Phylum: Apicomplexa

Class: Sporozoa

Family: Sarcocystidae

Genera:

Toxoplasma, Besnoitia, Sarcocystis, Hammondia, Neospora

- **In Final Host:**
  - Present asexual and sexual stage of life cycle
  - Non pathogenic for F/H.
- **In Intermediate Host:**
  - Present asexual stage of life cycle
  - Tissue stage of lifecycle (Extra intestinal)
  - Pathogenic for I/H
  - Cyst formation occur

Toxoplasma gondii

- *A*n obligate intracellular protozoan that causes the disease toxoplasmosis.
- Found worldwide
- *T. gondii* is capable of infecting virtually all warm-blooded animals, but felids such as domestic cats are the only known definitive hosts in which the parasite can undergo sexual reproduction.
- It is a cyst forming coccidia.
- It has been recognized as a serious pathogen for many years.
- It has a high zoonotic significance.

Host	Location
Final host: All felids (Domestic cat is the most important.)	Schizonts and Gamonts in the SI.
Intermediate host: All mammals (Including man, bird). Cat may also be intermediate host and bears extra intestinal stage.	Tachyzoites and Bradyzoites in extra intestinal tissues including muscle, liver, lung, brain, reproductive system.

Oocysts:

- Unsporulated oocysts in faces of final host (F/H).
- 12□10 µm & spherical shaped.
- Sporulation takes 1 to 5 days.
- Sporulated oocysts contain 2 ellipsoidal sporocysts each with 4 sporozoites.

Transmission:

a) Vertical transmission -from parents to offspring

- Transplacental transmission from mother to fetus, particularly when *T. gondii* is contracted during pregnancy.

b) Horizontal transmission -by contaminated feed & water

- By consuming raw or undercooked meat containing *T. gondii* tissue cysts.
- By ingesting water, soil, vegetables, or anything contaminated with oocysts shed in the feces of an infected animal.
- From a blood transfusion or organ transplant.
- By contact with soil.
- From eating unwashed raw vegetables or fruits

A. Intestinal stage:

1. Schizonts:

- Common in the jejunum and Ileum.
- 4 - 17 µm in diameter & Contain up to 32 merozoites.

2. Gamonts :

- Common in the ileum & approximately 10 µm in diameter.

B. Extra intestinal stage:

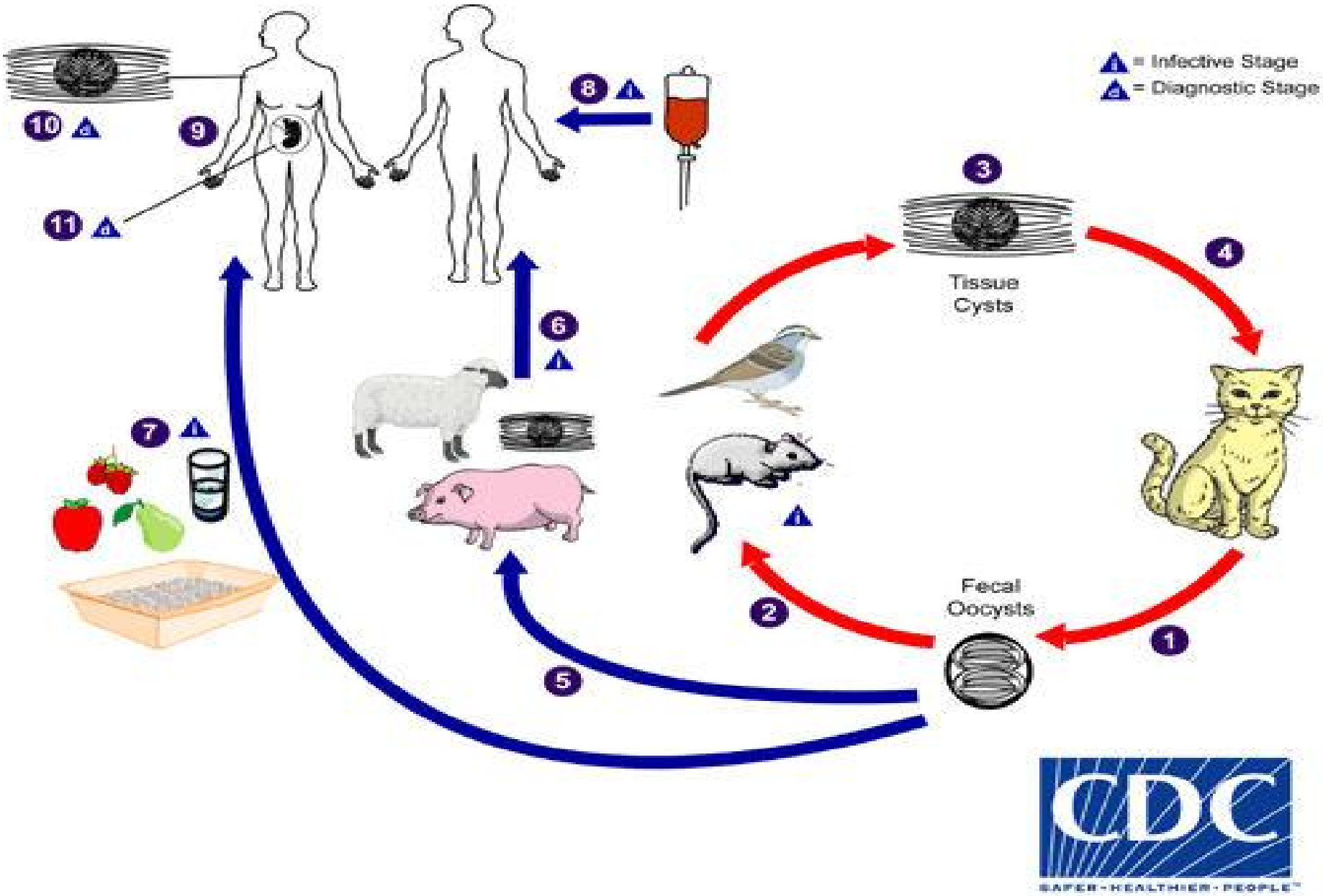
1. Tachyzoites:

- They are found developing in vacuoles in many cell types, for example, fibroblasts, hepatocytes, reticular cells, and myocardial cells.
- In any one cell there may be 8-16 organisms, each measuring 6-8 µm.

2. Bradyzoites:

- They are contained in cysts and occur mainly in the muscle, liver, lung, and brain.
- The Bradyzoites are lancet shaped.
- Several thousand may be present in one cyst which can measure up to 100 µm in diameter.

Life cycle of *T. gondii*



1. Unsporulated oocysts are shed in the cat's feces. Although oocysts are usually only shed for 1-2 weeks, large numbers may be shed. Oocysts take 1-5 days to sporulate in the environment and become infective.
2. Intermediate hosts in nature (including birds and rodents) become infected after ingesting soil, water or plant material contaminated with oocysts.
3. Oocysts transform into tachyzoites shortly after ingestion. These tachyzoites localize in neural and muscle tissue and develop into tissue cyst bradyzoites.
4. Cats become infected after consuming intermediate hosts harboring tissue cysts.
5. Cats may also become infected directly by ingestion of sporulated oocysts. Animals bred for human consumption and wild game may also become infected with tissue cysts after ingestion of sporulated oocysts in the environment.
6. Humans can become infected by any of several routes:
    I. eating undercooked meat of animals harboring tissue cysts.
    II. consuming food or water contaminated with cat feces.
    III. blood transfusion or organ transplantation.
    IV. transplacentally from mother to fetus .

Note:

In the human host, the parasites form tissue cysts, most commonly in skeletal muscle, myocardium, brain, and eyes; these cysts may remain throughout the life of the host.

Diagnosis is usually achieved by serology, although tissue cysts may be observed in stained biopsy specimens.

Diagnosis of congenital infections can be achieved by detecting *T. gondii* DNA in amniotic fluid using molecular methods such as PCR.

Epidemiology:

- Cat plays the central role in the epidemiology. About 60% cats are sero-positive and following infection cats shed oocysts for only 1-2 weeks after which they become resistant to re-infection (self limiting) and some remain as carrier especially during the periparturient period in queens or following corticosteroid therapy.
- Clinical outbreaks in ewes are sporadic. Pregnant ewes are most commonly infected during period of concentrate feeding prior to tupping or lambing if the stored feed having contaminated with cat faeces. Coprophagus insects may contaminate animal fodder, vegetables, meat, etc. Venereal transmission may occur in sheep.
- In human, serological titer reveals 25% infection. In some areas prevalence is high in veterinarian, abattoir workers of those who handle cats.

Harmful effects: Non pathogenic for F/H and pathogenic for I/H (including cat) and mostly due to extra intestinal stages.

- **Cat:** Intestinal phase: Enteritis may occur.
  - Extra-intestinal phase:
  - Enlarged mesenteric lymphnodes, Pneumonia, Degenerative changes in CNS and encephalitis in extra intestinal infection/phase.
- **Sheep:**
  - Most infections are asymptomatic but fever, dyspnea, nervous signs may occur in serious cases.
  - Fetal death & resorption (Barren ewe), Fetal death, abortion & mummified fetus, causes stillbirth or & weak lambs
  - In case of abortion there is a focal placentitis with typical white lesions in cotyledons and fetal tissues. Bradyzoites in brain with focal necrosis in acute cases and Glial nodules in chronic cases.
  - Diagnosis can be confirmed by impression smears stained with Giemsa. Fetal thoracic fluid or blood from ewes may be used for serology.

- **Human:** Infection may be acquired or congenital.

- **In acquired infection:**
  - Low grade fever, malaise, generalized lymphadenopathy.
  - Rarely myocarditis, encephalitis, and retinochoroiditis.
- **In congenital infection:**
  - Up to 10% results in abortion, stillbirth, or damage to CNS of fetus.
  - If infection occurs during first trimester of pregnancy, frequency of disease increases.
  - Congenital infection occurs if exposed to infection for the 1st time.
  - Severely affected infants show retinochoroiditis, cerebral necrosis, hepatosplenomegaly, liver failure, convulsions and hydrocephalus.

- **Dog:**
  - Fever, anemia, respiratory distress and hemorrhagic diarrhea.
  - Serosanguinous exudates in body cavities, small nodules in lungs, numerous ulcers in intestine, paralysis and other neurological manifestations.
  - Toxoplasmosis is a common complication of canine distemper, causing pneumonia and encephalitis.

- **Cattle/Horse:**
  - Opthalmitis.

Prevention & Control:

- **Cat:**
  - Impossible if cat allowed outdoors (hunting).
  - If kept indoors, feed only tinned or dry food; control vermin's.
- **Human:**
  - Avoid ingestion of tissue cysts:
  - Wash potentially contaminated raw food thoroughly. (e.g. Salad crops).
  - Wash hand after gardening or handling cats and especially before eating.
  - Cleanout cat litter trays every day (before oocysts sporulates).
  - Do not eat undercooked meat. Wash hand after handling raw meat.
  - Care when lambing or dealing with sheep abortion or steel births.
  - Women should avoid such activities altogether if they might be pregnant.
- **Sheep:**
  - There is a vaccine (Toxovax) based on a live, avirulent strain
  - All the attenuated organisms are therefore killed by immune response
  - A single dose is given by injection at least 6 weeks before tupping
  - Protects for at least two years in absence of natural challenge.
  - Immunity is boosted by exposure to natural challenge.
  - Medicated feed (decoquinate) can be given, if practicable, during the risk period (i.e. for 14 weeks prior to lambing.)
  - Animal feedstuffs should be covered to avoid cat fecal contamination.

Genus: *Neospora*

Species: *Neospora caninum*

- The protozoan is very similar to *T. gondii.*
- Morphologically the tissue cyst is very similar to those of *Toxoplasma*, although *Neospora* cyst often has a thicker wall.
- *Neospora* does not give positive result with *Toxoplasma* immunodiagnostic kits.
- The most reliable way of differentiating the two infections is by immunoperoxidase staining of the tissue stages but serological tests using IFAT or ELISA are also in use. Specific PCR reactions have been developed.

Definite hosts: Dog, coyots and dingoes

Intermediate hosts: Cattle, other ruminant animals, pig, canine and horse

Site in final host: Small intestine

Distribution: North America, Europe, Australia, South Africa, Japan.

