Plasmodium spp. (Malaria)

Parasitology (MA 301)

Medical Analysis Department

Summer School

Lecture 5

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Case Study

A 19-year-old man presented with recurrent episodes of fever, chills, sweating, headache, and weakness after returning from a malaria-endemic region. Examination of a peripheral blood smear revealed **Plasmodium** parasites within red blood cells. He was treated with an appropriate antimalarial regimen according to the infecting *Plasmodium* species and local resistance patterns. He was also advised to use mosquito nets and insect repellents and avoid mosquito bites, resulting in complete recovery.

Introduction

- They live intracellularly, at least during part of their life cycle.
- At some stages in their life cycle, they possess a structure called the apical complex, by means of which they attach to and penetrate host cells.
- These protozoa are therefore grouped under the Phylum Apicomplexa.
- The disease they cause is called Malaria

Causative agents of human malaria

- *Plasmodium vivax*: Benign tertian malaria
- *Plasmodium falciparum*: Malignant tertian malaria
- *Plasmodium malariae*: Benign quartan malaria
- *Plasmodium ovale*: Benign tertian malaria.

Distribution

Malaria is more common in poor rural populations and urban areas with poor sanitation. Epidemics may arise due to environmental, economic, or social changes, such as migration or heavy rains after droughts. The prevalence of the four malaria species differs by region.

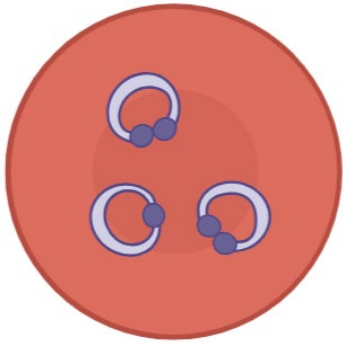
- *P. vivax* is the most widely distributed, being most common in Asia, North Africa, and Central and South America.
- *P. falciparum*, the predominant species in Africa, Papua New Guinea and Haiti, is rapidly spreading in Southeast Asia and India.
- *P. malariae* is present in most places but is rare, except in Africa.
- *P. ovale* is virtually confined to West Africa, where it ranks second after P. falciparum.


