TRENDS IN VETERINARY PARASITOLOGY

A TWO-DAYS COURSE
DEPARTMENT OF VETERINARY PATHOLOGY, MICROBIOLOGY & PARASITOLOGY
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PROTOZOA

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By Prof. Kanyari

PROTOZOA:

INTRODUCTION

They are **unicellular** organisms with organelles that carry out various activities such as locomotion, metabolism etc.
They are comparable to unicellular plants but lack chlorophyll which is contained in chromatophores.
They have a well-defined nucleus but don't have a rigid cell wall.
They have considerable variations in size and shape.
Certain protozoan forms share characteristics common to both plants and animal forms (hence the term **protista** – not generally adopted).
Up to 45,000 species of protozoa have been described, majority are free-living, occupying both land and water.
Parasitic protozoa are usually small in number but diseases they cause have a global impact as they result in:

- Deaths and deformity

- Sapping energy and initiative from mankind
- Cause death/decay in the moral fibre in many parts of the world.

STRUCTURE

- **NUCLEUS**

Protozoa are eukaryotic i.e. the nucleus is enclosed in a membrane cf. Bacteria are prokaryotic i.e. the nucleus is spread/dispersed in the cytoplasm.
Usually one nucleus is present but in some forms more than one nucleus may be present in some or all stages of development.
Two types of nucleus are recognized i.e.
Vesicular and Compact type.

➢Vesicular Nucleus

Consists of a nuclear membrane which encloses the nucleoplasm with an intranuclear body called

- Endosome (karyosome) – devoid of DNA

- Nucleolus – has DNA – it is found in the Sub-phyla Mastigophora and Sarcodina of the Phylum Sarcomastigophora.

➢**Compact Nucleus**

It contains a large amount of chromatin and a small amount of nucleoplasm.
It is found in Ciliates as Macronucleus.

Divides mitotically and regulates the cytoplasmic function of the cell.

- **Cytoplasm**

It is the extra-nuclear part of the organism

Divisible into:

- Ecto-plasm – outer homogenous and hyaline
- Endo-cytoplasm – inner, contains granules vacuoles and sometimes pigments

Locomotion

It is achieved by either of the following

- Gliding

or by use of:

- Pseudopodia
- Flagella or
- Cilia

Gliding: Achieved without the aid of cilia or flagella
Common in *Toxoplasma* spp, *Sarcocystis* spp

Pseudopodia: Used by amoeba-like organisms
They are temporary locomotory organelles which are formed when required and retracted when not required

Flagella: These are flamentous structures arising from a basal granule (Blepharoplast) in the cytoplasm.
It is composed of an axoneme (a central axial filament) and a contractile cytoplasmic sheath.
Ultrastructurally, the axoneme has two (2) central filaments surrounded by nine (9) peripheral filaments
In some forms, the flagella is attached to the protozoan body by a

membrane (undulating membrane)
e.g.
with Mastigophora – Trypanosomes

Cilia: Short flagella-like structures
Originate from a basal granule embedded in the pellicle or ectoplasm
Ultrastructurally, they resemble flagella, occur in large numbers and are arranged in rows over the protozoan body.
They can be used for :
Locomotion in Ciliates
Ingestion of food
Tactile purposes

Nutritional Organelles:

Protozoa are either:

- Holophytic
- Holozoic
- Saprozoic in their feeding/nutritional habits.

- Holophytic:

These are forms possessing characteristics of plants. Carbohydrates

are synthesized by chlorophyll which is carried in chromatophores
None of these forms are of Medical or Veterinary importance.

- Holozoic:

They utilize performed food materials derived from living animals or plants
The food material is ingested through a cytostome or pseudopodia then passes to a food vacuole for digestion.

Examples: *Entamoeba; Balantidium* spp. (they ingest tissue cells from their hosts)

- Saprozoic:

They absorb nutrients through the cell wall and these are utilized directly by the organism.
Stored materials are visible as glycogen granules or chromatoid material e.g. Coccidian parasites.

Reproduction

It may be asexual or sexual

Forms of Asexual Reproduction

1. Binary Fission:
   Two daughter cells result from a parent cell
   Division is along the longitudinal axis (but it is transverse in ciliates). The nucleus first divides and is then followed by the cytoplasm.

2. Schizogony:
   The nucleus divides several times before the cytoplasm divides.
   The new progeny forms along the plasmalemma of the parasite.

   Note: in some parasites e.g. *Eimeria* (coccidia), the nuclear body divides mitotically giving rise to several bodies

each of which associates with a portion of the cytoplasm – usually little or nothing of the parent cell remains. In this case the parent cell is called "Schizont" and the daughter forms are called "merozoites"

3. Budding:
   Two or many daughter cells are formed from the parent cell.
   There is usually an equal fragmentation of the nucleus and cytoplasm and the budded forms separate off and grow to full size.
   - Endopolygeny – An internal budding in which the resultant daughter cells are within the parent cell.
   - Endodyogeny – Simplified endopolyogeny resulting in 2 (two) daughter cell e.g. *Toxoplasma, Sarcosystis* spp.

Sexual Reproduction (Forms)

1. Conjugation:
   Two organisms pair and exchange their nuclear material (from the micronucleus).
   The individuals separate and nuclear reorganization takes place e.g. Ciliates

2. Syngamy:
   Two gametes fuse to form a zygote
   Male gametes form from
   Microgametocyte
   Female gametes form from
   Macrogametocyte by gametogony. The gametes may be of a similar size (isogamy) or differ markedly (anisogamy)

3. Sporogony
   An asexual mode of reproduction usually following syngamy.
   A number of sporozoites are formed within the cyst wall (Sporocyst) e.g. Coccidian oocysts – *Eimeria, Isospora.*

CLASSIFICATION OF PROTOZOA
(After Levine 1980)

Sub-Kingdom: Protozoa

Phylum

1. Sarcomastigophora
2. Apicomplexa
3. Ciliophora

1. **Sarcomastigophora**
   Have: flagella, Psedopodia or both and a single nucleus

Sub-Phylum:

a) Mastigophora "Flagellata"
b) Sarcodina "Amoebae"

a) Sub-Phylum: Mastigophora "Flagellata"
   One or more flagella present in the trophozoites
   Reproduction is by binary fission

Class:
Zoomastigophorea – these are predominantly parasitic

Orders:

- Kinetoplastida
  - Family: Trypanosomatidae
  - Genera: *Trypanosoma, Leishmania*
- Diplomonadida
  - Family: Hexamitidae
  - Genera: *Hexamita; Giardia*
- Trichomonadida
  - Family: Trichomonadiidae
  - Genera: *Pentatrichomonas*
    Trichomonas
    Tritrichomonas
  - Family: Monocercomonadiida
  - Genera *Histomonas*
    Parahistomonas

b) **Sub-phylum: Sarcodina**

Pseudopodia typically present
Flagella when present are restricted to developmental stages
Mostly free-living
The following genera are parasitic in the digestive tract of animals:

