PREFACE

With its grounding in the "guiding pillars of Access, Equity, Equality, Affordability and Accountability," the New Education Policy (NEP 2020) envisions flexible curricular structures and creative combinations for studies across disciplines. Accordingly, the UGC has revised the CBCS with a new Curriculum and Credit Framework for Undergraduate Programmes (CCFUP) to further empower the flexible choice based credit system with a multidisciplinary approach and multiple/ lateral entry-exit options. It is held that this entire exercise shall leverage the potential of higher education in three-fold ways– learner's personal enlightenment; her/his constructive public engagement; productive social contribution. Cumulatively therefore, all academic endeavours taken up under the NEP 2020 framework are aimed at synergising individual attainments towards the enhancement of our national goals.

In this epochal moment of a paradigmatic transformation in the higher education scenario, the role of an Open University is crucial, not just in terms of improving the Gross Enrolment Ratio (GER) but also in upholding the qualitative parameters. It is time to acknowledge that the implementation of the National Higher Education Qualifications Framework (NHEQF) National Credit Framework (NCrF) and its syncing with the National Skills Qualification Framework (NSQF) are best optimised in the arena of Open and Distance Learning that is truly seamless in its horizons. As one of the largest Open Universities in Eastern India that has been accredited with 'A' grade by NAAC in 2021, has ranked second among Open Universities in the NIRF in 2024, and attained the much required UGC 12B status, Netaji Subhas Open University is committed to both quantity and quality in its mission to spread higher education. It was therefore imperative upon us to embrace NEP 2020, bring in dynamic revisions to our Undergraduate syllabi, and formulate these Self Learning Materials anew. Our new offering is synchronised with the CCFUP in integrating domain specific knowledge with multidisciplinary fields, honing of skills that are relevant to each domain, enhancement of abilities, and of course deep-diving into Indian Knowledge Systems.

Self Learning Materials (SLM's) are the mainstay of Student Support Services (SSS) of an Open University. It is with a futuristic thought that we now offer our learners the choice of print or e-slm's. From our mandate of offering quality higher education in the mother tongue, and from the logistic viewpoint of balancing scholastic needs, we strive to bring out learning materials in Bengali and English. All our faculty members are constantly engaged in this academic exercise that combines subject specific academic research with educational pedagogy.We are privileged in that the expertise of academics across institutions on a national level also comes together to augment our own faculty strength in developing these learning materials. We look forward to proactive feedback from all stakeholders whose participatory zeal in the teaching-learning process based on these study materials will enable us to only get better. On the whole it has been a very challenging task, and I congratulate everyone in the preparation of these SLM's.

I wish the venture all success.

Professor Indrajit Lahiri
Vice Chancellor

Netaji Subhas Open University

Four Year Undergraduate Degree Programme

Under National Higher Education Qualifications Framework (NHEQF) & Curriculum and Credit Framework for Undergraduate Programmes

Course Type: Discipline Specific Elective (DSE)

Course Title: Medical and Forensic Entomology

Course Code: NSE-ZO-03

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UG Zoology

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Netaji Subhas
Open University

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Unit-1 □ Introduction-Impact of arthropods on human health

Arthropods and humans have been interacting for centuries. Disease-producing organisms transmitted to humans by arthropods have significantly influenced the history and demography of the human race. Among arthropods, different group of insects play a major role in transmission of diseases. Mosquitoes are the most important vector of some lethal diseases like, malaria, filariasis, yellow fever, dengue etc. Flies, like Sandflies, Tsetse flies, House flies act as vectors and also as carrier of many severe diseases. Fleas, ticks, mites and lice, among other arthropods are responsible for causing many viral and bacterial borne diseases in human. All these arthropods pose a huge threat to the public health and may cause explosive epidemics affecting millions of people.

Unit-2 □ Concept of vectors-Mechanical vectors and biological vectors; General ideas of vector-borne disease and their control

Biologically a vector is an organism that transmits a disease-causing agent from one organism to another. There are two types of relationship are established in between the vector and the organisms parasitic to human during transmission. The simplest relationship is one in which the arthropod is a **mechanical vector**, functioning merely as a passive carrier of the disease agent. Example of this type are typhoid organism and cyst of *Entamoeba histolytica* from contaminated excreta, those adhere to the body parts of the vector insects and subsequently transferred to food or drink touched by the insects.

Another type, the **biological vectors**, are the arthropods which are used by the parasitic agents not only by the vehicles of transmission but as the environment for their development and/or reproduction prior to their infective stages.

Types of Biological vectors:

i. **Propagative Biological vector**: There is no cyclical change in the life history of the parasite but its multiplication will occur in the arthropod host.

   Example: In the gut of sandfly, *Phlebotomus argentipes,* the protozoan parasite, *Leishmania donovani,* causing Kala-azar or visceral leishmaniasis, only propagates by binary fission without undergoing any cyclical change. *Plague bacillus* in fleas, and yellow fever virus in mosquito also avail propagative transmission.

ii. **Cyclopropagative Biological vector**: The parasitic organism not only reproduces but undergoes cyclical changes in the arthropod host as well.

   Examples: *Anopheles spp*. Are vector for *Plasmodium spp.* causing malaria, Tsetse flies for *Trypanosoma spp*. causing sleeping sickness diseases.

iii. **Cyclodevelopmental Biological vector**: The disease producing organism does not multiply but undergoes vital cyclical changes within the arthropod vector.

   Example: In the gut of the mosquito, Culex spp. the filarial worms undergo metamorphosis without multiplication.

iv. **Transovarial Biological vector**: Certain disease-producing organisms, such as rickettsiae that cause Rocky Mountain Spotted fever and Scrub Typhus, are transmitted from infected parent arthropods (Ticks, Mites) to their offsprings.

Vector-borne diseases are infections transmitted to humans and animals by vectors such as mosquitoes, ticks, fleas, and sandflies. These vectors carry pathogens like viruses, bacteria, and parasites, spreading them through bites or other forms of contact.

Common Vector-Borne Diseases

- **Mosquito-borne:** Malaria, Dengue, Zika virus, Chikungunya, Yellow Fever
- **Tick-borne:** Lyme disease, Rocky Mountain spotted fever
- **Flea-borne:** Plague
- **Sandfly-borne:** Leishmaniasis
- **Tsetse fly-borne:** African Trypanosomiasis (Sleeping sickness)

Control and Prevention Strategies

1. Vector Control

i. **Chemical Control:** Use of insecticides, larvicides

ii. **Biological Control:** Introduction of natural predators like fish that eat mosquito larvae

iii. **Environmental Management:** Eliminating stagnant water, maintaining sanitation

2. Personal Protection

i. Use of insect repellents, bed nets, and protective clothing

ii. Avoiding outdoor exposure during peak vector activity

3. Vaccination & Prophylaxis

i. Vaccines for diseases like Yellow Fever and Japanese Encephalitis

ii. Preventive drugs for malaria

4. Public Health Measures

i. Surveillance and early detection

ii. Community education and awareness programs

iii. Integrated vector management combining multiple strategies

5. Research & Innovation

- Genetic modification of vectors (e.g., sterile mosquito techniques)
- Development of new vaccines and treatments

Vector control requires a multi-pronged approach involving governments, communities, and health organizations to effectively reduce disease transmission.

Unit-3 □ Mosquitoes as vector-Mosquito borne diseases: Malaria, Dengue, Chikungunya and Filariasis (Biology, Symptoms and Treatments); Mosquito control measures

Malaria

The word **"malaria"** means bad air (Ital, mala = bad; area = air). A connection between swamps and fevers was long recognized and it was a common belief that the disease was contracted by breathing bad air. Another name, paludism, meaning marsh disease is still used for malaria. It is one of the most widespread and most devastating disease of the mankind and the World Health Organization has recognized malaria as one of the six major tropical diseases of the World. It has played a significant role in the history of human civilization as large areas of the earth have repeatedly been subjected to the ruinous effects of the disease.

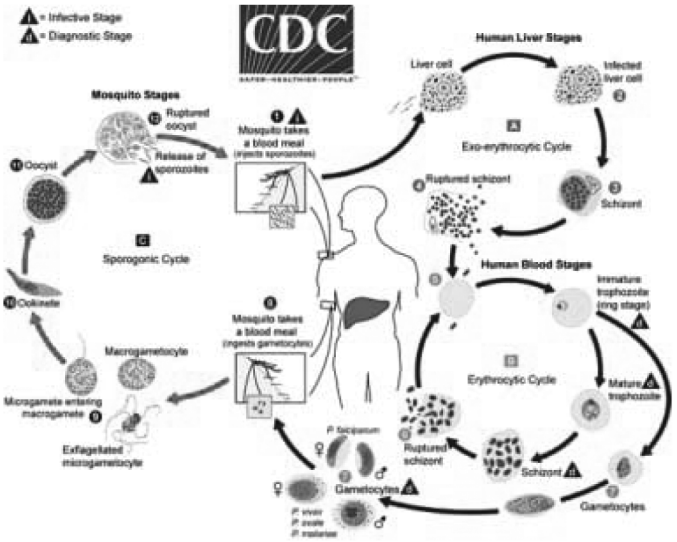
According to WHO, globally in 2023, there were an estimated 263 million cases and 597000 malaria deaths in 83 countries. In 2023, in WHO African Region 94% of malaria cases and 95% of malaria death were recorded. Children under 5 accounted for about 76% of all malaria deaths in the region.

Malaria is a life-threatening disease spread to humans by female *Anopheles* mosquitoes. It is mostly found in tropical countries. Malaria can be prevented by avoiding mosquito bites and with medicines. Treatments can stop mild cases from getting worse.

Female *Anopheles* mosquito

There are 5 *species* of *Plasmodium* parasite those cause malaria in humans and 2 of these specie-*P. falciparum* and *P. vivax*, pose the greatest threat. *P. falciparum* is the deadliest malaria parasite and the most prevalent on the African continent. P. vivax is the is the dominant malaria parasite

in most countries outside of sub-Saharan Africa. The other malaria species which can infect humans are *P. malariae, P. ovale*and *P. Knowlesi*.



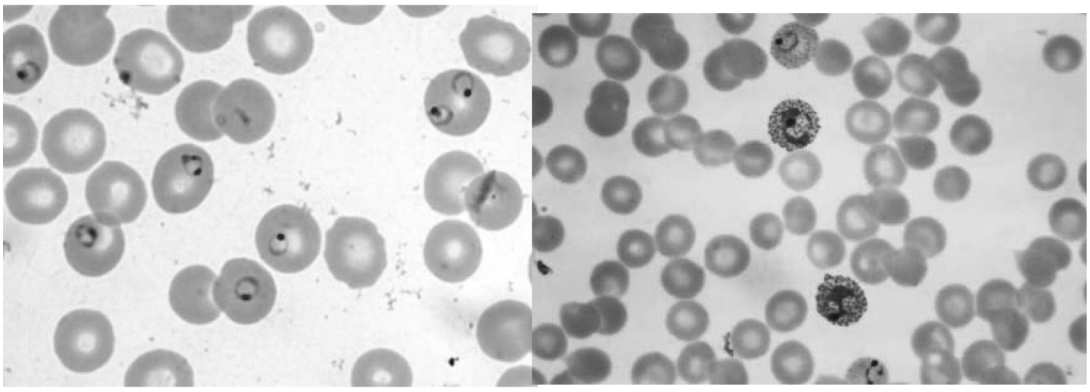
Biology:

Although, in 1902, **Ronald Ross** received the Nobel prize for the studies on *Plasmodium* parasite, **Manson, Lavern, Bignami, Grassi** and others, are also credited for their significant contribution on this parasite. The entire life span of the five species of Plasmodium that infect humans is spent in two hosts: the insect vector, a female Anopheles mosquito, and a human host. Technically, the invertebrate is the definitive host because sexual reproduction occurs there. Asexual reproduction takes place in the tissues of a vertebrate, which thus is the intermediate host. The asexual phase, termed schizogony, occurs in human, whereas the sexual phase, gamogony, occurs in the mosquito, subsequently after sexual phase another asexual reproductive phase, sporogony, occurs in mosquito.

Vertebrate Phases or sporogony:

When an infected mosquito takes blood from a vertebrate, she injects saliva containing tiny, elongated **sporozoites** into the bloodstream. Sporozoites are about 10 μm to 15 μm long by 1 μm in diameter. After being injected into the bloodstream, sporozoites disappear from the circulating blood within an hour. Their immediate fate was a great mystery until the mid-1940s, when it was shown that within one or two days, they enter the parenchyma of the liver. Where they are the first 24 hours

still is unknown. A protein covering the surface of the sporozoite (circumsporozoite protein) bears a ligand that specifically binds to receptors on the basolateral domain of the hepatocyte cell membrane. That is why sporozoites enter liver cells and no other cells in the body. Entry into a hepatocyte initiates a series of asexual reproductions known as the **pre-erythrocytic cycle** or **primary exoerythrocytic schizogony**. Once within a hepatic cell, the parasite metamorphoses into a feeding **trophozoite**, that feeds on the cytoplasm of the host cell. After about a week, trophozoites are mature and begin schizogony. Numerous daughter nuclei are first formed, transforming the parasite into a schizont, also known as a **cryptozoite**. By multiple fission process thousands of merozoites are produced. Merozoites are much smaller than trophozoites, approximately 2.5 μm in length and 1.5 μm in breadth. The merozoites rupture from the host cell, enter the blood circulation, and invade red blood cells, initiating the **erythrocytic cycle** or **erythrocytic schizogony.** However, as early as 1913 it was postulated that some sporozoites become dormant for an indefinite time after entering the body.6 Such dormant cells, called hypnozoites, have now been demonstrated. However, as early as 1913 it was postulated that some sporozoites become dormant for an indefinite time after entering the body. Such dormant cells, called **hypnozoite**s, have now been demonstrated.



Signet ring stage in *P. falciparum*

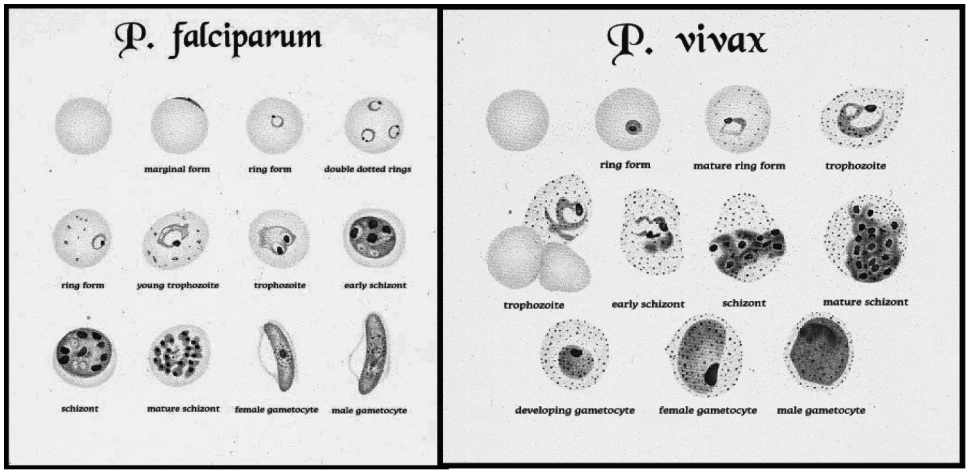
Signet ring stage in *P. viva*

On entry into an erythrocyte, the merozoite again transforms into a **trophozoite**. Host cytoplasm ingested by a trophozoite forms a large food vacuole, giving the young Plasmodium the appearance of a ring of cytoplasm with the nucleus conspicuously displayed at one edge. Due to its resemblance to a finger ring, this stage is called the **signet ring stage.** As the trophozoite grows, its food vacuoles become less noticeable by light microscopy, but pigment granules of

hemozoin in the vacuoles become apparent. Hemozoin is an end product of the parasite's digestion of the host's hemoglobin.

