driven by the gut microbiome in altering parasite infection outcomes. Elevated levels of *P. copri* have been associated with severe inflammation and an increased risk of autoimmune disease and colitis, suggesting that the organism is proinflammatory.

*Cryptosporidium*, *Giardia*, *Blastocystis*, and *Trichomonas* infections may also be influenced by the gut microbiota. A retrospective study of volunteers who were originally enrolled in *Cryptosporidium* infectivity studies examined the relationship between the relative abundances of several bacterial taxa commonly found in adults prior to or within 48 h of infection and infection outcomes. The patients that were protected from infection had a greater abundance of *Proteobacteria* and lower *Bacteriodetes* and *Verrucomicrobia* levels than infected subjects. There was a higher ratio of Firmicutes to Bacteriodetes in uninfected subjects than in infected subjects. Seven specific taxa had differences of at least 2.5-fold between the two groups. Specifically, uninfected subjects had increased relative abundances of the indole-producing bacteria *Escherichia coli* CFT073 and *Bacillus* spp., as well as *Clostridium* spp. In contrast, infected subjects had increased relative abundances of *Bacteroides fragilis*, *Bacteroides pyogenes*, and *Prevotella bryantii*, as well as *Akkermansia muciniphila*. Presently, the mechanism by which increased indole production may protect from *Cryptosporidium* is unknown.

## Alteration of the microbiota as a therapy for protozoan infections?

Patient cohorts and future microbiome epidemiological studies will establish a more complete understanding of variation in clinical presentations of infection with parasitic protozoa. However, population-based studies do not allow us to test the effects of the microbiota on parasite survival and proliferation. Therefore, in vitro and in vivo disease models provide a useful tool to understand how the intestinal bacterial microbiota may influence severity and progression of infection and what mechanisms might underlie that progression.

In vitro culture models allow interactions between infecting agents and individual components of the microbiota to be analyzed. A study of the in vitro effects of six *Lactobacillus acidophilus* strains and *Lactobacillus johnsonii* La1 on *Giardia duodenalis* survival, for example, demonstrated that *L. johnsonii* La1 significantly inhibited the

proliferation of *Giardia* trophozoites. The potential protective role of *L. johnsonii* La1 (NCC533) was independently confirmed by in vivo experiments with La1-treated gerbils, which were protected against Giardia infection and mucosal damage. In another in vitro study, common human commensal bacteria were cocultured with *E. histolytica*. Culture of *Lactobacillus casei* and *Enterococcus faecium* alone with amoebae reduced parasite survival by 71%. When both bacteria were used in combination survival was reduced by 80%. A previous study demonstrated a link between decreased *Lactobacillus and amebiasis* in Indian patients, further supporting a potential link between these bacteria and resistance to amoeba infection.

As mentioned previously, lactobacilli may impact susceptibility to *T. vaginalis* infection in women. Mechanisms underlying this effect are still being studied; however, inhibition of adhesion of the parasite might help explain protection. In one study, adhesion assays were carried out by incubating vaginal epithelial cells (VECs) with *T. vaginalis* and lactobacilli together and by comparing levels of parasite adhesion to non-lactobacillus recipient controls. *Lactobacillus gasseri* ATCC 9857 and CBI3 caused significant parasite adhesion inhibition in a dose-dependent manner.

## Probable questions:

1. Explain parasite-host specificity in the light of first and second intermediate host.
2. State the criteria of host resistance to parasitic infection.
3. What do you mean by amoebapore protein?
4. How mucosal parasites interact with microbiota in human?
5. Explain novel technique of microbiota alteration as a therapy for protozoan infection.

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# Unit-XII
# Genome organization in *Plasmodium*

## Objectives:

In this section we will discuss on Genome organization in *Plasmodium*

## Introduction

The parasite *Plasmodium falciparum* is responsible for hundreds of millions of cases of malaria, and kills more than one million African children annually. *Plasmodium falciparum* PfEMP1 is a malaria virulence protein whose repression is epigenetically regulated. The parasite's ability to express exclusively only one of the sixty var genes that encode PfEmp1 is essential for disease pathogenesis. Malaria is a life-threatening disease caused by *Plasmodium* parasites, with 241 million cases and an estimated 627,000 deaths worldwide in 2020. *P. falciparum* is the deadliest species of five *Plasmodium* species that cause malaria in humans. *Plasmodium* parasites expressed variant antigen encoding erythrocyte membrane protein 1 (PfEMP1) at the surface of host red blood cells to escape the human immune system. PfEMP1 is encoded by approximately 60 *var* genes and only one *var* gene is expressed during the 48h replication cycle in red blood cells

### PfEMP1 AND var GENES

- **PfEMP1**

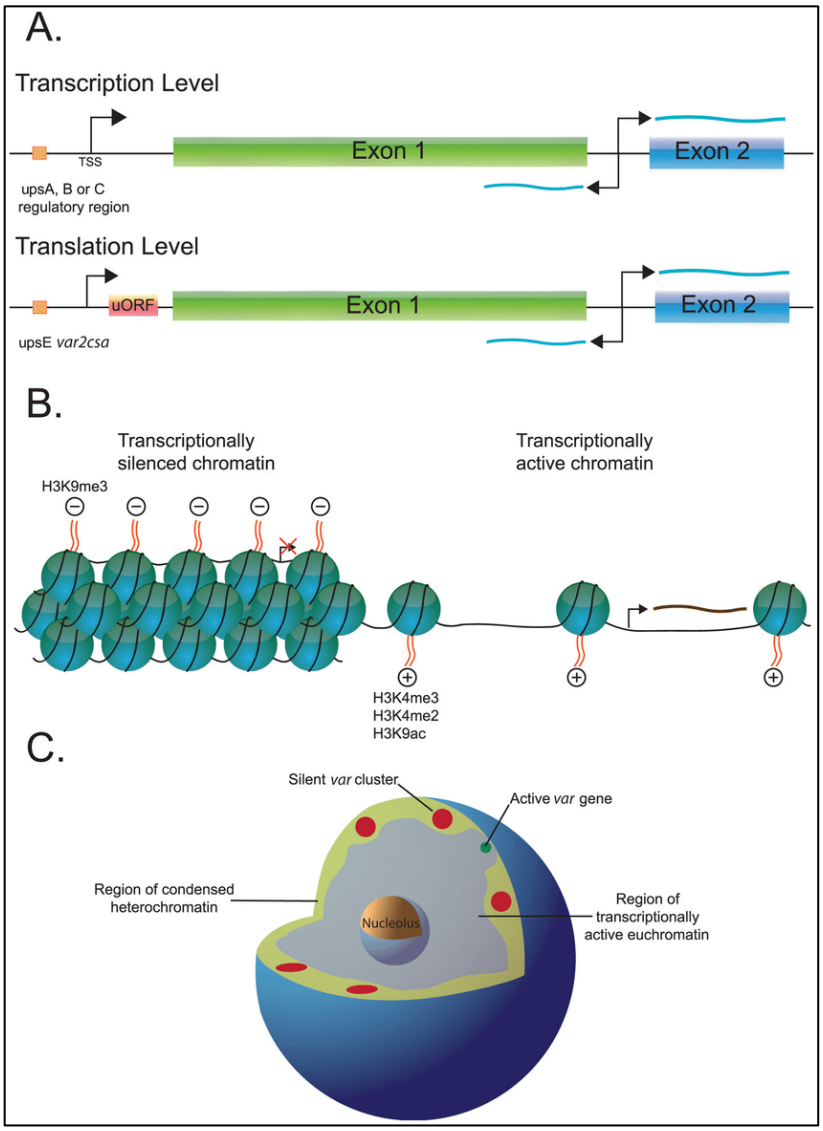
pfEMP1, the most important variant surface antigen exposed on the surface of infected erythrocytes, is responsible for cytoadherence of infected erythrocytes to the vascular endothelium and is encoded by a family of approximately 60 var genes. The sequential, exclusive expression of a single var prolongs the infection cycle within the human host, and PfEMP1 is linked to the lethal complications of malaria infection including cerebral malaria and anemia. Although it is known that the activation of expressed var gene and the silencing of an expressed var gene and the silencing of the others are epigenetically regulated, the molecular basis of the activation of an expressed var gene and the silencing of the others are epigenetically regulated, the molecular basis of the mutually exclusive expression of var genes has been an enigma.

Silenced var genes are marked with the conserved H3k9me3 heterochromatin mark.

**Heterochromatin mark**: Heterochromatin is found in the nuclear periphery and silenced var genes localize to 3-10 distinct puncta corresponding to heterochromatin regions. Active var mRNA peaks in ring stages, but the active locus needs to retain the

euchromatin epigenetic marks including the general gene activation mark H3K4me3 that allow access of the transcriptional machinery.

## EFFECT of *var* GENE




PfEMP1 was encoded by a gene family, called *var.* There are 40-50 *var* genes per haploid genome and these are predominantly localized in subtelomeric regions of all 14 chromosomes. The *var* genes vary in molecular size from 6 to 15 kb, are extremely variability separated from a smaller highly conserved 3' exon encoding an acidic region low in cysteine residues. The sequence is also referred to as ATS (Acidic Terminal Sequence) or Var C (for Conserved). Since fragments of the ATS were shown to bind to the knob-associated histidine-rich protein (KAHRP), the ATS has been presumed to anchor PfEMP1 to parasite protein in the knob. The 5' exon is predicted to be exposed on the surface of the PE and contains two to five copies of a motif denoted DBL, Duffy binding ligand. Members of this family, previously described from the merozoite stage, have the ability to bind to diverse cell receptor such as the Duffy blood group and glycoprotein A. The nomenclature of DBL is to number them in order from the 5' end and to identify the homology group by α-ε. The first DBL, DBLα is the most conserved domain. The second motif common to all *var* genes is the cysteine-rich interdomain region (CIDR). They also have sequence homologies termed α, β and γ. The first CIDR, CIDR1, is found immediately after DBL1α and the second CIDR, CIDR2, follows DBLγ. Different isolates contain different numbers of *var* genes in different chromosomal locations and may have

different sequences. Many *var* genes are transcribed at the early ring stage, but a single dominant mRNA coding for the surface-expressed PfEMP1 appears to be selected at later developmental stages.

## PfEMP1 RECEPTORS

The *var* genes show high sequence diversity and, therefore, it is impossible at the time to predict the adhesion phenotype of a *var* gene from its primary sequence. Nonetheless, several adhesion domains have been defined.