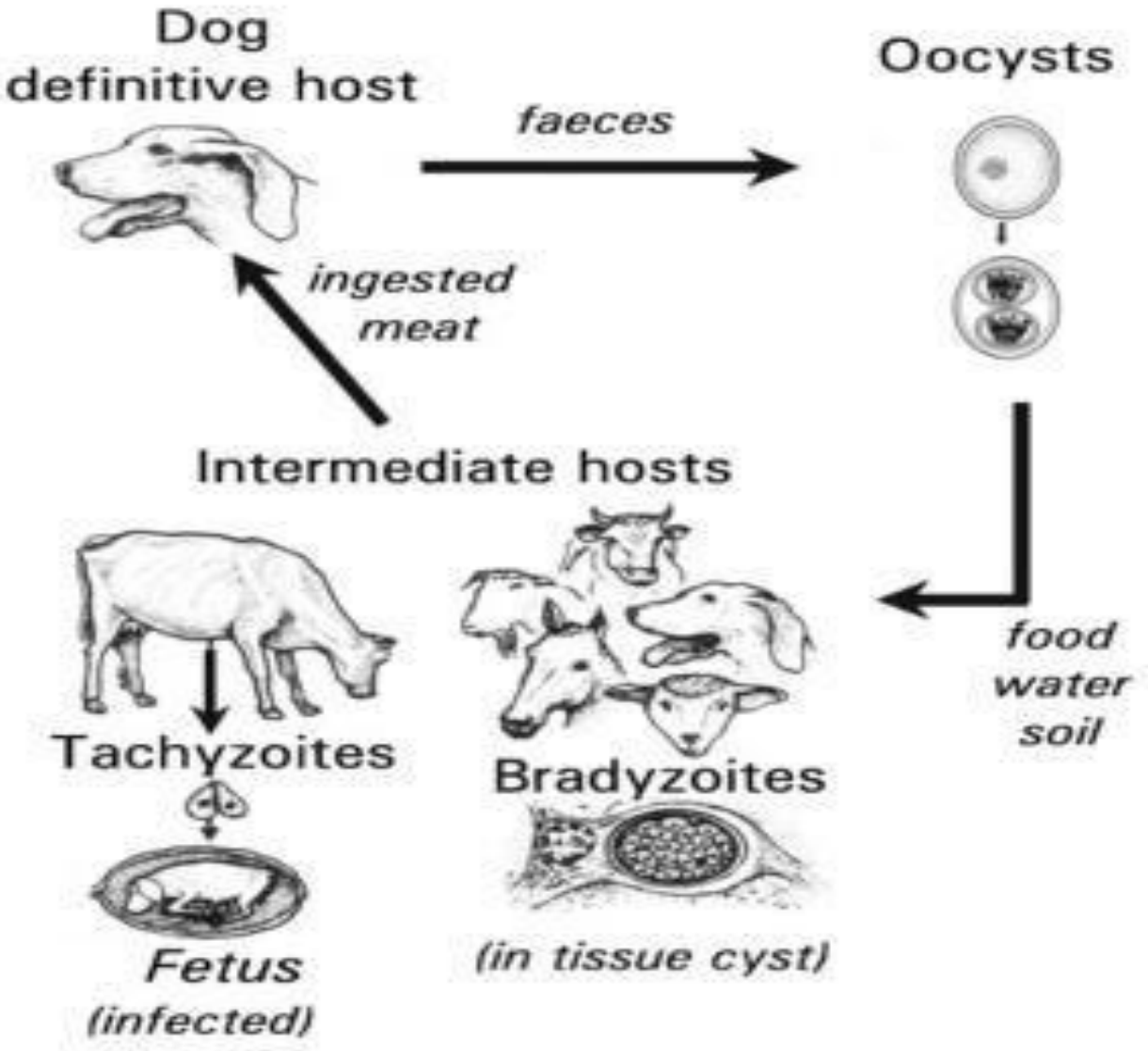
Epidemiology & Transmission

- Transplacental transmission occur in all I/H.
- An important difference from *Toxoplasma* is that, with *Neospora,* Transplacental transmission can happen in successive pregnancies.
- In cattle, the most common route of transmission is from dam to calf (vertical transmission); post natal infection appears to be uncommon.
- The infection has been transmitted experimentally to mice and cattle by feeding tissue cysts.
- Oocyst is thin, colorless, ovoid or ellipsoidal.
- Tissue cyst is round and oval & Cyst wall is smooth and irregular.

Life cycle:

- Indirect life cycle, where
  - F/H: dog, coyot and dingoes
  - I/H: Dog, cattle (deer, goat sheep, horse)
- Oocysts:
  - similar to *Toxoplasma* (10µm, 2 sporocysts each containing 4 sporozoites).
- Unsporulated oocysts in faeces of F/H
- Sporulated oocysts
- Through food and water
- I/H (Dog, cattle, sheep, goat, horse, deer, mule) in cattle several generations.
- Cyst formation in I/H (Bradyzoites, and Tachyzoites)
- Infect Dog i.e.F/H

Life cycle of *N. caninum*



Harmful effects:

Dog:

- Occurs mainly in pups, but often dog can be affected.
- Progressive ascending paralysis, especially of hind limbs often with muscle wasting.
- Multifocal necrosis in muscle, mainly diaphragm.
- Hepatitis may occur, Enlargement of liver.
- Sudden collapse due to myocarditis (sometimes).
- Pneumonia, Cervical weakness, dysphagia, death may occur.

Cattle:

- Significant or major cause of abortion in many countries (up to 42.5% of total abortion).
- Cows of any age, may abort from 3-7 months of gestation.
- Death in uterus mummified fetus autolyzed
- Still birth. ataxia, reduce milk production .
- effects on fertility in dairy cattle .
- Neuromuscular degeneration which led to hind limb paralysis.
- Born alive but diseased under weight, unable to rise or with neurological signs.

Control

- Disposal of aborted fetus and placenta by incineration or other means so that they cannot be available to definitive host.
- Prevent contamination of livestock feed with canine feces.
- Do not allow dogs to ingest bovine placental tissues, fetal membranes, or other raw meats.

Genus: Besnoitia

Species: *Besnoitia besnoiti*

- **F/H:** Cat.
- **I/H:** Cattle.
- Worldwide distribution, important in Africa
- It differs from other members of the family Sarcocystidae that cysts containing Bradyzoites are found in fibroblast in or under the skin (dermis, s/c tissue, fascia) and in laryngeal, nasal and other mucosa –in the I/H (Cattle).
- The cell enlarges and become multinucleated as the cyst grows in parasitophorus vacuoles.
- Cat shed unsporulated oocysts. Cattle get infection by ingesting sporulated oocysts. *
  - Also mechanically by blood sucking insects. (*Glossina palpalis*).
- Lymphadenopathy and edematous swelling in lower parts of the body. Bradyzoites development in skin leads to subcutaneous swelling, skin thickening, hair loss and necrosis.
- Condemnation of hides.

Genus: Hammondia

Species: *Hammondia hammondi*

Hammondia is an obligate heteroxenous protozoan of domestic cats (final host). Intracellular cysts develop mainly in striated muscle

- **Final host (F/H):** Cat
- **Intermediate host (I/H):** Rodents, Guinea pig, Dog.
- Unsporulated oocysts are colourless, spherical to sub-spherical, without a micropyle or residuum,
- Sporulated oocysts contain 2 ellipsoid sporocysts each with 4 sporozoites.
- I/H become infected by ingestion of sporulated oocysts (cat's feces).
- The multiplication of Tachyzoites in the lamina propria of intestinal wall and are followed by the production of cysts containing Bradyzoites in the skeletal muscle; Occasionally in heart muscle and brain.
- The cyst wall develops from the limiting membrane of the parasitophorus vacuole in which the Bradyzoites occur.
- Infective stage – Bradyzoites.
- Cyst stages which appear two weeks after infection are infective only for the definitive host.

Genus: *Sarcocystis*

- Cyst forming Coccidia.
- The infective form in the intermediate host (I/H) is called “Sarcocyst”.

Species	F/H	I/H
S. bovifelis/S. hirsute	Cat	Cattle
S. ovifelis		sheep
S. cruzi/S. bovicanis/S. fusiformix	Dog	Cattle, dog ,fox, hyena, wolves
S. hominis/S. bovihominis	Man, baboon, rhesus monkey	Ox
S. porcihominis	Man	Pig
S. ovicanis	Dog	lamb
S. porcicanis	Dog	Pig
S. porcifelis	Cat	Pig
S. equicanis/S. bertrami	Dog	Horse, ass, mule
S. fayeri	Dog	Horse

Oocyst:

- Sporulated when passed in faeces & contain 2 sporocysts each with 4 sporozoites
- Usually the sporulated sporocysts is found in the faeces (i.e. naked sporocysts in faeces)
- The sporocysts are difficult to find on faecal examination because they are very small & very few in number.
- The oocyst has a very fragile, thin wall, containing two strictly paired ellipsoidal sporocysts

Tissue stages:

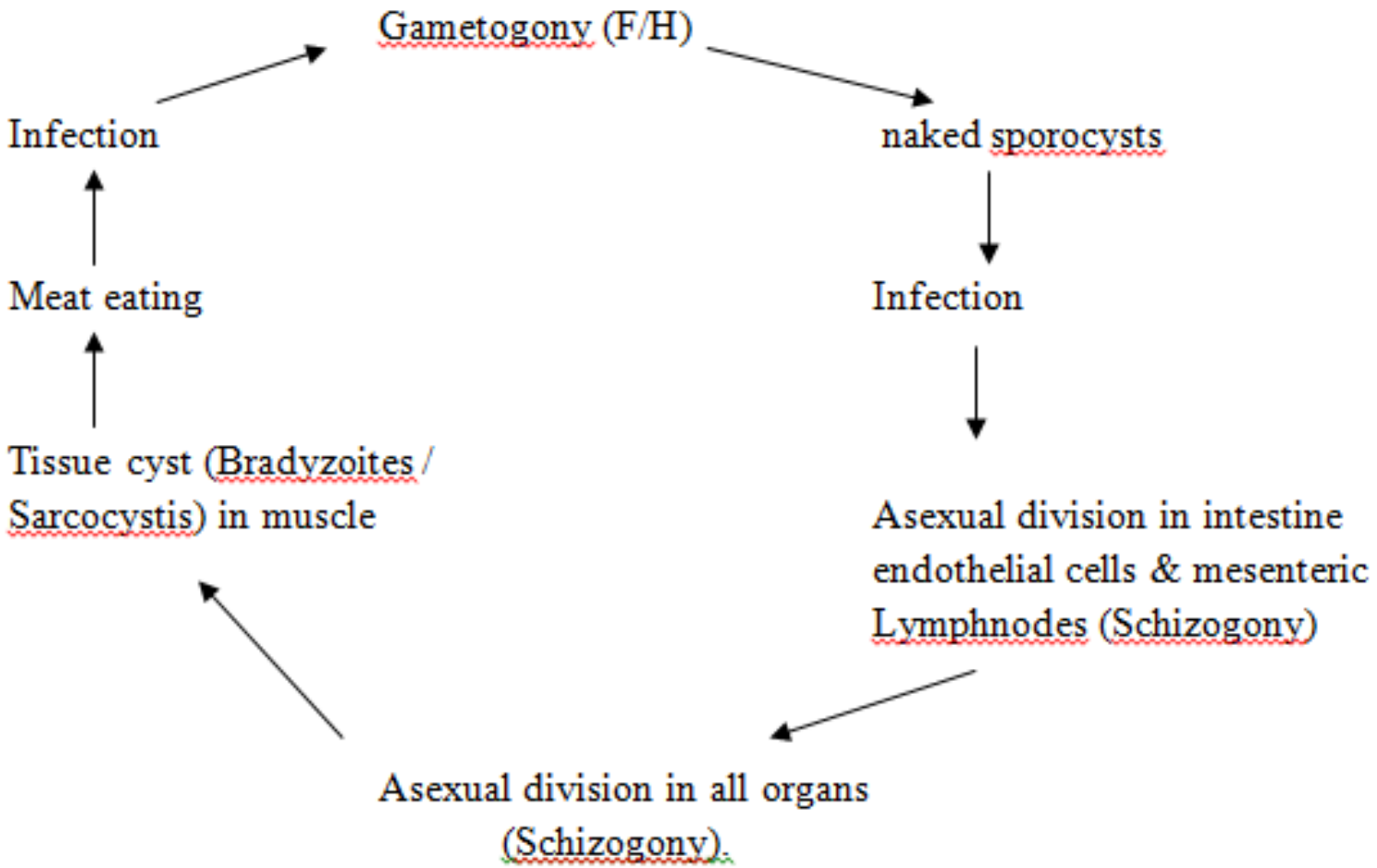
- In I/H
  1. Schizonts: in endothelial cells.
  2. Bradyzoites: can be very large & visible to the naked eyes

Epidemiology:

- The high prevalence in abattoirs, it is clear that where dogs/cats are kept in close association with farm animal or their feed.
- Sheep, dogs play an important part in the transmission of *S. ovicanis*.
- Feeding of uncooked meat. F/H are carnivores & omnivores.
- I/H are herbivores & omnivores.
- Humans are F/H for some species, I/H for others.