After multiple fission, the parasite rapidly develops into a schizont (or meront), producing a characteristic number of a new generation of merozoites in each infected RBC. When development of merozoites is completed, the host cell ruptures, releasing parasite metabolic wastes and residual body, including hemozoin. A great many of the merozoites are ingested and destroyed by reticuloendothelial cells and leukocytes, other merozoites are capable of infecting new erythrocytes

After an indeterminate number of asexual generations, some merozoites enter erythrocytes and become **macrogamonts (macrogametocytes)** and **microgamonts (microgametocytes)**. The size and shape of these cells are characteristic for each species; they also contain hemozoin. Unless they are ingested by a mosquito, gametocytes soon die and are phagocytized by the reticuloendothelial system.



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Invertebrate Stages:

The sexual phase occurs inthe susceptible female *Anopheles* mosquito, when the mosquito takes a blood meal that contains macrogametocytes and

microgametocytes. After release from its enclosing erythrocyte, a macrogametocyte matures to a macrogamete in a process involving little obvious change. In contrast, the microgametocyte displays a process of transformation, exflagellation. As a microgametocyte becomes extracellular, within 10 to 12 minutes its nucleus divides repeatedly to form six to eight daughter nuclei, each of which is associated with elements of a developing axoneme. Flageller budswith their associated nuclei move peripherally between the interrupted outer membrane of gametocyte. Ultimately continue outward and then break free, forming microgamets.

A microgamete swims about until it finds a macrogamete, which it penetrates and fertilizes. The resultant diploid zygote quickly elongates to become a motile ookinete. The ookinete penetrates the peritrophic membrane in the mosquito's gut and migrates intracellularly and intercellularly to the hemocoel side of the gut. There it begins its transformation into an oocyst. An oocyst is covered by an electron-dense capsule and soon extends out into the insect's hemocoel. The initial division of its nucleus is reductional; meiosis takes place immediately after zygote formation. The oocyst reorganizes internally into a number of haploid nucleated masses called sporoblasts. Sporoblasts, in turn, divide repeatedly to form thousands of sporozoites. The break out of the oocyst causes releasing of sporozoites into the hemocoel and then these migrate throughout the mosquito's body. On contacting the salivary gland, sporozoites enter its channels and can be injected into a new host at the next feeding.

Sporozoite development takes from 10 days to two weeks, depending on the species of *Plasmodium* and temperature. Once infected, a mosquito remains infective for life, capable of transmitting malaria to every susceptible vertebrate it bites. Anopheles spp. that are good vectors for human malaria live long enough to feed on human blood repeatedly.

Symptoms:

Pathology in human malaria is generally manifested in two basic forms:

i. Host inflammatory reactions

ii. Anemia

Of the five species of malaria, P. falciparum is the most virulent and causes, by far the highest mortality.

The most common early symptoms of malaria are nausea, fatigue, a slight rise in temperature, mild diarrhea, and muscular pains, headache and chills. Symptoms usually start within 10–15 days of getting bitten by an infected mosquito. A typical attack of benign tertian or quartan malaria begins with a feeling of intense cold as the hypothalamus, the body's thermostat, is activated, and the temperature then

rises rapidly to 104°F to 106°F. The teeth chatter, and the bed may rattle from the victim's shivering.The hot stage begins within one half to one hour later, with intense headache and feeling of intense heat. Often a mild delirium stage lasts for several hours. As copious perspiration signals the end of the hot stage, the temperature drops back to normal within two to three hours, and the entire paroxysm is over within 8 to 12 hours. A person may sleep for a while after an episode and feel fairly well until the next paroxysm.

Because synchrony in falciparum malaria is much less marked, the onset is often more gradual, and the hot stage is extended. Fever episodes may be continuous or fluctuating, but a patient does not feel well. Falciparum malaria is always serious, and sometimes severe complications occur.

Symptoms may be mild for some people, especially for those who have had a malaria infection before. Infants, children under 5 years, pregnant women, travelers and people with HIV or AIDS are at higher risk.

Severe symptoms include:

- extreme tiredness and fatigue
- impaired consciousness
- multiple convulsions
- difficulty breathing
- dark or bloody urine
- jaundice (yellowing of the eyes and skin)
- abnormal bleeding.

Falciparum malaria is always serious, and sometimes severe complications occur. Severe malaria traditionally has been understood as caused by two major syndromes: (1) severe anemia resulting from destruction of red blood cells, and (2) cerebral malaria, primarily a result of blockage of small blood vessels in the brain by sequestration of infected red blood cells.

The main causes of the anemia are destruction of both parasitized and nonparasitized erythrocytes, inability of the body to recycle the iron bound in hemozoin, and an inadequate erythropoietic response of the bone marrow. Destruction of erythrocytes leads to an increase in blood bilirubin, a breakdown product of hemoglobin. When excretion cannot keep up with formation of bilirubin, jaundice yellows the skin. Hemozoin is taken up by circulating leukocytes and deposited in the reticuloendothelial system. In severe cases the viscera, especially the liver, spleen, and brain, become blackish or slaty as the result of pigment

deposition. Hypoglycemia (reduced concentration of blood glucose) is a common symptom in falciparum malaria. Coma produced by hypoglycemia has commonly been misdiagnosed as cerebral malaria. This condition is usually associated with quinine treatment. Pancreatic islet cells are stimulated by quinine to increase insulin secretion, thus lowering blood glucose.

Malaria infection during pregnancy can also cause premature delivery or delivery of a baby with low birth weight.

People with severe symptoms should get emergency care right away. Getting treatment early for mild malaria can stop the infection from becoming severe.

Treatments:

Appropriate drug treatment of persons with the disease as well as prophylactic drug treatment of newcomers to malarious areas are integral parts of malaria control. The first known effective antimalarial drug was **Quinine**, an extract from the bark of the cinchona tree of tropical countries. Before World War II, a number of effective drugs were introduced. The most important of these was **chloroquine**. Subsequently a number of valuable drugs have been developed, including **primaquine, mefloquine, pyrimethamine, proguanil, sulfonamides such as sulfadoxine**, and **antibiotics such as tetracycline**. Drugs of choice are **chloroquine and primaquine** for P. vivax and P. ovale malarias and **chloroquine a**lone for P. malariae infections**. Chloroquine** is still recommended for strains of P. falciparum sensitive to that drug.

Resistance of P. falciparum to chloroquine has now spread through Asia, Africa, and South America. A combination of **sulfadoxine** and **pyrimethamine** (Fansidar) was used for chloroquine-resistant falciparum malaria. For multidrug-resistant P. falciparum, **mefloquine** (Lariam) is still effective, but resistance to mefloquine is established in several endemic areas. **Artemisinin and its derivatives** are effective for drug-resistant P. falciparum, both in severe and uncomplicated malaria.

World Health Organization, since October 2021, has recommended the use of RTS, S/AS01 vaccine to prevent malaria among children living in regions with moderate to high P. falciparum malaria transmission. The vaccine significantly reduces deadly severe malaria among young children. In October 2023, WHO recommended a second safe and effective malaria vaccine, R21/Matrix-M. Vaccines are now being rolled out in routine childhood immunization programmes across Africa.

Dengue

Dengue is a mosquito-borne viral illness caused by the dengue virus (DENV), a member of the *Flavivirus* genus. Annually, it affects approximately 400 million individuals worldwide, resulting in about 22,000 deaths. The disease is prevalent in over 100 countries across tropical and subtropical regions. DENV is a positive—stranded, enveloped RNA virus primarily transmitted by *Aedes* mosquitoes, especially *Aedes aegypti*. It comprises four distinct serotypes, DENV-1 through DENV-4, each with various genotypes, and encodes three structural and seven non-structural proteins. Clinical manifestations range from mild fever to severe forms like dengue hemorrhagic fever (DHF) or dengue shock syndrome (DSS), characterized by thrombocytopenia, leucopenia, and increased vascular permeability. Although primary infection causes activation of immune responses against DENV serotypes, the severity of the disease is enhanced via heterotypic infection by various serotypes as well as antibody-dependent enhancement (ADE).

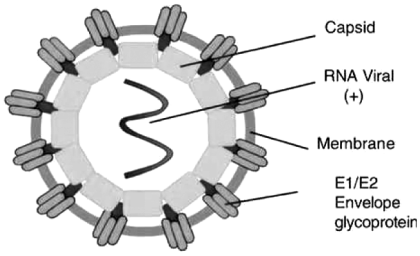
During the last 50 years, the incidence of dengue has increased 30-fold (CDC 2014). DENV epidemics occur annually in the Americas, Asia, Africa, and Australia, and also affect travelers from endemic regions. Apart from the effects on public health, these epidemics have a massive economic impact in the affected countries, including India. The Indian subcontinent, owing to its suitable environment, has several reports of dengue outbreaks involving all serotypes but DENV-5 (Dar et al. 2006; Mustafa et al. 2015) During 2019–2020, the incidence of infection with all DENV serotypes was detected in the northern part of West Bengal, especially in the Siliguri, Darjeeling, Jalpaiguri, and Alipurduar regions. Co-infections with at least three serotypes have been recorded in different cities in India.

Biology:

Electron micrographs have provided detailed insights into the structure of the dengue virus (DENV). These studies reveal that DENV virions are spherical particles, approximately 50 nanometers in diameter, featuring a relatively smooth surface. The virion's architecture includes a well-organized outer protein layer enveloping a lipid bilayer, with an inner nucleocapsid core. The virus comprises three structural proteins: capsid (C), membrane (M) (with its precursor, prM), and envelope (E), along with seven non-structural proteins: NS1, NS2A, NS2B, NS3, NS4A, NS4B, and NS5.

High-resolution cryo-electron microscopy has further elucidated the organization of these structural proteins. The external icosahedral scaffold consists of 90 glycoprotein E dimers, which play a crucial role in host cell recognition and entry. Beneath this scaffold lies the lipid bilayer, incorporating the membrane (M)

proteins. The nucleocapsid core, composed of capsid (C) proteins, houses the viral RNA genome.



The structural transformation of Dengue virus (DENV) from its immature to mature form is a critical process that enables the virus to become infectious. This maturation involves significant conformational changes in the envelope (E) and precursor membrane (prM) proteins, influenced by the pH variations encountered as the virus transits through the host cell's secretory pathway.

Immature Dengue Virus:

In the endoplasmic reticulum (ER), DENV assembles as immature, non-infectious particles characterized by a spiky surface. This appearance results from 60 trimeric prM-E heterodimers protruding from the viral membrane. The prM protein serves as a protective chaperone, preventing premature fusion of the virus with host membranes by covering the fusion loop of the E protein.

Maturation Process:

As the immature virion progresses through the acidic environment of the trans-Golgi network (TGN), the lowered pH induces conformational rearrangements in the E and prM proteins. These changes facilitate the cleavage of prM by the host protease furin, separating it into pr (precursor) and M (membrane) proteins. This cleavage is essential for viral maturation, transitioning the E proteins from trimeric spikes to dimers that lie flat against the viral surface, resulting in a smooth, mature virion. The pr peptide remains associated with the E protein in the acidic TGN, preventing premature fusion. Upon release into the neutral pH of the extracellular space, the pr peptide dissociates, rendering the virus infectious.

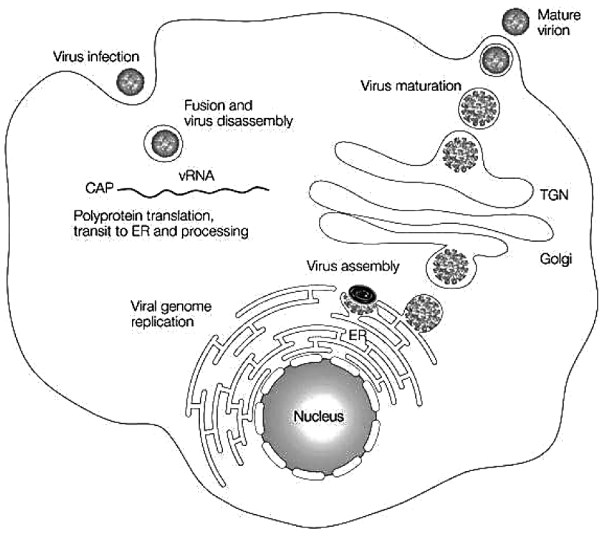
This maturation process is crucial for the infectivity of DENV, as only mature virions with rearranged E proteins and cleaved prM are capable of efficient attachment and entry into host cells.

The DENV genome is a positive (+) single-stranded RNA, approximately 11 kilobases in length (Miller et al. 2006). The RNA genome is divided into three parts: the 52 UTR region (untranslated region), ORF (open reading frame), and the 32 UTR region (Fig. 4). The genome has a type I cap (m7GpppAmp) at the

52 end with a single ORF that encodes a polyprotein and lacks a poly (A) tail at the 32 end.

Dengue virus (DENV) replication within host cells involves several critical steps:

1. **Attachment and Entry:** DENV targets dendritic cells, macrophages, monocytes, and lymphocytes. The virus binds to host cell receptors such as Fc receptors, glycosaminoglycans (GAGs), CD14-associated molecules, heparan sulfate, and lectin-like receptors like DC-SIGN. This binding facilitates receptor-mediated endocytosis, allowing the virus to enter the cell via clathrin-coated vesicles.
2. **Fusion and Uncoating:** Within the acidic environment of late endosomes, the viral envelope (E) protein undergoes conformational changes, leading to the fusion of the viral and endosomal membranes. This process releases the nucleocapsid into the cytoplasm, where the viral RNA genome is uncoated.
3. **Translation and Polyprotein Processing:** The positive-sense RNA genome is translated into a single polyprotein at the rough endoplasmic reticulum (ER). This polyprotein is subsequently cleaved by host and viral proteases into three structural proteins (capsid [C], membrane [M], and envelope [E]) and seven nonstructural proteins (NS1, NS2A, NS2B, NS3, NS4A, NS4B, and NS5).



DENV replication within host cell

4. **RNA Replication:** Replication occurs on the ER membrane, where the viral RNA-dependent RNA polymerase (NS5) synthesizes a negative-strand RNA intermediate. This intermediate serves as a template for producing new positive-sense RNA genomes

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5. **Virion Assembly and Maturation:** Newly synthesized genomic RNA is encapsidated by capsid proteins, and the resulting nucleocapsid buds into the lumen of the ER, acquiring its envelope containing the prM and E proteins. As the immature virions transit through the trans-Golgi network, the prM protein is cleaved by the host protease furin, resulting in mature, infectious virions.
6. **Release:** Mature virions are transported to the cell surface via the secretory pathway and released into the extracellular space through exocytosis, ready to infect new cells.