Distribution
of malaria

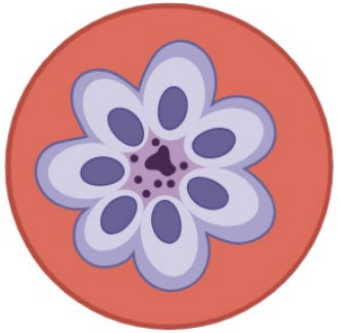
Morphology

The followings are the stages of plasmodium sp,

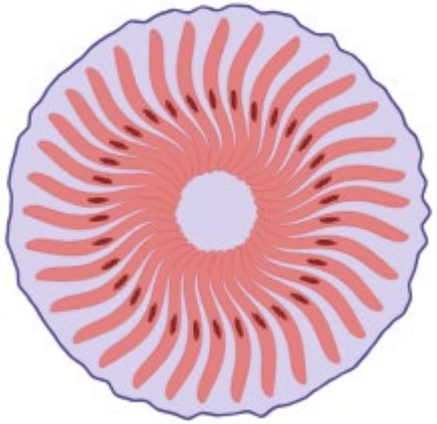
Sporozoite



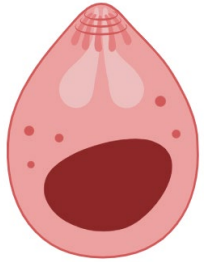
Ring stage



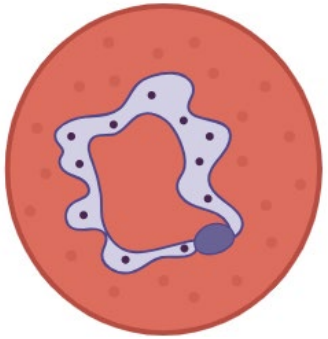
Schizont



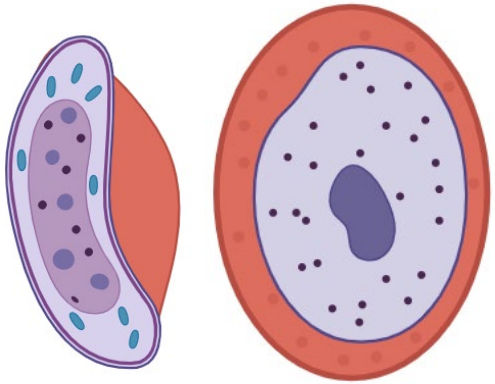
Oocyst



Merozoite

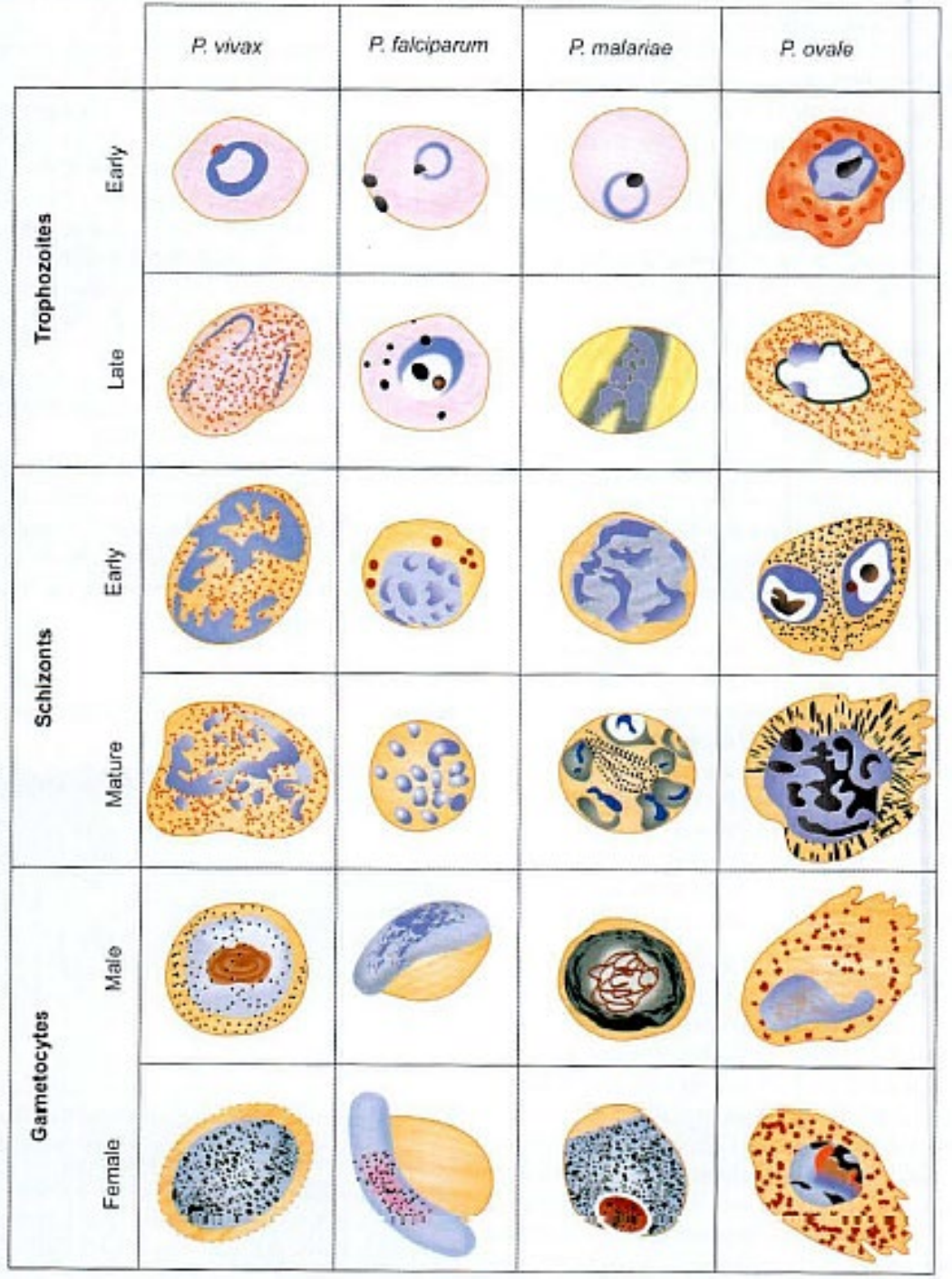


Trophozoite



Gametocyte

Morphology


P. vivax
P. falciparum
P. malariae