General life cycle of ***Sarcocystis***

Life cycle:

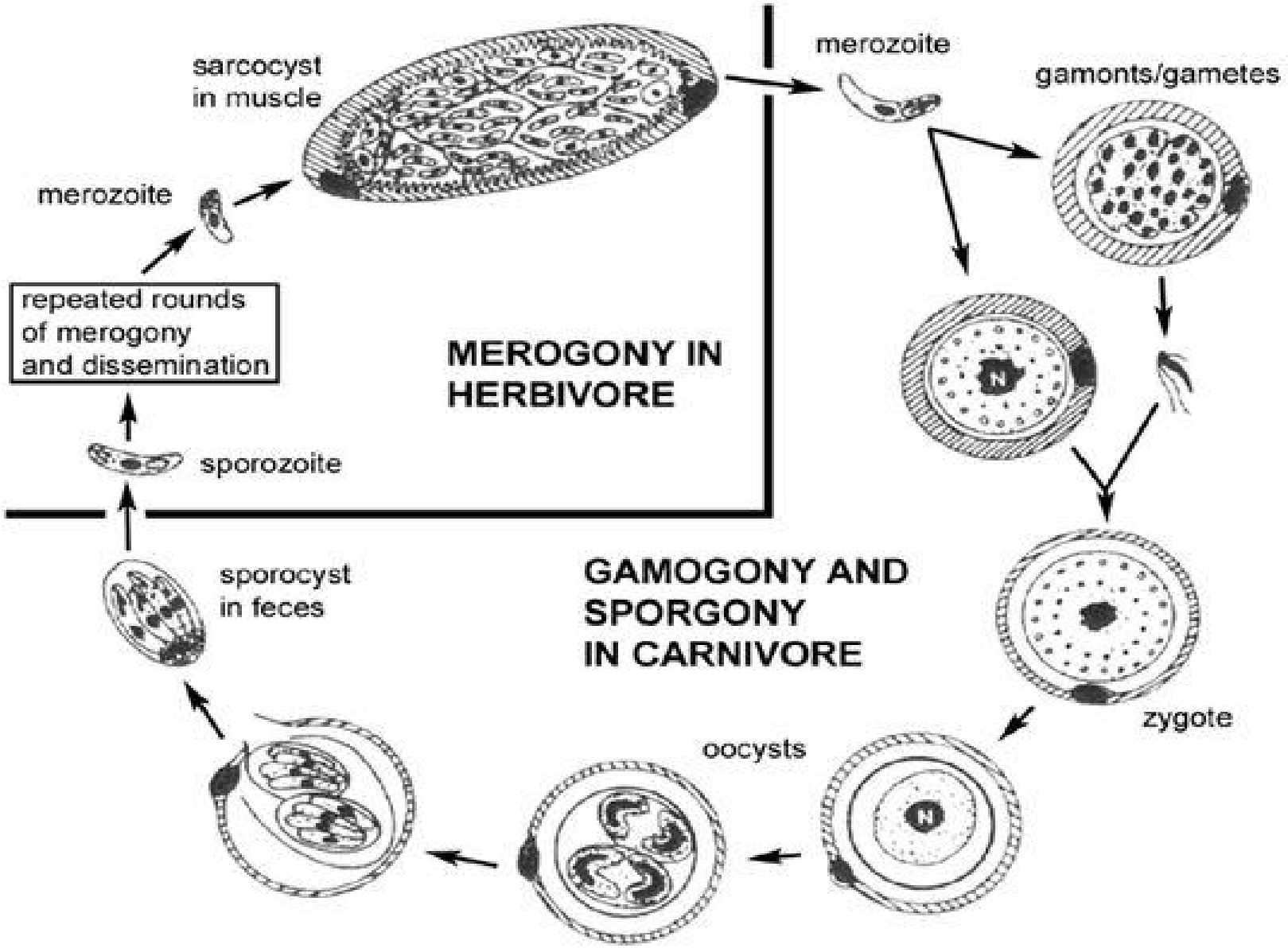


Gametogony (F/H)

- The infection of F/H is by ingestion of Bradyzoites cysts (Sarcocyst) in the muscles of the I/H.
- The Bradyzoites are liberated in the intestine of the F/H.
- Pass to the sub epitheloid lamina propria
- Differentiate into micro & macro gametocytes
- Formation of oocysts.
- **Sporulation of oocysts within the body.
- 2 sporocysts are formed each containing 4 sporozoites
- Usually the fragile oocysts wall ruptures & free sporocysts are found in the faeces.

Schizogony (I/H)

- The infection of I/H is by ingestion of sporocysts
- Followed by at least 3 asexual generations first
- Sporozoite are released from the sporocysts
- Invade the intestinal wall & enter the capillaries where they locate in endothelial cells.
- Under 2 schizonts cycle.
- Then, the 3rd asexual cycle occurs in the circulating lymphocytes.
- The resulting merozoites penetrate muscle cells.
- There they encyst & then divided by a process of budding or endodyogeny giving rise to broad banana shaped Bradyzoites (Brady means slow) contained within a cysts (this is the mature Sarcocyst & is the infective stage for the carnivorous final host.)


sarcocyst
in muscle
merozoite
gamonts/gametes
merozoite
repeated rounds
of merogony
and dissemination
MEROGONY IN
HERBIVORE
N
sporozoite
sporocyst
in feces
GAMOGONY AND
SPORGONY
IN CARNIVORE
zygote
oocysts
N

Harmful effects:

- In I/H, the principle pathogenic effect is attributable to the 2nd stage of schizogony
- In experimental infection of calves, death after one month
- Petechial hemorrhages in every organ (heart), generalized lymphadenopathy.
- In experimental infection in adult cows- abortion
- A naturally occurring chronic disease of cattle **"Dalmeny disease"** has been recognized in Canada, USA, & Britain. This is characterized by emaciation, submandibular edema, recumbancy & exophthalmia.
- *S. ovicanis* causes abortion in ewes
- Severe myositis and encephalitis in lambs.
- In horse – **"Equine protozoal myeloencephalitis".**it is characterized by ataxia, muscular weakness, caused by *S. neumona.*
- Presence of cysts in the muscle of food animals resulting in condemnation of carcasses.
- Anorexia, fever, anemia, loss of weight
- Disinclination to move and sometimes recumbency.
- In lamb – Dog sitting posture.
- In cattle – marked loss of hair at the end of tail.
- Abortion may occur in breeding stock.

Control:

- Hygienic management.
- Farm dogs and cats should not be housed in or allowed to defecate in pens where livestock are housed.
- It is also important that they are not fed uncooked meat.
- The introduction of Amprolium into the diet of the animals has a prophylactic effect.
- Carnivores should not be allowed to eat raw meat, offal, or dead animals.
- Infected pork and beef could be made safe by cooking at 70°C (158°F) for 15 min or by freezing at –4°C (24.8°F) for 2 days or –20°C (-4°F) for 1 day.

Subclass: Piroplasmea (Blood borne protozoa)

Order: Piroplasmidea

Family: 1. Babesiidae 2. Theileriidae

Genus: **1.** *Babesia* 2. *Theileria*

Genus: Babesia

Host: All domestic animals

Vector: Ixodidae ticks (Hard ticks)

Site: single/pair in RBC (Major link with haemopoeitic and cardiovascular system).

Distribution: Worldwide.

Transmission:

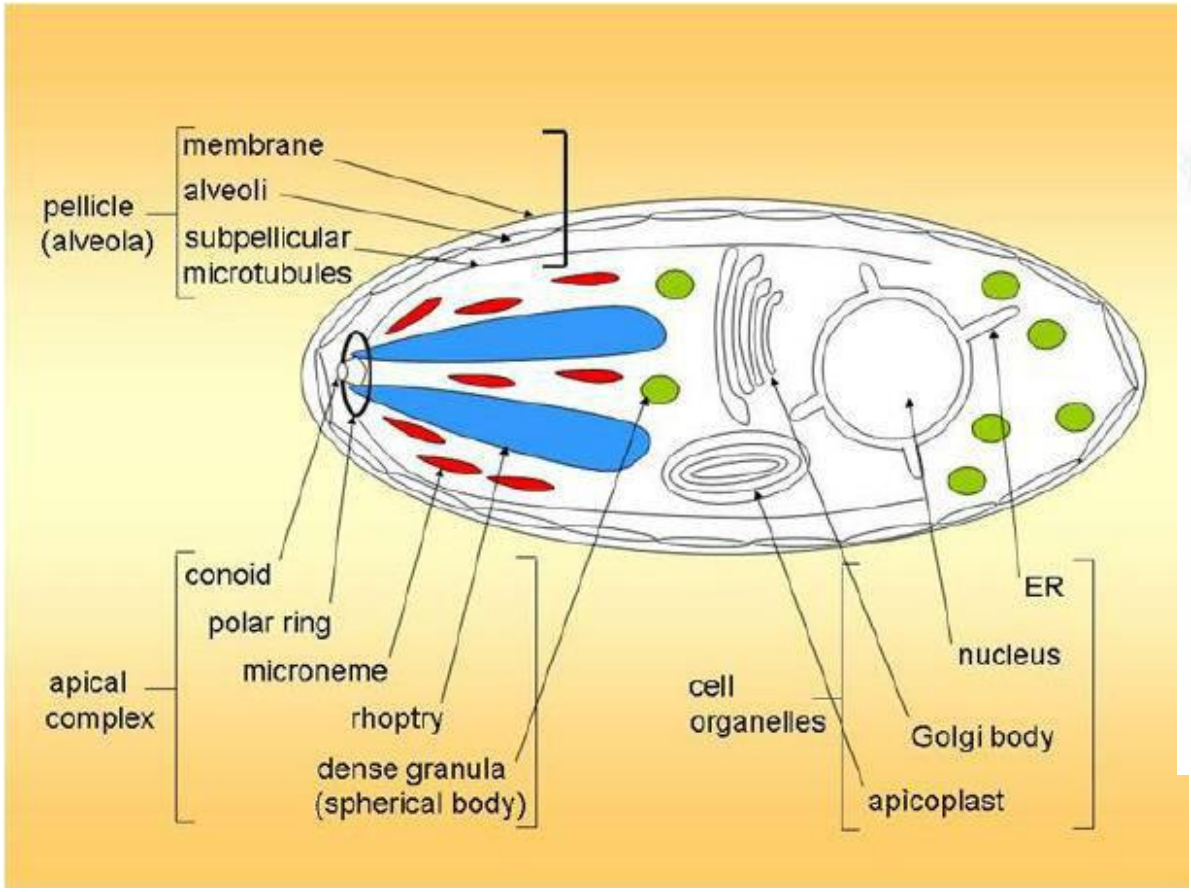
Vector, hard ticks of the family Ixodidae, in which trans-ovarian transmission infection ensures that *Babesia* are transmitted by the stages of the next generation of tick. Depending on the species, transmission may be by the larval, nymphal or adult stages or even all three

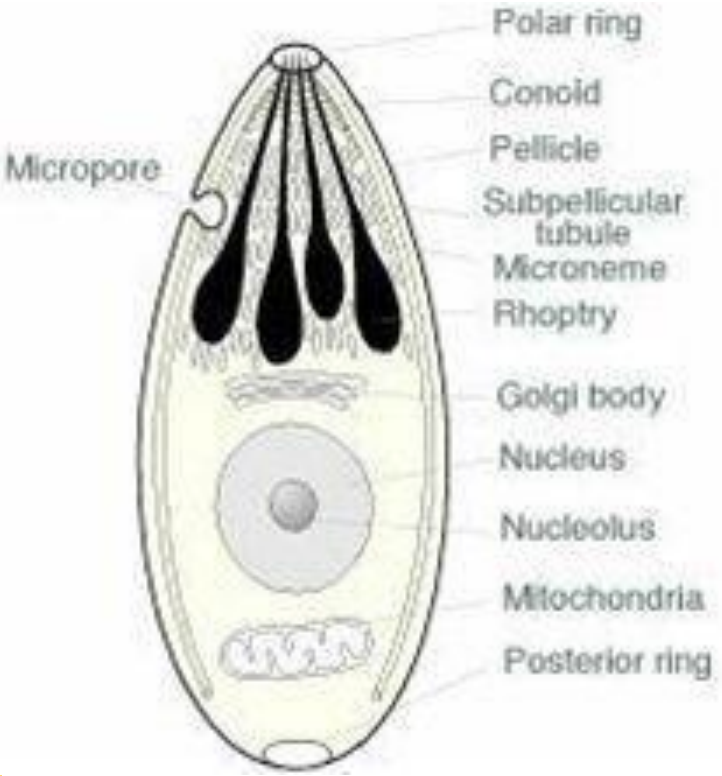
When infection persists from one stage to the next in two or three host ticks feeding on different hosts, transmission is said to be **transtadial**