Transmission of Dengue virus Dengue virus (DENV) transmission primarily involves two mosquito species: *Aedes aegypti* and *Aedes albopictus*

Primary Vector: *Aedes aegypti*

- **Habitat:** Predominantly found in tropical and subtropical regions worldwide, *Ae. aegypti* thrives in urban environments, often residing indoors.
- **Breeding Sites:** These mosquitoes lay eggs in artificial containers holding water, such as buckets, bowls, animal dishes, flowerpots, and vases.
- **Feeding Behavior:***Ae. aegypti* prefers human blood and is known for biting during daytime hours.

Secondary Vector: *Aedes albopictus*

- **Habitat:** Originally native to Asia, *Ae. albopictus* has expanded its range to various continents. It is commonly found outdoors in both rural and urban settings.
- **Breeding Sites:** Similar to *Ae. aegypti*, this species breeds in natural and artificial water-filled containers.
- **Feeding Behavior:***Ae. albopictus* is an aggressive daytime biter with a diverse host range, feeding on humans and animals.

Transmission Cycles of DENV:

1. Human Transmission Cycle:

- **Urban Cycle:** Involves transmission between humans and mosquitoes, primarily *Ae. aegypti*. Infected mosquitoes transmit the virus to humans through bites, and subsequently, mosquitoes acquire the virus from infected individuals.

2. Sylvatic (Zoonotic) Cycle:

- **Wildlife Reservoirs:** In certain regions, DENV circulates between non-human primates and forest-dwelling mosquitoes.
- **Geographical Occurrence:** This cycle has been documented in the forests of Southeast Asia and West Africa.
- **Transmission Dynamics:** Mosquitoes acquire the virus from infected primates and can transmit it to other primates or, occasionally, humans entering these forested areas.

Symptoms of Dengue fever:

Dengue virus (DENV) infection manifests across a spectrum of clinical presentations, from mild febrile illness to severe, life-threatening conditions.

Dengue Fever (DF):DF is the most common and typically milder form of dengue virus infection. It progresses through three distinct phases:

1. **Febrile Phase:** Characterized by a sudden onset of high fever lasting 2–7 days, often accompanied by severe headache, retro-orbital pain, myalgia, arthralgia, and a macular or maculopapular rash. Minor hemorrhagic manifestations, such as petechiae or gingival bleeding, may also occur.
2. **Critical Phase:** Typically occurring near the time when the fever subsides, this phase may involve increased capillary permeability, leading to plasma leakage. While many patients recover without complications, some may progress to more severe forms.

3. **Convalescent Phase:** This recovery phase involves the reabsorption of leaked fluids and improvement of symptoms. Patients may experience a rash, itching, and a return of appetite.

Dengue Hemorrhagic Fever (DHF) and Dengue Shock Syndrome (DSS):

DHF is a severe manifestation of dengue infection, primarily affecting children under 10 years in endemic regions. It is characterized by increased vascular permeability, thrombocytopenia, and hemorrhagic phenomena. DSS, the most severe form, involves significant plasma leakage leading to shock. The World Health Organization (WHO) outlines the following criteria for diagnosing DHF:

- **Fever:** Acute onset, lasting 2–7 days.
- **Hemorrhagic Manifestations:** Such as a positive tourniquet test, petechiae, ecchymoses, or bleeding from mucosa.
- **Thrombocytopenia:** Platelet count d”100,000 cells per mm^3.
- **Evidence of Plasma Leakage:** Manifested by hemoconcentration (hematocrit increase >20%), pleural effusion, ascites, or hypoproteinemia.

DSS includes all DHF criteria plus signs of circulatory failure, such as rapid and weak pulse, narrow pulse pressure (<20 mm Hg), or hypotension accompanied by cold, clammy skin and restlessness.

Early recognition and appropriate medical management are crucial to reduce morbidity and prevent mortality associated with severe dengue manifestations.

Treatment:

Dengue virus (DENV) infection currently lacks a specific antiviral treatment; management primarily focuses on supportive care, including fever reduction and pain management. However, research into antiviral agents has identified several promising compounds, particularly those derived from natural sources.

Sulfated Polysaccharides from Seaweeds:

Studies have demonstrated that sulfated polysaccharides extracted from various seaweeds exhibit antiviral activity against DENV.

- **Fucoidan:** Derived from brown algae, fucoidan has shown potent inhibition of DENV-2 by interfering with virus internalization into host cells.
- **Carrageenans and dL-Galactan Hybrids:** Polysaccharides such as kappa/iota/nu carrageenan G3d and the dL-galactan hybrid C2S-3 have demonstrated antiviral activity against all DENV serotypes by inhibiting virus internalization through interaction with host cell receptors like heparan sulfate.

Curcumin and Its Derivatives: Curcumin, the active compound in turmeric (*Curcuma longa*), along with its analogs, has been investigated for anti-dengue properties.

- **Curcumin:** This compound has been found to inhibit DENV replication by affecting various stages of the viral life cycle, including entry and replication.
- **Curcuminoids:**Analogs of curcumin have shown enhanced antiviral activity against DENV, potentially by inhibiting viral proteases and affecting cellular pathways essential for viral replication.

Other Investigational Antiviral Agents:

Several other compounds are under investigation for their potential anti-dengue effects:

- **Ribavirin and Nucleoside Analogs:** These compounds inhibit nucleoside biosynthesis, thereby reducing DENV replication within host cells.
- **Glycyrrhizin:** Derived from licorice root, glycyrrhizin and its derivatives may exert antiviral effects by modulating interferon secretion and inhibiting viral protein processing.
- **6-Azauridine:** This uridine analog inhibits pyrimidine synthesis, disrupting viral RNA and protein synthesis.
- **NITD008:** A nucleoside analog that has demonstrated broad-spectrum antiviral activity against DENV and other flaviviruses in both in vitro and in vivo studies.

Development of vaccine

Developing an effective dengue vaccine presents significant challenges due to the virus's complex nature. Dengue virus (DENV) exists in four distinct serotypes (DENV1–4), each capable of causing infection. A major concern in vaccine development is Antibody-Dependent Enhancement (ADE), where antibodies from a previous infection with one serotype may enhance infection with another serotype, potentially leading to more severe disease manifestations. This phenomenon necessitates a vaccine that provides balanced immunity against all four serotypes to avoid the risk of ADE.

The first dengue vaccine, Dengvaxia® (CYD-TDV), developed by Sanofi Pasteur, was licensed in several countries and is approved for use in individuals aged 9–16 years with a confirmed previous dengue infection and residing in endemic areas. However, its use is limited to those with prior exposure to the virus, as

vaccination in seronegative individuals has been associated with an increased risk of severe dengue upon subsequent infection.

Another vaccine, Qdenga® (TAK-003), developed by Takeda, received prequalification from the World Health Organization (WHO) in May 2024. This live-attenuated vaccine targets all four dengue serotypes and has shown promise in clinical trials.

In India, efforts are underway to develop and commercialize dengue vaccines. The live-attenuated tetravalent vaccine TV003/TV005 has been licensed for clinical development by Indian vaccine manufacturers, with clinical trials planned to evaluate its safety and efficacy in the local population.Despite these advancements, the development of a universally effective dengue vaccine remains challenging due to factors such as the virus's genetic variability, the need for balanced immunity against all serotypes, and the potential for ADE. Ongoing research continues to address these issues to achieve a safe and effective solution for dengue prevention.

Chikungunya

Chikungunya is a viral disease spread by mosquitoes, caused by the chikungunya virus (CHIKV). This virus belongs to the alphavirus genuswithin the Togaviridae family. The name "chikungunya" comes from the Kimakonde language, describing the characteristic contorted posture of those affected.

CHIKV was first identified in Tanzania in 1952 and later detected in other parts of Africa and Asia. Urban outbreaks were first recorded in Thailand in 1967 and in India during the 1970s. Since 2004, CHIKV outbreaks have become more frequent and widespread, partly due to viral adaptations that enhance its transmission by Aedes albopictus mosquitoes. The virus has now been identified in over 110 countries across Asia, Africa, Europe, and the Americas. On islands where a large portion of the population has been infected and developed immunity, transmission has been interrupted. However, in countries with many susceptible individuals, the virus continues to spread.

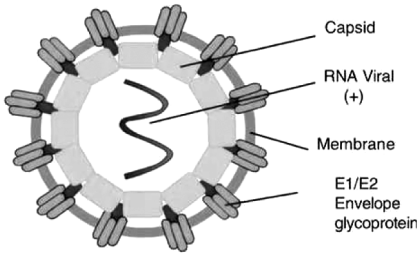
Biology

Chikungunya virus is primarily transmitted by mosquitoes, mainly *Aedes aegypti* and *Aedes albopictus*, which also spread dengue and Zika viruses. The virus may circulate within a number of animals, including birds and rodents.

Virology

Chikungunya virus (CHIKV) is a member of the *Alphavirus* genus within the *Togaviridae* family. It was first isolated in Tanzania in 1953 and is an RNA virus

with a positive-sense, single-stranded genome of approximately 11.6 kb. Since it is transmitted by mosquitoes,



CHIKV is classified as an *arbovirus* (arthropod-borne virus).

Three distinct genotypes of the chikungunya virus (CHIKV) have been identified, each with unique genetic and antigenic characteristics:

- **West African genotype**
- **East/Central/South African (ECSA) genotype**
- **Asian genotype**

The **Asian lineage**, which emerged in 1952, later diverged into two separate clades:

- **Indian Ocean Lineage (IOL)**
- **Southeast Asian clade**

Aedes mosquitoes are most active during daylight hours and lay their eggs in containers with standing water. Both species feed outdoors, while *Ae. Aegypti* also bites indoors. When a mosquito feeds on an infected person with CHIKV in their blood, it can ingest the virus. Over several days, the virus replicates within the mosquito, reaches its salivary glands, and can be transmitted to a new human host when the mosquito bites. The virus then replicates in the newly infected person, spreading through their bloodstream. Once the virus reaches high concentrations, they can infect other mosquitoes, continuing the transmission cycle.

The virus can circulate among various animals, including birds and rodents. Diagnosis is confirmed by testing blood samples for viral RNA or antibodies. Chikungunya symptoms may be mistaken for dengue fever or Zika fever, as they share the same mosquito vectors. It is believed that most people develop immunity after a single infection.

Symptoms:

In symptomatic patients, chikungunya virus (CHIKV) disease typically develops **4–8 days** after being bitten by an infected mosquito, with a range of **2–12 days**.

Key Symptoms:

- **Abrupt fever onset**, often accompanied by

severe, debilitating joint pain

- **Joint swelling** and **muscle pain**
- **Headache, nausea, fatigue, and rash**

Joint pain usually lasts a few days but can persist for **weeks, months, or even years**.

Because symptoms overlap with **dengue and Zika virus infections**, CHIKV cases are sometimes misdiagnosed. If significant joint pain is absent,

symptoms are generally mild and may go unnoticed.

Complications and Risk Factors:

- Most patients **fully recover**,

but some may develop **eye, heart, or neurological issues**.

- **Newborns infected during delivery**

and **older adults with underlying conditions**

face a higher risk of severe illness.

- In some cases, CHIKV infection can **increase mortality risk**.

After recovery, current evidence suggests that individuals likely develop **long-term immunity** against future infections.

Treatment:

Chikungunya virus (CHIKV) can be detected in blood samples during the **first week of illness** using tests like:

- **Reverse transcriptase–polymerase chain reaction (RT–PCR)**

  -Identifies viral RNA in the blood.

After the first week, other tests detect the body's **immune response** to the infection by measuring **antibodies**:

- **Antibody tests**

  -Detect **IgM and IgG antibodies** against CHIKV.

  -Antibodies usually appear within the **first week** of illness.

  -They can remain detectable for **up to 2 months**.

Currently, there is **no specific antiviral treatment** for chikungunya.

Management focuses on **supportive care** and **symptom relief**.

Symptomatic Treatment:

- **Fever and joint swelling relief:**
  - **Nonsteroidal anti-inflammatory drugs (NSAIDs)** like **naproxen**
  - **Non-aspirin analgesics** such as **paracetamol (acetaminophen)**
  - **Adequate hydration** with fluids
- **Avoid:**
  - **Aspirin** due to increased risk of **bleeding**
  - **Corticosteroids** during the acute phase, as they may cause **immunosuppression** and worsen the infection

Passive Immunotherapy (Experimental Treatment):

- Animal studies suggest that **passive immunotherapy** could be beneficial.
- Clinical trials are ongoing for **high-risk patients**.
- This therapy involves administering **anti-CHIKV hyperimmune human antibodies** (*immunoglobulins*) to those at high risk of severe disease.

Although no **approved antiviral drugs** exist, some medications have shown effectiveness **in vitro** (laboratory studies).

While there are several vaccines currently in different stages of development (as of Dec 2022) they are yet to be licensed.

A Chikungunya vaccine is a vaccine intended to provide acquired immunity against the chikungunya virus.

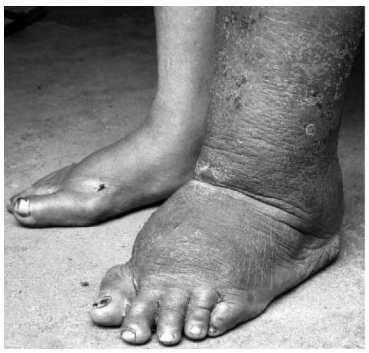
The most commonly reported side effects include headache, fatigue, muscle pain, joint pain, fever, nausea and tenderness at the injection site.[1]

The first chikungunya vaccine was approved for medical use in the United States in November 2023.

Filariasia:

Filariasis is an infection caused by parasitic nematodes (roundworms) transmitted by various insect vectors. One of the most significant forms of this disease is **lymphatic filariasis**, a leading cause of permanent disability worldwide. It affects over a hundred million people and is typically acquired in childhood, although symptoms may take years to appear.

While most infected individuals remain asymptomatic, some develop **elephantiasis**, a condition characterized by severe swelling in the arms, legs, breasts, or genitals due to lymphatic system damage.



Lymphatic filariasis is primarily caused by the filarial worm *Wuchereriabancrofti*, though two other species, *Brugiamalayi* and *Brugiatimori*, can also cause the disease. These parasites are transmitted by mosquitoes, including species from the *Culex*, *Anopheles*, *Aedes*, and *Mansonia* genera. *W. bancrofti* is the most widespread, affecting over 120 million people, particularly in Central Africa, the Nile Delta, South and Central America, tropical Asia (including southern China), and the Pacific Islands.