- *Endolimax*

- *Acanthamoeba*
- *Entamoeba*

2. **Apicomplexa** (Phylum)
   Apical complex made up of conoid, micronemes and rhoptries are present at some stage.
   Simple nucleus present
   Cilia and flagella absent (except in microgametes)
   Syngamy and cysts often present
   All are parasitic

Important Genera

Family:i). Eimeriidae:
Genus- *Eimera, Isospora*
ii). Cryptosporeidiidae: " *Cryptosporidium*
iii). Sarcocystidae: " *Toxoplasma,* " *Besnoitia, Sarcocystis*
iv) Plasmodiidae " *Haemoproteus, Leukocytozoon, Plasmodium*
v) Babesiidae *Babesia*

vi) Theileriidae *Theileria*

3. **Phylum :Ciliophora**

They have two types of nucleus
Have single cilia or compound ciliary organelles in at least one stage of the life cycle
They divide by transverse binary fission
Sexuality involves conjugation
Usually are free-living
Genus: *Balantidium* spp. – of the digestive tract

Part II: Protozoan Diseases

1.
East Coast Fever [E.C.F]and Other Theileriases

Theileriases are diseases of the haemopoietic system but more precisely of the lymphopoietic system and are not characterized by haemolysis and it's consequencies.
Schizogony occurs in the lymphocytes and the cells of the lymphoreticular system.
The forms that occur in the erythrocytes are gametocytes which reproduce by fission.

Significant species

- *Theileria parva parva*- cause of ECF

- *T. parva lawrenci* - cause of corridor disease
- *T. annulata*
- *T. mutans* > all are parasites of cattle
- *T. ovis*
- *T. hirci* > Parasites of sheep & goats

Account of the Life Cycle

Best known for *T. parva parva*
Transmission is by *Rhipicephalus appendiculatus* (Brown ear tick) found in Central, East and southern Africa.
The earliest stages in the life cycle and the lesions are in the Parotid and smaller Cervical Lymph nodes
The infective stage is the sporozoite in the saliva of the tick

The sporozoites invade the lymphoid cells, undergo schizogony to produce macroschizonts (agamonts) of which there may be more than one in one lymphocyte cells (size 2-12 μm average 8 μm.)
Each macroschizont contains several nucleated particles(hence multinucleate).
The macroschizonts are also referred to as Koch's Blue Bodies [KBB's].
In the case of *T. parva* and *T. annulata*, the parasite divides at the same time as the cell undergoes mitosis and it is distributed to both daughter cells
The infected cells may die releasing the macroschizont
Following macroschizont replication, microschizont (has

small chromatic granules) formation commences in the cytoplasm of lymphocytes, reticular cells and macrophages. The appearance of the microschizont coincides with the appearance of piroplasms in the erythrocytes(rbc's). Piroplasms are shaped variously as:
rod, round, oval, comma or ring-shaped.

The morphological similarities between micromerozoites(of the microschizonts) and piroplasms lead to the conclusion that piroplasms are derived from the microschizonts.
It is probable that piroplasms are the end forms of the infection in the mammalian host (no multiplication in the rbc's)

A sexual cycle of development has been demonstrated in *T.annulata* whereby comma-shaped and ovoid forms have been recognized in the erythrocytes. They are believed to represent the macrogamonts and microgamonts.

Development in the Tick

Following ingestion of the intraerythrocytic stages(piroplasms), erythrocytes are lysed and merozoites released which diffentiate into sexual stages in the tick.
They develop in the lumen of the gut into spindle -shaped microgamonts, these break up into several thread-like microgametes after nuclear division.

Microgametes and macrogametes undergo fertilization to form a zygote in the epithelial cells of the gut (of the tick)
The zygote then develops into a motile ookinete which passes on to the salivary glands and rounds up into a sporont which undergoes sporogony to produce infective particles.
This takes place within 2-4 days of engorgement by an adult or nymph tick.
The infective particles(sporozoites) occur in the acinar cells of the tick salivary glands.
An infected cell is recognized by its hypertrophied nucleus.

Pathogenesis and Clinical Presentation - *T. parva parva*

Clinical Signs

Incubation period - 2 weeks

The disease is marked by

3. High fever (40-41.7° C) followed by
4. Parasitaemia
5. Salivation and rapid heart beat
6. Lacrymation with swelling of the eye lids and ears,
7. Corneal opacity (sometimes)
8. Depression, diarrhoea with blood and mucous
9. Petechiations of the mucous membranes (such areas as under the tongue, vulva lips)
10. Marked emaciation.
11. Progressive and prominent enlargement of superficial lymph nodes(esp. of the neck and pre-scapulars).

This gives a close resemblance to malignant catarrhal fever[MCF].

12. Severe pulmonary oedema often terminates the infection
13. Other infected animals become comatose and prostrate
The disease course is 1 month
Mortality - 95%

During the acute phase of the infection, > 60% of the lymphocytes contain KBB's

An outstanding feature of ECF is leukopenia (cf. AIDS)

The macroschizonts are the pathogenic forms

Post-Mortem Changes

Acute Form:

Enlargement of the spleen and the liver which is also yellowish-brown, friable and degenerate

The lymph nodes are swollen and hyperaemic (but may also shrink

to about the normal size terminally)

Kidneys show haemorrhagic or grayish white infarcts- representing aggregations of lymphocytes

The lungs are congested and oedematous with floath in the trachea.

There may be fluid in the thorax, pericardium and underneath the kidney capsule.

Ulceration of the abomasum, small and large intestine occurs. The ulcers consist of a central necrotic area surrounded by a haemorrhagic zone.

Haemosiderosis is seen in the bone marrow suggesting a certain degree of haemolysis.

Turning Sickness

It is associated with *T. parva* and *T. mutans*

The affected animals make circling movements and there is abduction of the limbs

It is thought to result from cerebral localization of the infection in partially resistant cattle re-exposed to heavy infestations of the infected ticks.

Others consider it as a result of local or generalized breakdown in the immunity.

Parasitised lymphocytes containing KBB's localize by embolism or sequestration in cerebral vessels producing haemorrhagic infarcts.

In these, there are parenchymal and meningeal haemorrhages with lymphocytic cuffing of the vessels.

There is increased cerebral spinal fluid.

Other Theilerias

T. annulata: 'Tropical piroplasmosis'

Causes a disease of cattle resembling ECF in Africa, Mediterranean Coastal area and Middle East
Transmission: Ticks- *Hyalloma* spp.
The signs are generally milder and mortality lower than with ECF

T. mutans:
Causes 'Tzaneen Disease' of cattle in Africa. It is a mild infection with low mortality and KBB's are rare or impossible to find.
It has been recognized in Europe, Asia, Australia and USA. It is usually considered non-pathogenic in these areas.
Transmission: *Rhipicephalus* & *Boophilus* Spp.