## ROSETTING DOMAIN

In vitro mature-stage-infected red cells can adhere to uninfected red cells to form spontaneous rosettes. DBLα contains clusters of glycosaminoglycan (GAG) binding motifs which could play a role in resetting, since different strains have

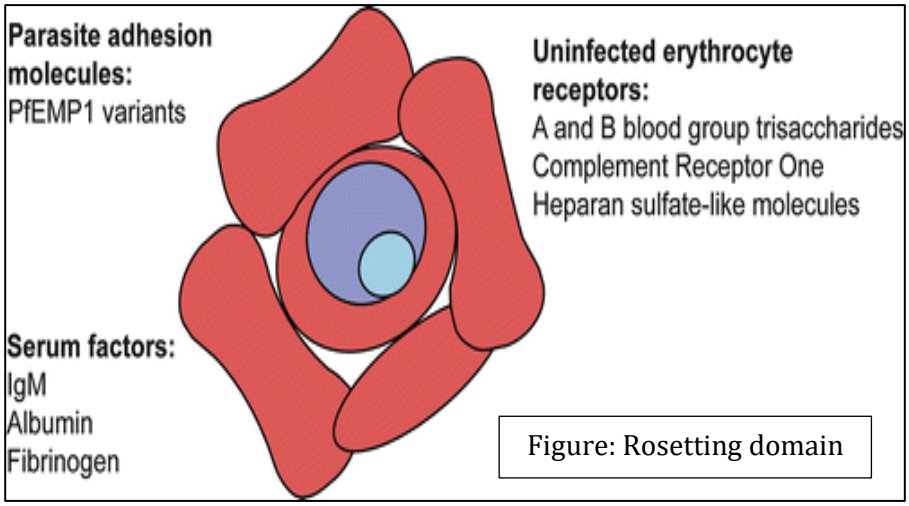
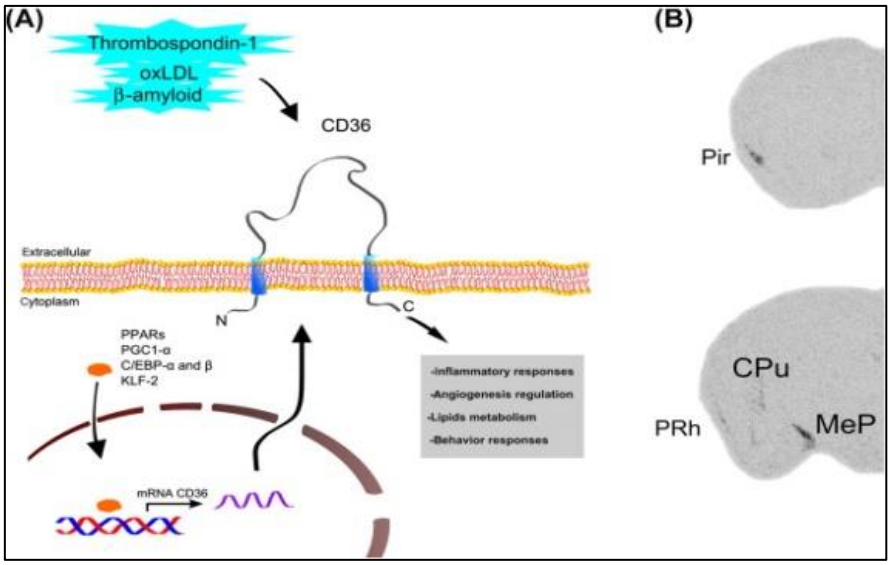


Figure: Rosetting domain

been shown to have variable sensitivity to rosette-disrupting agents such as heparin, heparin sulfate, fucoidin and dextran sulfate. DBLα, from a resetting property can vary between strains, it has been suggested that resetting may be prompted by host receptors acting in concert with DBLα. For example, resetting was shown to be influenced by immunoglobulin, glycoconjugates, blood group antigen A, and complement receptor (CR1).In the case of CR1, resetting was found to be impaired when RBC were deficient in this receptor and soluble CR1 blocked resetting formation and IgM binding and this resulted in a serum strengthening effect. It has been claimed that for the FCR3S1.2 clone, the CIDR1 domain



of PfEMP1 mediates IgM binding, whereas with the TM284S2 clone, it is the DBL2β domain that binds IgM.

## CD36 ADHESION DOMAIN

CD36 (also called GPIIIb, GPIV and GP88) is an 88-kDa glycoprotein found on the surface of several cell types including platelets, monocytes, dendritic cells and microvascular EC. The vast majority of clinical isolates and laboratory lines bind to CD36. It is generally believed that CD36 provides a stable and long-lasting anchor for PE to bind to the EC that line the post capillary venules. The PE-binding domain of CD36 has been reported to reside in amino acids 139-184, an immunodominant region, although other regions may also contribute to adherence. The MAb OKM5, which has as its epitope the immunodominant region, blocks cytoadherence of PE to CD36.

## CSA BINDING DOMAIN

Chondroitin sulfate A (CSA) is a GAG linked to the cell surface via a membrane associated protein. In general, it consists of a heteropolymer of alternating glucuronic acid and 4-sulfated N-acetyl glucosamine residues. CSA from different sources may differ substantially in sulfation patterns. PE adhesion is strongly dependent on sulfation in the fourth position as well as the specific saccharide chains. Most often those PE that bind to CSA do not bind to CD36 and vice versa. This dichotomy has been explained by conformational attributes. Some PE can also bind to hyaluronic acid (HA) a non-sulfated high molecular-weight GAG composed of alternating N-acetyl glucosamine and glucuronic residues.

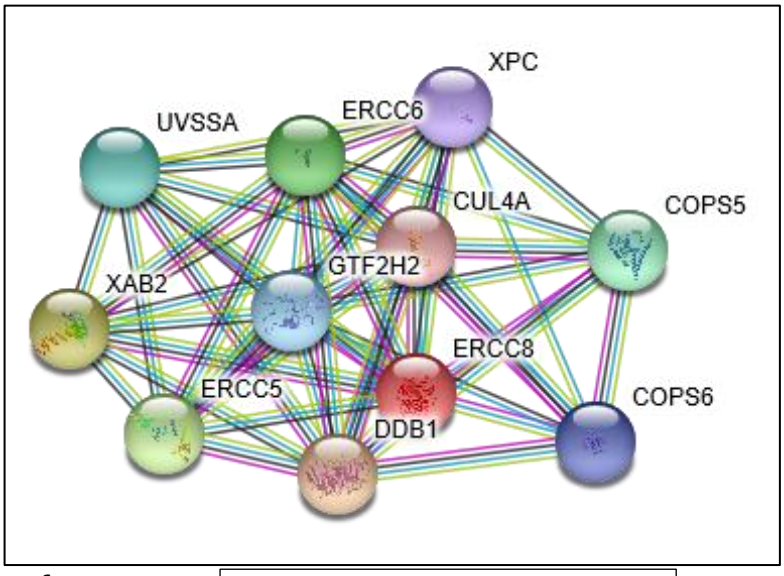



Figure: CSA binding domain

## ICAM-I AND TSP ADHESIVE DOMAIN

ICAM-I, member of the immunoglobulin superfamily, contains five immunoglobulin-like domains and is expressed on a variety of cells including lymphocytes, monocytes, macrophages and human umbilical vein endothelial cells (HUVEC). ICAM-1 expression, up-regulated by several inflammatory cytokines including TNF-a, acts as a receptor for

rhinovirus and the leukocyte integrin LFA-1, and mediates lymphocyte adhesion to ICAM-1. Many parasite strains do not bind to ICAM-1. But by in vitro selection on HUVEC it was possible to produce an ICAM-1-binding line (A4), and using this line the binding site for PE was localized to the first immunoglobulin domain. When PE were selected for ICAM-1 binding there was expression of particular *var* genes that differed from non-ICAM-1-binding lines. Since antibodies to DBLβ from the A4var line blocked adhesion of both A4var and A4tres strains, it was concluded that the DBLβ domain mediates adhesion to ICAM-1. In addition, transiently transfected COS cells expressing a combination of the DBLβ and CIDR2 domains from the A4tres clone bound to ICAM-1; however, in similar transfection experiments, using a cDNA construct from the A4VAR clone, ICAM-1 binding did not occur.

- **PfSET10**

PfSET10 is one of four methyltransferases with predicted H3K4me3 activity, report its unique localization to single nuclear region that colocalizes with the active var expression locus, suggesting a specialized function. The binding specificity of the PfSET10 PHD domain for naked H3 suggest that this methyltransferase binds to a freshly deposited histone within a nucleosome to mark it as the active var. PfSET10 is expressed in trophozoites and schizonts, after peak var mRNA levels, consistent with a role in the maintenance of the "poised" state of the expressed var gene locus.

## STEVOR AND RIFHNS

STEVOR (Subtelomeric Variant Open Reading Frame) was selected from an expression library by MAb7H8; the 1-kb gene is transcribed in both asexual and sexual stages and can be localized in Maurer's clefs. The rifhns (repetitive interspersed family of genes, rif) encode a group of 30- to 45-kDa proteins. The rif genes, of which there may be 200-500 per haploid genome, are expressed at 14-16 h post-invasion (i.e. early trophozoite stage), and although originally referred to as rosettins because they were associated with rosetting lines, subsequent studies have shown they are not always associated with this phenotype and are found in all clinical isolates. Rifhns are trypsin resistant, and antisera raised against the deduced amino acid sequence of rifhns immunoprecipitated proteins of similar molecular size. More recent evidence suggests that rifhns are not exposed on the surface of PE, since antibodies against rifhns can recognize antigens present on permeabilized PE but the same antibodies do not bind to intact infected erythrocytes. Furthermore, mass spectrometric analysis indicates that genes encoding rifhns are preferentially expressed at the sporozoite stage. The precise function of rifhns is unknown.

## CLAG

Some lines of *P. falciparum* maintained in vitro may spontaneously lose their capacity to adhere, and this has been mapped to a deletion on chromosome 9, the region termed clag 9 (cytoadherence linked asexual gene). It is transcribed in mature-stage parasites and translated into a 220-kDa protein distinct from PEMP1. Artificially disrupting this gene by transfection abolished adherence. Its hydrophobicity profile predicts four putative transmembrane domains and suggests that it is membrane associated and presumably exposed on the PE surface. This hypothesis is supported by the fact that antibodies to clag 9 protein inhibited adhesion to CD36 and labelled the PE surface.

## SEQUESTRIN

Sequestrin, a 270-kDa protein, was identified in a knobless cytoadherent line using an anti-idiotype antibody to the anti-CD36 MAb OKM8. It is similar to PEMPI in that it can be surface labelled with radioactive iodine, is PE specific, and of similar molecular size. However, unlike PEMP1 it is conserved among several isolates.

## MODIFIED BAND 3 PROTEIN

Several MAbs prepared against live PE that were found to be reactive only with the surface of knobby PE also had the capacity to block cytoadherence to C32 amelanotic melanoma cells and BB19 human brain microvascular endothelial cells. Using western blotting, immunoprecipitation and peptide of surface radioiodinated PE, it was determined that these MAbs did not recognize a parasite-encoded protein, but instead the neoantigens represented modified forms of the intrinsic red blood cell membrane protein band3, the anion transporter.

## PHOSPHATIDYLSERINE

Phosphatidylserine (PS) is a plasma membrane phospholipid exclusively localized in the inner leaflet of the lipid bilayer. PS molecules are "flipped" to the outer surface of activated platelets, apoptotic cells and red blood cells under some pathological conditions including sickle cell anaemia, thalassemia, and diabetes. PS exposure on red cells has been implicated in abnormal adherence of such red cells to TSP. CD36 was reported to liposomes. PS bind to PS exposure on PE has been controversial and attributed to ATP depletion or mechanical stress during in vitro manipulation of PE. However, PS exposure could be detected even when PE were collected from in vitro without centrifugation of cultures to avoid mechanical stress and in presence of glucose to avoid ATP depletion. Cytoadherence of PE to CD36 and TSP, but not to be inhibited by ICAM-1 and CSA, was annexin V (containing PS-specific probe) and PS liposomes, demonstrating PE adherence to CD36 and TSP.