Biology:

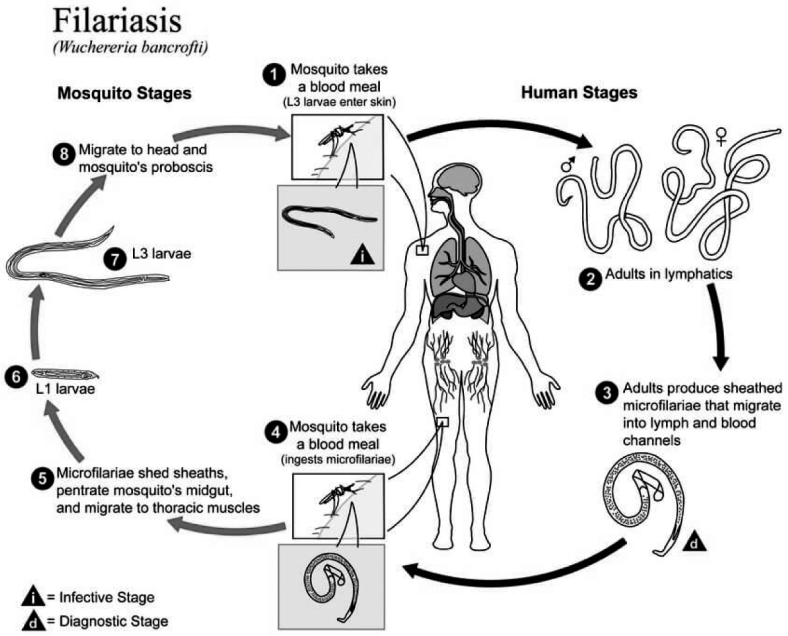
Adult worms are long and slender with a smooth cuticle and bluntly rounded ends. Their head is slightly swollen and bears two circles of well-defined papillae. Their mouth is small; a buccal capsule is lacking. Males are about 40 mm long and 100 μm wide. Their tail is fingerlike. Females are 6 cm to 10 cm long and 300 μm wide. Their vulva is near the level of the middle of their esophagus.

Adult *Wuchereriabancrofti* reside in the lymphatic ducts of humans, primarily in the afferent lymph channels near major lymph nodes in the lower half of the body. In rare cases, they may invade a vein. Females are ovoviviparous, giving birth to thousands of juveniles known as microfilariae. These microfilariae retain their egg membrane as a delicate, close-fitting sheath, which is visible at both their anterior and posterior ends. When stained, several internal nuclei and organ primordia can be observed within the microfilariae.

Females release microfilariae into the lymphatic system. While some may migrate into nearby tissues, most are carried into the bloodstream via the thoracic duct. In many regions where this parasite is prevalent, microfilariae exhibit distinct periodicity in peripheral blood, meaning they are detectable at specific times of the day and nearly absent at others. Their highest concentration typically occurs between 10 P.M. and 2 A.M., making night-feeding mosquitoes the primary vectors in areas where this periodicity is observed. During the daytime, microfilariae accumulate primarily in the blood vessels of deep tissues, especially within the pulmonary capillaries.

Reversal of a patient's sleep schedule causes reversal of periodicity so that microfilaremia becomes diurnal.

When mosquitoes take a blood meal, they ingest microfilariae along with it. The microfilariae may shed their sheath either before or during penetration of the mosquito's stomach, a process that typically occurs within two hours. Once inside, they migrate to the mosquito's thoracic muscles, where they develop into first-stage juveniles (J1s) over approximately eight days. These then molt into second-stage juveniles (J2s), which are short, sausage-shaped worms. At this stage, most organ systems are present, but the juveniles retain an anal plug and lack a functional anus. Despite this, they actively feed, causing some damage to the mosquito host. After two to four days, the gut of the J2 juveniles fully develops, and they molt into elongated, slender filariform J3s. At this stage, their development halts. These infective filariform juveniles measure between 1.4 mm and 2.0 mm in length. They migrate through the mosquito's hemocoel and eventually reach the labium or proboscis sheath. When the mosquito feeds, the J3s escape and enter the human host through the bite wound. From there, they travel through the peripheral lymphatics before settling in larger lymphatic vessels, where they mature into adult worms.



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Symptoms:

Symptoms and Effects of Lymphatic Filariasis

Elephantiasis

- The most noticeable symptom, characterized by extreme edema and thickening of the skin and underlying tissues.
- First disease identified as being transmitted by mosquito bites.
- Occurs when parasites obstruct the lymphatic system, leading to impaired drainage.

Acute Inflammatory Filariasis

- Presents with fever episodes lasting 5 to 7 days.
- Causes inflammation of lymph nodes, often accompanied by:
  - Epididymitis
  - Spermatic cord inflammation
- Secondary bacterial infections are common, especially severe in previously unexposed immigrants. It is probable that Wolbachia antigens, either from dead and degenerating worms or in worm excretory-secretory products, play a role in inflammation.

Chronic Filarial Disease

- Develops gradually over several years.
- Many patients show no physical symptoms despite lymphatic dilation.
- Inflammatory reactions to dying adult worms may cause:
  - Chronic lymphedema, progressing to elephantiasis
  - Hydrocele and scrotal elephantiasis (*Wuchereriabancrofti*)
  - Chyluria and chyloceles due to disrupted lymphatic drainage

Affected Body Parts

- **Elephantiasis primarily affects** the lower extremities.
- Other affected areas (less common):
  - Ears, mucous membranes, and amputation stumps.
- **Different species target different body parts:**
  - *Wuchereriabancrofti*-legs, arms, vulva, breasts, scrotum (hydrocele).
  - *Brugiatimori*-rarely affects the genitals.

Immune Response in Chronic Cases

- Individuals with chronic elephantiasis are usually **amicrofilaraemic** (lacking detectable microfilariae).
- Often exhibit **strong immune reactions** against both microfilariae and adult worms.

Treatments:

Treatment of Lymphatic Filariasis

Primary Drug: Diethylcarbamazine (DEC)

- **Used for over 40 years** as the primary treatment.
- Eliminates microfilariae from the blood and, with careful administration, can kill adult worms.

Standard Treatment Regimen

- **Dose:** 6 mg/kg over **7 to 12 days** to reach a **total of 72 mg/kg**.
- **Disadvantages:**
  - Significant side effects.
  - Difficulty ensuring patient compliance, especially in field settings.

Improved Treatment Approach

- A **single dose of 6 mg/kg**, given **annually or semiannually**, provides effective control.
- **Benefits:**
  - Fewer side effects.
  - Easier logistics for mass treatment programs.

Combination Therapy for Better Results

- **Ivermectin + DEC or Albendazole** provides additive effects, improving treatment efficacy.
- **DEC-fortified table salt** has also proven effective for control.

Edematous limbs in lymphatic filariasis can sometimes be treated successfully with pressure bandages, which help force lymph out of the swollen area. This method may gradually reduce the limb size to near normal. However, any connective tissue proliferation that has developed remains unaffected. In severe cases, surgical removal of elephantoid tissue is often a viable option.

Prevention focuses primarily on protecting against mosquito bites in endemic areas. Temporary visitors should rigorously use insect repellents, mosquito netting,

and other protective measures. Long-term prevention requires sustained mosquito control and mass chemotherapy for local populations to eliminate microfilariae from the bloodstream, thereby reducing transmission. Successful implementation of these strategies depends on educating communities in endemic regions. Studies have shown that awareness is often low—less than half of the affected population had heard of filariasis, and only 6% knew it was transmitted by mosquitoes.

Mosquito control measures

Understanding the Mosquito Life Cycle

- Mosquitoes go through **four stages**: egg, larva, pupa, and adult.
- The entire cycle can range from **four days to one month**, depending on species and temperature.
- **Water is essential** for mosquito development.
- Most eggs hatch within **48 hours**.

Mosquito Control and Prevention

- The most effective control method is **eliminating potential egg-laying sites** by removing standing water.
- **Prevent water accumulation:**
    - Add drainage holes to containers like barrels and old tires.
    - Change or circulate water in pet bowls and birdbaths weekly.
    - Fill tree holes with sand or mortar, or add drainage holes.
    - Clean roof gutters to prevent water buildup.
    - Dispose of trash that can collect water.
- **Reduce adult mosquito shelter:**
    - Keep weeds and vegetation trimmed.
- **For ponds and water bodies:**
    - Consider stocking fish that feed on mosquito larvae.

Control of Mosquito Larvae

- **Larvicides** can be used but should only serve as a supplemental measure.
- Use **only approved larvicides**—check with your local extension office or pest management department.
- **Do not apply pesticides to moving water** (e.g., streams).

- Always **read and follow label directions** when using pesticides.
- **Non-chemical options** like *Bacillus thuringiensis israelensis* (Bti) may be effective.
- **Methoprene products** can be used to treat water-collecting areas such as:
    - Birdbaths
    - Urns
    - Old tires
    - Flower pots
    - Abandoned swimming pool

Control of Adult Mosquitoes

- **Least efficient method** for mosquito control.
- Requires **specialized equipment** to apply adulticides (pesticides targeting adult mosquitoes).
- **Small droplets** of insecticide drift through the air to kill mosquitoes on contact.
- Consult **local extension offices or pest management departments** for approved pesticides.
- Pesticide use should be **supplemental** to reducing mosquito breeding sites.
- **Outdoor fogging** with **pyrethrin or 5% malathion** is an option—always follow label instructions.
- Seek **expert guidance** from local extension agents to develop an effective mosquito management plan.

Personal Protection Against Mosquito Bites

- **Avoid outdoor exposure** during peak mosquito activity times (dusk and dawn).
- Use **insect repellents** containing:
    - DEET (note: toxic to pets, avoid use on animals)
    - Picaridin
    - Oil of lemon-eucalyptus
- Ensure **windows, doors, and porches are tightly screened** to prevent mosquito entry.

Important Legal Notice:

- Using pesticides in a manner inconsistent with their label is a **violation of state and federal law**.
- Always **follow label directions** to prevent environmental damage and avoid harm to humans or animals.

Unit-4 □ Houseflies as vector-Role in food-borne illness, Controlling the spread of houseflies and disease

Flies are significant medical and veterinary pests and serve as major vectors of human diseases worldwide. Houseflies, in particular, are nuisance pests to both humans and domestic animals and are primary vectors of foodborne and animal pathogens. Houseflies (*Musca domestica*) are nonbiting muscoids of importance because they can be mechanical vectors of many kinds of pathogens such as bacteria, protozoa, viruses, and helminth eggs. Their indiscriminate movement, long flight range, and attraction to both decaying organic matter and food preparation areas increase the risk of human exposure to harmful microorganisms. Houseflies can transport microbial pathogens from low-risk reservoirs, such as animal manure, to high-risk environments like food sources, thereby amplifying disease transmission.

Similarly, stable flies are blood-feeding insects that pose a serious nuisance to humans and domestic animals, leading to significant economic losses in the livestock industry. Additionally, they contribute to the ecology of various bacteria originating from animal manure and other larval developmental habitats.

Most bacteria associated with insects include foodborne pathogens such as *Escherichia coli*, *Salmonella* spp., *Shigella* spp., and others. The ability of adult houseflies to transmit pathogens like *Campylobacter*, *E. coli*, *Salmonella* spp., and *Shigella* spp. including strains resistant to antibiotics, has been well documented. For instance, studies have shown that houseflies can transmit *E. coli* to cattle, which serve as the primary reservoir for this major human foodborne pathogen.

Flies are also a concern in the transmission of avian influenza. confirmed that *Musca domestica*, the dominant housefly species, can act as a vector for circovirus. This role may extend to other non-enveloped viruses as well.

Houseflies in dairy farms can be a concern since they could play a crucial role in increased bacterial counts in milk and the potential occurrence of transmitted diseases that affect public and animal health.

Surveillance of pathogens in flies can help identify persistent pathogens in farm environments and their potential impact on both public and animal health. Houseflies also serve as vectors for multidrug-resistant bacteria, facilitating the spread of antimicrobial resistance between farm animals and humans.

Despite their significant role in disease transmission, little is known about the prevalence of pathogens on houseflies in different areas of dairy farms. This

knowledge gap is critical, as it may have implications for udder health, human health, and the shelf life of dairy products.

Researchers estimate that houseflies (*Musca domestica*) can harbor and transmit at least 65 human diseases, making them one of the most significant insect vectors of pathogens worldwide. These flies are particularly concerning due to their ability to thrive in both urban and rural environments, their frequent contact with human food sources, and their attraction to decaying organic matter, animal waste, and garbage.

Common Diseases Transmitted by Houseflies

In the United States, houseflies are known to spread several foodborne and gastrointestinal illnesses, including:

- **Food poisoning**-Caused by bacteria such as *Escherichia coli*, *Salmonella*, and *Clostridium perfringens*, leading to nausea, vomiting, and diarrhea.
- **Dysentery**-A severe intestinal infection, often caused by *Shigella* spp., resulting in bloody diarrhea and dehydration.
- **Diarrhea**-Linked to various pathogens, including *Campylobacter* and *Vibrio* spp.

Global Health Threats Associated with Houseflies

Beyond common foodborne illnesses, houseflies are implicated in the transmission of serious bacterial, viral, and parasitic infections, including:

- **Anthrax** (*Bacillus anthracis*)-A rare but severe bacterial infection affecting the skin, lungs, or digestive system.
- **Cholera** (*Vibrio cholerae*)-A potentially fatal diarrheal disease spread through contaminated food and water.
- **Tuberculosis** (*Mycobacterium tuberculosis*)-While primarily transmitted via respiratory droplets, houseflies may contribute to bacterial spread in unsanitary conditions.
- **Typhoid fever** (*Salmonella enterica* serovar *Typhi*)-A systemic bacterial infection causing high fever, weakness, and digestive issues.

Parasitic Infections

In addition to bacterial and viral diseases, houseflies can also serve as mechanical vectors for the eggs and larvae of parasitic worms, such as:

- **Hookworms** (*Ancylostoma* spp.)-Parasitic nematodes that infect the intestines, causing anemia and malnutrition.
- **Roundworms** (*Ascaris lumbricoides*)-A common intestinal parasite that can lead to malabsorption and growth issues, especially in children.
- **Tapeworms** (*Taenia* spp.)-Can cause digestive discomfort and, in severe cases, neurological complications.

Mechanisms of Disease Transmission

Houseflies spread pathogens through several routes:

1. **Mechanical Transmission**-Bacteria and viruses adhere to the fly's exoskeleton, legs, and bristles as they move between contaminated surfaces and human food.
2. **Regurgitation**-Flies feed by regurgitating digestive enzymes onto food, potentially introducing pathogens.
3. **Fecal Contamination**-Houseflies frequently defecate, contaminating food, water, and surfaces with harmful microorganisms.

Houseflies (*Musca domestica*) thrive in close association with humans and domestic animals, as both urban and rural environments support their development. Their primary sources of nutrition and oviposition include human excrement, garbage, animal manure, and bedding. With a dispersal range of 5 to 32 km, houseflies can carry bacteria on the surface of their exoskeleton and within their alimentary canal, spreading them through mechanical translocation, defecation, and regurgitation.

The presence of bristles and glandular hairs on their legs enhances bacterial adhesion to their exterior surface. Additionally, the high viscosity of feces improves the efficiency of these structures in trapping bacteria suspended in manure. Bacteria can also be stored in the fly's crop, where they may multiply before being regurgitated or excreted, a process known as "bioenhanced transmission".

Given their ability to transport and amplify infectious agents, houseflies remain a serious public health concern, particularly in areas with poor sanitation and food safety practices. Controlling their populations through proper waste management, improved hygiene, and targeted pest control measures is crucial in reducing the risk of disease transmission.