P. ovale
Trophozoites
Early
Late
Schizonts
Early
Mature
Gametocytes
Male
Female

Vector

Human malaria is transmitted by over 60 species of female Anopheles mosquitoes.

The male mosquito feeds exclusively on fruits and juices, but the female needs at least two blood meals before the first batch of eggs can be laid.

Life Cycle

Malaria parasite passes its life cycle in two hosts:

1. Definitive host:

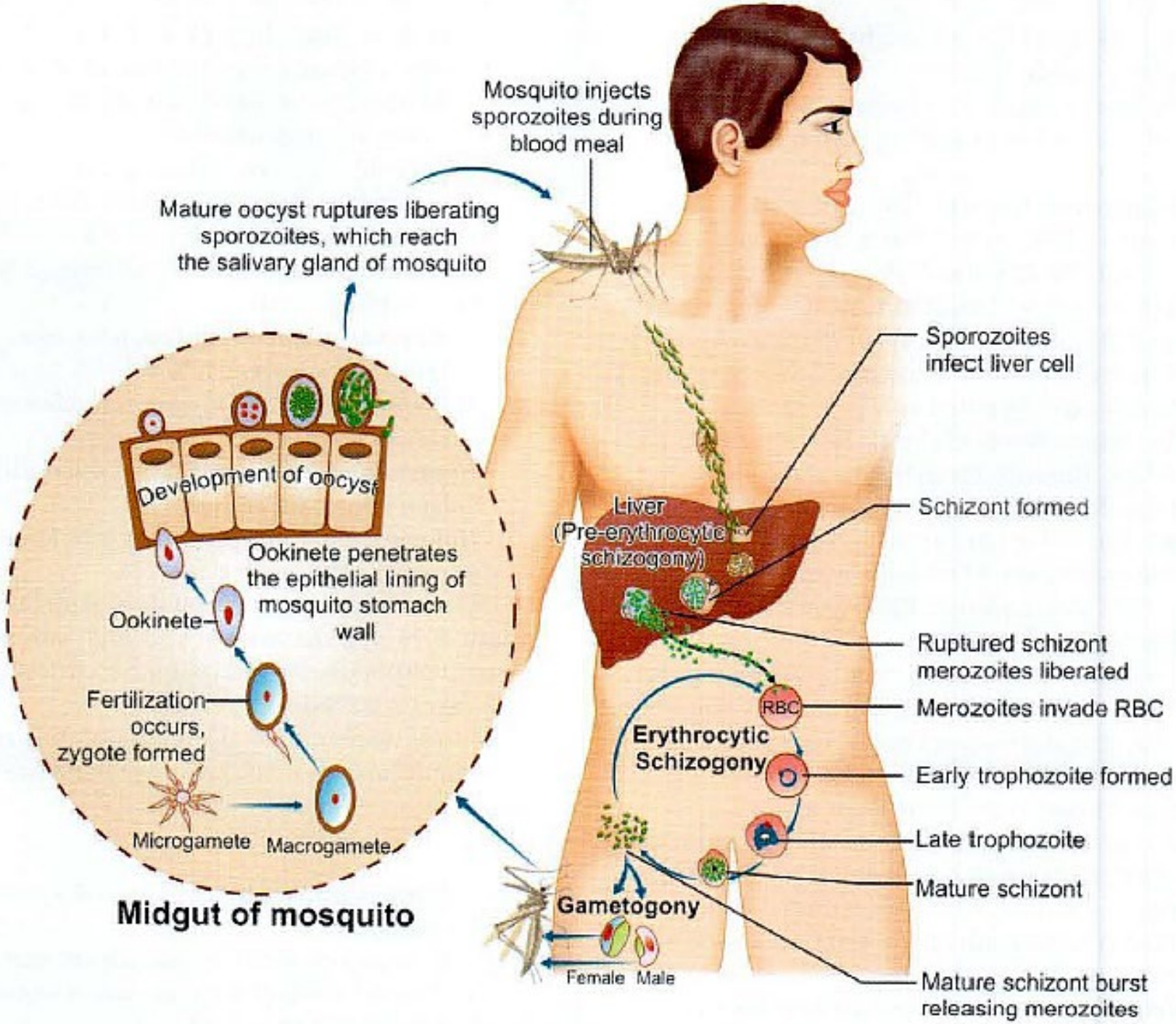
Female Anopheles mosquito.

