ARTHROPOD OR INTERMEDIATE VECTOR-BORNE DISEASES

a vector is any carrier of disease, but in the case of the ‘vector-borne diseases’ we restrict the word to those invertebrate hosts (insects or snails), which are an essential part of the life cycle of the disease organism.

A housefly just carrying bacteria or amoebic cysts on its feet to food is not regarded as a vector: this would be simple mechanical spread.

Insect vectors usually acquire the disease organism by sucking blood from infected persons, and pass it on, later, by the same route. There are other routes, however; infection may enter skin cracks or abrasions either from infected feces deposited when feeding, or from body fluid when an insect is crushed.

By definition the disease organism undergoes a period of development inside the vector, and the time taken for this is called the extrinsic incubation period.

Malaria

Alternative names:

- Quartan malaria
- Falciparum malaria

- Blackwater fever
- Tertian malaria

A vector-borne infectious disease caused by protozoan parasites.
It is widespread in tropical and subtropical regions

A bite from an infective female Anopheles mosquito.

Anopheles must be infected through a previous blood meal taken on an infected person to transmit malaria

At risk for malaria:
40% of the world's population

more than 500 million are ill of malaria yearly

If treated in the early stages, malaria can be cured.

Plasmodium falciparum - most common and deadly type of malaria infection

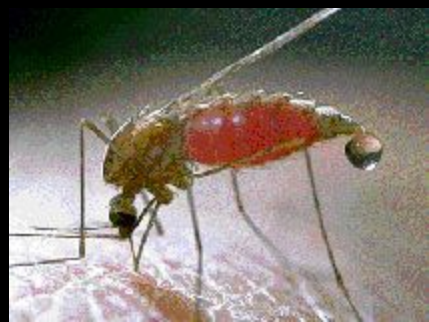
- can lead to cerebral malaria

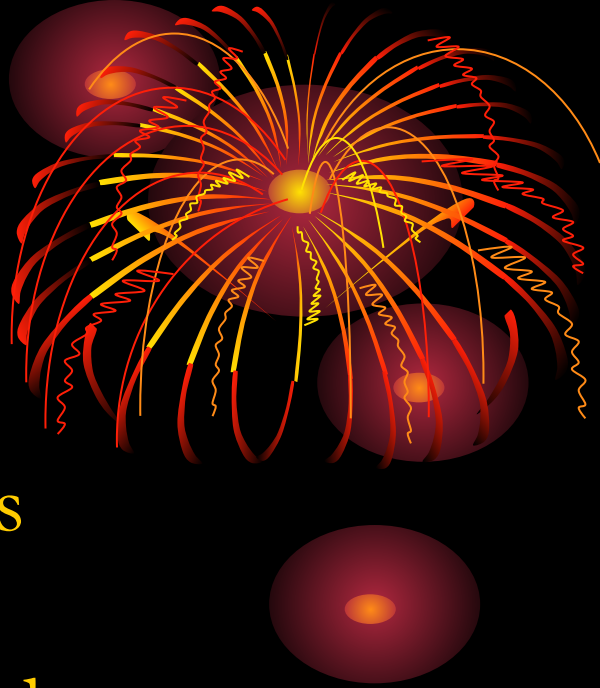
P.vivax - most common

- causes relapse if treatment was not completed.

P.ovale.

P.malaria





Female Anopheles are:

Anthropophilic : from humans
Zoophilic : from animals
Endophagic : prefer to bite indoors
Exophagic : prefer outdoor biting

PATHOPHYSIOLOGY:

Liver Stage. Human infection is initiated when sporozoites are injected with the saliva during mosquito feeding. The sporozoites enter the circulatory system and within 30-60 minutes will invade a liver cell. Host cell entry, as in all apicomplexa, is facilitated by the apical organelles. After invading the hepatocyte, the parasite undergoes an asexual replication. This replicative stage is often called exoerythrocytic (or pre-erythrocytic) schizogony.

In *P. vivax* and *P. ovale* some of the sporozoites do not immediately undergo asexual replication, but enter a dormant phase known as the hypnozoite. This hypnozoite can reactivate and undergo schizogony at a later time resulting in a relapse.

Blood Stage. Merozoites released from the infected liver cells invade erythrocytes. The merozoites recognize specific proteins on the surface of the erythrocyte and actively invade the cell in a manner similar to other apicomplexan parasites.