Control measures

Houseflies (*Musca domestica*) are major disease vectors, necessitating effective control measures to reduce their populations and minimize health risks. Control strategies include:

1. **Prevention of Breeding**-Proper waste disposal, sanitation, manure management, and covering outdoor toilets to eliminate breeding grounds.
2. **Chemical Treatments**-Disinfecting breeding sites with lime, borax, copper sulfate, crude oil, or formaldehyde to prevent egg-laying.
3. **Food Protection**-Keeping edibles covered and avoiding consumption of fly-contaminated food.
4. **Destruction of Adult Flies**-Manual killing, baiting, sticky wires, chemical sprays (Baygon, DDT, malathion, diazinon), and residual insecticides.
5. **Biological Control**-

I. **Natural enemies** (fungi, nematodes, beetles, mites, parasitic wasps, birds) that prey on or infect flies.

II. **Viral control** (*MdSGHV virus*) disrupts fly reproduction and shortens lifespan.

III. **Bacterial control** (*Bacillus thuringiensis*) targets larvae, while new bacterial strains disrupt fly development.

IV. **Fungal control** (*Entomophthora muscae*, *E. schizophorae*) infects and kills houseflies.

V. **Essential oils** (menthol, limonene, cineole) act as natural repellents and fumigant insecticides.

VI. **Insect predators and parasitoids** (histerid beetles, macrochelid mites, pteromalid wasps) target fly eggs, larvae, and pupae.

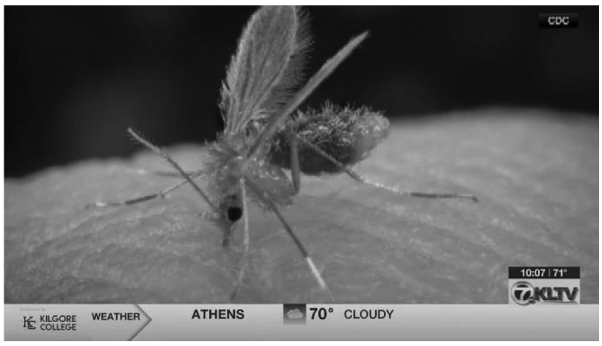
A combination of these methods provides the most effective and sustainable housefly control.

Unit-5 □ Other flies-Sandflies and Leishmaniasis; Tsetse flies and Trypanosomiasis (diagnosis/types, risk-factors, treatment and prevention)

Sand Fly and Leishmaniasis

Sand Fly (Phlebotomine Flies)

Sand flies are tiny, blood-feeding insects belonging to the *Phlebotomus* (Old World) and *Lutzomyia* (New World) genera. These nocturnal insects thrive in warm, humid environments, such as tropical and subtropical regions, and are known for transmitting **Leishmaniasis**, a serious parasitic disease.



https://www.google.com/url?sa=i&url=https%3A%2F%2Fwww.kltv.com%2F2023%2F10%2F27%2Feast-texas-expert-says-sand-flies-may-be-causing-skin-infection-epidemic%2F&psig=AOvVaw02AyxKz09mpUjWZBjp-ds0&ust=1742503599620000&source=images&cd=vfe&opi=89978449&ved=0CBQQj RxqFwoTCLip0piCl4wDFQAAAAAdAAAAABAE

Leishmaniasis Disease

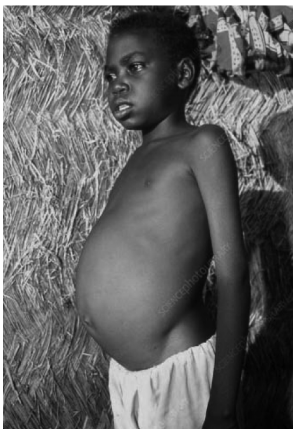
Leishmaniasis is caused by *Leishmania* parasites, which are transmitted through the bite of infected female sand flies. It affects humans and animals and occurs in three main forms:

1. **Cutaneous Leishmaniasis**-The most common form, causing skin ulcers, sores, and scars.
2. **Visceral Leishmaniasis (Kala-azar)**-The most severe type, affecting internal organs like the liver, spleen, and bone marrow, leading to fever, weight loss, and anemia. If untreated, it can be fatal.

3. **Mucocutaneous Leishmaniasis**-Affects mucous membranes, causing disfigurement, especially in the nose, mouth, and throat.

Leishmaniasis is prevalent in regions such as South America, the Middle East, Africa, and Asia. In India, the disease is named as Kala-azar.

Prevention includes vector control (using insecticides, nets, and repellents), reducing human-sand fly contact, and improving sanitation. There is no universal vaccine, but treatments include antimonial drugs, amphotericin B, and miltefosine.



Leishmaniasis: Diagnosis, Risk Factors, Treatment, and Prevention

1. Diagnosis of Leishmaniasis

Diagnosing leishmaniasis depends on the type of infection and may involve:

a) **Microscopic Examination**-Detecting *Leishmania* parasites in skin or tissue samples using Giemsa staining.

b) **Culture Tests**-Growing *Leishmania* in specialized media for confirmation.

c) **Molecular Tests (PCR)**-Identifying parasite DNA for high sensitivity and accuracy.

d) **Serological Tests**-Detecting antibodies (especially for visceral leishmaniasis).

2. Risk Factors for Leishmaniasis

Several factors increase susceptibility to *Leishmania* infection:

(i) **Geographical Location**-Common in tropical, subtropical, and Mediterranean regions (South America, Africa, Middle East, Asia).

(ii) **Poor Living Conditions**-Crowded housing, poor sanitation, and proximity to sand fly habitats.

(iii) **Weakened Immune System**-HIV/AIDS, malnutrition, or immunosuppressive therapy increases disease severity.

(iv) **Outdoor Activities**-Farmers, soldiers, travelers, and those working in endemic areas are at higher risk.

(v) **Climate Change & Deforestation**-Expanding sand fly habitats increase transmission.

3. Treatment of Leishmaniasis

Treatment depends on the form and severity of the disease:

- **Cutaneous Leishmaniasis**:

Local treatment measures are: Antiseptics, cryotherapy, or heat therapy. Systemic treatment: Pentavalent antimonials (sodium stibogluconate, meglumine antimoniate). There are some alternative drugs: Miltefosine, amphotericin B, or fluconazole in resistant cases.

- **Visceral Leishmaniasis (Kala-azar)**:

First-line drugs: Liposomal amphotericin B, miltefosine.

Second-line drugs: Pentavalent antimonials, paromomycin, or combination therapy.

Supportive care includes fluid management and treatment of secondary infections.

Mucocutaneous Leishmaniasis:Systemic treatment with amphotericin B, pentavalent antimonials, or miltefosine.

4. Prevention of Leishmaniasis

Since no universal vaccine exists, prevention focuses on controlling sand flies and reducing human exposure:

Vector Control:i. Use of insecticide-treated bed nets (ITNs).

ii. Indoor residual spraying with insecticides.

iii. Environmental management (clearing vegetation, controlling rodent populations).

Personal Protection:

iv. Wearing long-sleeved clothing and insect repellents (DEET, permethrin).

v Avoiding outdoor activities at dusk and dawn when sand flies are most active.

Public Health Measures:

vi. Early detection and treatment to prevent parasite reservoirs.

vii. Surveillance and control programs in endemic areas.

viii. Research on potential vaccines and ne

Tsetse Flies and Trypanosomiasis

Tsetse Flies (Glossina spp.)

Tsetse flies are blood-feeding insects found in sub-Saharan Africa. They belong to the *Glossina* genus and are primary vectors of **Trypanosomiasis** (sleeping sickness in humans). These flies thrive in warm, humid regions, particularly near rivers, forests, and savannas. Both male and female tsetse flies feed on blood, making them effective disease transmitters.

Trypanosomiasis (Sleeping Sickness)

Trypanosomiasis is caused by *Trypanosoma* parasites, which are transmitted through the bite of an infected tsetse fly.

1. Human African Trypanosomiasis (Sleeping Sickness)

Caused by:

- *Trypanosoma brucei gambiense* (chronic form, West & Central Africa)
- *Trypanosoma brucei rhodesiense* (acute form, East & Southern Africa)

Symptoms: Fever, headaches, swollen lymph nodes, muscle pain, sleep disturbances, confusion, coma, and death if untreated.

Sleeping Sickness (Human African Trypanosomiasis or HAT)

Sleeping sickness, or **Human African Trypanosomiasis**, is a protozoan parasitic disease caused by *Trypanosoma brucei* and transmitted through the bite of infected **tsetse flies** (*Glossina* spp.). The disease is fatal if left untreated and is endemic to **sub-Saharan Africa**, particularly in rural areas.

Causes and Types

Sleeping sickness is caused by two subspecies of *Trypanosoma brucei*:

1. Trypanosoma brucei gambiense (West & Central Africa)

This parasite causes **chronic** sleeping sickness.Symptoms develop slowly over months or years. The parasite is responsible for over **95% of cases**.

2. Trypanosoma brucei rhodesiense (East & Southern Africa)

The parasite causes **acute** sleeping sickness.Symptoms appear rapidly within weeks. This disease is less common but more severe.

Symptoms

Sleeping sickness progresses in two stages:

1. Early Stage (Hemolymphatic Phase)

i. Fever, headaches, muscle pain, swollen lymph nodes.
ii. Joint pain, weakness, and weight loss.

2. Late Stage (Neurological Phase)

i. Parasites invade the central nervous system (CNS).
ii. Sleep disturbances (daytime sleepiness, insomnia at night).
iii. Behavioral changes, confusion, mood swings.
iv. Seizures, paralysis, coma, and death if untreated.

Diagnosis:

a) **Microscopic Examination** of blood, lymph, or cerebrospinal fluid (CSF).
b) **Serological Tests** (Card Agglutination Test for Trypanosomiasis-CATT).
c) **Polymerase Chain Reaction (PCR)** for parasite DNA detection.

Treatment:

a) **Early Stage:** Pentamidine (*T.b.gambiense*), Suramin (*T.b. rhodesiense*).
b) **Late Stage:** Eflornithine, Melarsoprol (crosses blood-brain barrier).
c) **New Drug:** Fexinidazole (oral drug for *T.b.gambiense*).

Prevention & Control

a) **Vector Control:** Insecticide-treated tsetse traps, aerial spraying, and releasing sterile male flies.
b) **Personal Protection:** Wearing protective clothing, using insect repellents, and avoiding fly-infested areas.
c) **Public Health Measures:** Surveillance, early diagnosis, and treatment to prevent outbreaks.

Sleeping sickness is a major public health concern, but control programs by WHO and partners have significantly reduced cases, with the goal of **eliminating HAT as a public health threat by 2030**.

Control and Prevention

a) **Vector Control:** Insecticide-treated traps, aerial spraying, sterile insect technique (releasing sterile males to reduce populations).

b) **Personal Protection:** Wearing long clothing, insect repellents, and avoiding fly-infested areas.

c) **Treatment:** Drugs like pentamidine, suramin (early-stage), melarsoprol, and eflornithine (late-stage). For animals, drugs like isometamidium and diminazene are used.

d) **Surveillance & Early Diagnosis:** Screening at-risk populations to detect and treat infections before symptoms worsen.

Trypanosomiasis remains a major public health and economic challenge in Africa, requiring sustained control efforts.

I. **Vector Control:** Insecticide-treated tsetse traps, aerial spraying, and releasing sterile male flies.

II. **Personal Protection:** Wearing protective clothing, using insect repellents, and avoiding fly-infested areas.

III. **Public Health Measures:** Surveillance, early diagnosis, and treatment to prevent outbreaks.

Sleeping sickness is a major public health concern, but control programs by WHO and partners have significantly reduced cases, with the goal of **eliminating HAT as a public health threat by 2030**.

Unit-6 □ Ticks and Mites-General features; Differences between ticks and mites; Soft ticks and Hardticks

6.0 Objective

This unit clearly explains Ticks and mites, their general features, how they differ from each other, and the types of ticks. After completing the topic, readers will gain some knowledge about the *ticks and mites belonging to the class Arachnida.*

6.1 Introduction

Ticks and mites are types of arachnids, making them distantly related to spiders. They belong to the same class of organisms called Arachnida and order Acarina. Ticks can transmit several pathogens and cause direct damage (e.g., blood depletion, skin lesions, and paralysis) to their host. Mites can cause lesions in the skin (e.g., mange-causing mites) of their hosts and affect their respiratory tract.

6.2 General Features

Mites and ticks belong to one of the most diverse groups of all arachnids and can range in size from a minute up to 1 centimeter. Due to their diverse appearance a general description is difficult to give, however, all have the following characteristics:

- 4 pairs of legs, although some juveniles only have 3 pairs gaining a fourth pair with their first molt.
- No external segmentation of the abdomen, individuals appear as a single body mass.

- Never have antennae
- Life span varies from 2-20 years
- Males die after fertilizing females.

6.2.1 Differences between ticks and mites

- The most obvious difference between ticks and mites is their size. Ticks can be seen with the naked eye and are generally one-millimeter-long, but can expand up to three centimetres in length after feeding. Mites are microscopic creatures that are generally less than a millimeter and are difficult to see with the naked eye.
- Mites are found in almost all habitat types including terrestrial, freshwater, and marine environments. Terrestrial mites are commonly found in soil or leaf litter, under the bark of trees or feeding on the leaves and stems of plants. Most ticks are only discovered after they have attached themselves to a host animal. Both mites and ticks can be found living as parasites of other invertebrate and vertebrate animals.
- Ticks have short hair or none at all on their bodies, while mites have long hair on their bodies.
- The hypostome is the structure on ticks and mite's capitulum that allows them to attach to their host.
- Most species of mites are predatory and will feed on a variety of small invertebrates, while others are more herbivorous and often feed on plant sap, sometimes causing damage to crops and garden plants. Ticks are adapted to feeding on the blood of vertebrate animals such as humans, dogs, or livestock.

6.2.2 Reproduction and Life cycle

- Reproduction in mites and ticks is very variable with some species mating through the direct transfer of sperm via coupling of the genital regions. Other species transfer sperm indirectly with the male placing a sperm droplet on the genital opening of the female with his legs.
- After fertilisation the female will usually lay her eggs in the substrate where they are left to hatch. The first free-living stage is called a larva

and has only 3 pairs of legs. After the first moult, it will gain its fourth pair of legs and will molt several more times before becoming a mature adult.