Species

Animal	Species involved	Vector
Cattle, Buffalo	***Babesia bigemina*** (Large *Babesia* size 4.5 µm, distribution in Asia, Africa, Australia, America, South Europe)	1. *Boophilus microplus* *2. Boophilus annulatus,* *B.calcaratus* *4. Rhipicephalus evertsi, R.bursa* *6. Rhipicephalus appendiculatus* *7. Haemaphysalis punctata*
	2. *Babesia bovis* (Small Babesia, size 2 µm, distribution: same, available in Bangladesh)	1.*Boophilus microplus* *2. B. calcaratus* *3. Rhipicephalus bursa*
	3. *Babesia divergens* (size-1.5 µm)	1. *Ixodes ricinus*
	4. *Babesia major*	1. *Haemaphysalis punctata*
Sheep, Goat.	***Babesia ovis, Babesia motasi*** (Distribution: Tropical, subtropical areas and Southern Europe)	1. *Rhipicephalus spp.* *2. Haemaphysalis spp.* *3. Dermacentor spp.* *4. Ixodes spp.*
Horse	***Babesia equi, Babesia caballi*** (Distribution: Asia, Europe, America, and Africa.)	1. *Hyaloma spp.* *2. Rhipicephalus spp.* 3. *Dermacentor spp.*
Dog	***Babesia canis, Babesia gibsoni*** (Distribution: Same as horse)	1. *Rhipicephalus sanguineus* *2. Dermacentor spp.*
Cat	***Babesia felis***	1. Hard tick
Pig	***Babesia trautmanni***	1 *Rhipicephalus sanguineus*

Morphology


membrane
alveoli
pellicle (alveola)
subpellicular microtubules
conoid
polar ring
microneme
rhoptry
dense granula (spherical body)
apical complex
ER
nucleus
cell organelles
Golgi body
apicoplast


Polar ring
Conoid
Micropore
Pellicle
Subpellicular tubule
Microneme
Rhoptry
Golgi body
Nucleus
Nucleolus
Mitochondria
Posterior ring

Identification and morphology:

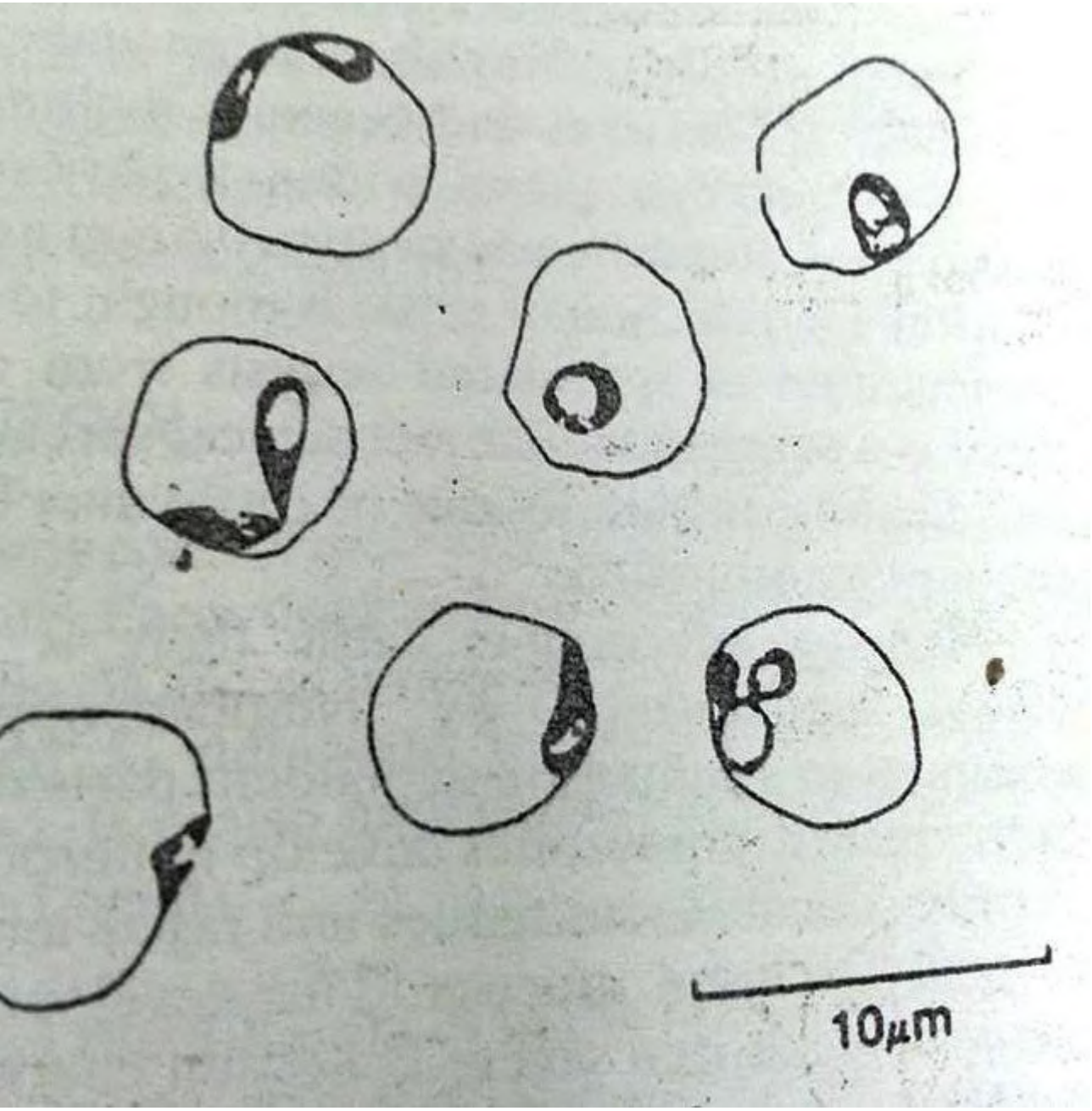
The organisms are found within red cells, almost always single or as pairs, often arranged at a characteristic angle with their narrow ends opposed.

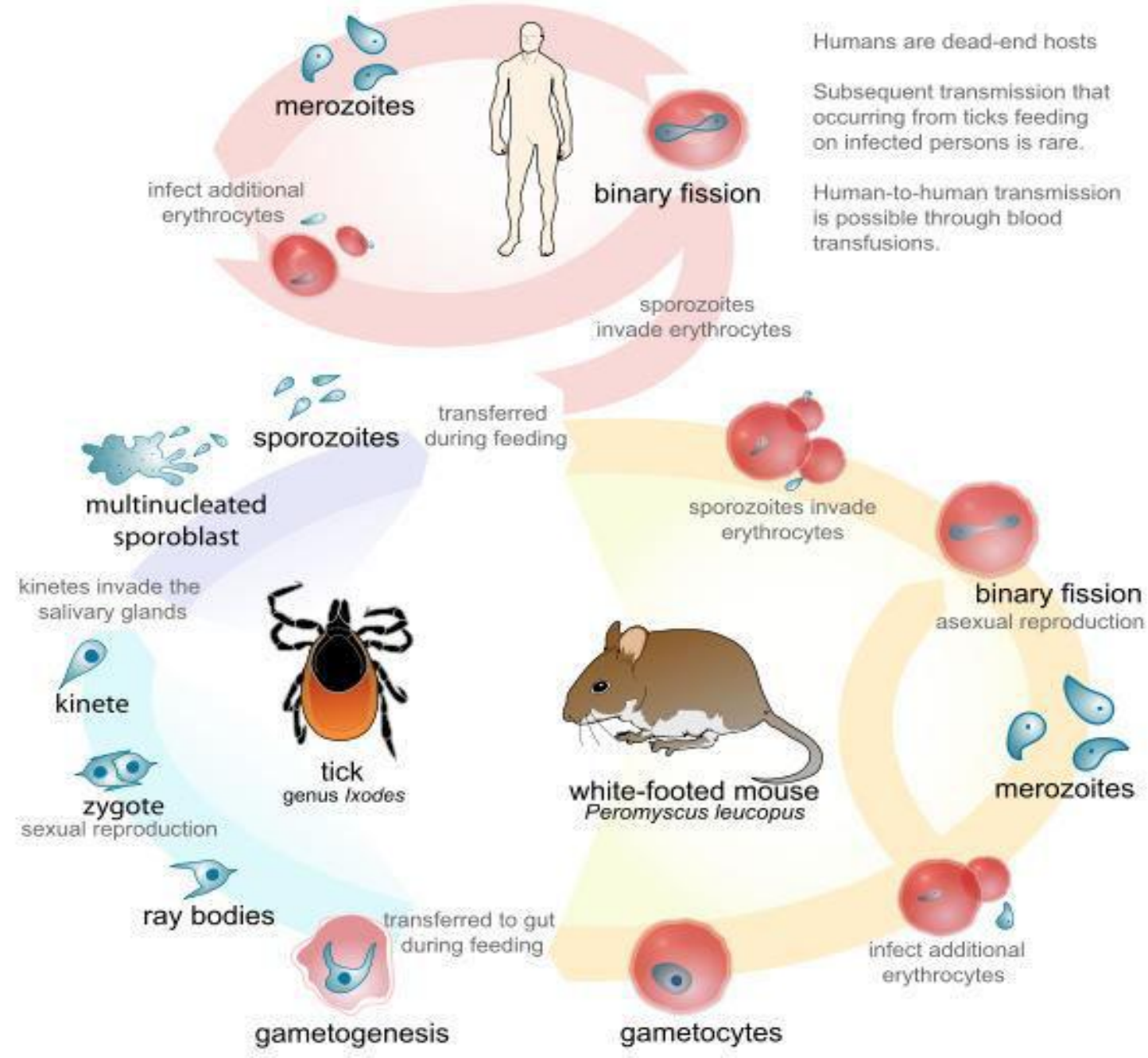
Typically they are pyriform but may be round, elongated or cigar shaped.

Small *Babesia* whose pyriform small bodies are 1-2.5 μm long and the large *Babesia* which are 2.5-5.0 μm long; with Romanowsky dyes the cytoplasm appears blue and nucleus red.

Under EM the parasite is seen to possess at its blunt end and electron dense "apical complex" which is thought to be concerned with assisting penetration of the erythrocytes.

Babesia sp. enter RBC at the sporozoite stage. Within the RBC, the protozoa become cyclical and develop into a **trophozoite ring**. The intra-erythrocytic trophozoites multiply by binary fission, forming two to four separate merozoites which have a tetrad structure **coined a Maltese-cross form**. The tetrad morphology, which can be seen with Giemsa staining of a thin blood smear, is unique to *Babesia*, and serves as a distinguishing feature from *Plasmodium falciparum*, a protozoan of similar morphology that causes malaria.


10μm


merozoites
binary fission
infect additional erythrocytes
sporozoites invade erythrocytes
Humans are dead-end hosts
Subsequent transmission that occurring from ticks feeding on infected persons is rare.
Human-to-human transmission is possible through blood transfusions.
sporozoites
transferred during feeding
multinucleated sporoblast
sporozoites invade erythrocytes
binary fission
asexual reproduction
kinetes invade the salivary glands
kinete
tick
genus Ixodes
white-footed mouse
Peromyscus leucopus
merozoites
zygote
sexual reproduction
ray bodies
transferred to gut during feeding
infect additional erythrocytes
gametogenesis
gametocytes

Life cycle

- *Babesia* organism divides asexually by binary fission to form 2 or sometimes 4 individuals (merozoites). Eventually the host cell ruptures & the organisms are liberated from erythrocytes to invade other new red cells.

- When the parasitemic blood is ingested by the Ixodid tick, a sexual phase occurs in the tick gut followed by Schizogony

- Schizogony results in the production of elongated, motile, club-shaped bodies-**vermicules**

- Vermicules migrate to the tissues of the tick, specially the ovary and undergo further multiplication to form more vermicules. (7 days required to reach at this stage)

- In the ovary, the vermicules invade the eggs and subsequently, continue to multiply in the tissues of the hatched larva

- When the larva first feed, the vermicules enter the salivary acini and form infective sporozoites, which are inoculated into the new hosts before feeding ceases

- When stage to stage transmission (**transtadial**) occurs, vermicules again reach the salivary glands of the next stage of ticks when feeding commences, and mature to become infective stage

- Some species of *Babesia* may be transmitted through the ovary for two or more generations of female ticks—**transovarian/vertical transmission**

Epidemiology

The epidemiology depends on the interplay of a number of following factors-

1) **The virulence of particular species of *Babesia***

Babesia divergens in cattle and *B. canis* in dogs are relatively pathogenic while *B. major* in cattle and *B. ovis* in sheep usually produce only mild and transient anaemia.

2) The age of the host: It is frequently stated that there is an inverse age resistance to *Babesia* infection. Young animals are less susceptible to **Babesiosis** than older animals.

3) The immune status of the host: In endemic areas, the young animal first acquire immunity passively, in the colostrums of dam and as a result, often suffers only transient infection with mild clinical signs. However, these infections are apparently sufficient to stimulate active immunity, although recovery is followed by a long period during which they act as carriers when, although showing no clinical signs, their blood remains infective to ticks for many months. It is thought that this active immunity is dependent on the persistence of the carrier state and the phenomenon is termed as **premunity**.

4) Level of the tick challenge:
In endemic areas, where there are many infected ticks, the immunity of the host is maintained at a high level though repeated challenge and overt disease is rare.