After entering the erythrocyte the parasite undergoes a trophic period followed by an asexual replication. The young trophozoite is often called a ring form due to its morphology in Geimsa-stained blood smears. As the parasite increases in size this 'ring' morphology disappears and it is called a trophozoite. During the trophic period the parasite ingests the host cell cytoplasm and breaks down the hemoglobin into amino acids. A by-product of the hemoglobin digestion is the malaria pigment, or hemozoin. These golden-brown to black granules have been long recognized as a distinctive feature of blood-stage parasites.

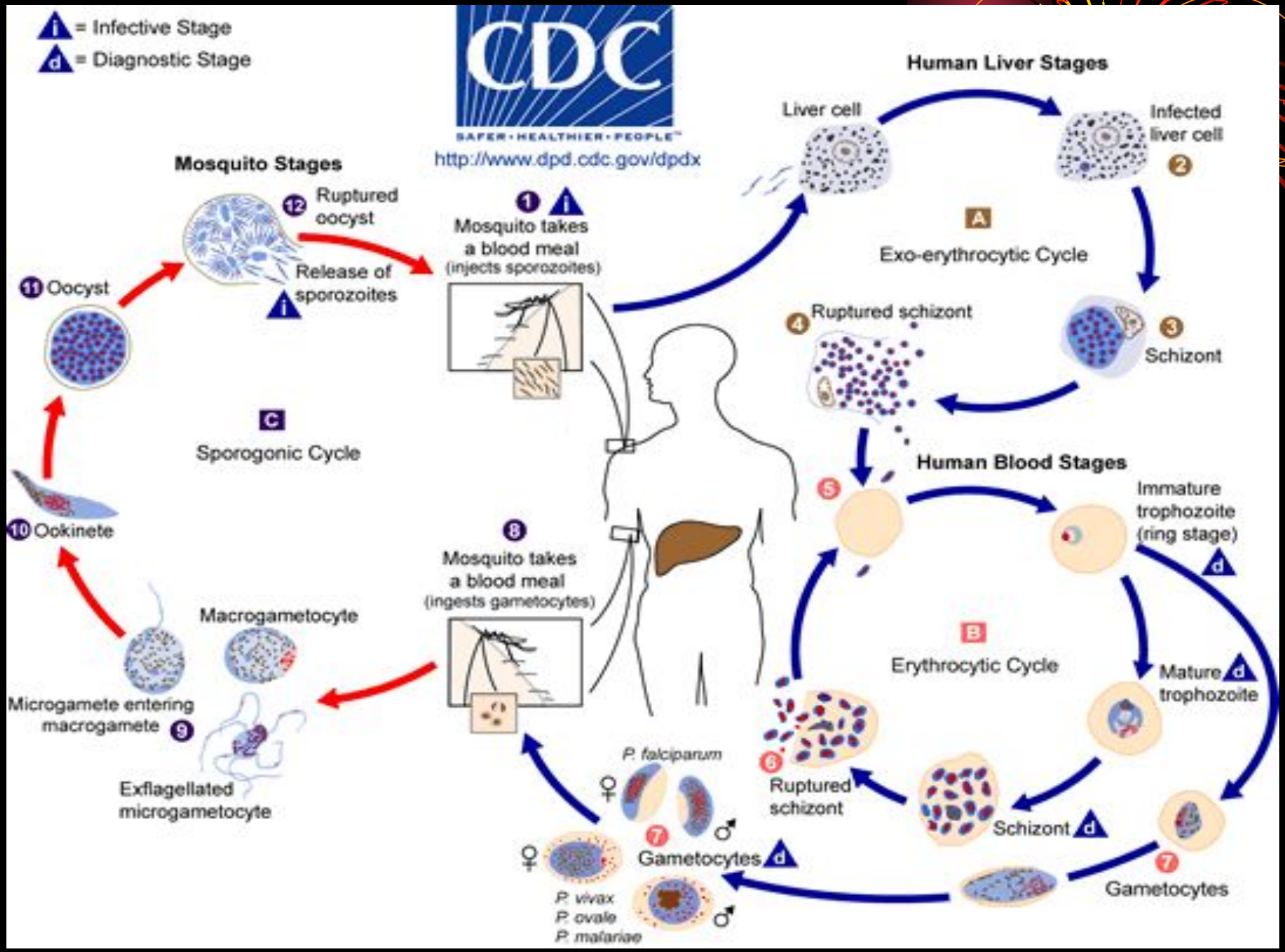
Nuclear division marks the end of the trophozoite stage and the beginning of the **schizont** stage. Erythrocytic schizogongy consists of 3-5 rounds (depending on species) of nuclear replication followed by a budding process. Late stage schizonts in which the individual merozoites become discernable are called **segmenters**. The host erythrocyte ruptures and releases the merozoites. These merozoites invade new erythrocytes and initiate another round of schizogony. The blood-stage parasites within a host usually undergo a synchronous schizogony.