Ticks **Red-legged earth mite**

6.3 Soft ticks and Hard ticks

- Order Acarina has two families

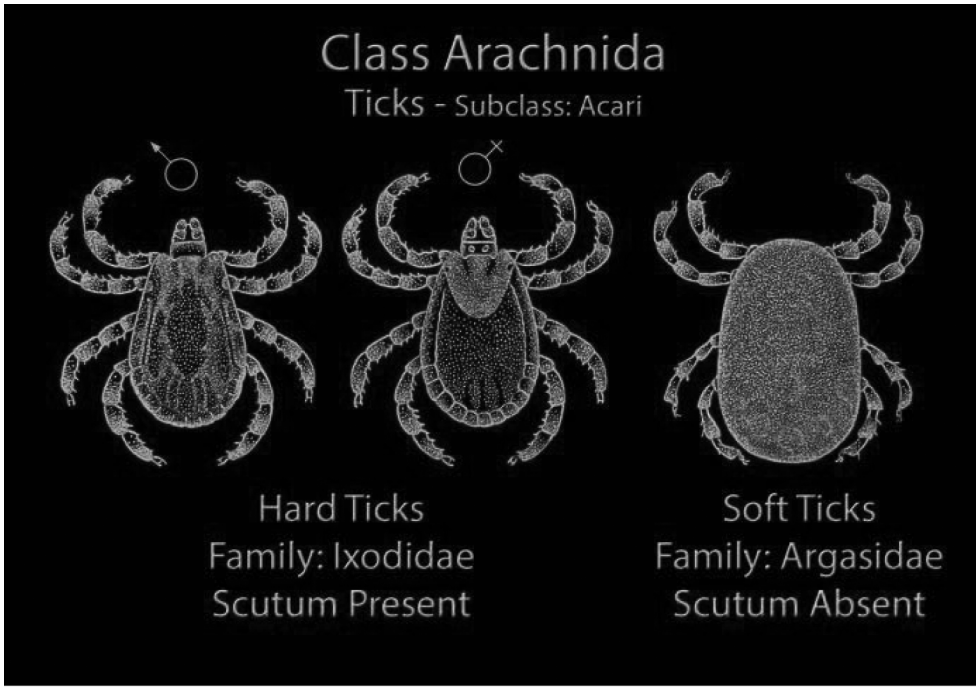
IXODIDAE : Hard tick

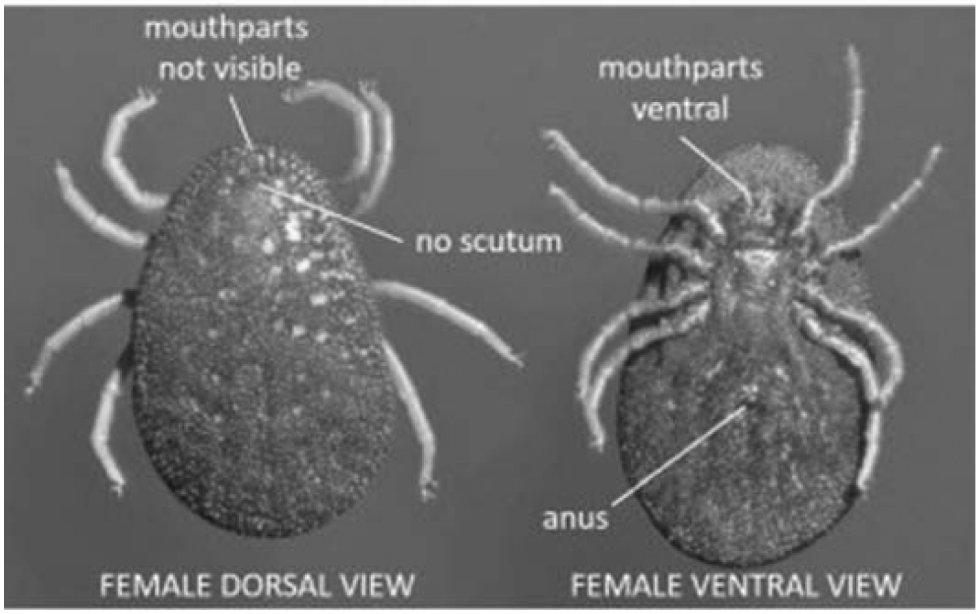
ARGASIDAE: Soft tick

- **HARD TICK (Ixodidae)**

Around 700 species in 20 genera. It has a Sac-like body varying in size from 2-10 mm when unfed and almost double when fed. They are dorsoventrally flattened.

- The male has a chitinized shield (SCUTUM). The capitulum or false head projects from the body and is visible from above differentiating these hard ticks from soft ticks.
- In females, only the anterior dorsal surface is chitinized, accommodating large blood meal.
- The genital opening of both sexes is close to the base of mouthparts on the ventral surface. Gravid females often lay around 20000 eggs and die. Egg hatch within a few days giving rise to 6-legged larvae. It waits for the host. Once on the host, it crawls to the soft areas like the ear and axillary region. It takes a blood meal and moults to form Nymphs.
- The species that leave the host at each stage of moulting (Larva-Nymph & Nymph-Adult) are called 3 host Ticks because they parasitize three hosts as adult, larva and nymph. Example :*I.ricinus and D. andersoni*.


Class Arachnida
Ticks - Subclass: Acari
Hard Ticks
Family: Ixodidae
Scutum Present
Soft Ticks
Family: Argasidae
Scutum Absent


mouthparts
not visible
no scutum
mouthparts
ventral
anus
FEMALE DORSAL VIEW
FEMALE VENTRAL VIEW

- Some ticks remain on the same host after first moulting i.e. from larva to nymph but change host from nymph to adult are called 2 host ticks. e.g*Hyalomma*and *Rhipicephalus*.
- Tocks that do not leave the host at the end of adult life are called single host tick. e.g. *Boophilus sp*p.

6.3.1 SOFT TICK (ARGASIDAE):

- Any of various small parasitic arachnids of the family Argasidae, typically living on the skin of warm-blooded animals and feeding on the blood and tissues of their hosts. They lack a hard dorsal chitin shield (Scutum) The integument is soft and leathery and is round in shape.

Important Species:*Ornithodorusmoubata*. It is vector of tick borne relapsing fever caused by *Borrelia duttoni*.

Unit-7 □ Tick-borne diseases-Rickettsiosis and Tick-borne encephalitis

7.0 Objective

This unit describes tick-borne diseases. After completing the topic, readers will have some depth of knowledge regarding rickettsiosis and tick-borne encephalitis.

7.1. Introduction

Tick-borne diseases and conditions are transmitted through the bite of an infected tick. These include Alpha-gal syndrome (AGS), Lyme disease, Anaplasmosis, Ehrlichiosis, Babesiosis, Powassan (POW), Rocky Mountain Spotted Fever, and Tularemia. Ticks can be infected with bacteria, viruses, or parasites. When an infected tick bites the human host, the human may become infected. Ticks can spread bacteria, viruses, and parasites (pathogens) that cause human diseases.Many tick-borne diseases can have similar signs and symptoms. Ticks transmit pathogens that cause disease through the process of feeding.

7.2 Rickettsiosis

Rickettsia is a group of vector-borne organisms that cause acute febrile illnesses throughout the world. While the clinical presentation of rickettsial infection is similar, the causative species and epidemiology can vary depending on the region. It is important to recognize both the typical symptoms and the epidemiology of a given region to correctly diagnose and treat these infections promptly, as they can be associated with significant morbidity and mortality.

7.2.1 Epidemiology

Rickettsia is typically vector-borne, transmitted by ticks, body lice, mites, and fleas. In most cases, humans are thought to be accidental hosts. The transmitting ticks vary depending on the region and organism, with Dermacentor variabilis (American dog tick), Dermacentor andersoni (Rocky Mountain wood tick), and Amblyommaamericanum (Lone Star tick) associated with most cases of Rocky Mountain spotted fever in the United States.

Alternatively, Amblyomma cajennense has been associated with spotted fever in South America. Because of the association with ticks and other vectors, infections with rickettsiae are more commonduring warmer months and in people exposed to outdoor activities. Epidemic typhus, R. prowazekii, is transmitted by body lice and is associated with crowded conditions and poor hygiene. Murine typhus (R. typhi) is most commonly reported in tropical and subtropical areas and is associated with flea bites.

7.2.2 How rickettsia infections are spread

Rickettsiae are usually injected directly from the saliva of ticks and mites as they feed on humans and, in the case of fleas, by contamination of bite sites by faeces.

7.2.3 Signs and symptoms of rickettsia infections

There is great variation in the range and severity of symptoms experienced. Commonly a small, hard, black sore (called an eschar) first appears at the bite site where the infection was introduced.

Other typical symptoms may include:

- Fever
- headache
- muscle aches
- swollen lymph glands
- cough
- rash.

Less common severe infections can be associated with confusion and breathing difficulties.

7.2.4 Diagnosis of rickettsiainfections

These infections are not common and usually mild they can be difficult to diagnose. While signs and symptoms can suggest the diagnosis, a definite diagnosis is made with a blood test or skin biopsy (for example a skin sample) of the bite site.

Incubation period

(time between becoming infected and developing symptoms)Between 3 to 10 days.

Infectious period

(the time during which an infected person can infect others)Infections are not transmitted from person to person.

7.2.5 Treatment for rickettsia infections

Treatment is usually with the tetracycline antibiotic doxycycline which reduces the duration and severity of infection.

Prevention of rickettsia infections

- Exclusion of people with rickettsia infections from childcare, preschool, school, and work is not necessary.
- There is no vaccine available to prevent infection.
- Wear long-sleeved protective clothing and a broad-brimmed hat to reduce the risk of infection when undertaking activities where human contact with ticks, lice, mites, or fleas may occur, such as bushwalking and camping in infected areas.
- Use an insect repellent containing DEET or picaridin and examine your skin for possible bites (especially behind the ears, on the back of the head, in the groin, armpits, and behind the knees) following these activities.

7.3 Tick-borne encephalitis

Tick-borne encephalitis (TBE) is a human viral infectious disease involving the central nervous system, and occurring in many parts of Europe and Asia. The virus is transmitted by the bite of infected ticks, found in woodland habitats.

TBE is most often manifested as a two-phased illness. The first phase is associated with symptoms like fever, fatigue, headache, muscular aches, and nausea. The second phase involves the neurological system with symptoms of meningitis (inflammation of the membrane that surrounds the brain and spinal cord) and/or encephalitis (inflammation of the brain).

Like other tick-borne infectious diseases, the risk from TBE can be reduced by using insect repellents and protective clothing to prevent tick bites. A vaccine is available in some disease-endemic areas.

7.3.1 Pathogen

Tick-borne encephalitis (TBE) is a viral infectious disease that attacks the central nervous system and can result in long-term neurological symptoms, and

even death. Tick-borne encephalitis is caused by a virus (Flavivirus genus, family Flaviviridae) which includes three subtypes:

1. European subtype, transmitted by Ixodes ricinus ticks, endemic in rural and forested areas of central, eastern and northern Europe;
2. Far eastern subtype, transmitted mainly by I. persulcatus, endemic in far-eastern Russia and forested regions of China and Japan; and
3. Siberian subtype, transmitted by I. persulcatus, endemic in the Urals region, Siberia and far-eastern Russia, and also in some areas in north-eastern Europe.

TBE has become a growing public health challenge in Europe and other parts of the world. The number of human cases of TBE in all endemic regions of Europe has increased by almost 400% in the last 30 years; the risk areas have spread and new foci have been discovered.

7.3.2 Mode of transmission:

The TBE virus is transmitted by the bite of infected ticks. Humans may acquire infection by consumption of infected unpasteurized dairy products. The TBE virus is not directly transmitted from human to human, apart from the possibility of transmission from an infected mother to a breastfed child. Laboratory accidents from needle-stick injuries or those associated with aerosol infection have been reported.

Infected ticks can be found in woodland habitats—deciduous forests and transition zones between forests and grasslands. When infected, ticks can transmit the virus throughout their life (mainly nymphs and adults). Tick activity and life cycle depend on climatic factors (temperature, soil moisture, and relative humidity). Wet summers and mild winters tend to increase tick population density.

7.3.3 Prevention measures

TBE virus infection can be prevented by avoiding tick bites through the following methods:

1. vaccination against TBE (inactivated vaccine) is considered to be the most effective means of preventing TBE in endemic countries;
2. application of tick repellents;
3. wearing protective clothing, with long sleeves and long trousers tucked into socks treated with an appropriate insecticide
4. inspecting the body for ticks after outdoor activities and removing ticks with tweezers or forceps; and

By avoiding consumption of unpasteurized dairy products in risk areas.

Unit-8 □ Fleas, Mites, and Lice-Rat flea, Itch mite, Lice born disease-disease relationship and controlmeasures

8.0 Objective

This unit particularly involves the study of Fleas, Mites, and Liceas well as lice-borne diseases and their relationship with control measures. After completion of the whole topic, readers will be acquainted with Fleas, mites, and their mode of transmission of diseases.

8.1 Introduction

Fleas and lice are different parasitic insects that can cause an itchy reaction on the skin. Though similar, they possess unique traits. For example, fleas can jump, but lice can only crawl.

Fleas are wingless parasitic insects that consume the blood of a host animal, including humans. They move from one host to another through jumping.

Head lice are another form of parasitic insect that feeds on human blood. They also do not have wings and cannot fly. Instead, they move from host to host through crawling. Some people refer to them simply as “lice.” Besides being much smaller, mites have a generally rounded body shape and lack any obvious body segmentation. Also, mites are arachnids, not insects, so an adult mite has eight legs while an adult louse (an insect) has only six. Mites are not as host-specific as lice and may parasitize many animal species.

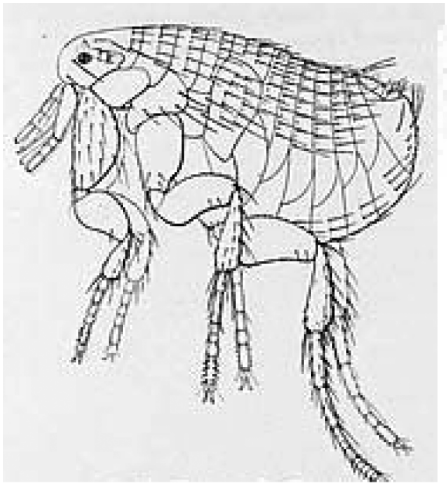
8.2 Rat flea

The rat flea (*Xenopsyllacheopis*) is a small parasite that feeds on the blood of rodents. They are known carriers of a variety of diseases and are considered the main vector of bubonic plague. Infection is transmitted after a flea feeds from an infected rodent and then bites a human.

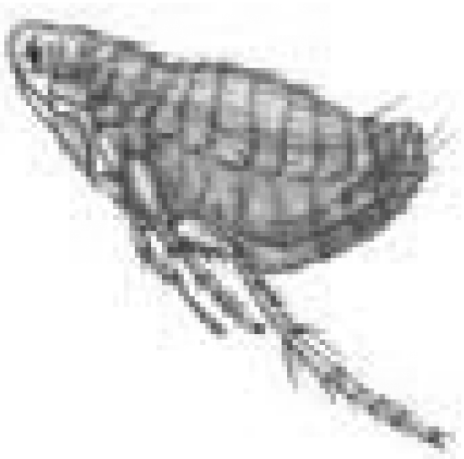
Rat fleas begin as white eggs, which drop from the female and hatch on the ground or are laid on the ground in the animals bedding. Emerging larvae are approximately 3 to 5 mm in length and appear similar to small, legless worms.

Unlike adult rat fleas, larvae do not consume blood, but instead eat flea droppings, dead skin cells and animal hair. Larvae spin white, silken cocoons within which they pupate. After emerging from the pupae, rat fleas are capable of drawing blood and reproducing. Adults can live up one year and prefer to inhabit warm environments.

Adult rat fleas have two eyes but are only able to register light. The mouth of the rat flea is used to inject saliva and draw blood. Fleas are incapable of flight, but can jump up to 200 times the length of their bodies and 130 times their own height.