In contrast, where there are few ticks or when they are confined to limited areas, the immune status of the population is low and the young animals receive little, if any colostral protection. In this circumstances, if the number of ticks suddenly increase due to favorable climatic conditions or to a reduction in dipping frequency, the incidence of clinical cases may rise sharply. This situation is known as **enzootic instability**.

5) Stress: In endemic areas the occasional outbreak of clinical disease, particularly in adult animals is often associated with some form of stress, such as parturition or presence of another disease such as tick borne fever.

Pathology (Babesiosis /Tick fever / Piroplasmosis / Red fever / Splenic fever / Biliary fever/ red water fever.)

The incubation period varies depending on the species of *Babesia* involved & it ranges from 5-10 days following infection multiplication of protozoa occurs in peripheral vessels and there is intravascular hemolysis. It has been pointed out that proteolytic enzyme are liberate from the infected erythrocytes & these enzymes interact with components of blood & thus lead to increased erythrocytic fragility, hypotensive shock & disseminated intravascular coagulation. Parasitaemia, fever, hemoglobinuria, haemolytic anaemia, and hemoglobinaemia are the main clinical attributes. PVC falls bellow 20%. Parasitemia is detectable when clinical signs appear, and may involve 0.2% -45% RBC.

At necropsy, carcass is pale and jaundiced , the bile is thick and granular. There may be sub-epicardial or sub-endocardial haemorrhage

Death occurs due to anaemic anoxia. The Pathogenicity depends on the species of *Babesia* involved. In *B. bovis* & *B. canis* infections clumping of RBCs may also occur in the capillaries of the brain, producing nervous signs **"Cerebral Babesiosis".**

- Hyperexcitability
- Incoordination, Convulsion
- Locomotion disturbance
- Ascities, dyspnea
- Enlarged tonsil
- Bleeding from muzzle

Clinical signs:

•High fever, anemia anoxia, depression & weakness.

• Increased heart & respiratory rate.

•The conjunctival mucous membrane is brick red in the initial stage & pale in the terminal stage.

•In terminal stage - severe jaundice & hemoglobinuria (**Coffee color urine**).

• In young animals, the disease is associated with sub clinical manifestation due to age resistance.

•The febrile period is characterized by tremors of skeletal muscles, when the temperature recedes, the animal assume either lateral recumbency or prostration, cessation of ruminal movement & abortion may occur in cattle, intermittent temperature in chronic infection.

•In *B. bovis* infection spasm of anal sphincter and **"pipe stem faeces"** are observed .in fatal cases, the animals are unable to stand or their feet in spite of support .A few hours before death tremors of the neck muscles, involuntary movements of the facial muscles accompanied by shallow, jerky respiration are observed .The infection may involved the central nervous system leading to ataxia, paddling of limbs, hyper excitability, incoordination, comma etc. The condition known as "**Cerebral form of babesiosis**".

•Haemoglobinuria in such cases last for a day or two.

Canine Babesiosis: Canine Babesiosis is similar to bovine Babesiosis. In dog, five forms of the disease may be encountered.

•Alimentary form - stomatitis, gastritis, enteritis.

• Respiratory form - respiratory distress (Dyspnea).

•Circulatory form - edema.

•Ocular form - keratitis and Iritis.

•Muscular form - muscle weakness/pain.

•Fever, Anemia, Jaundice, haemoglobinuria - common. But jaundice is not common in ***B.gibsoni*** infection.

Per acute case: Collapse and profound anemia leading to death within 1-2 days in imported dogs. ***B.canis*** is responsible for "**Cerebral babesiosis**" characterized by various forms of nervous signs such as hyper excitability and incoordination. A typical signs like convulsion, locomotion disturbance, ascitis, dyspnea, cough, enlarged tonsils and bleeding from muzzle may be observed in some cases. Recovered dog acts as carrier.

Equine Babesiosis: In Horse, haemoglobinuria is rare in occurrence and jaundice is more common, hence it is known as "**Biliary fever"** *B. equi* is more pathogenic than *B.caballi*. Fever, Anemia is common.

Control

A. For infected animal

•Firstly isolate the animal and give proper specific and supportive therapy.
•Proper hygienic management.
•Protect from ticks.

B. For healthy or noninfected animal

1) Tick control:

•Programme is to be taken to control tick population.
•A herd can be made tick free by dipping or spraying at periodical interval with Acaricide

2) Vaccination: Following vaccines have been used-

•Live vaccine: e.g. *B.bigemina.*
• Killed vaccine: e.g. *B.bigemina*.
•Inactivated from cell culture.
•Irradiated vaccine: e.g. Irradiated *B.bigemina*. Vaccine. The vaccine is capable of triggering optimum and protective immune response.
•Vaccination by attenuated blood :(attenuation of virulence of *Babesia spp* by repeated passage through splenectomized calves).
•Vaccination by blood from recently recovered animal.(by exchange transfusion rather exsanguinations.)
•In cattle selection and breeding of cattle which acquire a high degree of resistance to ticks, which is practiced in Australia.

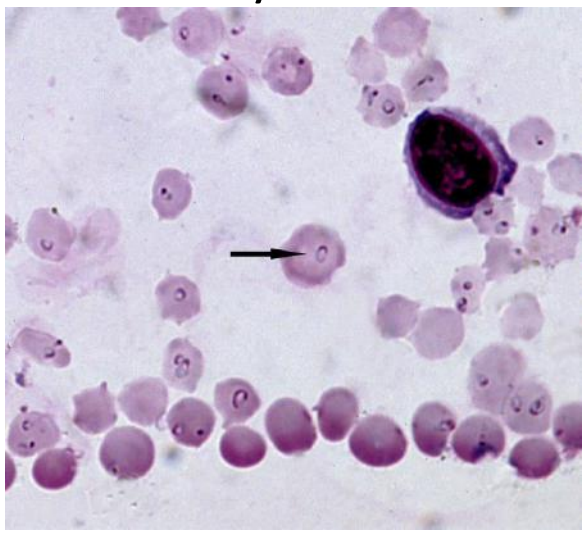
Immunity:

- Inverse age resistance.
- Breed of animals:-*Bos taurus* more susceptible than *Bos indicus.*
- Infection leads to immunity.
- Spleen plays a vital role in the development of immunity.
- Antigenic strain variation.
- Colostral acquired immunity.

Zoonotic aspect: Human cases of Babesiosis have been recorded and some of human cases have died. Since 1957; B. *divergens*: Jugoslavia, Russia, Ireland, Scottland. More recently; *B.microti* (Rodent parasite); USA (In most cases, the clinical syndrome and presence of intraerythrocytic parasites had suggested an initial diagnosis of Malaria).

Genus: *Theileria*

Theileria is an obligatory intracellular protozoa of wild and domestic animals. Members of this family are round, ovoid, rod like or irregular forms found in lymphocytes, histiocytes & RBC. They are transmitted by tick (vector).



Non pathogenic:

1. *Theileria mutans* in Cattle
2. *Theileria sergenti* in Cattle
3. *Theileria ovis* in sheep
4. *Theileria cervi* in deer

- (Cross immunity between *Theileria parva & Theileria lawrenci*)
- **Disease:** Theileriosis /Rhodesian tick fever / East coast fever.

Species	Host	Vector(Tick)	Disease	Distribution
T. parva	Cattle, Buffalo	*Rhipicephalus appendiculatus*	East coast fever	East & Central Africa
T. annulata	Cattle, Buffalo	*Hyalomma* spp	Mediterranean coast fever or tropical theileriosis	North Africa, South Europe, Middle East, Asia, Tropical & Sub tropical areas.
T. hirci	Sheep, Goat	*Hyalomma* spp	Malignant ovine (caprine) theileriosis	North Africa, South Europe, Middle east, Asia
Others				
T. lawrenci	Buffalo, Cattle	*R. appendiculatus*		East, Central & South Africa.
T. mutans	Cattle	*R. appendiculatus, R. evertsi, Haemophysalis punctata.*		Africa,Asia,Australia, Sovietunion, Tropical or Subtropical Asia
T. camelensis	Camel	*Hyalomma* spp		Egypt, Somalia, Turkestan
T. equi	horse		Equine piroplasmosis	

Theileria parva

Identification: Two forms:

1. Schizonts/Koch's blue bodies form &
2. Erythrocytic forms (piroplasms)

- In lymphocyte (Lymph gland & Spleen):
  - Circular or irregular shaped schizonts called "Koch's blue bodies" in the cytoplasm
  - With Giemsa stain- 2 types of Schizonts are seen-
    1. Macroschizont:
       - Blue, 8 µm in diameter & Contain 8 nuclei (purple particles).
    2. Microschizont:
       - Next stage of Macroschizont, mostly similar in size
       - Contain 36 small nuclei (purple particles) In RBC:
- In RBC
  - Rod (2µm×1µm), round, oval, comma or ring formed piroplasms.
  - With Giemsa stain - The cytoplasm of each is blue with a red chromatin dot at one end.

Host: Cattle, Buffalo

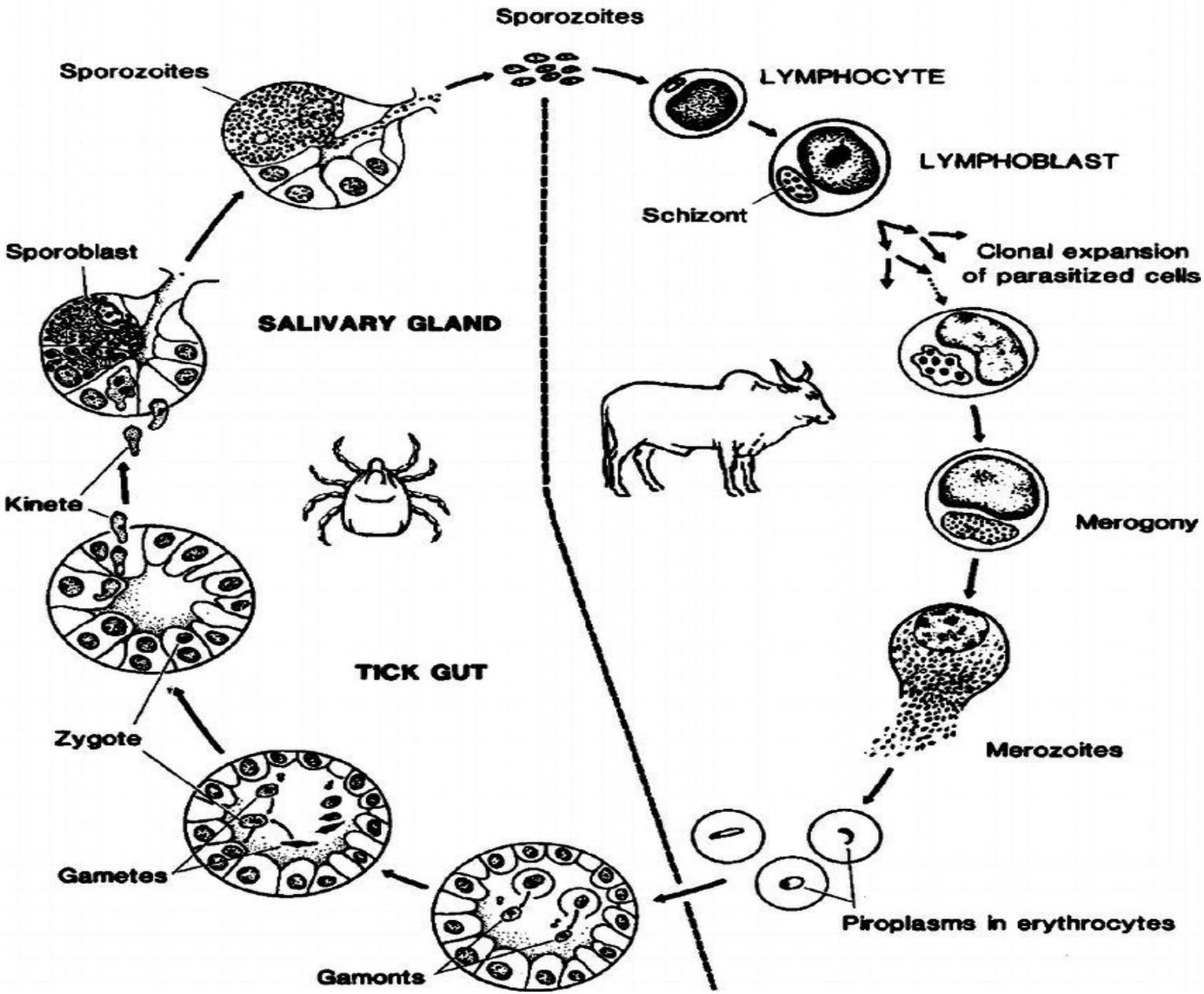
Vector: *Rhipicephalus appendiculatus* (Brown ear tick, 3 host Ixodid)

Transmission: Trans-stadial, No transovarian transmission.