T. lawrenci - Causes Corridor Disease of cattle in Zululand (southern Africa).
The disease derives it's name from the fact that it affects cattle being moved between game reserves where buffaloes were found. The buffaloes harbour the parasite as a silent infection. The signs and post-mortem lesions resemble those of East Coast Fever but the degree of parasitization is far much lower.

Theilerias of Small ruminants

T. ovis
Affects sheep and goats in Algeria, Egypt, Yugoslavia the Middle East and the Caucasus
Signs:

- High fever
- Anaemia, Icterus and sometimes haemoglobinuria

Mortality may be as high as 50%

T. hirci:
It has about the same geographic distribution as *T. ovis* but infection is mild and seldom fatal.

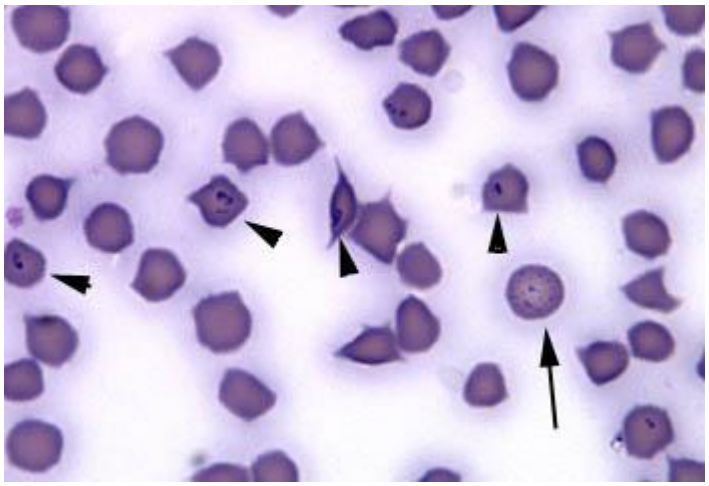


Figure 3: Peripheral blood smear from a white-tailed deer with *Theileria cervi.* Arrowheads denote piroplasms in erythrocytes, while the arrow indicates basophilic stippling.

Treatment

Note: Treatment of animals should only be performed by a licensed veterinarian. Veterinarians should consult the current literature and current pharmacological formularies before initiating any treatment protocol.

- Several drugs (parvaquone, buparvaquone and halofuginone lactate) are available for the treatment of East Coast fever.
- However, treatment must be initiated at the onset of clinical signs to be effective. These drugs are expensive and may be cost-prohibitive.
- Tetracyclines also can be used to treat animals with East Coast fever, but often

the parasites are resistant to these antimicrobials.
- As with the other drugs, treatment must be initiated at the onset of clinical signs.

Babesia-Disease Babesiosis

Order: Piroplasmia
Family: Babesidae
Genus: Babesia

The parasites in this genus appear mostly as single, round, oval, elongate, amoeboid forms arranged in pairs or as exponential multiples of two within the red blood cells (usually at one pole).
They multiply by binary fission or schizogony in the RBC's and characteristically, they are pear-shaped forms lying at an angle with the narrow ends in apposition.

The vectors of these parasites are Ixodid ticks and they cause a disease condition called Babesiosis (as opposed to Babesiasis which refers to all aspects of parasitism).

Synonyms:

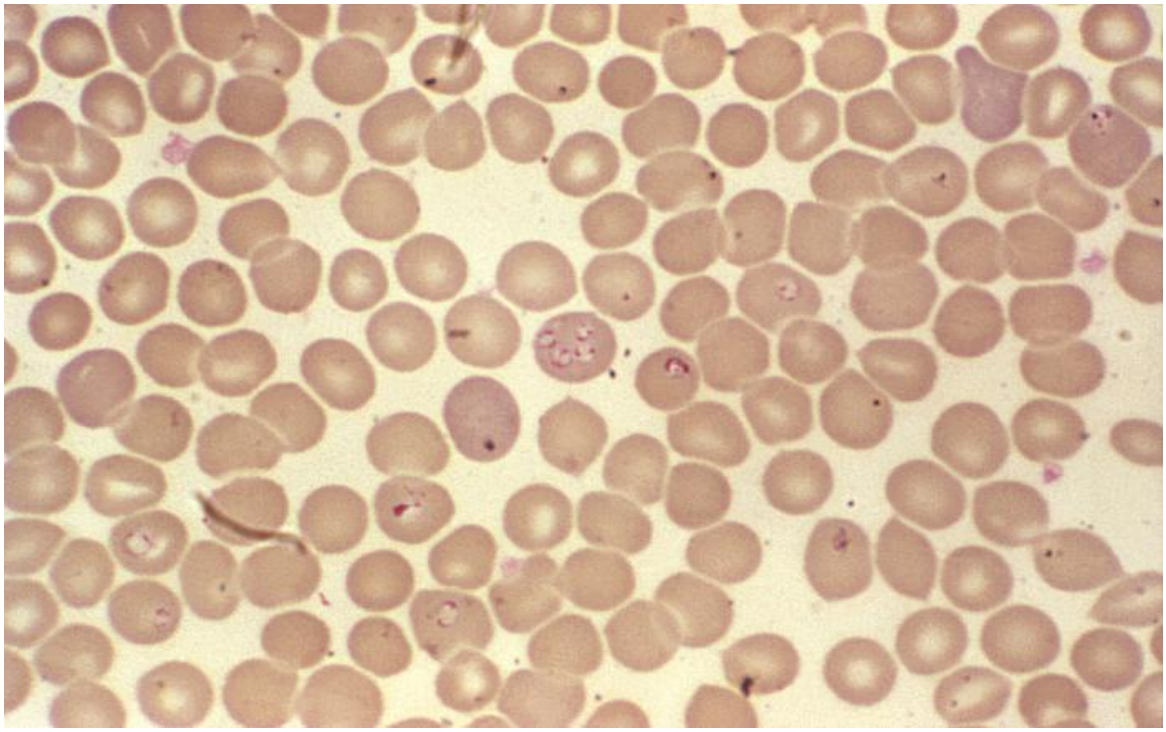
b) Bovine piroplasmosis
c) Tick Fever
d) Red-water/fever
e) Cattle tick fever
f) Southern Fever or Texas fever - in the USA

History:

Babesiosis was once an extremely important disease of cattle in the USA but it has now been eliminated and the only important species is *B.canis* which occurs in dogs in Florida, Virginia, Texas and other southern states.

Babesia is still important in livestock in Central & South America and Europe especially along the Mediterranean Sea, Africa and Australia.