Xenopsyllacheopis (Male)



Xenopsyllacheopis(Rat Flea)

8.2.1 Physical Description

Adult *Xenopsyllacheopis* are about 1.5 to 4mm in length and have a laterally compressed body. Like all fleas, *X. cheopis* adults are wingless. Adults vary from light brown to dark brown to camouflage themselves in the host's fur. Adult *Xenopsyllacheopis* lack both genal and pronotalctendium (combs of bristles in the front and back). Males and females are sexually dimorphic.

Females have dark-colored spermatheca that resemble small sacs, a distinguishing characteristic of this species. Males have complex genitalia that are easily distinguishable from females. Larvae are 4.5 mm long and resemble worms; they are slender, white, eyeless, and legless. Each has fourteen bristled segments. During the last larval instar, they molt and form cocoons that are silky and covered in debris from their surroundings.

8.2.2 Development

Fleas are holometabolous, which means they go through four life-cycle stages: egg (embryo), larva, pupa, and adult (imago). Eggs normally incubate for about two to twelve days. *Xenopsyllacheopis* passes through three molts during the larval stage, which usually lasts about nine to fifteen days, but can last up to 200 days in unfavorable conditions. Next, the larva spins a silk cocoon where it remains until it is finished pupating. During the pupal stage, the flea's development rate is greatly affected by its surroundings. Changes in temperature and humidity outside the cocoon can inhibit emerging for up to a full year.

8.3 Itch mite

Sarcoptesscabiei var. *hominis*, the human itch mite, is in the arthropod class Arachnida, subclass Acari, family Sarcoptidae. The mites burrow into the upper layer of the skin but never below the stratum corneum. The burrows appear as tiny raised serpentine lines that are grayish or skin-colored and can be a centimeter or more in length. Other races of scabies mites may cause infestations in other mammals, such as domestic cats, dogs, pigs, and horses. It should be noted that races of mites found on other animals may cause a self-limited infestation in humans with temporary itching due to dermatitis; however, they do not multiply on the human host.

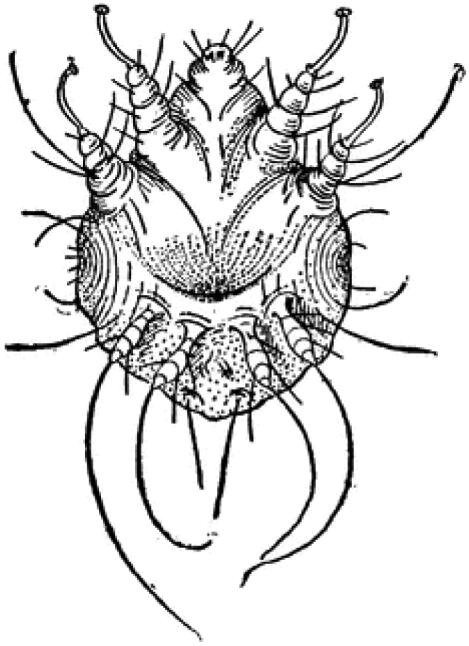
8.4 life cycle

Sarcoptesscabiei undergoes four stages in its life cycle: egg, larva, nymph and adult. Females deposit 2-3 eggs per day as they burrow under the skin . Eggs are oval and 0.10 to 0.15 mm in lengthand hatch in 3 to 4 days.

After the eggs hatch, the larvae migrate to the skin surface and burrow into the intact stratum corneum to construct almost invisible, short burrows called molting pouches. The larval stage, which emerges from the eggs, has only 3 pairs of legsand lasts about 3 to 4 days. After the larvae molt, the resulting nymphs have 4 pairs of legs. This form molts into slightly larger nymphs before molting into adults. Larvae and nymphs may often be found in molting pouches or hair follicles and look similar to adults, only smaller.

Adults are round, sac-like eyeless mites. Females are 0.30 to 0.45 mm long and 0.25 to 0.35 mm wide, and males are slightly more than half that size. Mating occurs after the active male penetrates the molting pouch of the adult female.

Mating takes place only once and leaves the female fertile for the rest of her life. Impregnated females leave their molting pouches and wander on the surface of the skin until they find a suitable site for a permanent burrow. While on the skin's surface, mites hold onto the skin using sucker-like pulvilli attached to the two most anterior pairs of legs. When the impregnated female mite finds a suitable location, it begins to make its characteristic serpentine burrow, laying eggs in the process.



Itch mite (*Sarcoptesscabiei*)

After the impregnated female burrows into the skin, she remains there and continues to lengthen her burrow and lay eggs for the rest of her life (1-2 months). Under the most favourable of conditions, about 10% of her eggs eventually give rise to adult mites. Males are rarely seen; they make temporary shallow pits in the skin to feed until they locate a female's burrow and mate.

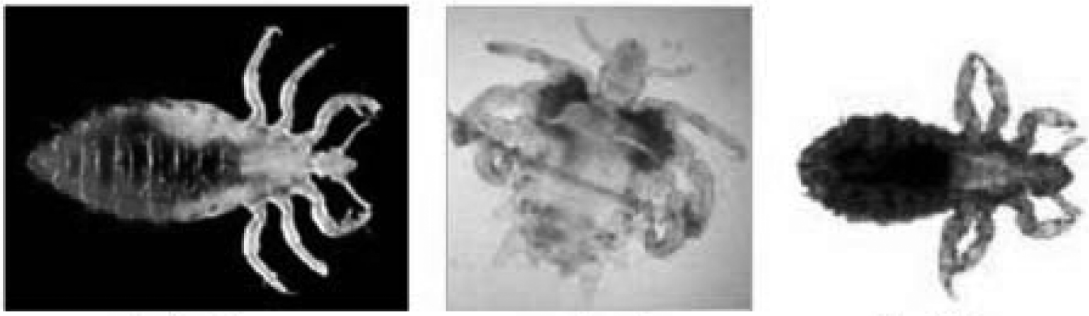
8.4.1 Transmission

Transmission occurs primarily by the transfer of the impregnated females during person-to-person, skin-to-skin contact. Occasionally transmission may occur via fomites (e.g., bedding or clothing). Human scabies mites often are found between the fingers and on the wrists.

8.5 Lice:

Lice infesting only humans belong to the sucking lice. Their mouthparts are anatomically distinct from those of chewing lice that are found in association with the feathers of birds or the fur of mammals.

Three louse species infesting humans are known, the head louse (*Pediculus humanuscapitis*) infesting human heads , the body louse (*P. humanushumanus*) infesting undergarments or clothing in contact with a human body, and the pubic louse (*Pthirus pubis*) mainly infesting the pubic hair.



Body Lice Pubic Lice Head Lice

8.5.1 Description:

The head louse and the body louse are almost morphologically indistinguishable from each other, though the former sucks blood more frequently, has slender body, and is slightly smaller in size. Adult head louse measures 2-3 mm in length (3 mm for female and 2-3 mm for male) and is ash-colored; the intestine is seen black on account of the sucked blood. It spends its life from the nymph to the adult holding onto the hair with hook-like claws found at the end of each of its 6 legs. Each female lays about 100 eggs in a month laying 3-4 eggs per day. The egg hatches and releases the nymph in a week, which sucks the blood at an interval of several hours. It sheds its skin three times and becomes an adult in 2 weeks. The infestation starts with a small number of lice but attains a large number through repeated ovulation. The saliva excreted during sucking sensitizes the host and causes unbearable itching about a month after the start of the infestation. The head louse is found throughout the year, though there are two epidemic peaks in a year, June-July and November.

The body louse is a size larger than the head louse. The female louse lays eggs along the seam of clothes and underclothes, stays there with its nymphs, and moves to the human body to suck the blood.

The pubic louse is 1-2 mm in length. It is morphologically different from the above two louse species, and often called "crab louse" on account of its morphology. It infests mainly pubic hairs but occasionally eyelashes of children (*Phthiriasispalpebrarum*). In such cases, infestation of the louse in the family should be suspected, and the carriers should be appropriately treated.

8.5.2 Modes of transmission of lice

The head louse is transmitted in a family or among children directly through contact of heads or indirectly through shared use of bedclothes, pillowcases, towels, caps, combs, brushes, lockers, etc. To avoid transmission, such practices should be avoided.

Infestation of body lice is often found among old persons living alone in a small room or persons without housing (a homeless wearing clothes infested by more than 1,000 body lice has been found among those who wore the same clothes for more than a month). Very often homeless persons acquire the body lice through clothes when they pick them up early autumn.

The pubic louse is transmitted through sexual contact. So, it is considered as a sexually transmitted disease. Chronically infested persons generally do not experience itching. Only newly infested persons experience intense itching several weeks after the acquisition, and come to know that the louse infested them.

8.6 Lice born disease-disease relationship and control measures

Due to their blood-feeding behavior, body lice can transmit a great variety of diseases, such as epidemic typhus (caused by *Rickettsia prowazekii*), louse-borne relapsing fever (caused by *Borrelia recurrentis*), or trench fever (caused by *Bartonella quintana*)

Lice generally have a high host-specificity, and within a host species there is sometimes also a specificity to certain body regions. They are widely distributed across the globe. Lice can be divided into two main groups: sucking lice (Anoplura) and chewing lice (Mallophaga). All active stages of lice are parasitic in warm-blooded vertebrates (birds and mammals). The two groups differ in their feeding habits, with Mallophaga feeding on skin and skin products and Anoplura generally feeding on blood. The groups can be easily differentiated morphologically. Anoplura is an important pest and disease vector affecting both public and veterinary health. Mallophaga are important pest species for animals, but not for humans.

8.6.1 Risk factors

There are certain risk factors for lice-borne diseases, especially when poor hygienic conditions prevail, such as in refugee camps, jails, and among homeless people. Lice are more likely to spread in crowded conditions and more likely to spread during social disruption, such as during war or famine.

Lice are spread by direct contact with an infested person or by sharing personal items, particularly those that touch the head, such as combs, brushes, hats, and hair accessories.

8.6.2 Diagnosis

There is no laboratory test to confirm body lice. Body lice can be diagnosed by finding eggs and crawling lice in the seams of clothes. Sometimes a body louse can be seen crawling on the skin during feeding.

8.6.3 Treatment

Wash clothing, bedding, and towels using hot water and dry with high heat. Improve personal hygiene for the infested person. Medicated lotions can be used on the body as well.

8.6.4 Prevention

Reduce the risk of catching body lice by bathing regularly, changing and washing clothes, and avoiding potentially infested bedding.

8.7 Control Measures

In the case of humans, the management of lice requires differing treatment methods, depending on the species. Delousing is considered the best way to control louse-borne diseases. To control the body lice infestation, regular showering or bathing together with regular washing and changing of clothes and linen/blankets is essential, particularly in crowded places such as refugee camps, shelters for the homeless, and prisons.

Confirmed cases of body louse-borne infections should be immediately treated using antibiotics. To control head lice, mechanical removal of lice and nits is necessary, combined with treatment of the scalp using various pediculicides. As products are generally only effective against adults and nymphs, treatment should be repeated after 10”14 days. Although certain products claim to have an ovicidal activity, this is generally limited, and a second treatment is recommended for all products, except for the topical 0.5% ivermectin .

Scalp shaving is also an efficient method of control and pubic lice can also be controlled by combing or shaving the pubic hair. An effective way to remove lice from clothes and linen is to heat them (i.e. air-drying) to 60°C or wash them for 30 minutes at 55°C, or for a shorter period at a higher temperature.

Unit-9 □ Forensic Entomology-A brief idea and status of Forensic Entomology, Subfields of forensic entomology

Forensic entomology is an emerging field in forensic sciences,that deals with the application of insects and arthropods in civil and criminal case investigation.This branch plays an important role in analyzing the stages of body decomposition thus helping to answer questions of the law enforcement agencies related to death.By studying the insect population and the developing larval stages, forensic scientists can estimate the postmortem index, any change in position of the corpse as well as the cause of death. Now a daysforensic entomology have to offer and use it as an adjunct to the conventional means of forensic investigation.

Apart from an early case report from China (13th century) by Sung Tzu and later artistic contributions, the first observations on insects and other arthropods as forensic indicators were documented in Germany and France during mass exhumations in the late 1880s by Reinhard and Hofmann, whom are proposed recognizing as co-founders of the discipline.However, only in the last 30 years has forensic entomology been systematically explored as a feasible source for evidence in criminal investigations.

Forensic Entomology subfields:

Urban Forensic Entomology: It concerns with litigations arising from Bedbugs and Termites affecting manmade structures like buildings, gardens etc. and other aspects of human environment.

Stored-Product Forensic Entomology: It covers the litigations arising from grains, flour, and packaged meals contamination by insects.

Medico-Legal Forensic Entomology: It involves the analysis of necrophagous insects to gain insight into Time of Death (TOD) of various incidents such as murder, suicide, rape, physical abuse and contraband trafficking.

Unit-10 □ Forensically relevant insects and other arthropods-tools used and case studies

Forensically relevant insects and other arthropods play a critical role in forensic entomology, the study of insects and their developmental stages to aid in legal investigations, particularly in estimating time of death. These organisms are among the first to colonize a decomposing body, providing vital clues about the postmortem interval (PMI) and the circumstances surrounding death. Here's an overview of the key groups of insects and other arthropods that are commonly involved in forensic cases:

1. Blow Flies (Family: Calliphoridae)

Blow flies are the most significant and commonly studied insects in forensic entomology due to their quick response to decomposing tissue. Adult blow flies are attracted to the odors of decaying flesh and will lay eggs within hours or days of death. The eggs hatch into larvae (maggots) that feed on the tissue. The life cycle of blow flies, from egg to adult, is temperature-dependent and can be used to estimate the PMI. Key species include:

- **Lucilia sericata** (common green bottle fly)
- **Calliphoravicina** (bluebottle fly)
- **Phormiaregina** (black blow fly)

2. Flesh Flies (Family: Sarcophagidae)

Flesh flies are another important group, though less common than blow flies. They typically deposit live larvae directly onto the corpse, which accelerates the decomposition process. Their maggots also provide clues about PMI, but their developmental timing differs from blow flies, making them an additional reference for investigators.

3. House Flies (Family: Muscidae)

While house flies are less commonly involved in decomposition than blow flies or flesh flies, they can still colonize a body, especially indoors. The common house fly (**Musca domestica**) lays eggs on both fresh and decomposed material, and its developmental stages can also be used to estimate PMI.

4. Beetles (Order: Coleoptera)

Beetles often arrive after flies in the decomposition process and play a significant role in the later stages of decay. They feed on both decaying tissue and other insects. Forensically relevant beetles include:

- **Dermestidae** (skin beetles): These beetles consume dried flesh and hair in the final stages of decomposition.
- **Silphidae** (carrion beetles): These scavengers feed on both the decaying corpse and maggots, influencing decomposition rates.
- **Staphylinidae** (rove beetles): They are commonly found in various stages of decomposition, often feeding on larvae of other insects.

5. Wasps and Ants (Order: Hymenoptera)

Various wasps and ants are known to be predators or scavengers on decomposing bodies. Wasps often feed on fly eggs and larvae, impacting the succession of insect species and thus influencing PMI estimations. Ants may feed on maggots and can even delay the colonization of other insects, making them relevant in forensic analyses.