Epidemiology:

- Exotic and cross bred dairy cattle are more susceptible than indigenous cattle.
- Distribution in Africa, USSR, part of China and Indian subcontinent
- All age group of exotic cattle are highly susceptible. The young indigenous calves are very much susceptible.
- Outbreak is seasonal as the tick vector most active after the onset of rain throughout the year where rainfall is constant.
- Indigenous cattle in endemic areas show a high degree of resistance, mortality negligible.
- Such cattle remain carrier.
- *Theileria parva lawrenci*: - In the African buffalo which act as carrier. Severe disease with high mortality in cattle.

LIFE CYCLE OF *THEILERIA PARVA*



- The sporozoites are (infective stage) inoculated into Cattle by the tick & quickly enter lymphocytes and associated lymph gland (usually parotid) and spleen.
- The parasitized lymphocyte transforms to a lymphoblast which divides rapidly as the macroschizont develops.
- This division is apparently stimulated by the parasite, which itself divides synchronously with the lymphoblast to produce 2 infected cells. The rate of proliferation is such that a 10 folds increase of infected cells may occur every 3 days.
- About 12 days after infection a population of the Macroschizont develops into Microschizonts and within a day or so these produce the Micromerozoites, which liberated by rupture of the Microschizonts, invade the RBCs to become piroplasms.
- The piroplasms do not multiply in the RBCs. For completion of the life cycle the Piroplasms ingested by the larvae or Nymphal stages of the 3 host tick.
- A second phage (Gametogony) occurs in the tick gut followed by the formation of sporoblast in the salivary glands.
- No further development occurs until the next stage of the tick starts to feed when the sporoblast produce infective sporozoites from about 4 days onwards.
- (Since female tick feed continuously for about 10 days and males intermittently over a longer period, this allows ample time for infection of the host).

Harmful effects: The sequences of events in a typical and fatal infection progress through three phages:-

- Piroplasms in RBCs of final host
- Tick feeds & become infected (larva or nymph stage)
- Gametogony in tick gut (sexual)
- Asexual multiplication in salivary gland
- Infective sporozoites in salivary glands
- Invades lymphocytes/ Lymphoblast
- Tick feeds & Transmits infection Schizogony

- **1st week:** Incubation period, neither parasitemia nor lesion.
- **2nd week:** Marked hyperplasia and expansion of the infected lymphoblast population, initially in the regional lymph node and then the whole body.
- **3rd week:**
  - Lymphoid depletion and disorganization massive lymphocytosis followed by exhaustion of the lymphoid tissues and leucopenia.
  - Atrophy of the cellular contents of the lymph nodes and spleen.
  - Pulmonary edema and emphysema.
  - Petechial and echymotic hemorrhages on the GIT mucosa also on the serosal and mucosal surface of the organ.
  - Occasionally nervous signs “Turning sickness” due to the accumulation of Schizonts in the “cerebral capillaries” (Cerebral Theileriosis).

Clinical signs:

- High rise of body temperature(40.5 - 41.5^{o}c)
- Enlargement of regional superficial lymph node.
- Increase in heart and respiratory rate.
- Slight anorexia, restlessness and rough coat.
- Tense eye ball along with watery lacrimation.
- Laboured respiration, serous nasal discharge and coughing.
- Depression and Petechial hemorrhage on conjunctiva
- Declination of fever and development anemia with high colored urine in later stage
- Bilirubinuria and jaundice in some cases
- Urticarial type skin lesions.
- Occasional nervous sign i.e. turning sickness.
- Weakness, Prostration and death.

Control:

- Control of tick population is necessary. Systemic application of Acaricide and rotational grazing may minimize the tick population. Exotic and crossbred cows should be kept in tick free shed as far as possible.
- Legislative control on animal movement.
- Fencing to control nomadic cattle.
- Vaccination:-

  i. "Rakshavac-T"

  ii. Current vaccine: - Frozen stabilate of ground up ticks (GUT) containing infective sporozoites i.e. a live unattenuated vaccine. Long acting OTC is administered at the same time as vaccine. Slow down Schizogony giving immune response time to develop.
- Isolation of infected animal and provide proper treatment with hygienic management and protect from ticks.

Theileria annulata

- In early stage- Similar clinical sign as *T. parva*
- In later stage- Hemolytic anemia, often icterus.

Theileria lawrenci

- Causes “corridor disease” in cattle in central Africa.
- Mortality reaches 80% or more.
- African buffalo resistant and act as carrier.
- Found in 100 miles corridors between Hluhluwe & Umfolozi game reserves in Zululand.

Theileria hirci

- Acute &high fatal disease caused by the protozoan in sheep/goat.
- All are same as *Theileria annulata* with fever, enlarged lymphocytes, anemia, and icterus in susceptible animals.

Class: Sporozoa

Suborder: Haemosporina

- Schizogony --- in vertebrate
- Gametogony & sporogony ---in bloodsucking arthropods
- Motile zygote (Ookinete)

Family: Plasmodiidae (Medical & Veterinary importance)

Genus:

- *Plasmodium* (Vector: Mosquito)
- *Haemoproteus* (Vector : Hippoboscidae & midges)
- *Leucocytozoon* (Vector: *Simulium*)

Genus: Plasmodium

Host: Human, other mammals, birds (vertebrate)

Vector: Mosquito (invertebrate)

Reproduction:

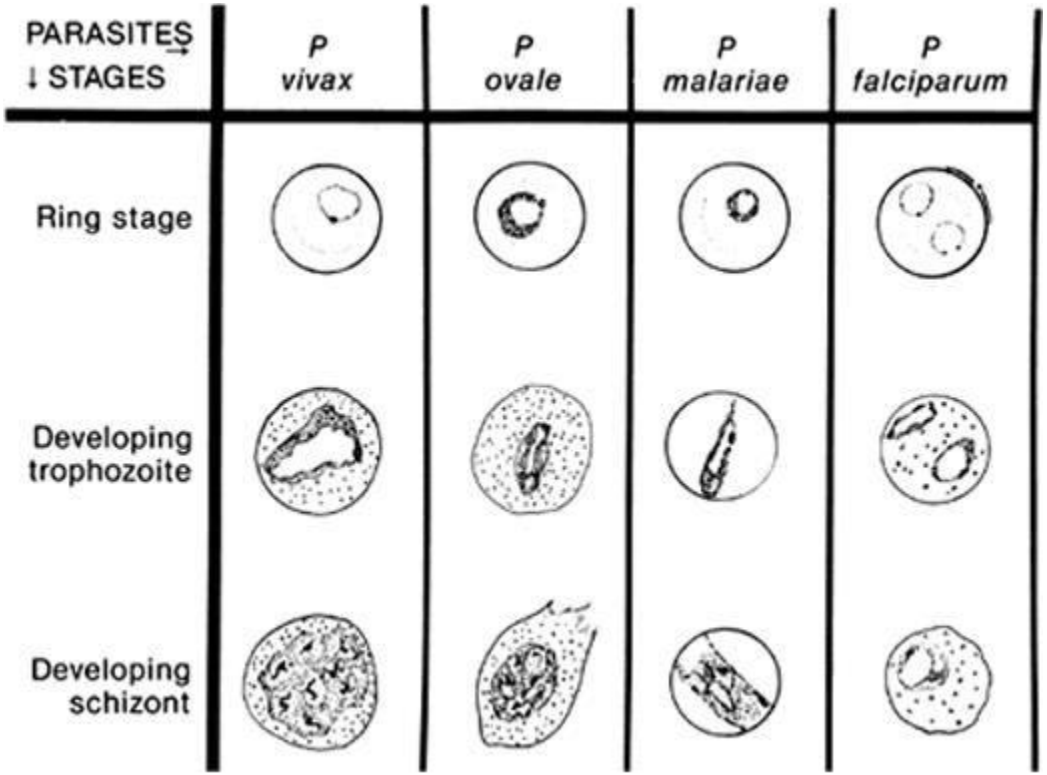
- Asexual (schizogony) in F/H (vertebrate), sexual in I/H
- RBC, Endothelial cells of internal organs
- Sexual (Gametogony /sporogony) in vector (invertebrate) - Blood sucking insects, where
- For mammalian - Anopheline mosquito
- For avian - Culicine mosquito/ *Aedes*
- But, Gametogony start in F/H & mutation of gamete occur in vector
- The major consideration is devoted to avian malaria

Avian species Vector: *Culex* & *Aedes*

A. Species with round or irregular gamonts which displace the nucleus of host cell.	
Species	Host
Plasmodium cathemerium	Common in passerine birds e.g.: English sparrow, red winged blackbird etc.
Plasmodium juxtanucleare Highly pathogenic	
Plasmodium relictum Highly pathogenic for pigeon.	Pigeon, mourning Drove, some passerines & Anatidae (worldwide)
Plasmodium gallinaceum	
B. Species with elongate gamonts which do not usually displace the host cell nucleus	
Species	Host
Plasmodium circumflexum	Passerines birds, ruffled grouse, Canada goose.
Plasmodium elongatum	English sparrow, carriers
Plasmodium fallax	Owl, African guinea fowl, chicken.

Identification:

- The trophozoite is a small rounded form containing a large vacuole
- The nucleus is situated at one of the poles, giving the young form a‘signet ring’ appearance when staine by Giemsa.
- Both gametocytes and meronts of *P. gallinaceum* can be round, oval or irregular in shape.



Human species Vector: Anopheles

Species	Disease	Distribution
Plasmodium falciparum The most malignant form of malaria in human.	Malignant tertian malaria/falciparum malaria/subtertian malaria. Paroxysms every 48 hours	Widely distributed in tropics relatively uncommon in temperate zones.
Plasmodium malariae	Quartan malaria Paroxysms every 72 hours	Tropical and subtropical areas.
Plasmodium ovale	Mild tertian malaria Paroxysms every 48 hours	India, Africa and Philippines.
Plasmodium vivax	Benign tertian or vivax malaria Paroxysms every 24 hours	commonest and the most widely distributed.

Non-human primate's species

A) Quotidian malaria: In this type, the schizogony cycle takes one day.

1.Plasmodium knowlesi

B) Tertian malaria: In this type the schizogony cycle takes 2 days.

1. *Plasmodium cynomolgi*

C) Quartan malaria: Schizogony cycle takes 3 days.

1. *Plasmodium inui*

Non-human primate's malaria as a zoonosis

True zoonotic species:

1) *Plasmodium cynomolgi.*

2) *Plasmodium knowlesi*

3) *Plasmodium inui*

4) *Plasmodium schwetzi*

Anthropozoonotic species (Human from infecting animals)

1) *Plasmodium schwetziis* anthropozoonotic form of *P. ovale*

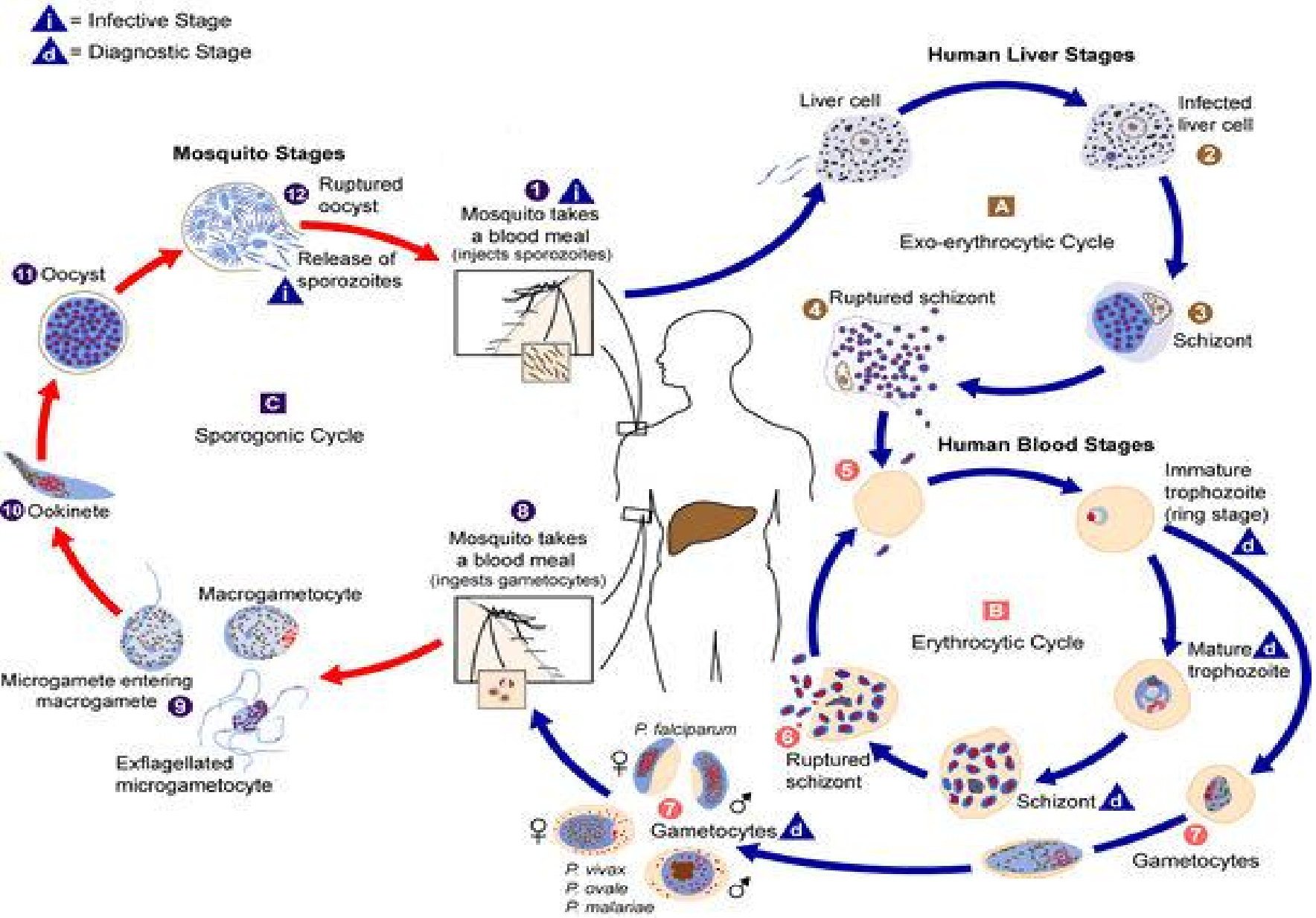
2) *Plasmodium simium i*s anthropozoonotic form of *P. vivax.*

3) *Plasmodium brasilianum* is anthropozoonotic form of *P.malariae.*

***Rodent's* species**

1. *Plasmodium vinckei*: Rat.