## MEMBRANE-ASSOCIATED PARASITE PROTEINS

The described knob-associated as an histidine protein, KAHRP, was first 80-kDa protein in knobby lines biosynthetically labelled with radioactive histidine; by microscopy, using an immunoelectron microscopy, and using an antiserum to HRP from the avian malaria *P. lophurae*, the knobs in *P.falciparum*-infected cells were red labelled.

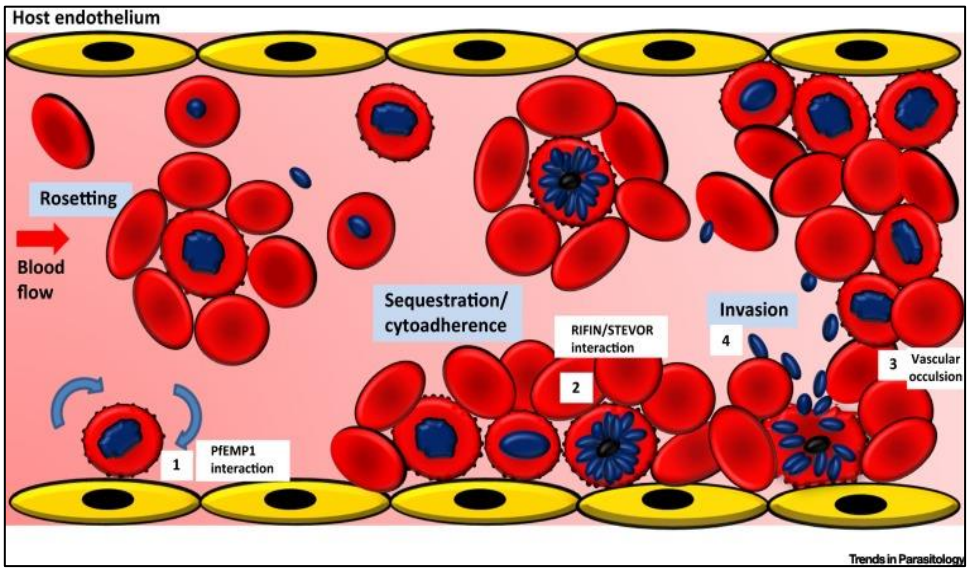
The interaction between KAHRP and PfEMP1 may have an electrostatic component. Indeed, it has been shown that the negatively charged cytoplasmic ATS domain of PfEMP1 interacts with three binding domains of KAHRP, with two mapped to the histidine-rich region and the 5 region, and a third in the carboxyl domain The dissociation constants for the histidine-rich and 5' domains were found to be 0.1 and 3.3 uM, respectively, with the carboxyl terminal having a much lower affinity. Although it is not known whether all the three regions interact with a single PfEMP1 molecule, the fact that the dissociation constant of full-length KAHRP was 10 nM suggests that the intact molecule has a higher affinity and could play a role in clustering of surface adhesions. KAHRP, PfEMP3 play secondary roles such as remodelling of the cytoskeleton, and/or altering the mechanical properties and morphology, thereby changing the exposed surface area for binding, or affecting the trafficking of PfEMP1 to the surface or its anchoring to the membrane skeleton. Although KAHRP is an internal protein, an antibody to it was found to disrupt rosettes; the basis for this effect is unknown, but it may be that sequences in the rifins are cross-reactive with KAHRP.

## TRAFFICKING

Morphologic studies of PE by TEM provide convincing evidence that some parasite-encoded proteins, particularly KAHRP, are exported to the cytoplasmic face of the red cell membrane. However, since the development of the Plasmodium takes place within a cell lacking the machinery for protein transport, it has been hypothesized that there must be a unique set of processes that selectively allows such proteins to traverse the parasite plasma membrane, the parasitophorous vacuolar membrane (PVM) and the erythrocyte cytosol to eventually reach the red cell surface and become exposed.

The transport of secreted plasmodial proteins is proposed to be accomplished by a multistep process: secretion, translocation across the PVM, movement and specific sorting in the red cell cytosol, followed by insertion into the target site. As the parasite matures, KAHRP appears to associate with Maurer's clefts. Since the 5′ region of KAHRP has been shown to bind to the ATS region of PfEMP1 it has been proposed that this enhances and strengthens their interaction. The transient co-localization of KAHRP and PfEMP1 in Maurer's clefts presumes that KAHRP recruitment involves the ATS of PfEMP1, exposed on the surface of Maurer's clefts. Thus, in this model, assembly of the knob structure is postulated to take place in Maurer's clefts before protein is inserted

below the erythrocyte cytoskeleton. Since trafficking of endogenous KAHRP, PfEMP3 and PfEMP1 is brefeldin A sensitive, and these proteins are retained within membrane-bound compartments, it is suggestive of protein export via a classical vesicle-mediated secretory pathway.




## HOST RECEPTORS

- **ENDOTHELIAL RECEPTORS**

### 1. CD36

CD36 is known to bind various ligands including TSP, collagen, PS, long-chain fatty acids, high-density lipoprotein, oxidized low-density lipoprotein, apoptotic cells, sickle red cells, and P. falciparum-infected red cells. CD36 is involved in the phagocytosis of red cells containing gametocyte-stage parasites by macrophages.

### 2. ICAM-1

ICAM-1, a member of the immunoglobulin superfamily, is expressed on several cell types including EC and monocytes. ICAM-1 expressed on EC binds to the lymphocyte function-associated antigen-1 (LFA-1) on lymphocytes and thereby mediates homing and extravasation of the leukocytes.

### 3. TSP

TSP, a 450-kDa trimeric glycoprotein binds to CD36, αIIbβ3/αvβ3 integrin, PS, heparin, histidine-rich glycoprotein, fibrinogen, fibronectin, plasminogen, collagen, LDL receptor-related protein, syndecan-1, CD47, and sulfatide.

### 4. Other receptors

P-selectin was shown to bind PE under in vitro flow conditions, and PfEMP1 was identified as a ligand for this protein.

PECAM (CD31) and αvβ3 integrin were reported to bind PE [23]; however, the expression of the receptors on the luminal surface of EC at the site of sequestration has not been confirmed.

## Receptors in PLACENTAL MALARIA

A severe form of malaria has been found in pregnant women living in holoendemic areas of the disease. Since such patients harbour a high burden of malaria parasites in their placenta, this form of malaria is called placental malaria, and the accumulation of parasites in the placenta is believed to contribute to low birth weight, abortion, and maternal mortality. A number of PE were found in the intervillous spaces of the placenta, and the specific binding of PE was observed on the surface of syncytiotrophoblast (cells lining the intervillous space). CSA as a novel receptor for PE in placental malaria, PE binding to CSA was found to be stationary under in vitro physiological flow conditions, as is the case with CD36-mediated PE binding. A CSA-containing glycosaminoglycan, thrombomodulin, was suggested to be a receptor on the syncytiotrophoblast.

Women at the first pregnancy face a higher risk of severe malaria, even if they have already experienced malaria infections, but become less susceptible to the infection after the second pregnancy. In vitro and field studies on CSA-mediated binding of PE showed that (1) PE isolated from the placenta bound to CSA but not to CD36, whereas peripheral PE from non-pregnant women showed an opposite binding specificity and (2) sera from women who survived the first pregnancy developed antibodies that agglutinated PE or inhibited PE binding to CSA. These findings suggest that CSA- and CD36-binding parasites express different surface antigens for receptor binding.

## ADHESINS, MECHANISMS OF CYTOADHERENCE

There is cooperation and redundancy in PE adhesins and EC receptors, that binding occurs through the sequential action of ligands, and the interactions between PE and the cells to which they bind are dynamic under both static and flow conditions. PE binding need not be dependent on an observable structural change, i.e. knob, but when such modifications do occur, adhesins are clustered and binding strengthened.

In the first step the electrostatic repulsion separating the cells must be overcome. Completion of the first step would provide for a relatively weak and reversible binding but would not allow for tissue specificity. The second step would involve a set of adhesins

that would allow for a more stable and functionally reversible attachment, and with involvement of a third set of adhesins cell binding would be stronger, essentially irreversible, and result in tissue-specific tropism. If any of these steps is missing or blocked, stable binding would not occur.

Step 1. Change in surface charge of the PE results from a depletion of sialic acid residues, adsorption of serum proteins, naturally occurring antibodies, and exofacial exposure of PS.

Step 2. Stable and functionally reversible attachment of PE occurs via alterations in band 3 protein, exposure of PfEMP1, and binding to TSP and HA.

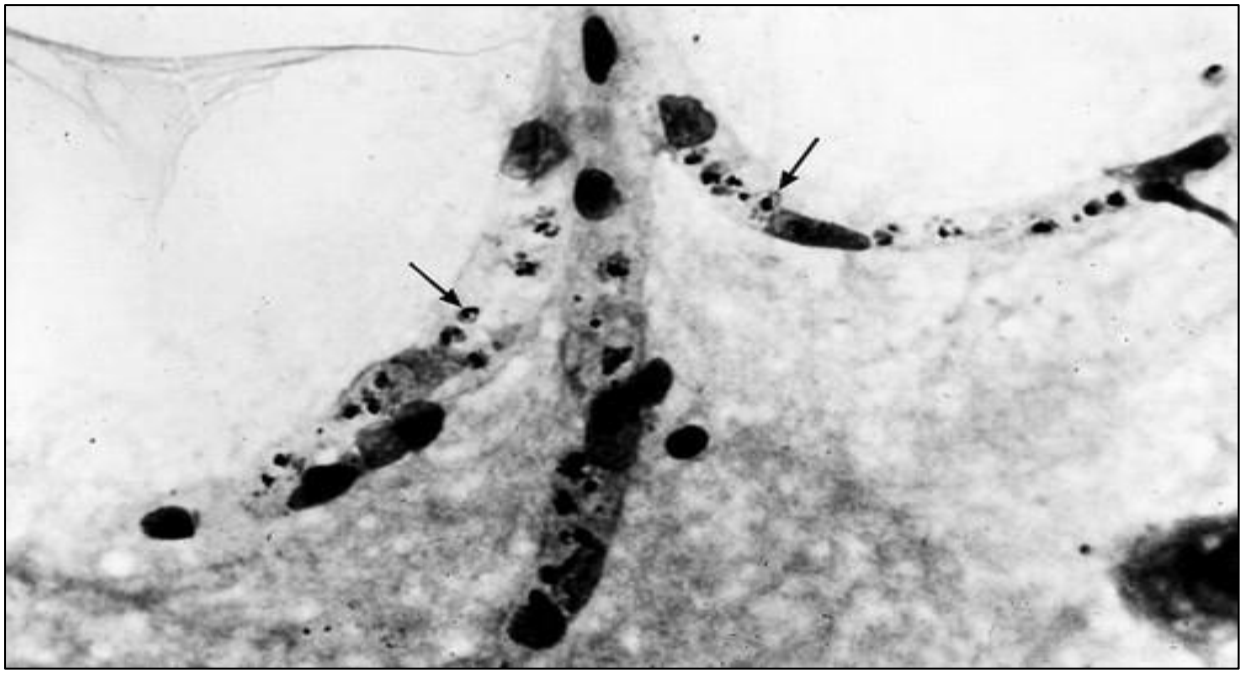
Step 3. Tissue tropism results from EC activation and binding of PE via modified band 3 and PfEMP1 to EC ligands such as CD36, ICAM-1, CSA, and IgM.