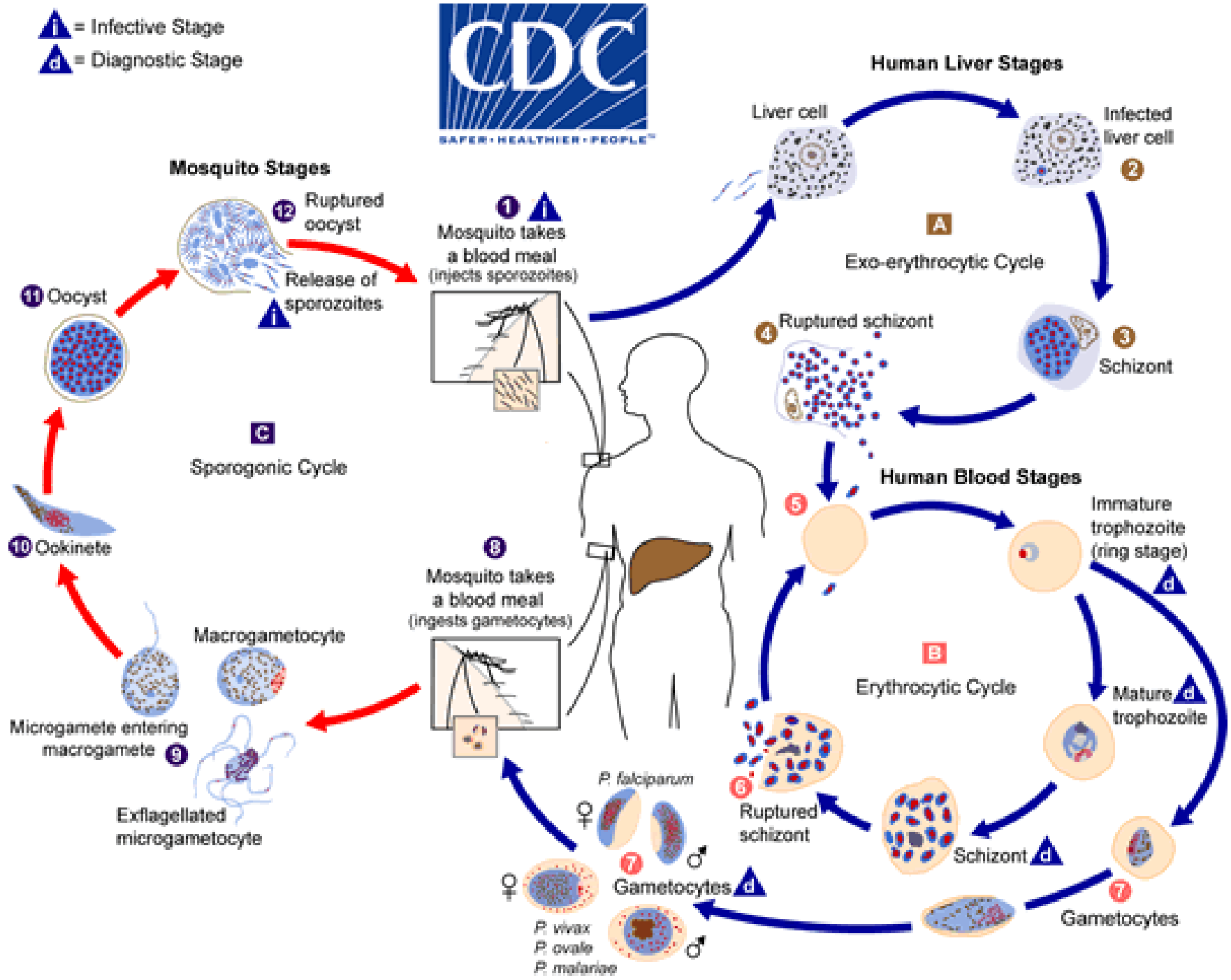
2. Intermediate host:.