Sexual Stage. As an alternative to schizogony some of the parasites will undergo a sexual cycle and terminally differentiate into either **micro- or macrogametocytes.** Gametocytes do not cause pathology in the human host and will disappear from the circulation if not taken up by a mosquito.

Gametogenesis, or the formation of **micro-** and **macrogametes**, is induced when the gametocytes are ingested by a mosquito. After ingestion by the mosquito, the microgametocyte undergoes three rounds of nuclear replication. The macrogametocytes mature into macrogametes.

The highly mobile microgametes will seek out and fuse with a macrogamete. Within 12-24 hours the resulting **zygote** develops into an **ookinete**. The ookinete is a motile invasive stage which will transverse both the peritrophic matrix and the midgut epithelium of the mosquito.

Sporogony. After reaching the extracellular space between the epithelial cells and the basal lamina, the ookinete develops into an oocyst. The oocysts undergo an asexual replication, called sporogony, which culminates in the production of several thousand sporozoites. This generally takes 10-28 days depending on species and temperature. Upon maturation the oocyst ruptures and releases the sporozoites which cross the basal lamina into the hemocoel (body cavity) of the mosquito.


i = Infective Stage
d = Diagnostic Stage
CDC
SAFER·HEALTHIER·PEOPLE™
http://www.dpd.cdc.gov/dpdx
Human Liver Stages
Liver cell
Infected liver cell
2
A
Exo-erythrocytic Cycle
Ruptured schizont
4
3
Schizont
Human Blood Stages
5
Immature trophozoite (ring stage)
d
Mature trophozoite
d
B
Erythrocytic Cycle
6
Ruptured schizont
Schizont
d
7
Gametocytes
P. falciparum
♀
♂
7
Gametocytes
d
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
C
Sporogonic Cycle
12
Ruptured oocyst
Release of sporozoites
i
Mosquito Stages
1
i
Mosquito takes a blood meal
(injects sporozoites)

Exoerythrocytic schizogony and prepatent and incubation periods

	P. falciparum	*P. vivax*	*P. ovale*	*P. malariae*
Prepatent period (days)	6-9	8-12	10-14	15-18
Incubation period (days)	7-14	12-17	16-18	18-40
Merozoite maturation (days)	5-7	6-8	9	12-16
Merozoites produced	40,000	10,000	15,000	2000

Signs & symptoms:

The pathology and clinical manifestations associated with malaria are almost exclusively due to the asexual erythrocytic stage parasites. Tissue schizonts and gametocytes cause little, if any, pathology. ***Plasmodium*** infection causes an acute febrile illness which is most notable for its periodic fever paroxysms occuring at either 48 or 72 hour intervals. The severity of the attack depends on the ***Plasmodium*** species as well as other circumstances .

Sometimes the incubation periods can be prolonged for several months in *P. vivax*, *P. ovale*, and *P. malariae*. All four species can exhibit non-specific prodromal symptoms a few days before the first febril attack. These prodromal symptoms are generally described as 'flu-like' and include: headache, slight fever, muscle pain, anorexia and nausea. The symptoms tend to correlate with increasing numbers of parasites.

In contrast to the other three species, *P. falciparum* can produce serious disease with mortal consequences. This increased morbidity and mortality is due in part to the high parasitemias associated with *P. falciparum* infections. These potentially high parasitemias are due in part to the large number of merozoites produced and the ability of *P. falciparum* to invade all erythrocytes.

Other Physical symptoms:

Fever: Fever can be very high from the first day. Temperatures of 40°C and higher are often observed. Fever is usually continuous or irregular. Classic periodicity may be established after some days.

Hepatomegaly: The liver may be slightly tender.

Splenomegaly: Splenomegaly takes many days, especially in the first attack in nonimmune children. In children from an endemic area, huge splenomegaly sometimes occurs. Anemia: Prolonged malaria can cause anemia, and malarial anemia causes significant mortality.

Jaundice: With heavy parasitemia and large-scale destruction of erythrocytes, mild jaundice may occur. This jaundice subsides with the treatment of malaria.

Dehydration: High fever, poor oral intake, and vomiting all contribute to dehydration.