## WHY CYTOADHERENCE?

Infected erythrocytes containing the more mature stages of the human malaria Plasmodium falciparum may adhere to endothelial cells and uninfected red cells. These phenomena, called **sequestration** and **rosetting**, respectively, are involved in both host pathogenesis and parasite survival.

This review provides a critical summary of recent advances in the characterization of the molecules of the infected red blood cell involved in adhesion, i.e. parasite-encoded molecules (**PfEMP1, MESA, rifins, stevor, clag 9, histidine-rich protein**), a modified host membrane protein (band 3) and exofacial exposure of phosphatidylserine, as well as receptors on the endothelium, i.e. thrombospondin, CD36, ICAM-1 (intercellular adhesion molecule), and chondroitin sulphate. Deep tissue sequestration of PE containing the trophozoite and schizont stages is generally believed to favour the survival of the parasite by preventing PE passage through the spleen, where they would be recognized as abnormal, and removed. Sequestration in microvasculature places the PE in a parasite-favouring microaerophilic environment thereby promoting more rapid asexual multiplication.

The adhesion of uninfected red cells to an infected red cell, PE are shielded from destruction by the immune mechanisms of the host. Sequestration and rosetting, under these scenarios, would favour parasite growth and reproduction, but would have untoward consequences for the host, resulting in microvessel occlusion, tissue infarction and cerebral malaria. Another hypothesis that has been advanced is that cytoadherence may have developed in order to ensure the survival of the gametocyte and only by chance has it become a virulence feature of the asexual parasite.



**PHOTOMICROGRAPH: cerebral venules packed with infected erythrocytes(arrows) in a fatal case of cerebral malaria.**

Finally, adhesive surface antigens may result in immunomodulation by inhibiting the maturation and activation of dendritic cells, there by interfering with antigen presentation.

The pathogenicity of *P. falciparum* results from its potential to multiply to high parasite burdens and the unique ability to adhere to capillary and postcapillary venular endothelium during the second half of the 48-h life cycle, a process that is called cytoadherence. The resulting sequestration of infected erythrocytes (IRBC) leads to alterations in microcirculatory blood flow, metabolic dysfunction, and, as a consequence, many of the manifestations of severe falciparum malaria.

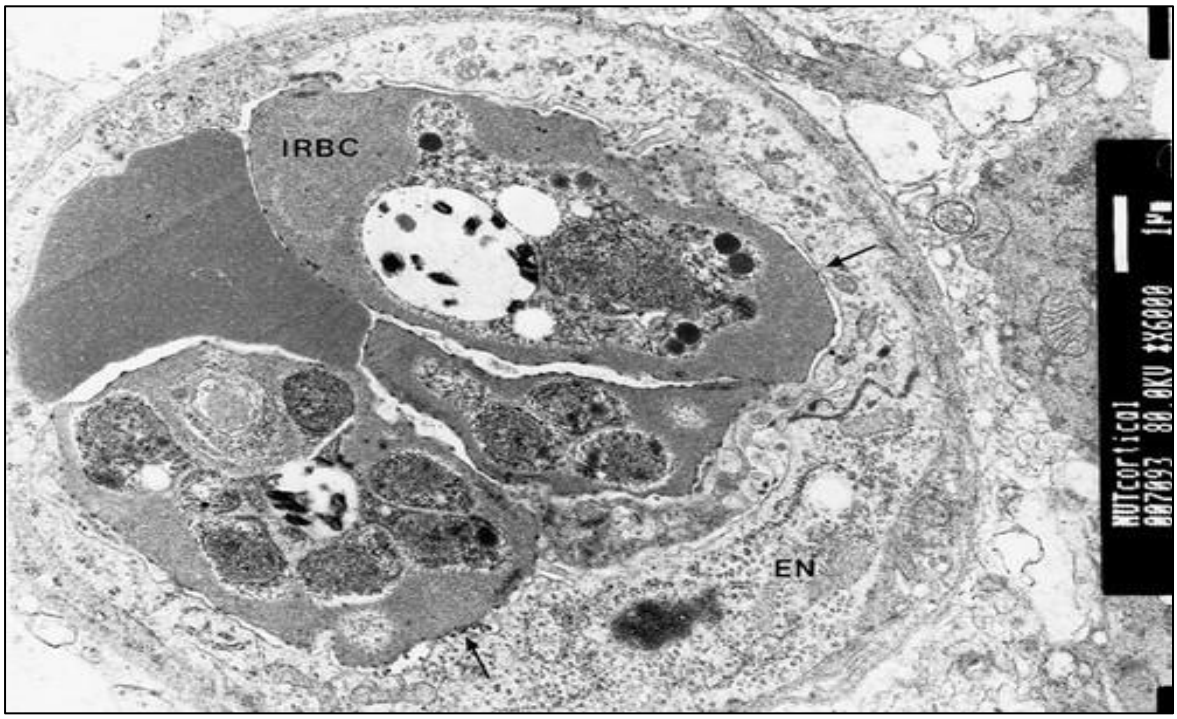
# Advantages of cytoadherence for the parasites:

## Cytoadherence confers at least two survival advantages for the parasites:

The microaerophilic venous environment is better suited for their maturation, and adhesion to endothelium allows them to escape clearance by the spleen, which recognizes their loss of deformability and opsonization. As a result of cytoadherence, patients who die in the acute phase of falciparum malaria have intense sequestration of erythrocytes containing mature forms of the parasite in the microvasculature of vital organs. The organ distribution of sequestration varies and tends to reflect the clinical features of the preceding clinical illness; for example, patients with coma (cerebral malaria) show

increased cerebral sequestration compared with that in other organs. Even within the brain, variation in the degree of sequestration is seen between cerebral and cerebellar vessels, and white and gray matter. At the microvascular level, there is considerable heterogeneity among the individual vessels (N. J. White and K. Silamut, unpublished observations). Some are packed with IRBC containing fully developed schizonts, others with mature trophozoites that have yet to undergo schizogony, and others contain no parasites. This synchronous clustering suggests that, once the erythrocytes have adhered, "unsticking" and recirculation do not occur.

The majority of parasites in fatal cases are at the mature trophozoite stage, but this probably represents antemortem drug effects that preferentially arrest at the ultrastructural level, electron-dense, knoblike protrusions of the erythrocytic membrane are seen at the points of contact between the IRBC and endothelial cells. These knobs are made up of parasite-encoded proteins that have been exported to the surface of the infected erythrocyte. They are essential for firm cytoadherence by facilitating the initial attachment of the infected erythrocyte to the endothelial cell and by concentrating the parasite ligands at a particular site. Although knobless IRBC can adhere to target cells in static binding assays in vitro, ultrastructural studies of human tissues from fatal malaria cases have not shown cytoadherence independent of knobs.




**Photomicrograph: Cytoadherence between infected erythrocytes (lRBC) and endothelial cells showing knobs(arrow) at the point of attachment.**

Furthermore, targeted disruption of the gene encoding one of the knob proteins, knob-associated histidine-rich protein (KAHRP or PfHRP1), results in the failure of knob formation and an inability of the IRBC to adhere under flow conditions in vitro. Thus, these knobs appear to serve the same function as microvilli on the surface of leukocytes, where ligands for interaction with endothelium are presented. Parasitism would continuously favour the selection of knob-positive organisms able to form a more stable union with host cells and thus to allow the parasites to evade splenic clearance, thereby increasing the probability of survival and transmission development at this stage.

In the acute phase of cerebral malaria there is remarkably little extravascular pathology and, occasional fibrin strands may be seen. Inflammatory cells are more prominent in patients who die many days after starting treatment and in whom parasites have largely cleared, and phagocytic cells are seen to be ingesting parasite pigment that has been released by ruptured IRBC or that remains in cytoadherent erythrocyte ghosts. There is no pathological evidence of acute vasculitis.

## CONCLUSION

In human *P. falciparum* malaria infection, IRBC either sequester or are removed from the circulation primarily by the spleen. The balance between splenic clearance and sequestration, which allows the parasite to survive to initiate a new life cycle, is a major determinant of the rate of increase and magnitude of the infecting parasite burden. Within this paradigm, pathogenicity is proportional to the size of the sequestered parasite burden and the pattern of vital organ sequestration. In the past decade, detailed molecular studies have provided exciting new insight into the process of cytoadherence. The next challenge lies in translating the advances in our understanding of pathogenesis into improved treatment for the many millions who are affected by falciparum malaria.

## Probable questions:

1. . Give the structure and diversity of PfEMPl. Mention it's importance in cerebral malaria. What ae the different types of *var genes*?
2. "var promoter" is sufficient for epigenetic silencing and mono-allelic transcription -Explain. Differentiate host specificity and host susceptibility.

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# Unit-XIII
# Molecular adaptation of antigenic variation in *Plasmodium*

## Objectives:

In this section we will discuss on molecular adaptation of antigenic variation in *Plasmodium*.

## Introduction:

Antigenic variation is a key survival strategy employed by a wide range of infectious organisms, allowing them to colonize and persist in a vertebrate host in the face of an evolving immune response. This subsequent chronic infection of the host maximizes the opportunity for these infectious organisms to be transmitted to a new host. Variant surface antigens (VSA) expressed on the surface of malaria-infected erythrocytes (IE) are targets of antibody-mediated immune responses, resulting in clearance of the recognized parasite population. Clonal switches in the VSA expressed by a subpopulation of parasites allow them to survive the mounting immune response against the previous variant, thus ensuring the chronicity of infection.

VSA of the parasite responsible for the most severe form of human malaria, *Plasmodium falciparum*, also contribute significantly to the pathology of disease. A strain-specific component of *P. falciparum* IE surface antigens was shown during the 1980s to be a family of large polymorphic proteins, termed *P. falciparum* erythrocyte membrane protein 1 (PfEMP1). Presence of VSA on the IE surface was shown to correlate with the ability of IE to adhere to host cells, with many studies since showing that PfEMP1 specifically mediates IE adhesion to a variety of different host ligands. Adhesion to microvascular endothelial cells results in the sequestration of IE within major organs which, among other complications, can lead to reduced blood perfusion through occlusion of the microvasculature. Specific host adhesion properties of PfEMP1 have been associated with severe disease pathologies. For example, adhesion by PfEMP1 to intercellular adhesion molecule 1 (ICAM-1; CD54) expressed on endothelial cells in the brain may play a role in cerebral malaria. Similarly, adhesion of IE to complement receptor 1 (CR1; CD35) on uninfected red blood cells leads to the formation of rosettes, a phenotype associated with severe disease outcome. Binding to chondroitin sulfate A (CSA) expressed on syncytiotrophoblasts in the placenta is a factor contributing to pregnancy associated malaria. Furthermore, PfEMP1-mediated adhesion of IE to dendritic cells modulates the activity of these important professional antigen-presenting cells. As PfEMP1 forms the target of protective immune responses, with acquired immunity developing following repeated infection with IE expressing different PfEMP1

variant types, PfEMP1 is thus central to both the pathogenesis of falciparum malaria and the induction of protective immunity.