Life cycle of *Plasmodium vivax*



1. The *P. vivax* life cycle involves two hosts. During a blood meal, a malaria-infected female Anopheles mosquito inoculates sporozoites into the human host.
2. Sporozoites infect liver cells and either enter a dormant hypnozoite state or mature into schizonts , which rupture and release merozoites.
3. After this initial replication in the liver (exo-erythrocytic schizogony A), the parasites undergo asexual multiplication in the erythrocytes (erythrocytic schizogony B).
4. Merozoites infect red blood cells. The ring stage trophozoites mature into schizonts, which rupture releasing merozoites.
5. Some parasites differentiate into sexual erythrocytic stages (gametocytes).
6. Blood stage parasites are responsible for the clinical manifestations of the disease. The gametocytes, male (microgametocytes) and female (macrogametocytes), are ingested by an Anopheles mosquito during a blood meal.
7. The parasites' multiplication in the mosquito is known as the sporogonic cycle (C). While in the mosquito's stomach, the microgametes penetrate the macrogametes generating zygotes.
8. The zygotes in turn become motile and elongated (ookinetes) which invade the midgut wall of the mosquito where they develop into oocysts.
9. 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.

Harmful effects:

- In birds, chickens are mainly susceptible.
- Sometimes adult birds may suffer a mortality of upto 80% in some areas.
- Progressive emaciation, Anemia, Enlargement of spleen & liver.
- Paralysis may occur due to massive number of Exo-erythrocytic form in the endothelial cells of Brain capillaries.
- Birds with acute infection may be lethargic, anaemic with pale combs, diarrhoeic.
- Show partial or total paralysis.
- Even sudden death
- Fever does not appear to be a significant part of the syndrome in avian hosts. (But in Human - Intermittent high rise of temperature. The release of merozoites from the Schizonts occurs synchronously in host & this is associated with a **paroxysm** (fever).

Control:

1. Eradication of mosquitoes (at least for temporary resident) e.g –

- Destroy of breeding sites.
- Repeated application of insecticides – synthetic pyrethroides.
- Mineral oils to breeding sites.

2. Regular use of prophylactic anti-malarial drugs.

3. Proper treatment of infected animal or birds.

4. Selection of suitable drug to overcome the drug resistance problem.

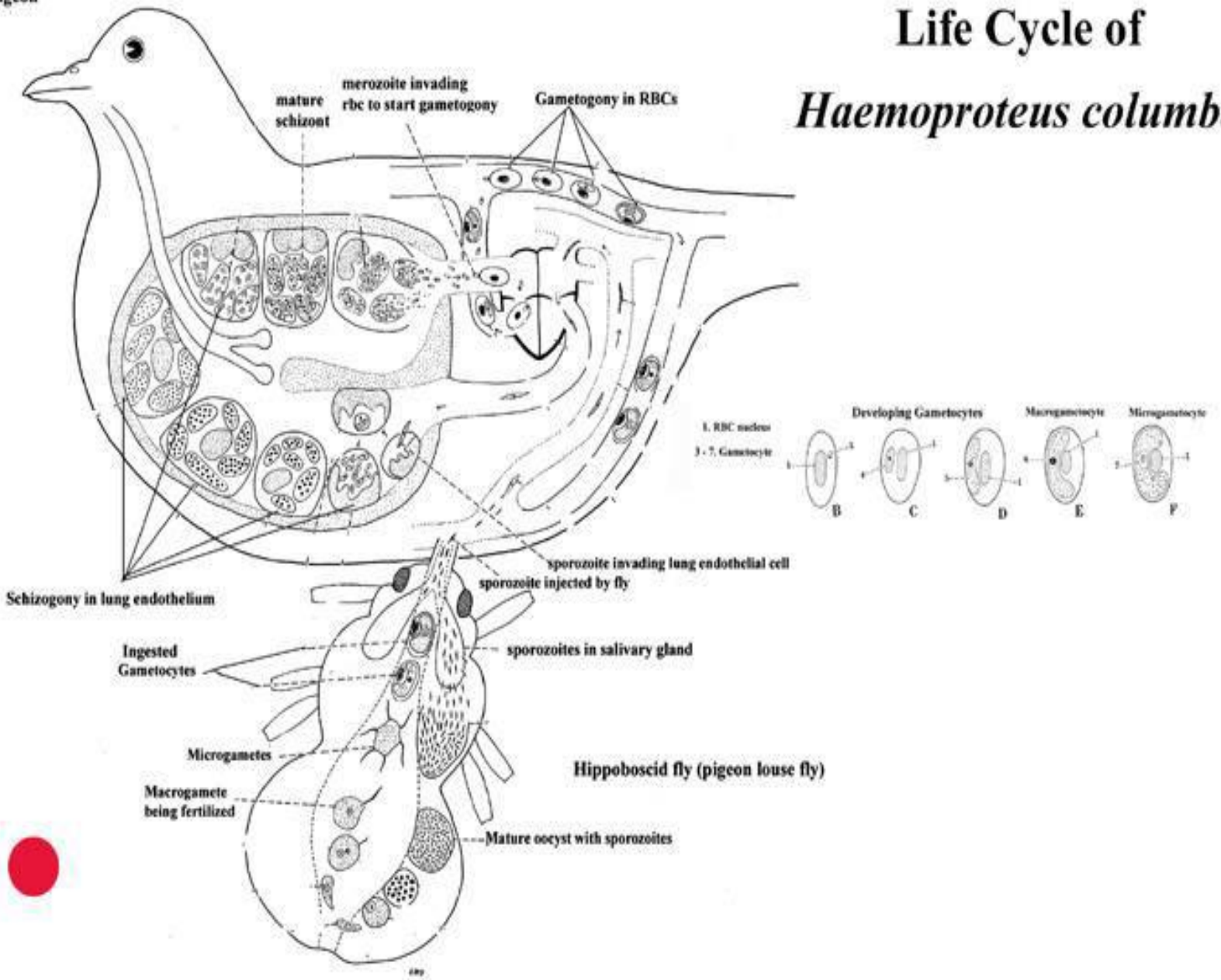
Genus: Haemoproteus

- The protozoa are intracellular parasites that infect the erythrocytes.
- Gametogony in the RBC of final host
- Schizogony in the epithelial cells of blood vessels of inner organs especially in lungs of final host.
- Syngamy in flies (hippoboscid/culicoides)

Species	Final host	Vector(Black fly)	Distribution
H. columbae	Pigeon, Dove	*Pseudolynchia canariensis*	Worldwide
H. meleagridis	Turkey		America
H. nettionis	Duck, Goose	*Culicoides spagnamensis*	Worldwide
H. laphortyx	Quail	*Lynchia hirsuta*	California, other countries
H. sacharovi	Pigeon, Dove	*Culicoides* spp	North America, Europe

Life Cycle of

Haemoproteus columbae



Harmful effects:

- Adult birds usually show no evidence of disease.
- Acute form in pigeon nestlings in which heavy mortality occurs.
- Clinically anorexia and anemia.
- Enlarged gizzard.
- Enlargement of the spleen, liver and kidneys
- Infected birds may suffer from reluctance to move,
- Ruffled appearance,
- The average cumulative mortality for flocks experiencing outbreaks may over 20%.
- Prostration and Death.

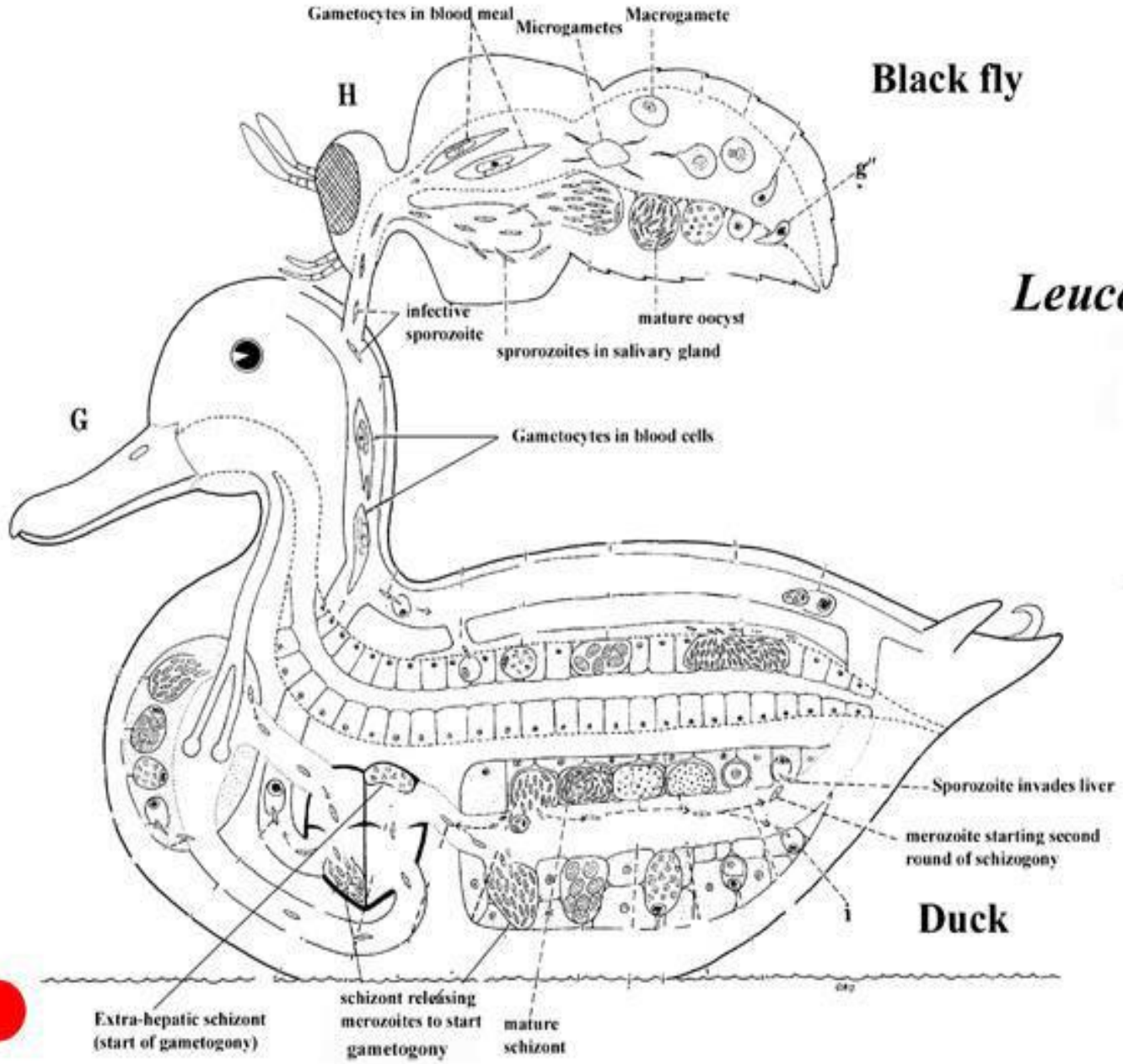
Control: Controlling the vector

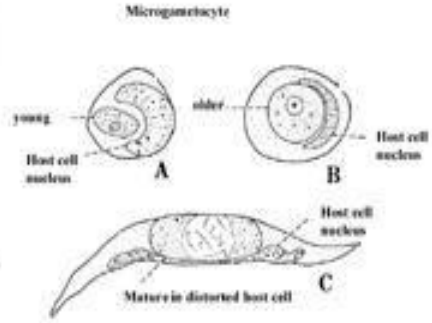
Genus: Leucocytozoon

- Schizogony in endothelial & parenchymatous cells of the liver, heart, kidney and other organs of avian hosts.
- Gametogony in circulating erythrocytes; infected cell become grossly distorted and assume a spindle shape.
- *Simulium* spp.are vectors.