Human

Life Cycle

- When an infected Anopheles mosquito bites a human, it injects sporozoites into the bloodstream.
- The sporozoites travel to the liver, invade liver cells, and multiply into merozoites.
- Merozoites are released into the blood, where they infect red blood cells. They multiply to develop to the Ring stage, then the Trophozoite stage, after that, schizonts and then, and then cause the cells to rupture, producing fever and chills.
- Some parasites in red blood cells develop into male and female gametocytes instead of merozoites.
- When another mosquito bites the infected person, it ingests these gametocytes.
- In the mosquito’s gut, the gametocytes fuse to form a zygote, which develops into an ookinete, then an oocyst.
- The oocyst releases new sporozoites that migrate to the mosquito’s salivary glands, ready to infect another human host.


Mosquito injects sporozoites during blood meal
Mature oocyst ruptures liberating sporozoites, which reach the salivary gland of mosquito
Development of oocyst
Ookinete penetrates the epithelial lining of mosquito stomach wall
Ookinete
Fertilization occurs, zygote formed
Microgamete
Macrogamete
Midgut of mosquito
Sporozoites infect liver cell
Liver (Pre-erythrocytic schizogony)
Schizont formed
Ruptured schizont merozoites liberated
RBC
Merozoites invade RBC
Erythrocytic Schizogony
Early trophozoite formed
Late trophozoite
Mature schizont
Gametogony
Female
Male
Mature schizont burst releasing merozoites


i = Infective Stage
d = Diagnostic Stage
CDC
SAFER·HEALTHIER·PEOPLE™
Human Liver Stages
Liver cell
Infected liver cell
2
A
Exo-erythrocytic Cycle
3
Schizont
4
Ruptured schizont
Human Blood Stages
5
Immature trophozoite (ring stage)
d
Mature trophozoite
d
B
Erythrocytic Cycle
6
Ruptured schizont
Schizont
d
7
Gametocytes
Gametocytes
d
P. falciparum
P. vivax
P. ovale
P. malariae
8
Mosquito takes a blood meal (ingests gametocytes)
Macrogametocyte
Microgamete entering macrogamete
9
Exflagellated microgametocyte
10
Ookinete
11
Oocyst
12
Ruptured oocyst
Release of sporozoites
Mosquito Stages
1
Mosquito takes a blood meal (injects sporozoites)
C
Sporogonic Cycle

Asexual Phase

In humans, the parasite multiplies by schizogony (splitting). This occurs in two sites: the liver (exoerythrocytic or pre-erythrocytic schizogony) and red blood cells (erythrocytic schizogony). The products of schizogony are called merozoites.