Aetiology:



The babesia organisms affecting cattle are:

- *B. bigemina*
- *B. bovis*
- *B. divergens* - N. Europe

- *B. major*- S. America, N. & W. Africa, Great Britain and the Soviet Union

B. bigemina and *B. major* are large being 2.5 - 5.0 μm while others are small being 1.0 2.5 μm long

B. bovis has been referred to as *B.argentina* and *B.berbera* in older literature

Hosts (of the 4 species of Babesia)

Bovine, Water buffalo and wild Ruminants

Man can be infected and affected

In North America, the disease exists mostly in Eastern Long-Island and the islands off the coast of Massachusetts. It is sometimes called "The Malaria of The North East."

Life Cycle:

Multiplication of Babesia organisms in the vertebrate host occurs in the erythrocytes by a budding process (schizogony) to form two, four or more **trophozoites**.

These are liberated from the RBC's and then invade other RBC's. The process is repeated until a large percentage of RBC's are parasitized.

Occassionally, a RBC shows **multiple infection** with a large number of trophozoites but it is considered that, it is a result of **binary fission** rather than multiple invasion of the cell.

The blood forms are readily transmissible by **mechanical means** to other animals and these initiate another cycle of asexual reproduction.

Young erythrocytes are preferentially invaded especially in acute babesiosis by *B. bigemina* and probably all babesial infections.

Penetration into the RBC commences with the identation of the erythrocyte membrane (by the blunt end of the merozoite). This is then followed by a rapid penetration of the cell by the parasite

Under natural conditions, Babesia spp. Are transmitted by ticks, this was first demonstrated by with *B. bigemina*, the casual agent for Texas fever. This observation was a major land-mark in the history of arthropod-borne diseases as it opened the way to the successful control of many diseases of both man and animals.

Whether or not sexual reproduction takes place in the tick is really not clear.

Essentially, development and transmission of Babesia spp.

In ticks is by either:

- Transovarian or
- Stage to stage

In one host tick such as *Boophilus*, the only mode of transmission is **Transovarial** since

following attachment of the larvae, the rest of the developmental stages of the tick occur on the same host animal.
With two or three-host ticks, the stage to stage transmission becomes of importance; the adults transmitting infections (to bovine hosts) acquired as nymphs and nymphs doing the same with infections acquired as larvae.

➢Trans-ovarian Transmission

The female tick ingests blood from an infected vertebrate host and acquires parasites most of which are destroyed in the intestine of the tick especially those ingested early in the engorgement.
Some of the organisms reproduce in the intestinal epithelial cells by binary fission or schizogony. These parasite stages initially multiply to form the ' **fission body**' which on maturation ruptures releasing club-

shaped bodies or '**vermicules**' into the lumen of the gut.

They then penetrate to other tissues of the tick such as ovaries, eggs and haemolymph where further binary fission takes place.

Thus typically, infection passes through the egg and transmission to a new host is effected by ticks of the next generation.

The parasites are particularly numerous in the malphigian tubules of the salivary glands. They are inoculated in the blood with the saliva when the tick feeds.

The development of the Babesia to the infective stage for the bovine is dependent on the moulting of the tick from the larval to the nymphal stage in the case of *B. bigemina* and B. *caballi* .

- **Transtadial Transmission:**

[Stage to Stage]

Organisms multiply in the larval phagocytes adjoining the hypodermis in the body cavity (of the larval tick).
By the nymphal stage, the organisms are referred to as pseudocysts within which are club-shaped organisms 9 x 12 μm.
These forms are liberated from the phagocytes to migrate into muscle sheaths and into the cells (of the muscle).
They round up and divide repeatedly to form a large number of small ovoid forms about 1.2 μm long.
Further development continues as the adult tick takes a blood meal.
Then the parasites migrate to the salivary glands and enter the cells of the acini. Here they undergo repeated binary fission to form a large number of small ovoid infective stages.
[This process has been demonstrated with *B.canis* in *Rhipicephalus sanguineus*]

Pathogenesis

B. bigemina is highly pathogenic for adult animals but less so for calves. However, in Australia *B.bovis* is the more pathogenic species.

The Incubation period is 8-15 days or less. After the introduction of the parasite (sporozoite) into the blood stream by tick bite, the parasites invade the circulating RBC's.

They become rounded or oval, they grow and divide by 'binary fission' into more characteristic pear-shaped forms.

The parasitised RBC's are destroyed **intravascularly** releasing the contents of the cell and the parasites into plasma.

The pear-shaped forms then invade other cells and repeat the process of multiplication and cell destruction.

Destruction of erythrocytes (including non-infected ones directly by phagocytosis) and

release of haemoglobin into blood stream leads to symptoms of:

- >fever
- >haemoglobinaemia
- >haemoglobinuria
- >anaemia and
- >icterus.

In typical acute cases in adult cattle, the RBCs may drop to 1 or 2 million/cc of blood from the normal 7 million/cc blood

In ***B. bovis*** infection, activation of kinin forming enzyme " Kallikrein" (a pharmacologically active substance) is more important than haemolysis.

The substance (Kallikrein) causes **increased vascular permeability** and **vasodilation** leading to **circulatory stasis** and **shock**.

Also the substance can trigger **intravascular coagulation** which can lead to blockage of capillaries of the brain and liver.

Clinical Signs

Most infections in calves are unnoticed but they (calves) may develop sub-acute diseases. Yearlings are more severely affected with mortalities of up to 25%.

Signs of acute tick fever occur in older cattle and it is characterized by:

- Sudden onset with temperatures of 40-41.7^{o}C[104-107^{0}F]
- Depression> anorexia> atony of the rumen> tachycardia> increased respiration > a drop in milk production.
- Diarrhoea may be present early in the infection but later the animal is constipated.
- Pregnant animals abort.

- RBC destruction leads to the mucous membranes being pale and icteric(yellowish)
- Urine is red to blackish red and death usually occurs 4-10 days following the onset of the signs.

Chronic tick fever may follow acute infections or may develop from the start as an insidious chronic infection. The mortality is low but recovery requires several weeks to months and is often incomplete. Brain involvement can cause convulsions, incoordination and coma eg. in case of *B. bovis* and *B. canis*.

Pathology

The main lesions in acute cases are:

c) Spenomegally - the splenic pulp is soft, dark and red.

d) The liver is enlarged and yellowish brown and

e) The gall bladder is distended with dark bile.

f) The mucous of the abomasums and the intestines is odematous and icteric with patches of haemorrhage.

g) The blood is thin and watery and the plasma may be tinged red (coloured slightly) and the urine in the bladder is usually red.

Diagnosis

Fever associated with haemoglobinuria, anaemia and icterus is suggestive of babesiosis.

Confirmation by microscopic examination of stained thin or thick smears from peripheral blood (a cornerstone in diagnostic procedures in acute cases).

Serological Tests:

CF-test

IFA-test

IHA

Rapid Agglutination Test

Differential Diagnosis

Anaplasmosis
Leptospirosis
East Coast Fever
Bacilliary Haemoglobinuria

Immunity

Some breeds of cattle are more susceptible than others eg. *Bos indicus* is more resistant that *Bos Taurus.*

Cattle recovered from attacks of babesiosis by *B.bigemina* remain infected for life and are immune to re-infection.