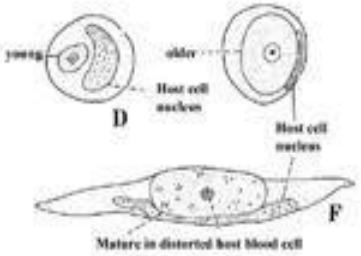
Species	**F/H**	**Vector(Black fly)**
***L. simondi* (common)**	Domestic duck & goose	*Simulium venustum* *S. croxtoni* *S. euryadminiculum*
L. smithi	Turkey (death upto 90%)	*S. occidentale* *S. nigroparvum* *S. slossonae*
L. caulleryi	Domestic chicken	*Culicoides arakawa*

Life cycle of *Leucocytozoon simondi*





Macrogametocyte



Harmful effects:

- Markedly pathogenic for young duck & geese.
- Ducklings are listless, Anorexia
- Dyspnea (due to large no. of megaloschizonts in lung capillaries)
- Nervous disorder prior to death, which occurs within a day or so.
- Slowly develop emaciation, listlessness, Seldom death

Order: Rickettsiales

Genus:

Anaplasma, Haemobartonella, Ehrlichia, Eperythrozoon, Aegyptianella

- These organisms were initially thought to be protozoa but are now known to be Rickettsia. They are confused with blood sporozoa.

Genus: *Anaplasma*

Host: Cattle, wild Ruminants and perhaps sheep (Reservoir host)

I/H: 20 tick species, e.g. *Boophilus spp*

Site: In RBCs of hosts.

Species:

1. ***Anaplasma marginale*** (causes South African Gall Sickness).
2. ***Anaplasma centrale***

Identification: In Giemsa stained blood films-

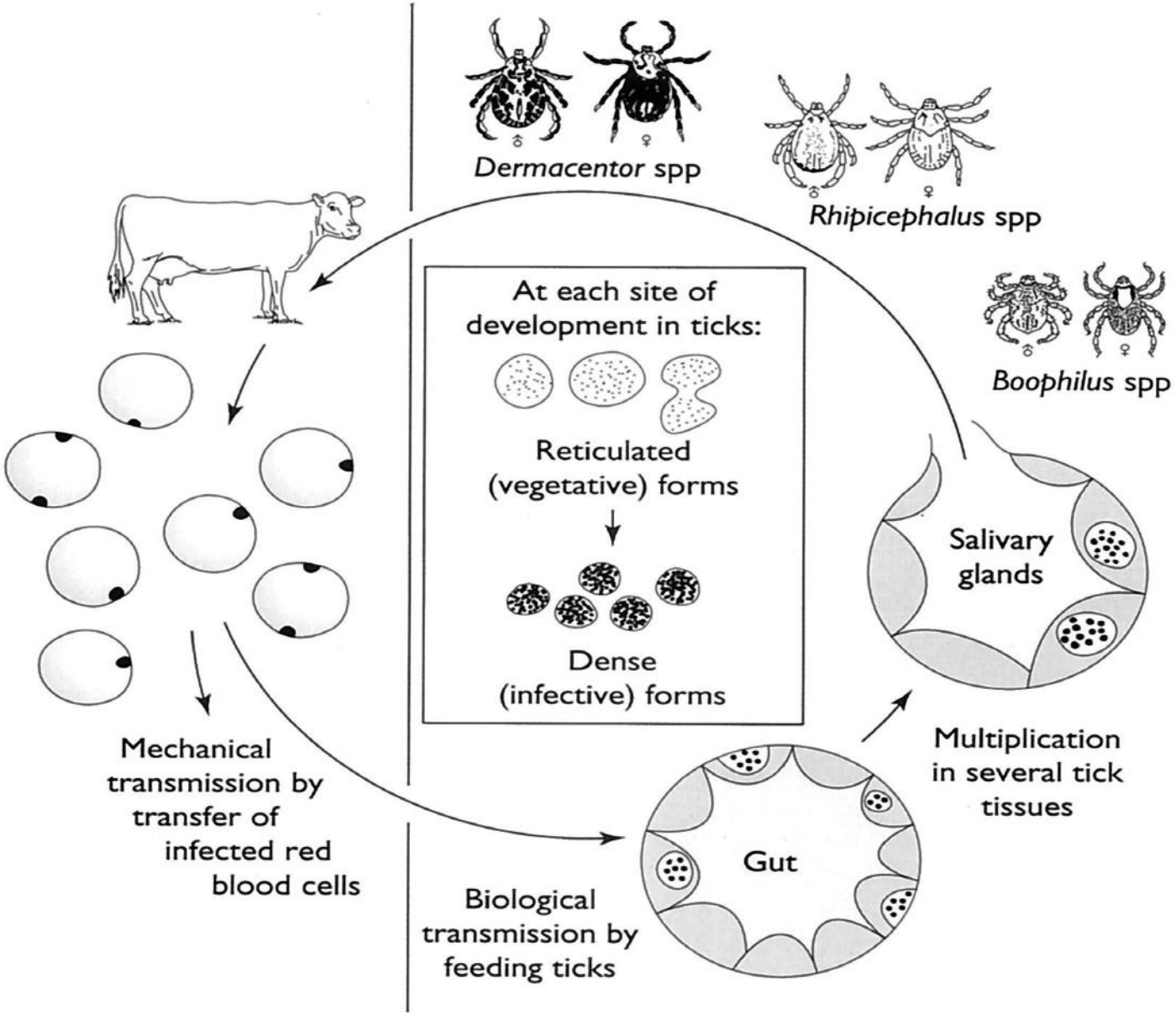
- ***Anaplasma marginale*:** Small, round, dark red "Inclusion bodies" within the RBCs, lies at the outer margin of the cell.
- ***Anaplasma centrale*:** Similar except that the organisms are commonly found in the centre of the RBC. (*Anaplasma centrale* - Mildly pathogenic)

Epidemiology:

- Widely distributed in tropical and sub-tropical countries including Southern Europe and USA.
- Exotic and crossbred cattle are highly susceptible. Sub clinical infection is a feature of indigenous cattle, buffalo, sheep, deer, camel, goat, and bison may be infected. It has been recorded in 15 days old Jersey calf.
- The infection spreads through ticks mostly by means of transovarian. Besides tick, *Tabanas* spp., *Stomoxys* spp. and mosquitoes have been found to transmit the disease.
- Carrier animals like cattle and other wild ruminants play vital roles in the transmission of the disease.
- Mechanical transmission through dehorning, castration, vaccination, ear marking, surgical instruments, hypodermic needle.
- Cattle, especially adults introduced into endemic areas are particularly susceptible, the mortality rate being up to 80%. In contrast cattle reared in endemic areas are much less susceptible presumably due to previous exposure when young although their acquired immunity usually co-exists with a carrier state.
- Clinical anaplasmosis supervenes when cattle are stressed by other diseases such as Babesiosis

Life cycle:

- **In tick (Vector):** Development is not clear. Organisms are found in different tissues (Transovarian transmission). Besides tick, the organisms can be transmitted mechanically by biting flies or contaminated surgical instruments.
- **In host:** Once in blood, the organisms enter the RBC by invaginating the cell membrane so that a vacuole is formed, there after it divides to form an inclusion body containing up to 8 **"initial bodies"** packed together. The inclusion bodies are most numerous during the acute phase of the infection, but some persist for years afterwards.


Dermacentor spp
Rhipicephalus spp
Boophilus spp
At each site of development in ticks:
Reticulated (vegetative) forms
Dense (infective) forms
Salivary glands
Multiplication in several tick tissues
Gut
Biological transmission by feeding ticks
Mechanical transmission by transfer of infected red blood cells

Harmful effects:

- The erythrocytes are parasitized by Anaplasma organisms. The infected erythrocytes are engulfed by the cells of reticuloendothelial system (Spleen, Bone marrow). This process of erythrophagocytosis cause **extra** vascular hemolysis leading to anemia.
- The spleen is an important organ to limit the establishment of the infection.
- Incubation period: 4 days.
- Up to 70% RBCs may be destroyed.

Control:

- Strict control of insects (Tick and biting fly) population should be made by Acaricidal spray or dips.
- Carrier animals should be isolated or disposed.
- Serological test of the herd should be made regularly and the positive one should be brought under treatment.

The clinical signs are usually very mild in native cattle under one year old. Thereafter, susceptibility increases so that cattle aged 2-3 years developed typical & often fatal anaplasmosis while in cattle over three years the disease is often per acute & frequently fatal.

- **Acute:**
  - High rise of body temperature
  - Loss of condition, Nasal discharge, Lacrimation, Inappetance
  - Coughing, dry rales, moist rales
  - Rumen atony, Dehydration, dyspnea
  - Rough body coat, Anemia, Pale / icteric mucous membrane
  - Muscles tremor, Enlargement of superficial lymphnodes
  - Cardiac dilatation, Grinding of teeth, Loud heart sound, Loss of vision
  - Abortion may be occurred
- **Per acute:**
  - Seen in pure & cross bred cattle
  - Excessive salivation, Rapid respiration & death
  - Nervous manifestation may be present.
- **Chronic:**
  - Inappetance, Variable temperature, Anemia
  - Loss of condition.

Genus: *Ehrlichia/ Cytoecetes*

Site: In blood leucocytes (as intracytoplasmic inclusions). Considered as leucocytophilic bacteria and they multiply within the cytoplasmic vacuoles of circulating monocytes and tissue macrophages.

Species	Host (F/H)	Vector (Tick)	Disease
Ehrlichia phagocytophila	Sheep, Cattle	*Ixodes ricinus*	Tick borne fever (Bovine Ehrlichiosis Nofel)
E. canis (Zoonotic significant*)*	Dog and other canine	*Rhipicephalus sanguineus*	Canine ehrlichiosis, Nairobi bleeding disease, Lahore canine fever, Tropical pancytopenia
E. bovis	Cattle	*Amblyomma, Rhipicephalus*	Bovine Ehrlichiosis Nofel
E. ovina	Sheep	*Rhipicephalus evertsi*	
E. risticii	Horse		Equine Ehrlichiosis, Equine Ehrlichial colitis, Potomac horse fever.

Epidemiology:

- Distributed in North & South Africa, N. America, Middle East, and Asia.
- The Ehrlichiosis is not a contagious disease and the disease is transmitted through the bites of ticks.
- In endemic areas the prevalence in young hill lambs is virtually 100%.
- Incubation period 7 days.

Harmful effects:

- On entry organisms invade the monocytes, macrophages and epithelial cells. Monocytes multiply in number and the entire cytoplasm is filled with them. As a result of destruction of leucocytes & thrombcytes, Leucopenia & thrombocytopenia takes place. There is bone marrow depression and anemia. In horse, the organism may invade the intestinal epithelial cells causing colitis and typhilitis.

Control:

- Tick control: e.g. in dog, tick collar may be applied. All out measures should be taken to
- remove the tick from the animal body.
- Animal from endemic area should not be introduced in a new herd.
- An inactivated whole cell adjuvant vaccine.

Genus: *Eperythrozoon*

- Minute rings or coccoid shaped granular bodies occurs relatively loosely on the surface of RBC and free in the plasma.
- 5-3 µm in diameter, Stain reddish- purple.
- Prokaryotic.

Transmission: By lice, flea, tick bites.

Species	Host
Eperythrozoon ovis	Sheep, Goat
Eperythrozoon suis	Pig
Eperythrozoon parvum	Pig
Eperythrozoon wenyoni	Cattle
Eperythrozoon felis	Cat

Genus: *Haemobartonella*

- **Morphology:**
- Like eperythrozoon but rarely occur as ring forms and are closely associated with cell surface.
- Prokaryotic.

Species	Host
Haemobartonella canis	Dog Transmitted by- *Rhipicephallus sanguineus* (Tick).
Haemobartonella felis	Cat
Haemobartonella muris	Rat, Mouse, Hamster

Genus: *Aegyptianella*

- **Morphology:**
  - Round to oval trophozoites or initial bodies containing chromatin granules occur in RBC.
  - Spherical bodies may occur.
- **Transmission:** By fowl tick *Argas persicus*.

1. Aegyptianella pullorum

- *Aegyptianella pullorum* causes a disease in domestic chickens, goose, duck and turkey in South
- East Asia, India, South and North Africa, Sudan, South Eastern Europe and Soviet Union which is somewhat similar to anaplasmosis in cattle.

2. Aegyptianella moshkovshii

- *Aegyptianella* moshkovshii causes disease in chicken, turkey and other birds in Indian subcontinent, South East Asia, Egypt.