The discovery in the mid-1990s of the gene family that encodes PfEMP1, termed *var*, opened up the potential for the molecular investigation of this molecule. More recently, the completion of the *P. falciparum* genome project has revealed the organization and distribution of the entire complement of the var gene family.

## Genomic Distribution of *var* Genes

PfEMP1 is a collective term for a family of variant surface antigens encoded by approximately 60 *var* genes per haploid genome. *var* genes are principally located near the sub-telomeric repetitive sequence elements of *P. falciparum*'s 14 chromosomes, along with members of two further multigene families, *stevor* and *rif* (Fig. 1A). However, more than a third of the *var* gene repertoire is located within chromosome-internal regions. Both the sub-telomeric and centrally located *var* gene variants contribute to antigenic variation. As *var* genes can be activated in situ, what role does this pattern of chromosomal distribution play? High meiotic and/or mitotic recombination rates within the more plastic sub-telomeric regions may serve as a mechanism to diversify the antigenic repertoire or perhaps assist gene regulation through physical clustering of chromosome ends at the nuclear periphery. Additionally, it has been proposed that the *var* genes located within the relatively stable internal chromosomal regions may serve as a library of basic *var* sequence information. However, the few highly conserved *var* genes identified to date lie within the sub-telomeric domains. Certainly, the significance of distinct chromosomal compartments for *var* genes remains unresolved.

## *var* Gene Structure within the Genomic Context

Characterization of *var* gene coding sequences is difficult due to their extreme diversity, but there are certain key conserved features in var gene structure. The first exon is highly variable in sequence and length (3.5 to 9.0 kb) and encodes the immunologically exposed portion of PfEMP1, as well as the transmembrane domain (Fig. 1B). The second exon is relatively short (1.0 to 1.5 kb) and highly conserved and encodes the cytoplasmic tail which anchors PfEMP1 to the IE cytoskeleton through noncovalent interactions with a number of parasite-encoded and host proteins. Exon 1 encodes between two and seven Duffy-binding-like (DBL) domains and, in most cases, at least one cysteine-rich interdomain region (CIDR). In striking contrast to exon 1 sequences, the noncoding regions 5 of *var* genes fit into highly conserved sequence groups which are specific to particular genomic locations. Analysis of the entire 3D7 genome sequence confirmed that most *var* gene upstream sequences belong to one of three groups: *upsA*, *upsB*, or *upsC*. *upsA var* genes are always sub-telomeric, *upsC var* genes are always chromosome internal, and *upsB* var genes can be in either location (Fig. 1A). Additionally,

at chromosome ends, the transcriptional orientation of *upsA var* genes (if present) is always towards the telomere, whereas *upsB var* genes are transcribed away from it. Chromosome-internal *upsB* and *upsC var* genes are usually organized in head-to-tail clusters, with no other obvious bias in orientation. Single copies of two further 5′ sequence types, *upsD* and *upsE*, are associated with two unusual conserved *var* genes which are sub-telomeric and transcribed towards the telomere (Fig.1A). These general rules in *var* promoter orientation seem to apply to other *P. falciparum* isolates, allowing

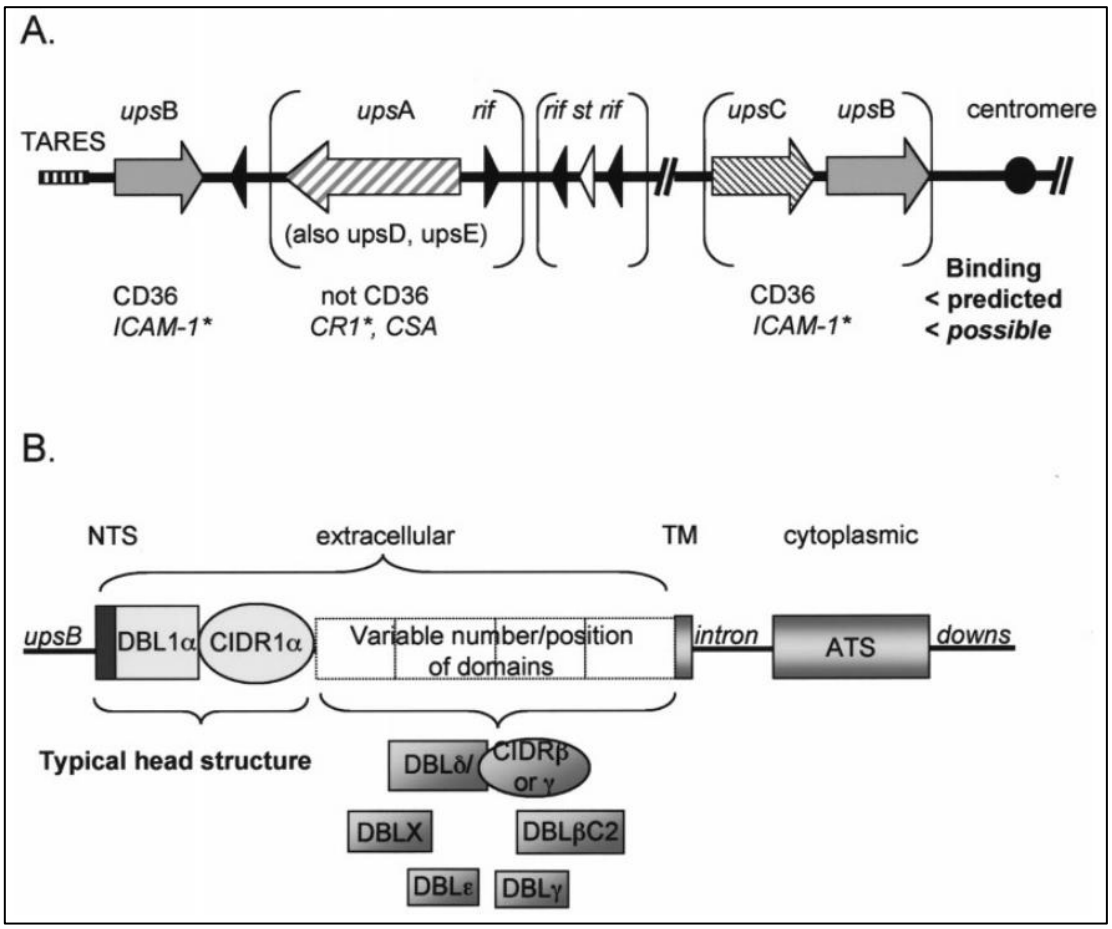


Figure 1: Chromosomal distribution and orientation of the *var* multigene family. *var* genes are indicated as block arrows with the upstream sequence type (*ups*) indicated using letters A, B, and C.

prediction of genomic location on the basis of 5′ noncoding sequence. Introns and sequences downstream of *var* genes exhibit a less-pronounced degree of conservation. Only those *var* genes with *upsA* type 5′ noncoding regions have similar 3′ noncoding sequences. Intron sequences vary greatly in length, ranging from ~150 bp up to 1.2 kbp. Although some introns can be divided into three regions of broad sequence conservation, any association of particular intron sequence types with chromosomal location remains to be tested. The conservation in upstream sequences, in terms of both sequence and organization, may indicate some evolutionary pressure that restricts recombination

between limited subsets of *var* genes. The identification of conserved *var* gene types, all located at chromosome ends and transcribed towards the telomere, supports this speculation (Fig. 1A). Comparison of these genes in unrelated isolates shows that they share an identical organization of DBL domains, each with a reasonably high degree of peptide sequence conservation (dependent on DBL type). These "conserved" *var* genes are *var1CSA*, *var2CSA*, type 3 *var*, and *var4*. Two of these genes have unique upstream sequences, *upsD* (*var2CSA*) and *upsE* (*var1CSA*), the other two being *upsA* types (Fig. 1B). Given the instability of sub-telomeric regions, there must be strong positive selection to maintain conservation within these var coding sequences, as well as their upstream sequences.

## Control of *var* Gene Expression

Clonal switching of *var* gene expression during chronic infection reflects the sum of several molecular processes, including temporal regulation of *var* gene expression during intraerythrocytic development, mutually exclusive expression of only one *var* variant per IE, and the ability to switch *var* expression in progeny parasites. Of necessity, we will consider these regulatory features separately, although a central role for transcriptional control suggests that these regulatory processes may actually be integrated.

PfEMP1 expression is developmentally regulated, first appearing at the IE surface some 16 to 18 h postinvasion (HPI) in late-ring stages, when IE cytoadherent phenotypes are first detected. Investigation of the control of *var* gene transcription has typically been carried out on long-term in vitro-adapted cultures that are highly selected in order to be homogeneous in their adhesion phenotype. Different experimental approaches taken in the molecular investigation of temporal *var* transcription patterns have led to two possible models of control.

Northern blot analysis indicates that the major *var* mRNA transcript encoding the PfEMP1 variant destined for the IE surface is present at its highest relative level in mid-ring-stage parasites, declining at steady-state level until it is barely detectable in pigmented trophozoites. Other *var* transcript types are undetectable by Northern blot analysis of ring stages from these highly selected parasites. The difference in timing of *var* transcript accumulation and PfEMP1 expression on the IE surface presumably reflects the time necessary for PfEMP1 to be translated and exported from the intracellular parasite to the IE surface. Together, these data suggest that temporal control of var gene expression is regulated at either the level of transcription initiation or a stage-specific targeted posttranscriptional degradation of mRNA. By contrast, studies using reverse transcription-PCR (RT-PCR) suggest that many var genes are transcribed in ring-stage parasites but that only one major *var* gene transcript is present in pigmented trophozoite stages. These data would imply a posttranscriptional mechanism for the control of temporal gene expression. Abortive or incomplete transcription has been invoked as a possible molecular control mechanism, since numerous ring-stage

transcripts are not detected by Northern blotting, although a report has described multiple spliced *var* transcript types in pigmented trophozoites.

How can we reconcile these conflicting data? A partial explanation may lie in the recent discovery that one *var* gene has an unusual temporal regulation: it is transcribed in pigmented trophozoite stages of most parasites regardless of their adhesive phenotype. Similarly, but equally important, the level of transcript detected by either experimental approach has to be meaningful in the context of the overall population or individual cell phenotype. For example, RT-PCR detects not only multiple *var* transcripts but also sporozoite specific transcripts in asexual stages. This suggests that RT-PCR may readily pick up background noise, present in either slightly heterogeneous parasite populations or transcripts present at extremely low levels, which are then interpreted as the real signal. By contrast, Northern blots, which detect only full-length RNA transcripts in large amounts, lack the sensitivity of detection necessary to investigate more subtle underlying mechanisms. The best investigative approach, described as yet only for the var gene exhibiting unusual temporal regulation and is therefore not representative, relies on nuclear run-on transcription studies that describe where and when the RNA polymerases are active over the *var* repertoire. Unlike antigenic variation in African trypanosomes where genes encoding variant surface glycoproteins can be activated through duplicative transposition into a limited number of specific expression sites, *var* genes are activated in situ. While it would appear that most members of the *var* repertoire are equally capable of transcriptional activation at any time, only one is expressed in any IE. The expression of only one *var* gene variant while the rest of the repertoire remains silent is termed mutually exclusive expression.