This type of immunity due to low grade infection is known as pre-munition and it is antibody mediated.

Acquired immunity can persist for several years in absence of demonstrable organisms and it is species-specific.

The spleen plays an important role in maintaining immunity so that

splenectomy is often followed by severe or fatal relapse in premunized animals.

Antigenic variation:

A well known phenomenon among protozoan parasites. It is their ability to change the specificity of important surface antigen(s) in response to unfavourable environmental stimuli such as exposure to specific antibody.

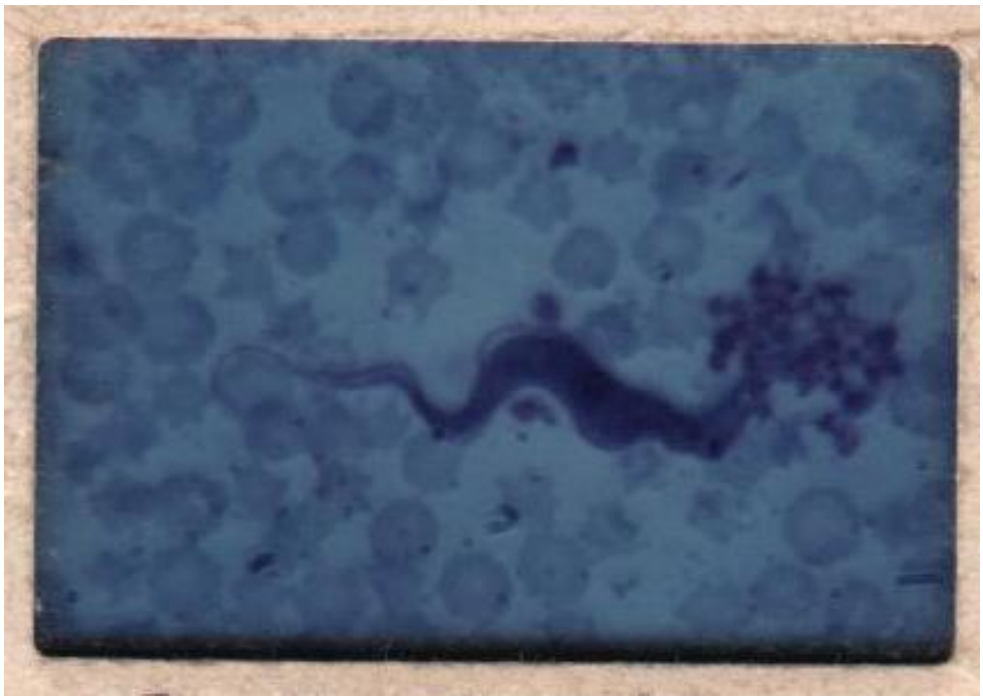
Among Babesia spp. **Antigenic Variation** (AV) is recognized and has been demonstrated experimentally in *B.rodhaini* in rodents.

B. bovis may have up to 100 different antigenic types.

3a. TRYPANOSOMOSIS

Caused by flagellated, motile protozoan parasites which frequent the blood of many vertebrate and invertebrate hosts and also

localize in tissues, sometimes in non-flagellated forms.



The effect of trypanosomes up on the host is as a result of hypoglycemia while chronic infections are thought to lower resistance to intercurrent disease.

The various diseases caused by trypanosomes are:

a) Nagana- A collective term for African trypanosomiasis of domestic animals

especially caused by *T. brucei*, *T. congolense* and *T. vivax.*

The disease occurs in all domesticated animals in tropical Africa and may be acute or chronic.

Manifestations are by:

irregular fever

anaemia

emaciation

subcutaneous oedema

weakness and

photophobia at times

Death may occur after an acute illness or after a prolonged course during which gradual wasting is a dominant feature.

Post-Mortem Lesions:

Severe emaciation with oedematous changes in all fatty tissues

Lymph nodes are swollen, oedematous and occasionally with haemorrhage in the medulla

The liver is enlarged and congested

The spleen is either normal or atrophic with large prominent malphigian corpuscles.
Kidney- necrosis
Lungs- emphysema
Haemorrhages are common especially at the sub-endocardial and epicardial locations
Pericardial fluid may be excessive
Congestion and haemorrhage may be a prominent feature in the gastro-intestinal tract.

3b) Dourine

A disease of equidae with a cosmopolitan distribution
Caused by *T. equiperdum.*
Transmitted by coitus and not biting flies
The disease is manifested by:
Odematous lesions in the genital tract and ventral body wall

Persistent ulcerous plaques in the genitalia and skin

Occasionally by anaemia, incoordination and paralysis.

The causative agent is demonstrable in the lesions particularly those of the genitalia.

3c) Surra

Occurs in Asia particularly in equidae but dogs and ruminants may be infected.

Wild ruminants and elephants can act as reservoirs

Caused by *T. evansi* which is transmitted mechanically by *Tabanus* and *Stomoxysis*.

The disease is recognised frequently in a sever form with:

intermittent fever associated with trypanosomes in the blood

Gradual emaciation in spite of good appetite

Serous nasal discharge

Patchy alopecia

Petechie and ecchymoses of visible mucosae

Weakness and incoordination

Oedema of the limbs and lower abdomen and thorax

Icterous and progressive anaemia and fatal termination.

3d) Mal de Caderas

Caused by *T. equinum*

Occurs in tropical and subtropical South America affecting equidae, ruminants and wild water hogs

The disease is an acute infection quite similar to Surra.

3e) Murrina de Caderas

Occurs in Central America

Caused by *T. hippicum* among horses and mules

It is esentially similar to Surra although it may have a more prolonged course manifested by:
weakness, emaciation, anaemia, ecchymoses splenomegally and paralysis.

3f) Chagas' Disease= South American Trypanosomiasis.

A human disease spread by the kissing bug= *Triatoma*
Cause: *T. cruzi*

The following hosts can harbour the infection:

dogs, cats,monkeys and small wild animals.

T. cruzi assumes a typical trypanosomal form in the blood and tissues of mammals and in the intestine and rectum of insects. It also occurs in leishmanial forms in both mammalian cells and tissue cultures and hence it is closely related to *Leishmania*. It's clinical manifestations are related to the damage in the brain and myocardium.

