Plasmodium species: **Sporozoan parasites that require two hosts for completion of their life cycle. The mosquito for the sexual reproductive stages and human for the asexual reproductive stage.**

Type of malare depend onbloodcycle -

***P. vivax* Tertian malare Blood cycle 48h -**

***P. ovale* Tertian malare Blood cycle 48h -**

***P. malariae*. Quatrain malare *Blood cycle 72h* -**

Plasmodium falciparum melegnant malare Blood cycle36h 36h

life cycle involves two hosts.

***Sporogenic Stage**

- During a blood meal, a malaria-infected female *Anopheles* mosquito inoculates sporozoites into the human host.

*** Hepatic Stage**

- Sporozoites infect liver cells and mature into schizonts , which rupture and release merozoites into the bloodstream.

*** Blood Stage**

- Merozoites infect red blood cells

- The parasites undergo asexual multiplication in the erythrocytes.

- The ring stage trophozoites mature into schizonts, which rupture releasing merozoites.

- Some parasites differentiate into male and female gametocytes.

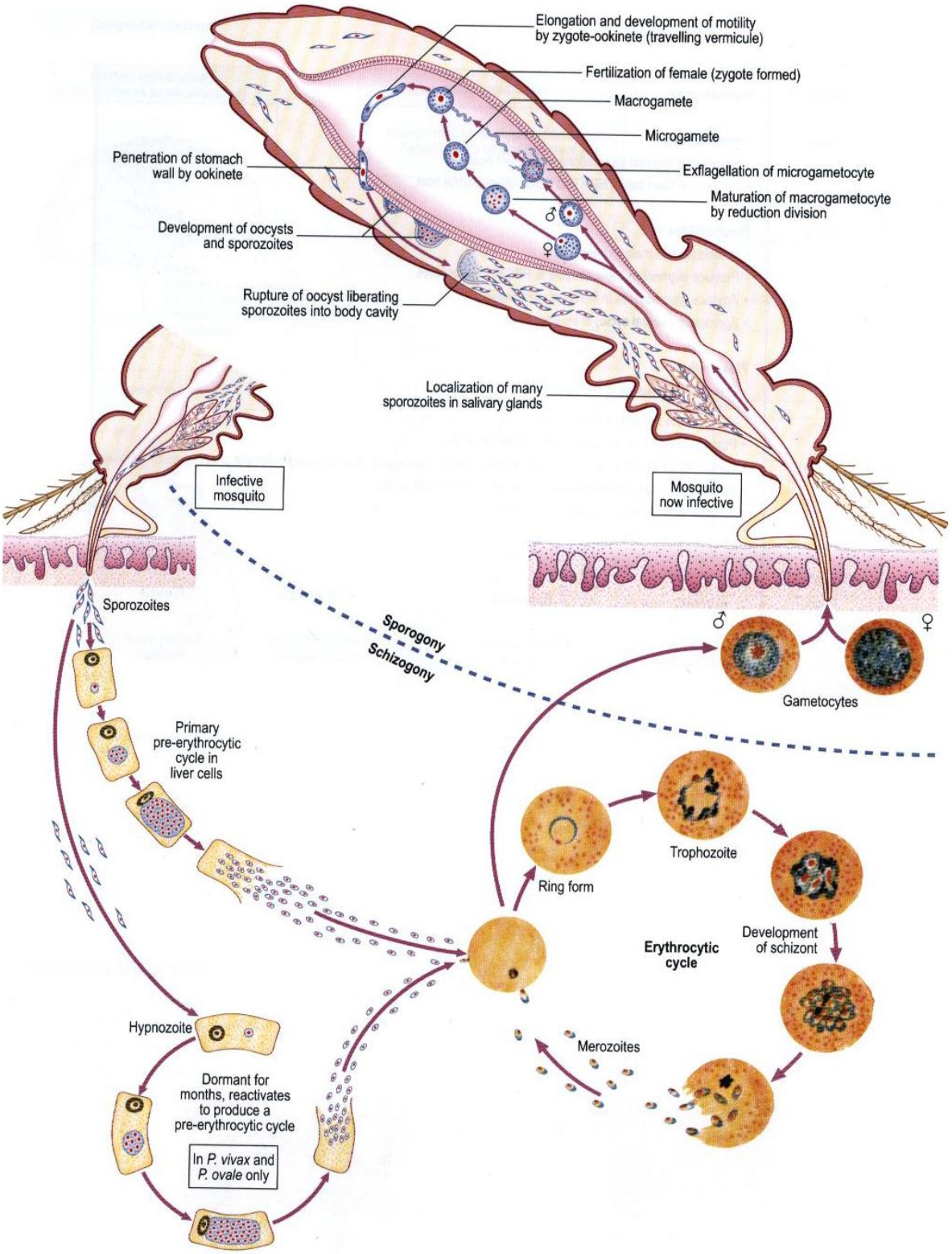
- Blood stage parasites are responsible for the clinical manifestations of the disease.