So, the key question is, how is only one *var* gene variant expressed while the rest of the repertoire is silent? Recent work characterizing a cooperative interaction between two regulatory regions within each *var* locus provides important clues to how *var* genes are silenced. Each *var* gene appears to have two promoters or, more specifically, two start sites of transcription. The first, approximately 1 kb from the start of exon I, presumably gives rise to the functional mRNA present in ring-stage parasites that is ultimately translated into PfEMP1. The second promoter is found within a relatively conserved sequence of the *var* intron and drives transcription of abundant, noncoding “sterile” RNA molecules that accumulate within pigmented trophozoite-stage IE. While a single *var* gene variant is apparently transcribed from its upstream promoter and the remainder of the *var* repertoire is silent, or at least not producing full-length mRNA, most or all of the intron promoters seem to be active at the same time.

The interaction of these promoters in regulating mutually exclusive expression has been investigated using transfection technology. The cooperative nature of upstream and intronic *var* sequences was first demonstrated when the constitutive activity of a *var* upstream sequence on an episomal plasmid was silenced by the addition of a *var* intron to the plasmid construct. The specificity of this cooperative activity was demonstrated when *var* intronic sequences were unable to silence the transcriptional activity of an unrelated promoter. Interestingly, the ability of the intron to induce silencing was

dependent on transition of the transfected parasites through S phase of the cell cycle. S-phase-dependent silencing is a characteristic of gene regulation based on both chromatin assembly and modification, which are thought to take place simultaneously with DNA replication during S phase. Electrophoretic mobility shift assays have identified a conserved element upstream of *var* genes that is bound by either a protein or protein complex found in nuclear extracts. While this element is likely to be important for regulating *var* gene expression, its exact role remains unclear, although it is noteworthy that this element appears to be bound during S phase of the cell cycle. Together, these data imply that cooperative interaction between sequence elements within the introns and the *var* upstream regions control the transcriptional status of each individual var gene.

*var* introns thus appear to be powerful, S-phase-dependent transcriptional silencing elements that possess an independent promoter activity. Deletions within the intron both disrupt transcriptional activity from this site and eliminate its ability to silence a *var* upstream promoter. The intron appears to exert its silencing effect through its promoter activity, with the sterile transcripts produced presumably affecting the assembly of a silent epigenetic state over the *var* upstream promoter. Such a model is consistent with mechanisms of imprinting and allelic exclusion in other organisms, including autosomal imprinting and X-inactivation in mammals. In these examples, the transcriptional state of the gene is determined by alterations in chromatin structure and the action of noncoding RNAs. Exactly how these RNA molecules affect chromatin assembly is not yet understood in detail, and it is not yet known whether transcriptionally active *var* genes also produce sterile transcripts. Aspects of epigenetic mechanisms that may contribute to regulating gene expression are considered below.

While duplications and rearrangements within the *var* gene family do not appear to play a role in the control of *var* gene switching, some role for deletions has been indicated. Deletions are unlikely, however, to represent a major switching mechanism, since this would result in loss of *var* repertoire. Moreover, the lack of any apparent alterations to DNA methylation or nucleosomal phasing over transcriptionally active *var* genes similarly eliminates them as potential regulatory mechanisms. Since the majority of parasites continue to express the same var gene through multiple cell cycles, this implies that there is some form of epigenetic imprinting or cellular memory that maintains a single *var* gene in the active state from one cell generation to the next, while the remaining *var* gene variants are silenced. Thus, molecular investigations are currently being directed toward understanding how silencing is lifted over the *var* gene variant that is being activated while simultaneously imposing it on the previously active copy.

**Probable questions:**

1. "Antigenic variation is a key survival strategy adopted by pathogens"- Justify.
2. Describe the structure of *var* gene with a labelled diagram.
3. Where do the *var* genes distributed in the genome of a pathogen?
4. Write a short note on the control of *var* gene expression in a pathogen.
5. What is clonal switching?

**Suggested reading:**


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# Unit-XIV
# Host parasite interaction

## Objectives:

In this section we will discuss on host parasite interaction.

## Introduction:

Parasitology has been broadly defined as “a study of symbiosis or literally speaking “living together”. Naturally speaking, parasitology is defined as the scientific study of parasites. What then are parasites? A parasite is defined by the advanced English dictionary as “an animal or plant living in or on another and getting its food from it”.

Taking the broad definition of parasitology into consideration, the word “symbiosis” as used, raises another question. The word itself is taken from Ancient Greek language meaning “living together” i.e., close and long-term interactions between two or more different biological species. In 1877, Albert Bernard Frank used the word symbiosis to describe the mutualistic relationship existing among lichens. This usage conforms to the definition of symbiosis given by the Oxford advanced English dictionary depicting people living together in a community. In 1879, Heinrich Anton de Bary, a German Mycologist defined symbiosis as “living together of unlike organisms”.

The meaning of the word symbiosis has become controversial among Scientists. Whereas some Scientists believe symbiosis should refer to relationships that are beneficial to both parties, (i.e. mutualistic relationships), others believe it should apply to any type of persistent biological interactions. Consequently, four different types of symbiotic relationships have emerged namely:

1. Parasitism
2. Mutualism
3. Commensalism
4. Phoresis

# Types of Symbiosis:

### *Obligate symbiosis*

This is the type of relationship where both symbionts entirely depend on each other for survival. For example, many lichens consist of fungal and synthetic symbionts that cannot live on their own.

### *Facultative symbiosis*

This is the type of relationship where the organisms may not necessarily live with each other in order to survive.

### *Ecosymbiosis*

Here, one organism lives on another eg mistletoe.

### *Endosymbiosis*

This is where one partner or symbiont lives inside the other e.g. lactobaccili and other bacteria on humans or symbiodinium in corals.

### *Conjunctive symbiosis*

This is the type of relationship in which the two organisms have bodily union i.e., attached to each other. If the opposite is the case, it is called disjunctive symbiosis.

## Hosts:

The word host as applied to parasitology has been defined as "an organism infected with or is fed upon by a parasitic or pathogenic organism (eg nematodes, fungi, virus etc). It is also described as an animal or plant that nourishes or supports a parasite. The host does not benefit but instead is harmed by the association.

## Types of Hosts:

### *Definitive or primary host*

An organism in which a parasite reaches sexual maturity e.g., the mosquito is the definitive host for the malaria parasite, *Plasmodium*.

### *Intermediate (alternative or secondary) host*

An organism in which a parasite develops but does not attend sexual maturity e.g., humans and other vertebrate animals are intermediate host for *Plasmodium*.

### *Paratenic host*

A host which may be required for the completion of a parasite's life cycle but in which no development of the parasite takes place e.g., the unhatched eggs of nematodes are sometimes carried in a paratenic host such as a bird or a rodent. When a predator eats the paratenic host, the eggs are ingested and it becomes infected.

### *Accidental host*

One that accidentally habours an organism that is not ordinarily parasitic in the particular species.

### *Dead-end host*

This is the host in which the disease cannot be transmitted to another animal. Any host organism from which a parasite cannot escape to continue its life cycle. E.g., humans are dead end hosts for trichinosis because the larvae encysted in the muscle and human flesh are unlikely to be a source of food for other animals susceptible to the parasite.

### *Predilection host*

Is the host most preferred by the parasite.

### Reservoir host

An animal or species that is infected by a parasite and which serves as a source of infection for humans or other species.

### *Transfer or transport host*

A host which is used until the appropriate host is reached, but is not necessarily to complete the life cycle of the parasite.

## Host-parasite specificity

A parasite can infect one or a limited number of hosts at a given time i.e. most parasites occur on a restricted number of hosts. This gives rise to the concept of specificity. Host specific parasites generally have a major host and then a few less frequently used hosts in the absence of the major one. Even among parasites that do not discriminate among hosts, there is preference for some species hosts above others. It is said that many parasite groups have a drift toward greater host specificity. Host specificity is the characteristic of a parasite that renders it capable of infecting only one or more specific hosts at a time.

### Host-parasite evolution/specificity

Natural selection tends to occur and favour the specialization of parasites to their local environment or hosts. The parasite ecosystem is a world of competition between organisms where there is survival of the fittest. Thus, the most adapted and fitted host or parasite exists in greater abundance than the least fitted. Host specialization is said to be promoted by host-dependent fitness trade-offs which is dependent on the relative availability and predictability of hosts. A parasite should specialize if the advantages of using one single host species in a profitable manner outweigh the benefits of interacting less profitably with several less frequent host species. In other words, lack of adequate hosts will promote parasite generalization, while abundance of hosts will make parasites to specialize to the specific environmental conditions. It is believed that host parasite interactions and thus host specificity take place simultaneously at several “host” levels.

This is probably while such interactions are especially difficult to explain. Studies carried out by Georgi et al. (2001), using ectoparasitic mites, (*Spinturnicidae*) which infest colonial bats, revealed that parasite specificity may be mediated by three main mechanisms:

1. Dispersal capacity of the parasite which depends on the number of hosts it can physically encounter during its life.

2. Host preference.

3. Ability to successfully transmit and establish a population on a new host. Considering the third mechanism, it is said that highly specific parasites are expected to exhibit a higher reproductive success or survival on traditional or native host species than on less closely related ones.

## Parasitism

Parasitism is defined as a relationship in which one of the participants (the parasite), either harms its host or in some sense lives at the expense of the host. Traditionally, a parasite referred primarily to organisms which were visible to the naked eye, otherwise known as macro-parasites (eg helminths), but nowadays, parasites include microscopic organisms such as viruses, and bacteria which are referred to as microparasites. The word "parasite" was derived from a Latin word "parasitus" which means "one who eats at the table of another". Although parasites may inflict harm on their hosts, it is not in the best interest of the parasite to kill its host. A parasite which kills its host has invariably committed "suicide". Some of the ways parasites inflict harm on their hosts include:

- Boring a hole into the host e.g., Schistosomes
- Digging into hosts skin or other tissues e.g., hookworm larvae
- Stimulation of damaging inflammatory or immune response eg microfilariae
- Robbing of the host of nutrients e.g., tapeworm, hookworm
- A combination of two or more of the above conditions

Unlike predators, parasites are usually smaller than their hosts and will often live in or on their hosts for an extended period of time. Both parasitism and predators are special cases of consumer resource interactions.

Parasites display a high degree of specialization and reproduce at a faster rate than their hosts. Examples of parasitism include interactions between vertebrate hosts and diverse animals such as tapeworm, flukes, the plasmodium species and fleas.

## Types of Parasites:

• **Ectoparasites:** Are parasites living on the surface of their hosts e.g., bed bugs, mites, ticks etc.

• **Endoparasites:** Are parasites living within the body of their hosts e.g., Schistosomes, tape worm, *Ascaris* etc.