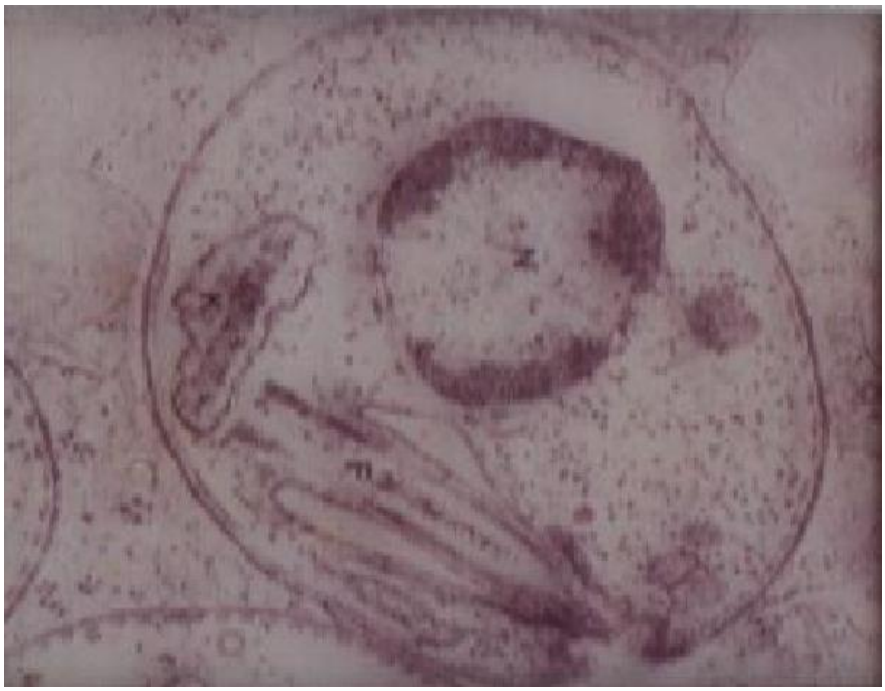
4. LEISHMANIASIS

An infection of man and animals caused by a protozoan organism namely:
L. donovani- visceral leishmaniasis= kala azar

L. tropica – cutaneous leishmaniasis- Of Southern Europe, North Africa and the Mediterranean countries.

L. braziliensis – mucocutaneous or American leishmaniasis=espundia Occurs in Mexico, Central and South America.

Animals infrequently affected in nature.



Visceral leishmaniasis

Occurs naturally in man, dogs,cats squirrels, cattle horses and sheep.
The disease is common in countries bordering the Mediterranean, large areas of Africa, India and China.

It occurs in animals as chronic debilatating disease with periods of fever and gradual weight loss with anaemia appearing in the terminal stages.
A history of persistent cutaneous ulcers may sometimes be obtained.
Splenomegally, hepatomegally and lymphadenopathy may be detected in dogs and ascites occurs in rare cases.

The skin may later get involved with a specific type of dermatitis.

Lesions:

The organisms stimulate production of large numbers of macrophages with cytoplasm filled with leishmanial forms
The lymph nodes and the spleen are particularly involved by reticulo-endothelial hyperplasia although the liver, bone marrow and kidney and less often, other viscera and skin may be infected.

Post-Mortem:

Gross findings are severe emaciation, enlarged lymph nodes, spleen and liver.
Ulcers can be found in the intestines.
Histologically, leishmania organisms can be found in the cytoplasm of enlarged macrophages.
Lymph nodes show moderate fibrosis and reticulo-endothelial hyperplasia.

Macrophages infiltrate large areas of the spleen and the liver.
The bone marrow may be hyperplastic or replaced largely with macrophages filled with leishmania.

Diagnosis:

Due to the presence of other diseases which cause reticulo-endothelial proliferation, the differential diagnosis includes:

- Histoplasmosis
- Toxoplasmosis
- Salmon Disease
- Blastomycosis
- Epizootic Lymphangitis

Confirmation:

Demonstration of causative organisms in tissue sections, smears or cultures.

5. BESNOITIOSIS= GLOBIDIOSIS

A chronic debilitating disease with cutaneous and systematic manifestations in cattle and horses.

May occasionally be fatal.

Cause= *Besnoitia besnoiti* (*Globidium besnoiti*)

B. jellisoni of deer mite, Idaho, USA.

The organisms in cattle and horses are encountered most frequently in cyst form which are unmistakable in appearance.

The cysts are smoothly spherical in well fixed tissues with a diameter of 0.1-0.5 mm.

Are surrounded a dense uniformly eosinophilic wall which is homogenous or concentrically laminated.

Inside the wall are one or more giant nuclei which become compressed

against the periphery by the enlarging mass of spores within.
The giant nuclei are surrounded by a narrow band of cytoplasm which encircles the inner wall and send one or more narrow dividing septa across the cyst.
The inner contents are made up of many tiny crescentic spores which are very similar to *Toxoplasma* in size and morphology.

Lesions:
The lesions are closely related to the organisms which may not only localize in the skin but may spread to all parts of the body.
Grossly, the cutaneous lesions are seen as thickened, rugose, partially hairless areas of skin particularly on legs, thighs and scrotum.

Microscopically, large spherical cysts which may also occur in deeper areas including small blood vessel walls. Invasion of the scrotum, epididymis and testis as well as the upper gastro-intestinal tract is common and may be accompanied by a severe granulomatous tissue reaction particularly when numerous spores are released into the tissue.

In contrast, the mature cysts with their wide hyaline walls are usually surrounded by little other inflammatory tissue reaction.

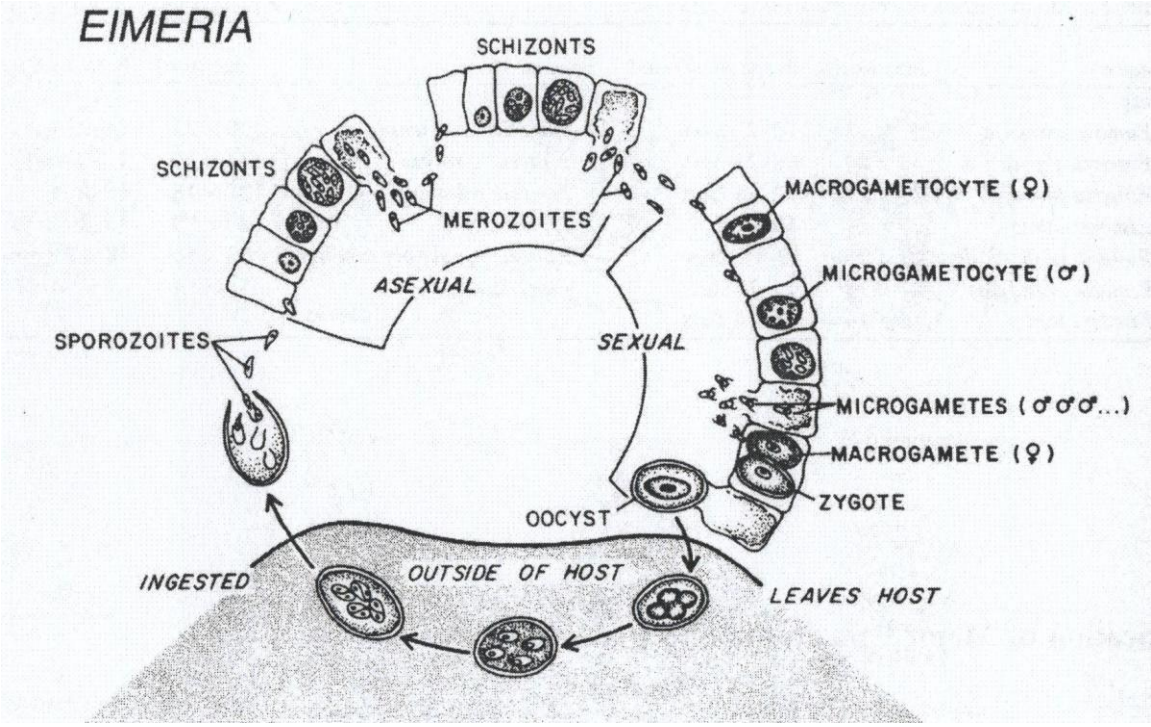
Coccidiosis:

Genus Eimeria:

Mainly parasites of both vertebrate and invertebrate hosts especially birds and mammals. They are intracellular and occupy mainly the gastrointestinal tract where they multiply and cause epithelial cell damage with associated pathogenic effects especially characterised by diarrhoea with blood and mucous.

Some extra-intestinal coccidians are
Eimeria stiedae of the rabbit

A Typical Life Cycle Pattern of *Eimeria* spp



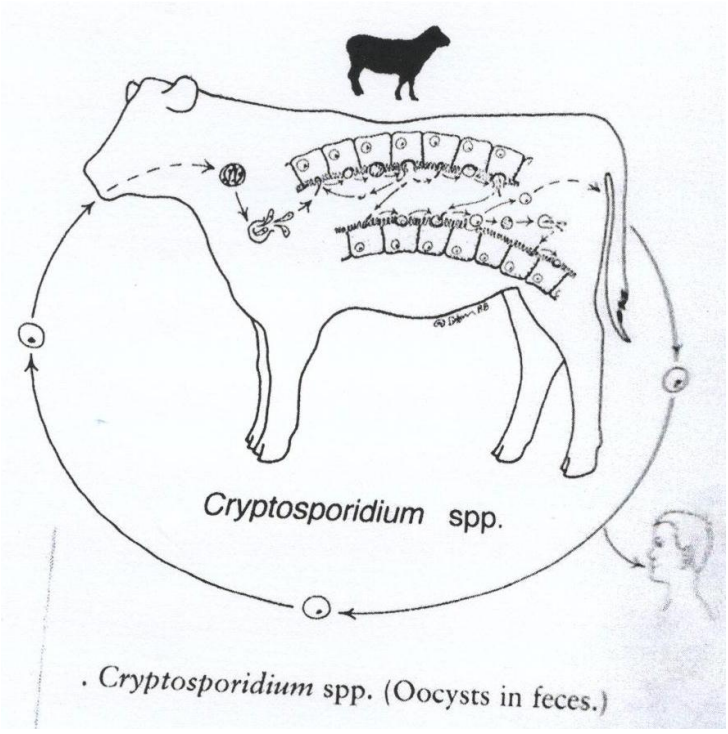
Diagnosis: Faecal floatation/smear and examination for oocysts.

Genus: *Cryptosporidium*:

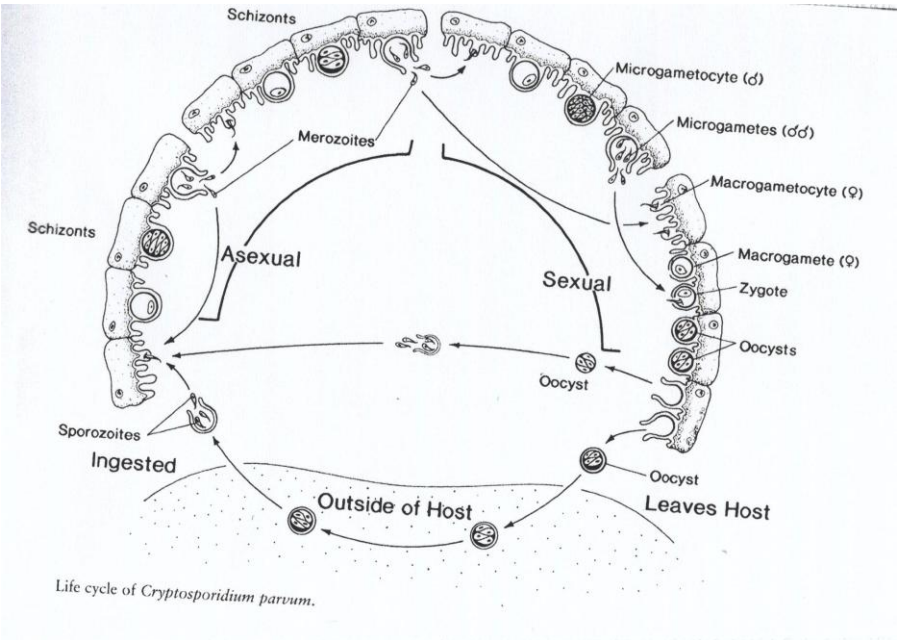
A coccidian parasite normally apathogenic but has assumed importance as an emerging zoonosis in the light of HIV and AIDs scourge among human populations. The organism is an opportunist taking advantage of the host's lowered immune status. The parasite occupies an intracellular but extra-cytoplasmic portion of the cell.

It has a direct life cycle with schizogony and gametogony taking place within the luminal border of the epithelial cells capacity for autoinfection. Studies by Kanyari et. al (2002) have shown that, among immunosuppressed mice, *in utero* infection is possible. This raises the possibility of this too occurring among HIV positive human hosts infected *Cryptosporidium* spp.

Diagrammatic Illustration of the Life Cycle of *Cryptosporidium* spp.



. *Cryptosporidium* spp. (Oocysts in feces.)