Black water fever

- Massive intravascular hemolysis
- Due to P. falciprum
- Severe acute hemolytic anemia
- RBC=1-2*106 /ml
- Hemoglobinuruia
- Increase bilirubin
- Acute tubcular necrosis& Hb casts

Medical intervention:

Examine blood under microscope (geimsa stain)

chest x-ray: helpful if respiratory symptoms are present

CT scan: to evaluate evidence of cerebral edema or hemorrhage

Polymerase chain reaction (PCR)

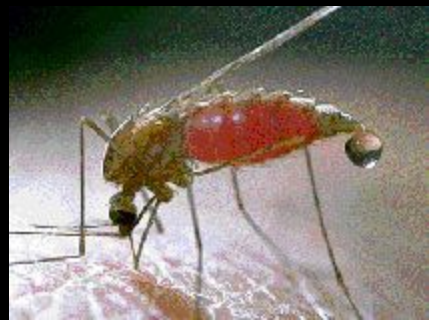
-determine the species of plasmodium

Dipstick test

- not as effective when parasite levels are below 100 parasites/mL of blood

Blood examination:

Thick and thin blood film



Other tests:

CBC:

-Lukopenia

-Thrombocytobenia

-Esinophilia

-monocytosis

Quntitative buffy coat techniqe

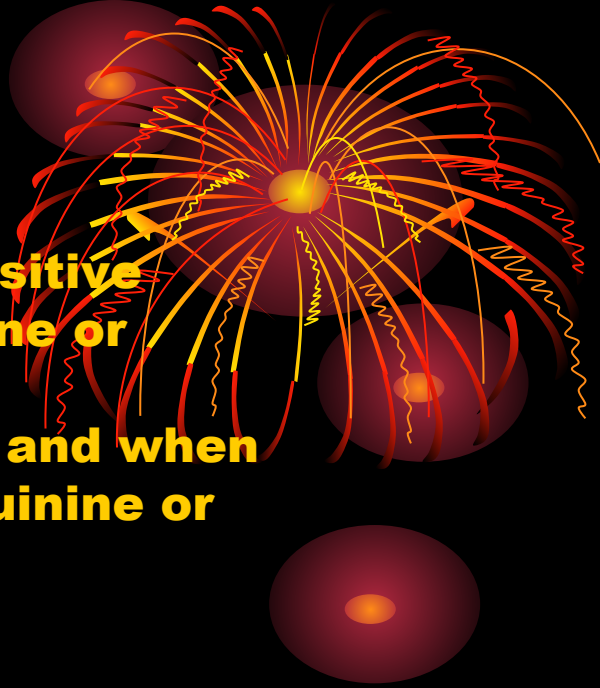
Urinalysis

Increase ESR

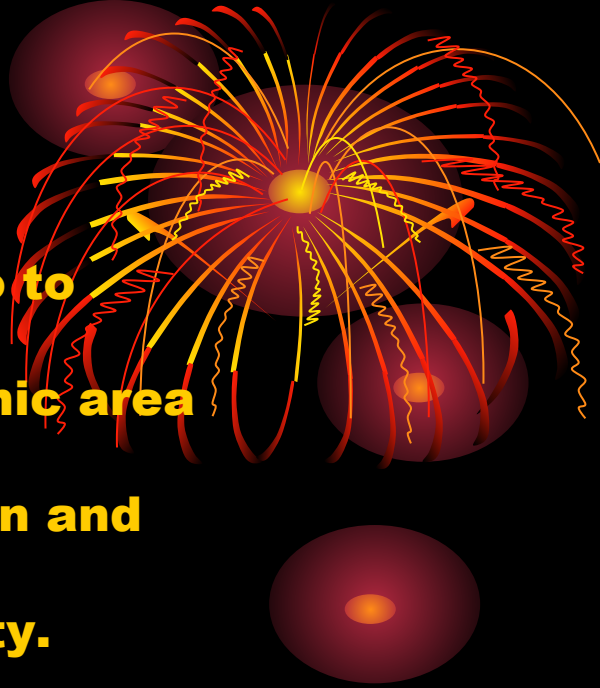
Treatment

1. Plasmodium vivax, ovale and sensitive plasmodium falciparum , Chloroquine or Fansidar

2. Chloroquine resistant falciparum and when sensitivity pattern is not known ,Quinine or Fansidar



Prevention and control



1. Chemoprophylaxis- for those who go to endemic areas
but not for those who live in the endemic area (travelers
and newcomers); for under-five children and pregnant
mothers who have not enough immunity.
2. Vector control
 Avoiding mosquito breeding sites
 Residual DDT spray or other chemicals
 Personal protection against mosquito bite (use of bed
nets, etc.)
3. Chemotherapy of cases