• **Obligate parasites:** Cannot complete their life cycle without spending at least part of the time in parasitic relationship e.g., *Plasmodium*. However, many obligate parasites have free living forms which can exist outside the host for some period of time in the external environment in a protective egg shell or cyst e.g., hookworm larvae, *Ascaris*, *Entamoeba histolytica*.

• **Facultative parasites:** These are not normally parasitic but become so when they are accidentally eaten or enter a wound or other body orifices e.g., certain free-living amoeba such as *Naegleria fowleri* and free-living Nematodes of the genus *Micronema*. Infection of humans by any of these facultative parasites is always very fatal.

• **Accidental/incidental parasites:** This occurs when a parasite enters or attaches to the body of species of host different from its usual preferred host e.g., Nematodes parasitic in insects can live for a short time in the intestines of a bird or rodent. Fleas can live for a while in dogs or humans. Accidental parasites usually do not survive in the wrong host but in some cases, they can be extremely pathogenic e.g. *Toxicara*, *Baylis ascaris*.

Parasitism usually results from a long history of evolutionary symbiosis between the parasites and the hosts in which both parties are fully adapted. It is no wonder then why accidental parasitism is fatal for both host and the parasites because neither of the two parties is adapted for the co-existence.

• **Permanent parasites:** These are parasites which live their entire adult lives within or on their hosts.

• **Temporary or intermittent parasites:** These feed on their hosts and then leave eg mosquitoes, bed bug. They are also called Micropredators because they also prey on several different hosts or the same host at several discrete times.

• **Mesoparasites:** Are those parasites living in an intermediate position i.e., half ectoparasites and half Endoparasites.

• **Epiparasites:** Are parasites which feed on other parasites. This is sometimes referred to as hyperparasitism e.g., a Protozoan living in the digestive tract of a flea living on a dog.

• **Social parasites:** Are parasites which take advantage of interactions between members of a social group of organisms such as ants or termites e.g., *Phengaris arion*, a butterfly whose larvae employ mimicry to parasitize certain species of ants.

# Types of Parasitism:

## *Kleptoparasitism*

In this type of relationship, parasites appropriate the food gathered by the host eg brood parasitism practiced by many species of cuckoo and cowbird which do not build nests of their own but rather deposit their eggs in nests of other species and abandon them there. The host behaves as a "baby sitter" as they raise the young ones as their own. If the host bird ventures to remove the Cuckoos eggs, some cuckoos will return to attack the nest to compel the host bird to comply with their wish. In the case of the cowbird, the host's brood is not necessarily harmed but this is not so with the cuckoo which may remove one or more of the host's eggs to avoid detection or the young cuckoo may heave the hosts eggs and nestlings out of the nest entirely.

## Intraspecific social parasitism

This may occur in the form of parasitic nursing where some members of the relationship take milk from unrelated females eg in wedge capped capuchins, higher ranking females sometimes take milk from low ranking females without any reciprocation. That is to say high ranking females benefit at the expense of the low-ranking ones.

## Cheating or exploitation

Parasitism can also occur as isolated cheating or exploitation among more generalized mutualistic interactions eg broad classes of plants and fungi exchange carbon and nutrients in common mutualistic mycorrhizal relationships. However, some plant species known as mycohetrotrophs "cheat" by taking carbon from a fungus without donating it.

# Parasitoids:

These are organisms whose larval development takes place inside or on the surface of another organism (the host) leading to the death of the later. This differentiates parasitoids from true parasites which normally do not kill their hosts. Thus, parasitoid relationship is similar to predation where the host is always killed. Parasitism differs from parasitoid relationship in the sense that parasitoids generally kill their hosts. Parasitoidism occurs in a similar variety of organisms to that in which parasitism occurs. A parasite can reduce the host's biological fitness in a variety of ways:

- parasitic castration of the host ie impairment of the hosts secondary sex characteristics
- modification of the hosts behavior

Parasites can also increase their own fitness by exploiting the host for resources necessary for their own survival such as food, water, heat, habitat and transmission.

### Adelpho-parasitism

An adelpho parasite is one in which the host species is closely related to the parasite, often being a member of the same family or genus eg the citrus blackfly parasitoid, *Encarsia perplexa* whose unmated females may lay haploid eggs in the fully developed larvae of their own species. These result in the production of male offsprings. Secondly, the marine worm *Bonellia viridis* has a similar reproductive strategy, although the larvae are planktonic.

### Autoinfection

Is the infection of a primary host with a parasite, particularly a helminth, in such a way that the complete life cycle of the parasite occurs in a single organism without passing through other hosts i.e the primary host is at the same time the secondary host. Examples include *Strongyloides stercoralis*, *Enterobius vermicularis*, *Taenia solium* and *Hymenolepis nana*. *Strongyloides* for example can cause premature transformation of a non-infective larva to infective lava, which can then penetrate the intestinal mucosa (internal autoinfection) or the skin of the perineal area (external autoinfection). Thus, infection can be maintained by repeated migratory cycle for the rest of the person's life.

## Host Defenses against Parasites

The host responds to parasitism in a variety of ways ranging from morphological to the behavioural. Some of these ways include:

1. Toxins: Some plants produce toxins which are antiparasitic to inhibit the growth of parasitic fungi and bacteria.

2. Immune systems: Vertebrate animals develop complex immune systems which fight parasitic organisms to get rid of them. In humans' parasitic immunity involves IgE.

3. Behavioural defenses: For example, sheep avoid open pastures during spring when roundworm eggs are known to accumulate *en masse* over the previous years. Secondly some infected fruit flies ingest alcohol as a form of self-medication against blood borne parasites.

## Evolution of Parasites

Biotrophic parasitism is said to be a common mode of life that has arisen independently many times in the course of evolution. It is also believed that as many as half of all animals have at least one parasitic phase in their life cycles and it is also frequent in plants and fungi. Secondly, almost all free-living animals are hosts to one or more parasitic organisms at one time or another.

Furthermore, parasites have been known to evolve in response to the defense mechanisms of their hosts. As a consequence of their host defenses, some parasites evolve adaptations that are specific to a particular host taxon, specializing to the point where they infect only a single species. Such parasites may pay dearly over time if the host species become extinct. Consequently, many parasites evolve to infect a variety of more or less closely related host species with different success rates.

Host defenses also evolve in response to parasitic attacks. In theory, parasites may have advantage in this evolutionary arms race because parasite generation time is commonly shorter i.e., hosts reproduce less quickly than parasites and therefore have fewer chances to adapt than their parasites do over a given range of time.

In some cases, a parasite may co-evolve with its host taxa. It is said that long term co-evolution may lead to a relatively stable relationship tending towards commensalism or mutualism since it is in the best interest of the parasite that the host remains alife. A parasite may evolve to become less harmful for its host or a host may evolve to cope with the unavoidable presence of a parasite-to the extent that the parasites absence causes the host harm. For example, it is known that animals infected with parasitic worms are often clearly harmed, such infections may also reduce the prevalence and effects of auto immune disorders in animal hosts, humans inclusive.

Competitions between parasites often occur and this tends to favour faster reproducing and hence more virulent parasites. Parasites which kill the host in the course of their life cycle, in order to enter a new host, evolve to be more virulent or even change the behavior or other properties of the host to make it more vulnerable to predators. Parasites that reproduce largely to the offspring of the previous host, tend to become less virulent or mutualist, so that its hosts reproduce more effectively.

The presumption of shared evolutionary history between parasites and hosts can sometimes explain how host taxa are related. For instance, the relationship between flamingos and storks or their relatives and ducks, geese and their relatives has been controversial. It has been said that the fact that flamingos share parasites in common with ducks and geese is evidence or proof that these groups may be more closely related to each other than either is to the storks.

Parasitism has been used to explain the evolution of secondary sex characteristics seen in breeding males throughout the animal kingdom eg the plumage of male peacocks and manes of male lions. According to this theory, female hosts select males for breeding based on such characteristics because they indicate resistance to parasites and other diseases.

## Parasites Adaptations

Parasites are adapted to infect hosts that exist within their same geographical area (sympatric host) more effectively than hosts found outside their own geographical area

(allopatric hosts). This phenomenon is said to support the so called "Red Queen hypothesis" which states that interactions between species (such as hosts and parasites) lead to constant natural selection for adaptation and counter adaptation. Experiments conducted by the later authors, using two snail populations from different sources substantiated the fact that parasites were more infective to sympatric hosts than they were to allopatric hosts i.e. although the allopatric snails were equally infected, by the digenetic Trematodes (parasites), the infectivity was much less when compared to the sympatric snails. Hence the parasites were found to have adapted to infecting local populations of snails.

## Parasitic transmission

Since parasites inhabit living organisms (hosts), they are faced with numerous problems emanating from the host which will mount many forces aimed at repelling or destroying these invaders. Consequently, parasites develop several strategies to evade these host defense mechanisms to ensure their movement from one host to the other. This is referred to as parasitic transmission or colonization. Some endoparasites infect their host by penetrating its external surface (eg hookworm larvae), while others must be ingested in food by the host (e.g. *Entamoeba histolytica*). Once they are inside the host, adult endoparasites (eg tapeworm, *Ascaris*) must shed their offspring to the external environment so as to infest other hosts. Many adult endoparasites live in the host's gastrointestinal tract, where the eggs can be shed along with the hosts excreta or faeces. Examples here include tapeworms, thorny headed worms and most flukes. Some other parasites like malaria parasites (*Plasmodium*) or trypanosomes use insect vectors to transmit their infective stages.

Furthermore, soe larval stages of endoparasites infect sites other than the blood or gastrointestinal tract e.g., muscle tissue. In such cases, larval endoparasites require their hosts to be consumed by the next host (predators) in the parasites life cycle in order to survive and to reproduce. On the alternative, some larval endoparasites may shed free living transmission stages that migrate through the host's tissue into the external environment where they actively search for or await ingestion by other hosts. The above-mentioned strategies are used variously by larval stages of tapeworms, thorny headed worms, flukes and parasitic round worms. Furthermore, some ectoparasites e.g., monogenian worms, depend on direct contact between hosts eg lice. Some ectoparasites may shed eggs which may survive off the host (e.g., fleas) or wait in the external environment for an encounter with a host (e.g., ticks). Some aquatic leeches locate hosts by sensing movements and only attach when certain temperatures and chemical cues are present.

## Host behavior

Some parasites modify hosts behavior to make transmission to other hosts more likely. For instance, in California salt marshes, the fluke *Euhaplorchis californienses* reduces the ability of its killifish host to avoid predators. This parasite matures in egrets which are more likely to feed on infected killifish than on uninfected fish. Another example is the protozoan *Toxoplasma gondii*, a parasite which matures in cats, though it can be carried by other animals. Uninfected rats avoid cat odours, whereas infected rats are attracted to cat odours which causes their being easily devoured and hence transmission.

### Roles of Parasites in the Ecosystem

Although parasites are often omitted in the depiction of food webs, they usually occupy the top position of every food web. Thus, they function like keystone species, thereby reducing the dominance of superior competitors and allowing competing species to co-exist. Many parasites require multiple hosts of different species to complete their life cycles and rely on predator-prey or other ecological interactions to get from one host to another. Thus, the parasite in an ecosystem reflects the health of that system.