6. Moths (Order: Lepidoptera)

Though less common, certain species of moths, particularly from the family **Tineidae** (clothes moths), may be found feeding on hair or other keratin-based materials in the later stages of decomposition.

7. Mites (Subclass: Acari)

Mites are arthropods that can be found in various stages of decomposition. Their presence can provide insight into the environment of the body and how long it has been exposed to certain conditions. Some mites feed on the decomposing tissue itself, while others are predators of insect larvae, which can further complicate the timeline of colonization.

8. Cockroaches (Order: Blattodea)

Cockroaches, particularly in indoor environments, may feed on decaying organic matter, including human remains. They are generally not primary decomposers but can still provide relevant forensic evidence, particularly in cases where a body has been left unattended in a building.

9. Spiders and Other Predators

Spiders, centipedes, and other arthropod predators may be found near decomposing bodies, feeding on insects attracted to the corpse. Their presence does not directly impact PMI estimation, but they can influence the local insect population and patterns of decomposition.

Role of Arthropods in Forensic Investigations

In addition to providing crucial information for PMI estimation, arthropods can offer insights into other aspects of a forensic investigation:

- **Geographical Location**: The presence of certain insect species can indicate whether a body has been moved postmortem or the location of death based on the known distribution of species.
- **Time of Death**: The progression of insect colonization stages allows forensic entomologists to establish a timeline, helping investigators approximate the time of death.
- **Toxicology**: Insects that feed on decomposing tissue may ingest any toxins or drugs present in the body, allowing for chemical analysis to detect the presence of substances in cases where human tissue is unavailable.

Tools used

Detecting and analyzing forensically relevant insects requires a range of tools and techniques to accurately assess the postmortem interval (PMI) and other factors relevant to a death investigation. Forensic entomologists use various **field, laboratory, and analytical tools** to collect, preserve, and examine insect evidence.

1. Collection Tools

In the field, forensic entomologists rely on basic collection tools to gather insect evidence from a body and its surrounding environment.

- **Forceps and Tweezers**: These are used to carefully collect insects, larvae, and eggs from the body without damaging them.
- **Insect Nets**: Used to capture adult flying insects (like blow flies) near the body.
- **Hand Lens**: A magnifying lens to inspect small insect larvae or eggs at the crime scene.

- **Insect Traps**: Sticky traps or baited traps are often used to catch live adult insects that are attracted to the body.
- **Specimen Containers and Vials**: Collected insects and larvae are stored in labeled vials for transport to the laboratory. Some specimens are preserved in **70-95% ethanol** to halt decomposition, while others are kept alive for developmental studies.

2. Temperature and Environmental Measurement

Because insect development is influenced by environmental factors, particularly temperature, forensic entomologists must gather detailed environmental data from the crime scene.

- **Thermometers and Temperature Loggers**: Used to record air temperature at the crime scene, as well as the temperature around the body and in the soil. Data loggers can continuously record temperatures over time, providing a detailed environmental profile.
- **Weather Data**: Local weather data (from nearby stations) is often obtained to correlate temperature fluctuations and estimate the impact on insect growth.

3. Laboratory Tools

In the lab, collected insect samples undergo further analysis to establish their species and stage of development.

- **Microscopes**: High-powered **dissecting microscopes** are used to identify the species of larvae or adult insects by closely examining their physical characteristics. This is crucial, as different species develop at different rates.
- **Scanning electron microscopy:** a technique that can use scanning electron microscopy (SEM) to identify key morphological features of eggs and maggots
- **Gene expression studies:** This is particularly useful in determining developmental stages that are not evidenced by change in size; such as the egg or pupa and where only a general time interval can be estimated based on the duration of the particular developmental stage. This is done by breaking the stages down into smaller units separated by predictable changed in gene expression.

- **DNA Analysis (PCR)**: In cases where visual identification is difficult, **polymerase chain reaction (PCR)** or other genetic techniques may be used to sequence the insect's DNA and accurately identify the species.
- **Developmental Rearing Chambers**: Some insect samples are kept alive and placed in temperature-controlled chambers to rear them through their full developmental cycle. Observing how long it takes for larvae to reach adulthood under controlled conditions helps estimate PMI more accurately.

4. Analytical Tools

Several models and software are used to analyze insect evidence and estimate the postmortem interval.

- **Accumulated Degree Hours (ADH)** and **Accumulated Degree Days (ADD)**: These methods calculate the total heat energy available to insects during their development. Using temperature data from the crime scene and known developmental rates of insect species, forensic entomologists can estimate the PMI.
- **Developmental Models and Growth Charts**: Databases and growth charts for different insect species are used to track their life cycles under varying conditions. These allow scientists to backtrack from the insect's current stage of development to estimate when colonization began.

Notable Case Studies

1. Case of Danielle Van Dam (2002, San Diego, California)

In this high-profile case, forensic entomology was crucial in determining the time of death of a seven-year-old girl. Insect evidence, particularly blow fly larvae, was collected from the body. By analyzing the developmental stage of the larvae and comparing it with environmental temperatures, forensic entomologists provided an estimated PMI. This evidence supported the prosecution's timeline and contributed to the conviction of David Westerfield.

2. The Caylee Anthony Case (2008, Orlando, Florida)

In the murder of two-year-old Caylee Anthony, insect evidence played a controversial role. Investigators found maggots in the trunk of her mother Casey Anthony's car. Forensic entomologists testified that the presence of these maggots suggested the decomposition of a human body in the trunk. Though the evidence

was debated in court, it highlighted how insects can link a body to a crime scene, even in cases where the body is not directly found.

3. The Body in the River Case (Europe, 2000s)

A body was discovered in a river, and forensic entomologists were called to determine the time of death. The unique insect species found on the body, including aquatic beetles and fly larvae, indicated that the body had been submerged in water for several days. The forensic team used this information to refute an initial estimate of PMI based solely on decomposition and helped establish the correct timeline.

4. The Body in the Desert (1970s, Nevada)

In a famous case, a partially decomposed body was found in the Nevada desert, with a large number of blow flies present. By analyzing the fly larvae and correlating the insect's development with temperature data, forensic entomologists concluded that the body had been exposed for a shorter time than initially thought. This led to a revised timeline of the victim's death and helped exonerate a suspect who had an alibi for that period.

Unit-11 □ Insect succession on corpse; Post-mortem interval (PMI) estimation

The **succession of arthropods in decomposition** follows a predictable and staged process, with different species of insects and arthropods arriving at specific times based on the body's condition and environmental factors. This succession is crucial in forensic entomology, as it helps estimate the postmortem interval (PMI) and provides insights into the timeline and circumstances of death.

1. Fresh Stage (0-3 Days Postmortem)

- **Initial colonizers**: Blow flies (family: Calliphoridae) are typically the first insects to arrive, often within minutes or hours after death. These flies are attracted to the gases released by the body, primarily those associated with the breakdown of soft tissues.
- Blow flies lay their eggs on natural body openings (e.g., mouth, nose, eyes) or wounds. These eggs hatch into larvae (maggots) within hours. The presence of blow flies is a strong indicator that a body has not been moved, as they are among the most consistent early colonizers.
- In some cases, **flesh flies** (family: Sarcophagidae) may also arrive early, though they tend to deposit live larvae instead of eggs, hastening the decomposition process.

2. Bloat Stage (3-6 Days Postmortem)

- As the body enters the bloat stage due to gas buildup from bacterial activity, the smell intensifies, attracting more insects.
- **Blow fly larvae** (maggots) actively feed on the tissue, accelerating decomposition. During this time, **house flies** (family: Muscidae) may also join in colonization, particularly if the body is indoors or in an urban environment.
- **Flesh flies** continue to lay larvae, and their maggots mix with those of blow flies. The competition between maggots from different species creates an intense feeding frenzy, speeding up the decay.

3. Active Decay Stage (5-11 Days Postmortem)

- This is one of the most intense periods of decomposition, characterized by the breakdown of soft tissues, fluid release, and strong odors. **Blow fly maggots** are in their most active feeding stage.
- At this point, **carrion beetles** (family: Silphidae) begin to arrive. These beetles feed on decaying flesh but also on fly larvae, which they see as competitors. Their presence indicates that the body is in an advanced state of decomposition.
- **Rove beetles** (family: Staphylinidae) and **Hister beetles** (family: Histeridae) also appear during this phase, feeding on both the body and the maggots.

4. Advanced Decay Stage (10-25 Days Postmortem)

- As most soft tissue has been consumed by maggots and other decomposers, the body dries out, and the rate of decomposition slows significantly. Fewer blow flies and house flies are present as they finish their life cycles and leave the body.
- **Beetles** dominate this stage. **Dermestid beetles** (family: Dermestidae) are particularly common, as they specialize in feeding on dry, desiccated remains such as skin, ligaments, and hair. Their arrival signals that the body has been exposed for a longer period.
- **Cheese flies** (family: Piophilidae) may also appear during this stage, attracted to the drier, protein-rich tissue left behind.

5. Dry/Skeletal Stage (25+ Days Postmortem)

- Once all the soft tissue has been consumed, the body consists primarily of bones, cartilage, and dry skin. The insects that now dominate are those that specialize in feeding on dry, tough materials.
- **Dermestid beetles** remain prominent, consuming hair, tendons, and dried skin. Their presence, along with that of **clothes moths** (family: Tineidae), which feed on keratin, indicates that decomposition has reached its final stages.
- Certain mites (order: Acari) also begin to appear, feeding on any remaining organic material. Their presence can further refine the PMI

estimate, as mites typically colonize during the very late stages of decomposition.

Ecological Significance of Succession

Insect succession in decomposition is highly consistent under natural conditions, but it can vary depending on environmental factors such as **temperature, humidity,** and the **location** of the body. For instance, **in warm environments**, decomposition and insect activity occur faster, shortening the life cycles of the colonizing species. In contrast, **cool or dry conditions** slow down both decomposition and insect development, potentially extending the PMI.

Environmental and Geographical Variations

The specific species involved in succession can also differ based on geographical location. For example, the types of blow flies found in tropical climates will be different from those found in temperate regions. Similarly, bodies left indoors may attract different arthropods compared to those exposed to the outdoors. Forensic entomologists must account for these variations when interpreting insect succession patterns to accurately estimate PMI.

Factors That Affect Arthropod Succession

Several factors can influence the natural progression of insect colonization:

- **Accessibility**: Bodies that are buried, submerged, or wrapped may experience delayed insect activity because flies and beetles cannot easily reach them.
- **Exposure to chemicals**: Pesticides or other toxins present at the crime scene may kill or repel insects, altering their normal succession patterns.
- **Presence of scavengers**: Larger animals, such as rodents or birds, can disturb the body and its insect population, which may influence the timing and species of colonization.

Conclusion

The succession of arthropods in decomposition is a reliable and scientifically grounded process that offers critical forensic insights, especially in determining PMI. By understanding the predictable arrival and development of different insect species, forensic entomologists can reconstruct the timeline of death, even when other physical evidence has degraded. The presence or absence of specific species

and their stages of development provides investigators with powerful tools to uncover the circumstances of death and estimate how long a body has been exposed to the environment.

Post-mortem interval (PMI) estimation

A key aspect of forensic entomology is estimating the **postmortem interval (PMI)**, which largely relies on analyzing insect activity on a corpse. By studying the life stages of insects found on or near the body,Forensic entomologists use insect activity on decomposing bodies to calculate PMI, as insects colonize a body in a predictable, time-dependent sequence. Thus forensic entomologists can make reasonable estimates of how long a person has been deceased.

The rate of insect colonization and development is affected by various factors, including **temperature, humidity, and the presence of other organisms**. One common method used by forensic entomologists to estimate PMI is the **accumulated degree hour (ADH) approach**, which calculates the total heat energy a body has absorbed since death, influencing insect development.

Although PMI estimation using insect evidence is generally reliable, it's important to consider additional factors, such as **body position, environmental conditions, and insect behavior**, as these can affect the timing and activity of insect colonization.

Unit-12 □ Application of Forensic Entomology and its limitations

Applications of Forensic Entomology

Forensic entomology is the study of insect evidence in legal investigations, particularly in criminal cases. It is primarily used in the estimation of time since death (post-mortem interval, or PMI), but it also has applications in other areas of forensic science.

1. Estimation of Post-Mortem Interval (PMI)

- Insects colonize a decomposing body in a predictable sequence, helping estimate the time since death.
- Blowflies (Calliphoridae) are among the first to arrive, followed by beetles (Coleoptera) and other scavenger species.
- The development stages of insect larvae help forensic entomologists determine how long they have been feeding on the body.

2. Determining Cause and Manner of Death

- Some insect species are attracted to specific types of wounds, which can indicate antemortem injuries or sites of trauma.
- Presence of toxic substances (e.g., drugs or poisons) in insect larvae feeding on human tissues can help toxicologists determine cause of death.

3. Locating Bodies and Movement of Remains

- Insect species vary by region, so identifying non-native species on a body can suggest that it was moved after death.
- Soil-dwelling insects may help locate buried remains.

4. Identifying Neglect and Abuse Cases

- Infestations of maggots or other insects on living individuals (e.g., elderly or disabled persons) can indicate neglect in cases of elder abuse, child abuse, or animal cruelty.

5. Environmental and Wildlife Forensics

- Insect evidence can be used to investigate illegal wildlife trade, poaching, and environmental crimes by identifying the time of death of animals or the conditions in which they were kept.

Limitations of Forensic Entomology: Despite its usefulness, forensic entomology has several limitations that can impact the accuracy of investigations:

1. Environmental Influences

- Temperature, humidity, and weather conditions affect insect activity and development rates.
- Rain or extreme heat can delay insect colonization, leading to errors in PMI estimation.

2. Insect Behavior Variability

- Insect colonization can vary due to the presence of clothing, body coverings, or toxic substances in the body.
- Some forensic insects may not colonize immediately if the body is in an inaccessible location (e.g., submerged in water, buried, or inside a building).

3. Species Identification Challenges

- Accurate identification of insect species is critical for PMI estimation, but misidentification can lead to incorrect conclusions.
- Some species look similar in their early developmental stages, requiring expert analysis.

4. Human and Animal Interference

- Scavenging animals (e.g., rats, birds) may consume insect larvae, disrupting the expected succession pattern.
- Human activities such as body relocation or pesticide use can alter insect evidence.

5. Legal and Ethical Challenges

- Forensic entomology is not always accepted as conclusive evidence in court due to variability in results.

- Lack of standardization in insect collection, preservation, and analysis can lead to inconsistent findings.

6. Limited Availability of Experts

- Requires specialized training, and there are relatively few experts in forensic entomology compared to other forensic disciplines.
- Expertise is essential for accurate species identification and interpretation of insect evidence.

Conclusion

Forensic entomology is a valuable tool in criminal investigations, particularly for estimating PMI and understanding the circumstances surrounding death. However, its effectiveness depends on environmental factors, insect behavior, and expert interpretation. To improve its reliability, forensic entomologists must work alongside pathologists, toxicologists, and law enforcement professionals, ensuring proper collection and analysis of insect evidence.