Sexual Phase

The female Anopheles mosquito is the definitive host where sexual reproduction occurs. Gametocytes formed in human RBCs mature and fertilize in the mosquito, producing many sporozoites. This phase, called sporogony, is the invertebrate or extrinsic phase, alternating with the asexual phase in humans.

Types of *Plasmodium* sp. reproduction

- **Schizogony** – Asexual multiplication of Plasmodium in humans, occurring in the liver (exoerythrocytic) and RBCs (erythrocytic), producing merozoites.
- **Gametogony** – Formation of male and female gametocytes in human RBCs, which are infective to mosquitoes.
- **Sporogony** – Sexual cycle in the mosquito, where gametocytes mature, fertilize, and produce oocyst and then sporozoites that migrate to salivary glands for transmission.

Plasmodium Vivax

P. vivax has the widest geographical distribution, extending through the tropics, subtropics and temperate regions. It is believed to account for 80% of all malaria infections. lt is the most common species of malaria parasite in Asia and America, but is much less common in Africa. **lt causes benign tertian malaria** with frequent relapses.

P. vivax sporozoites are narrow and curved. On entering the liver cells, the sporozoites initiate two types of infection: some form exoerythrocytic schizonts, while others remain dormant as **hypnozoites**.

Plasmodium falciparum

The name falciparum comes from the characteristic **sickle shape of the gametocytes** of this species. This is the highly pathogenic of all the plasmodia and hence, the name **malignant tertian or pernicious malaria** for its infection.

The disease has a high rate of complications and, unless treated, is often fatal. The species is responsible for almost all deaths caused by malaria.

Plasmodium malariae

It causes **quartan malaria**, in which febrile paroxysms occur every 4th day, with 72 hour interval between the bouts. The disease is generally mild, but is notorious for its long persistence in circulation at undetectable levels for 50 years or more. The old trophozoites are sometimes seen stretched across the erythrocyte as a broad band. These **band forms** are a unique feature of P. malariae.

Plasmodium ovale

This parasite produces **a tertian fever** resembling vivax malaria, but with milder symptoms, prolonged latency and fewer relapses.

It is the rarest of all plasmodia infecting humans and is seen mostly in tropical Africa, particularly along the West Coast.

Hypnozoites are present.

Pathogenesis

- Clinical manifestations of malaria result from products of erythrocytic schizogony and the host's immune response, along with tissue hypoxia due to obstructed blood flow by infected RBCs.
- Liver is enlarged and congested. Kupffer cells are increased and filled with parasites.

Clinical Features

Benign Malaria

Incubation period: 12–17 days.
Clinical features: Periodic fever with chills, anemia, splenomegaly, and hepatomegaly.

Febrile paroxysm (8–12 hrs), synchronized with erythrocytic schizogony) it comprises three distinct stages:

1. Cold stage: Intense chills, rigor, headache, nausea (15 min–1 hr).
2. Hot stage: High fever up to 41°C, headache persists, nausea usually improves (2–6 hrs).
3. Sweating stage: Profuse sweating, temperature returns to normal, patient feels refreshed.

Fever periodicity:

- 48 hours → Tertian malaria (P. vivax, P. falciparum, P. ovale).
- 72 hours → Quartan malaria (P. malariae).
- 24 hours (quotidian) → Two broods of tertian parasites or mixed infection.

Clinical Features

Malignant Tertian Malaria

- **Cerebral malaria**: Most common. Begins with fever, headache, nausea → progresses to confusion, seizures, coma. May cause retinal hemorrhages, hypoglycemia, renal failure, metabolic acidosis, pulmonary edema, or shock. Mortality: ~15% children, 20% adults.

  Pathogenesis: Infected RBCs develop knob-like structures (PfEMP-1) → adhere to endothelium → microvascular obstruction → brain anoxia and hemorrhage.