## Importance of parasites

- They account for as much as more than half of life's diversity.
- They perform an important ecological role (by weakening prey) that ecosystems would take some time to adapt to.
- Without parasites, organisms may eventually tend to asexual reproduction thereby diminishing the diversity of sexually dimorphic traits.
- They provide an opportunity for the transmission of genetic material between species. On rare occasions, this may facilitate evolutionary changes that would not otherwise occur or taken longer time to occur.

## Mutualism:

Mutualism is the type of relationship where two organisms of different species exist together with each one benefitting. A similar interaction between organisms of the same species is known as cooperation. Mutualism differs from interspecific competition in which each species experiences reduced fitness and exploitation or parasitism where one species benefits at the expense of the other. Mutualism is one aspect of symbiotic relationships. Examples of mutualism include:

- Relationship between ungulates (e.g., Bovines) and bacteria within their intestines. The ungulates benefit from the cellulose produced by the bacteria, which facilitates digestion, while the bacteria benefit from the abundant nutrient present in the host environment.

• Humming bird Hawkmoth and Dianthus. Here, the hawkmoth drinks from the dianthus and in the process helps to bring about pollination.

• The Oxypecker (a kind of bird) and the rhinoceros or zebra. Oxypeckers land on rhinos or zebras and eat ticks or other parasites that live on their skin. The birds get food while the beasts get pest control. Also, when there is danger, the oxypecker fly upward and scream a warning which helps the animal to run away.

• The bee and the flower. Bees fly from flower to flower sucking nectar which serves as food. In the process bees bring about cross pollination which benefits the plant.

• The spider crab and the algae. Spider crabs live in shallow areas of the ocean floor and green brown algae live on the crabs back, thus making the crabs blend in with their environment thereby becoming unnoticeable to predators. The algae get good place to live while the crab gets camouflage.

• Humans and bacteria. A certain kind of bacteria lives in the intestines of man and other animals. The bacteria eat the food humans cannot digest and partially digest it, allowing the human to complete the job. The bacteria benefit by getting food while the human benefits by being able to achieve full digestion.

### Importance of Mutualistic Relationships

1. Mutualistic relationships are important for terrestrial ecosystem function since more than 48% of land plants rely on mycorrhizal relationships with fungi to provide them with inorganic compounds and trace elements.

2. Mutualism is thought to have driven the evolution of much of the biological diversity we see, such as flower forms (which is important for pollination mutualism) and co-evolution between groups of species. Despite its importance in ecology, mutualism has received less attention from Scientist than other relationships such as predation and parasitism.

## Types of Mutualistic Relationships:

Mutualistic relationship has been described as a form of “biological barter” in which species trade resources (eg carbohydrates and inorganic compounds or services, such as gamete, offspring dispersal or protection from predators.

- ***Resource-resource mutualism***

This is probably the most common form of mutualism where one type of resource is traded for a different resource. Examples include:

a) Mycorrhizal association between plant roots and fungi in which the plant provides carbohydrates to the fungus while the later provides inorganic phosphates and nitrogenous compounds.

b) Rhizobia bacteria that fix nitrogen for leguminous plants (family fabaceae) in return for energy containing carbohydrates.

- ***Service-resource relationship***

These are also common. Examples include:

a) The Oxypecker eats ticks on the zebra's skin. Whereas the bird gets food, the zebra gets service of pest control.

b) Pollination in which nectar or pollen (food resource are traded for pollen dispersal (service)

c) Ant protection of aphids where the aphid trade sugar-rich honey dew, a by-product of their mode of feeding on plant sap) in return for defense against predators such as ladybugs.

d) Phagophiles feed (resource) on ectoparasites thereby providing anti pest service as in cleansing symbiosis.

e) *Elacatinus* and *Globiosoma*, genus of globies also feed on ectoparasites of their client while cleaning them.

f) Zoochory-an example where animals disperse the seeds of plants. This is similar to pollination in that the plant produces food resources (eg fleshy fruits, overabundance of seeds) for animals that disperse the seeds (service).

- ***Service-service relationship***

Strict service-service relationships are very rare for reasons which are not clear. Examples of service-to-service relationships include:

a) Relationship between sea anemones and anemone fish in the family Pomecetridae. The anemone provides the fish with protection from predators, while the fish defends the animal against butterfly fish which eats anemones. However, it is believed that there is more to this relationship than service-service mutualism. For instance, waste ammonia from the fish feed the symbiotic algae that are found in the anemone's tentacles. Thus, what appears as service-service relationship has a service-resource component.

b) Relationship between some ants in the genus *Pseudomyrmex* and trees in the genus Acacia such as the Whistling thorn and Bullhorn Acacia. The ant's nest inside the plants thorns thereby obtaining shelter whereas the plant gets protection from attacks by herbivores, which they frequently eat, thereby introducing service service relationship) and competition from other plants by trimming back vegetation that would shade the Acacia. In addition, another service-resource component is obvious since the ants regularly feed on lipid-rich food bodies called Beltian bodies that are found on the Acacia plant.

c) In the neotropics, the ant, *Myrmelachista* Schumani builds its nest in special cavities in Duroia hirsute. Plants in the vicinity that belong to other species are killed with

formic acid. This selective gardening can be so aggressive that small areas of the rain forest are dominated by Duroia hirsute. These peculiar perches are known by the local people as “devils gardens”.

d) *Cordia* species trees in the Amazonian rain forest have a kind of partnership with *Allomerus* species ants, which make their nests in modified leaves. The ants often destroy the trees flowerbud to make more living space available. As the flowers die, more leaves develop and take their place, thus creating more room for the ants.

e) Another type of *Allomerus* species ants live with the *Hirtella* sp tree in the same forest; but unlike in the former relationship, when the tree wants to make flowers, the leaves harbouring the ants dwellings begin to wither and shrink, thus forcing the ants to flea thereby leaving the trees flowers to flourish free from ants’ attack.

## Humans and Mutualism

Mutualistic relationships between humans and other species abound in life:

a) Humans and gut flora: The gut flora helps man to digest food efficiently.

b) Head lice and Man: It is apparent that head lice confer some immunity to man thereby helping to reduce the threat from body louse-borne lethal diseases.

c) Humans and domesticated animals: Dogs and sheep were among the first animals to be domesticated by man and they are beneficial to him.

d) Man, and some agricultural varieties of maize: The later are unable to reproduce without human intervention. First the leafy sheath does not fall open and secondly, the seed head (the “corn on the cob”) does not shatter to disperse the seeds naturally unless man intervenes.

e) In traditional agriculture, some plants have mutualists as companion plants, providing each other with shelter, soil fertility and or natural pest control. For example, beans may grow up corn stalks as trellis, while fixing nitrogen in the soil for the corn. This phenomenon is applied in the Three Sisters farming.

f) The Boran people of Ethiopia and Kenya traditionally use a whistle to call the honey guide bird. If the latter is hungry, it usually guides them to a bee’s nest where they (Boran) harvest the honey leaving some for the birds to eat.

g) In Laguna Brazil, a population of bottle nose dolphins communicates through body language with local net using fishermen in order for both to catch schools of mullet.

## Commensalism and Phoresis

Commensalism simply means “eating at the same table”. It is a type of symbiotic relationship where one partner benefits whereas the second partner (the host) is neither

helped nor harmed. Commensal relationships mainly involve feeding on food "wasted" or otherwise not consumed by the host. Examples of commensalism include:

a) Remora sharks and Whales: The remora sharks have adhesive disk on the dorsal surface of their head which they use to attach to larger animals such as whales which tend to be sloppy eaters. When food floats away from the whale's mouth, the remora shark can unhitch itself and collect the scraps of food from the host.

b) Barnacles and Whales: Barnacles are crustaceans whose adults are sedentary. The motile larvae find a suitable surface and then undergo metamorphosis to the sedentary form. The barnacles adhere to the skin of a whale or shell of a mollusc and are transported to areas with new sources of food.

c) The titan triggerfish (*Balistoides viridescens*) and smaller fish: The former fish creates feeding opportunities for smaller by moving large rocks which are too big for the smaller fish to shift.

d) Humans and prostistans: Humans harbour several species of commensal protistans such as *Entamoeba gingivalis* which lives in the mouth where it feeds on bacteria, food particles and dead epithelial cells but never harm healthy tissues. Adult tape worms though generally regarded as parasites may not have known ill effects on their hosts.

## Types of Commensalism:

### *Facultative commensalism*

This is a situation where the commensal may not necessarily participate in the relationship to live e.g., stalked ciliates of the genus *Verticella* are frequently found on small crustaceans but they can survive equally on sticks on the same pond.

### *Obligate commensalism*

This is a situation where the commensals necessarily need each other to survive e.g., some related ciliates such as *Epistylis* spp cannot survive without the presence of other organisms especially crustaceans.

## Phoresis:

This is the relationship in which two organisms are simply "travelling together" and there is no physiological or biochemical dependence on the part of each participant. The two organisms are known as phoronts. Usually, the smaller organism is usually carried by the larger organism (the host). Examples of phoresic relationship include:

a) Bacteria on the hairs of a fly

b) Fungus spores on the feet of beetle

c) Mites on insects such as beetles, flies or bees.

d) Pseudo scorpions on mammals

e) Millipedes on birds.

f) The Dermatollia hominis larvae usually live beneath the skin of warm-blooded animals including man. The eggs are usually carried by other insects such as mosquitoes and are deposited on the host's skin as the mosquito perches to feed. The eggs quickly hatch and the larvae burrow their way into the skin. Like commensalism, phoresis can be facultative or obligate depending on the existing environmental conditions.

# Other Relationships

## *Inquilinism*

This is a type of relationship where one organism uses the other as a permanent housing or place of abode. Examples include:

a) Epiphytic plants (e.g., Orchids) that grow on trees.

b) Birds that live in holes in trees.

## *Metabiosis*

This is a relationship in which one organism creates or prepares a suitable environment for the other. Examples include:

a) Maggots which feast and develop in corpses.

b) Hermit crabs which use gastropod shells to protect their bodies.

## *Amensalism*

This is the type of relationship that exists where one species is inhibited or completely obliterated and the other is not affected. This type of relationship is common in the natural world. An example is a sapling growing under the shadow of a mature tree. The mature tree usually robs the sapling of necessary sunlight and other nutrient (e.g., rain water). The mature tree remains unaffected while the sapling dwindles and dies. The mature tree will even make use of nutrients arising from the decaying sapling.

## *Synnecrosis*

This is a rare type of symbiosis in which the interaction between species is detrimental to both organisms involved. It is a temporal condition since the interaction will eventually lead to death of the two partners. Consequently, evolution selects against synnecrosis hence it is uncommon in life and the term is rarely used.

## Probable questions:

1. Define parasitism.
2. Explain the different types of symbiotic relationship between two organisms.
3. State the different types of hosts citing example in each case.
4. What are the different mechanisms by which parasite specificity may be achieved?
5. What do you mean by parasitoid? Give example.
6. Explain parasite host co-evolution.
7. What role does parasite play in an ecosystem?
8. What are the types of mutualistic relationship?
9. Differentiate commensalism and amensalism.
10. What is phoresis?

## Suggested reading:


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