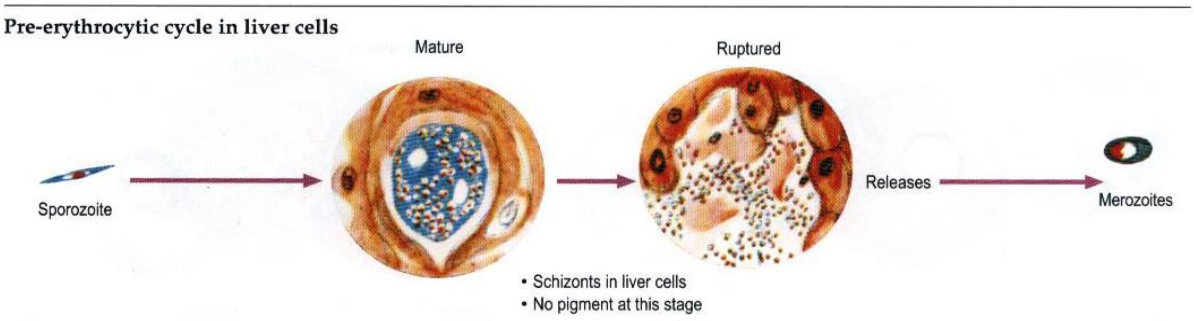
The male and female over maturation gametocytes are ingested by another *Anopheles* mosquito. And only newly gametocyte can survive and multiplication in the mosquito is known as the sporogonic cycle, generating zygotes.

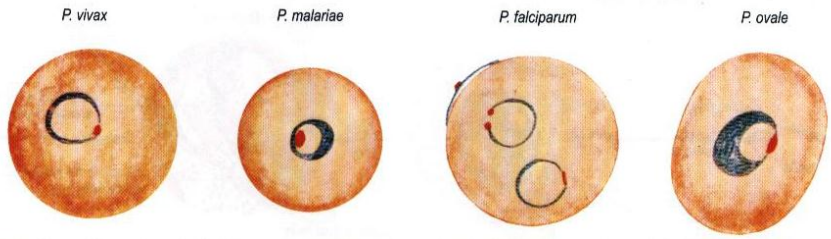
- The zygotes develop into oocysts. The oocysts grow, rupture, and release sporozoites, which make their way to the mosquito's salivary glands.

- Inoculation of the sporozoites into a new human host perpetuates the malaria life cycle .

Life cycle


Elongation and development of motility by zygote-ookinete (travelling vermicule)
Fertilization of female (zygote formed)
Macrogamete
Microgamete
Penetration of stomach wall by ookinete
Exflagellation of microgametocyte
Maturation of macrogametocyte by reduction division
Development of oocysts and sporozoites
Rupture of oocyst liberating sporozoites into body cavity
Localization of many sporozoites in salivary glands
Infective mosquito
Mosquito now infective
Sporozoites
Sporogony
Schizogony
Gametocytes
Primary pre-erythrocytic cycle in liver cells
Ring form
Trophozoite
Development of schizont
Erythrocytic cycle
Merozoites
Hypnozoite
Dormant for months, reactivates to produce a pre-erythrocytic cycle
In P. vivax and P. ovale only





Symptoms: The symptomatology of malaria depends on parasitemia, the presence of the organism in different organs and the parasite burden. The incubation period varies generally between 10-30 days. As the parasite load becomes significant, the patient develops headache, lassitude, vague pains in the bones and joints, chilly sensations and fever. As the disease progresses, the chills and fever become more prominent.

The chill and fever follows a cyclic pattern (paroxysm) with the symptomatic period lasting 8-12 hours.

In between the symptomatic period, there is a period of relative normalcy, the duration of which depends upon the species of the infecting parasite. This interval is about36-48 hours in the case of P. vivax and P. ovale (tertian malaria), and 60-70 hours in the case of P. malariae (quartan malaria). Classical tertian paroxysm is rarely seen in P. falciparum: persistent spiking or a daily paroxysm is more usual.

The malarial paroxysm is most dramatic and frightening: it begins with chilly sensation which progresses to teeth chattering overtly shaking chill, peripheral vasoconstriction resulting in cyanotic lips and nails (cold stage) which lasts for about an hour. At the end of this period the body temperature begins to climb and reaches (39- 41degrees C). Fever is associated with severe headache, nausea (vomiting) and convulsions.

The patient experiences euphoria, and profuse perspiration and the temperature begins to drop. Within a few hours the patient feels exhausted but symptomless and remains symptomatic until the next paroxysm**. Each paroxysm is due to the rupture of infected erythrocytes and release of parasites**

Without treatment, all species of human malaria may ultimately result in spontaneous cure except with P. falciparum which becomes more severe progressively and results in death. This organism causes sequestration of capillary vasculature in the brain, gastrointestinal and renal tissues.

Chronic malaria results in splenomegaly, hepatomegaly and nephritic syndromes

Pathology and immunology: Symptoms of malaria are due to release of massive number of merozoites into circulation. Infection results in the production of antibodies which are effective in containing the parasite load. These antibodies are against merozoites and schizonts. The infection also results in the activation of the reticuloendothelial system (phagocytes). The activated macrophages help in the destruction of infected (modified) erythrocytes and antibody coated merozoites. Cell mediated immunity also may develop and help in the elimination of infected erythrocytes. Malarial infection is associated with immunosuppression.

Diagnosis

Malaria parasites in the thin blood film. Stained by Leishman or Giemsa.

-It is also possible to use thick blood films stained by Field or Giemsa.

Bone marrow films may be examined -

-Serology (IFAT or ELISA) is not appropriate for the detection of acute malaria but is deployed as retrospective test for epidemiology use to establish the cause of nephrotic syndrome or hyperreactive malarial splenomegaly.

Treatment and Control:

Treatment is effective with various quinine derivatives (quinine sulphate, chloroquine, meflaquine and primaquine, etc.). Drug resistance, particularly in P. falciparum and to some extent in P. vivax is a major problem. Control measures are eradication of infected anopheline mosquitos. Vaccines are being developed and tried but none is available ye for routine use.