- **Septicemic malaria:** High continuous fever, parasite spread to multiple organs, causing multiorgan failure (80% fatal).

Merozoite-induced Malaria

Unlike natural sporozoite-induced malaria, here infection occurs directly by merozoites, causing only erythrocytic schizogony (no liver phase or relapse, short incubation).

- **Transfusion malaria:** From infected donor blood (parasites survive 1–2 weeks in stored blood).
- **Congenital malaria:** Transplacental transmission from mother to fetus.
- **Renal transplantation:** From a donor with parasitemia.
- **Shared syringes:** Among drug users.

	Mosquito-borne malaria	*Blood transfusion malaria*
Mode of transmission	Mosquito bite	Blood or blood products transfusion
Infective stage	Sporozoite	Trophozoite
Incubation period	Long	Short
Pre-erythrocytic schizogony	Present	Absent
Hypnozoites	May be present	Absent
Severity	Comparatively less	More complications seen
Relapse	May occur	Does not occur
Radical treatment	Required	Not required

Why *Plasmodium falciparum* is the most lethal species among the Plasmodium parasites?

Laboratory Diagnosis

Demonstration of Parasite by Microscopy

Diagnosis of malaria can be made by demonstration of malarial parasite in the blood.

Two types of smears are prepared from the peripheral blood. One is called thin smear and the other is called thick smear.

Demonstration of Parasite by Microscopy

Thin smears: They are prepared from capillary blood of finger tip and spread over a good quality slide by a second slide held at an angle of 30-45° from the horizontal such that a tail is formed.

- Thin film is examined first a t the tail end

Purpose

- single layer of red cells should be formed
- determining the species.

Demonstration of Parasite by Microscopy

- **thick smears:** In a thick film, usually **three drops** of blood are spread over a small area (about 10 mm).
- The amount of blood in thin smear is about **1- 1.5 µL,** while in a thick smear it is **3-4 µL.**
- If parasites are not detected in thin film, then thick film should be examined.

Laboratory Diagnosis

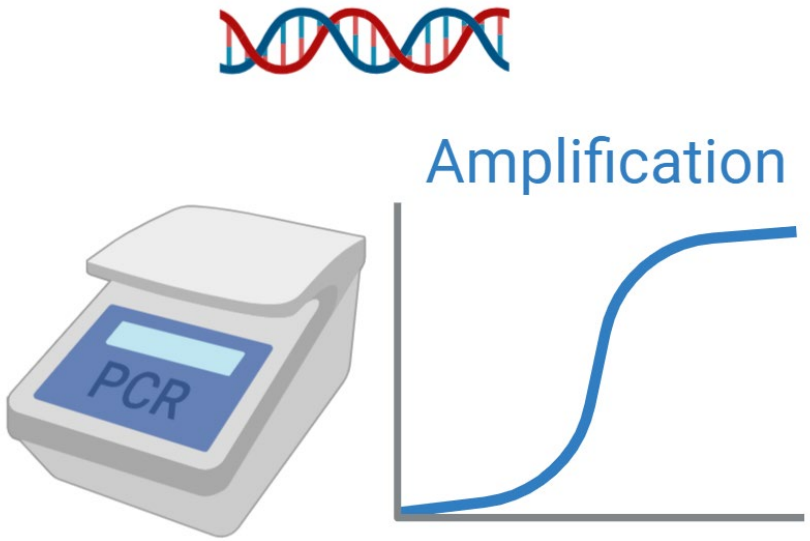
Fluorescence microscopy:

Kawamoto technique: Fluorescent dyes like acridine orange or benzothiocarboxy purine are used, which stain the parasites entering the RBCs but not white blood cells (WBCs).

Laboratory Diagnosis

3. Molecular Method

- DNA detection of the parasite



Treatment

The best available treatment for malaria, particularly for Plasmodium falciparum malaria, is **artemisinin-based combination therapy (ACT).**

(artemisinin + sulfadoxine - pyrimethamine)