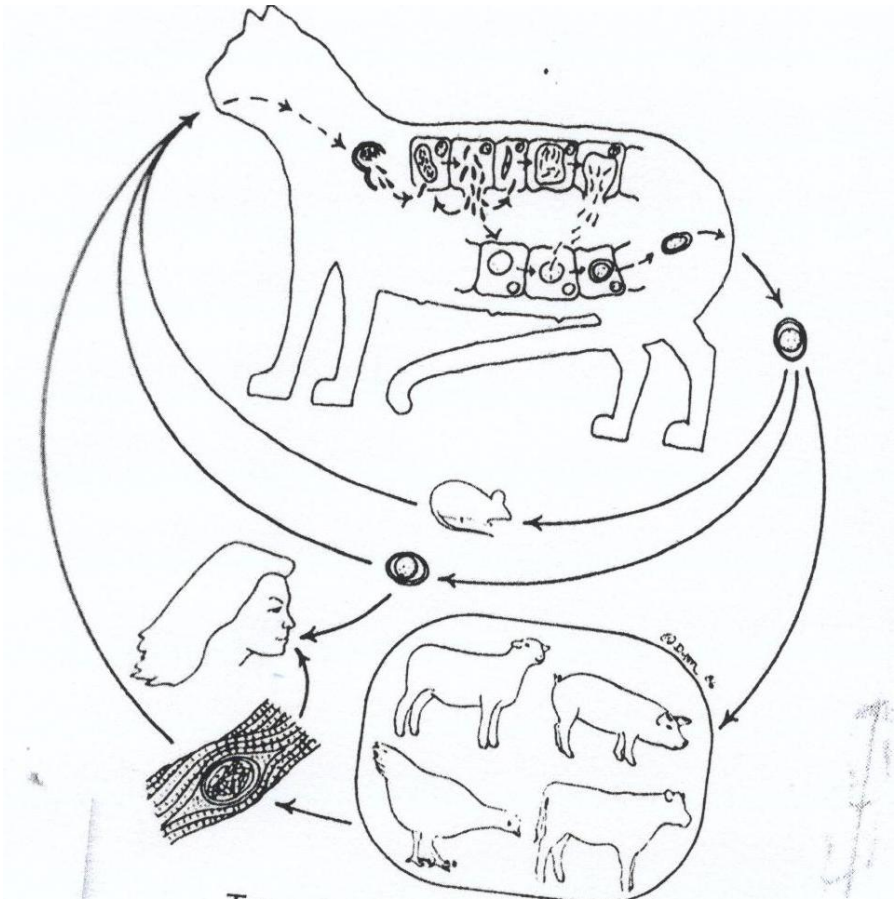
Life cycle of *Cryptosporidium parvum*.

Genus *Toxoplasma* Spp

Has a wide host range namely: cats, dogs, pigs, birds, humans and wild animals. Like majority of Eimeria Spp, it is an intra-cellular cellular intestinal protozoan. It’s importance in humans lies in the parasite’s ability to develop within deeper tissues in the phagosomal vacuoles

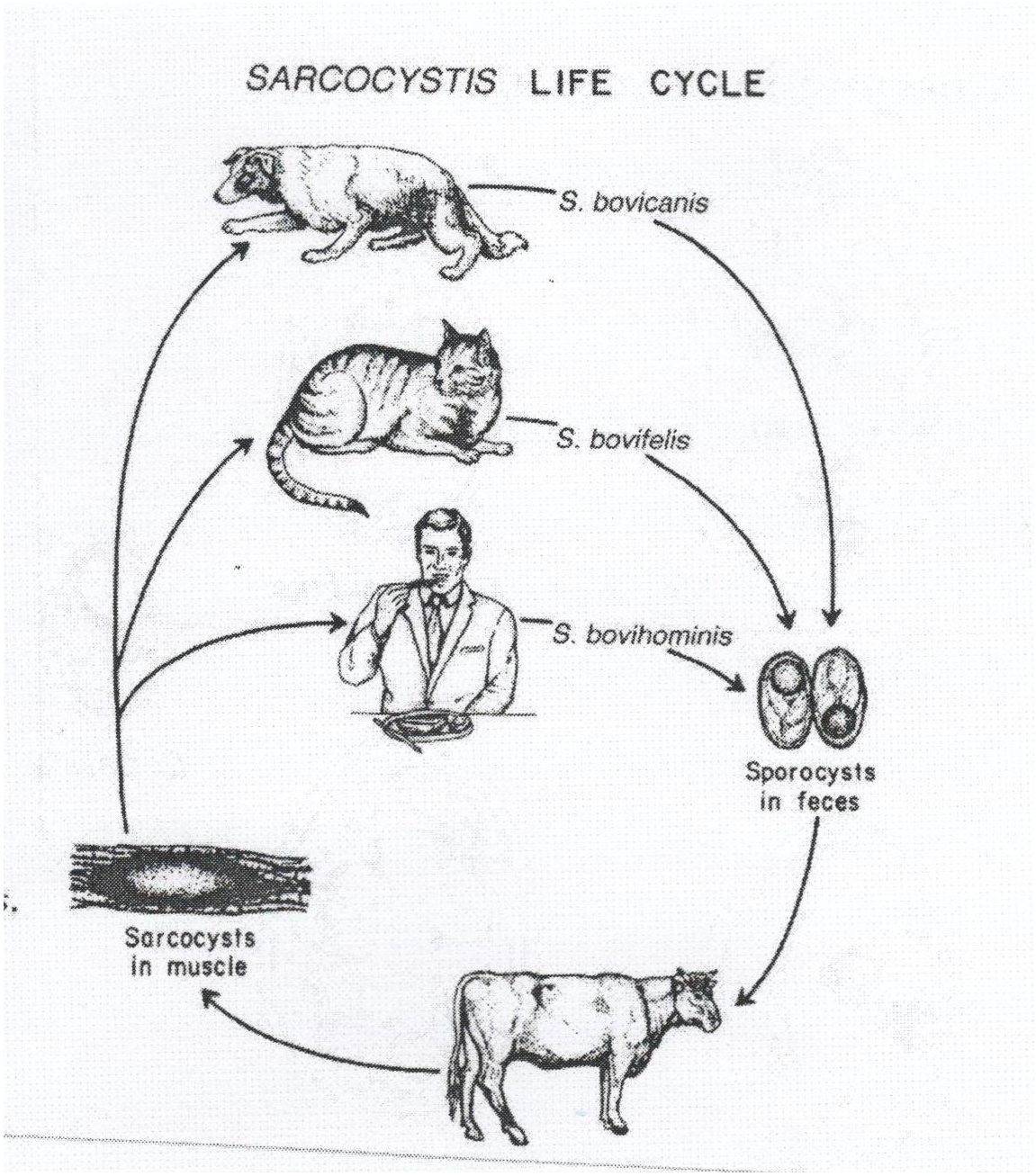
of macrophages thus evading the hosts's immune defences. It is a chronic infection in humans and can result in foetal death and abortion.

Toxoplasma gondii: Life cycle (pseudocyst in the brain of a sheep)



Sarcocystis

Infection of the definitive host (including man) involves eating flesh containing zoitocysts which then release bradyzoites after digestion. Gametes form giving rise to zygotes and then oocysts. These sporulate within the gut to be shed as fully sporulated oocysts in the faeces.
Host range: Cattle, sheep, pigs, horses, buffaloes, camels and in game animals.
The economic losses due to sarcocystis are related to abortion and reduced life weight gain.


SARCOCYSTIS LIFE CYCLE
S. bovicanis
S. bovifelis
S. bovihominis
Sporocysts
in feces
Sarcocysts
in muscle

TRICHOMONAS Spp

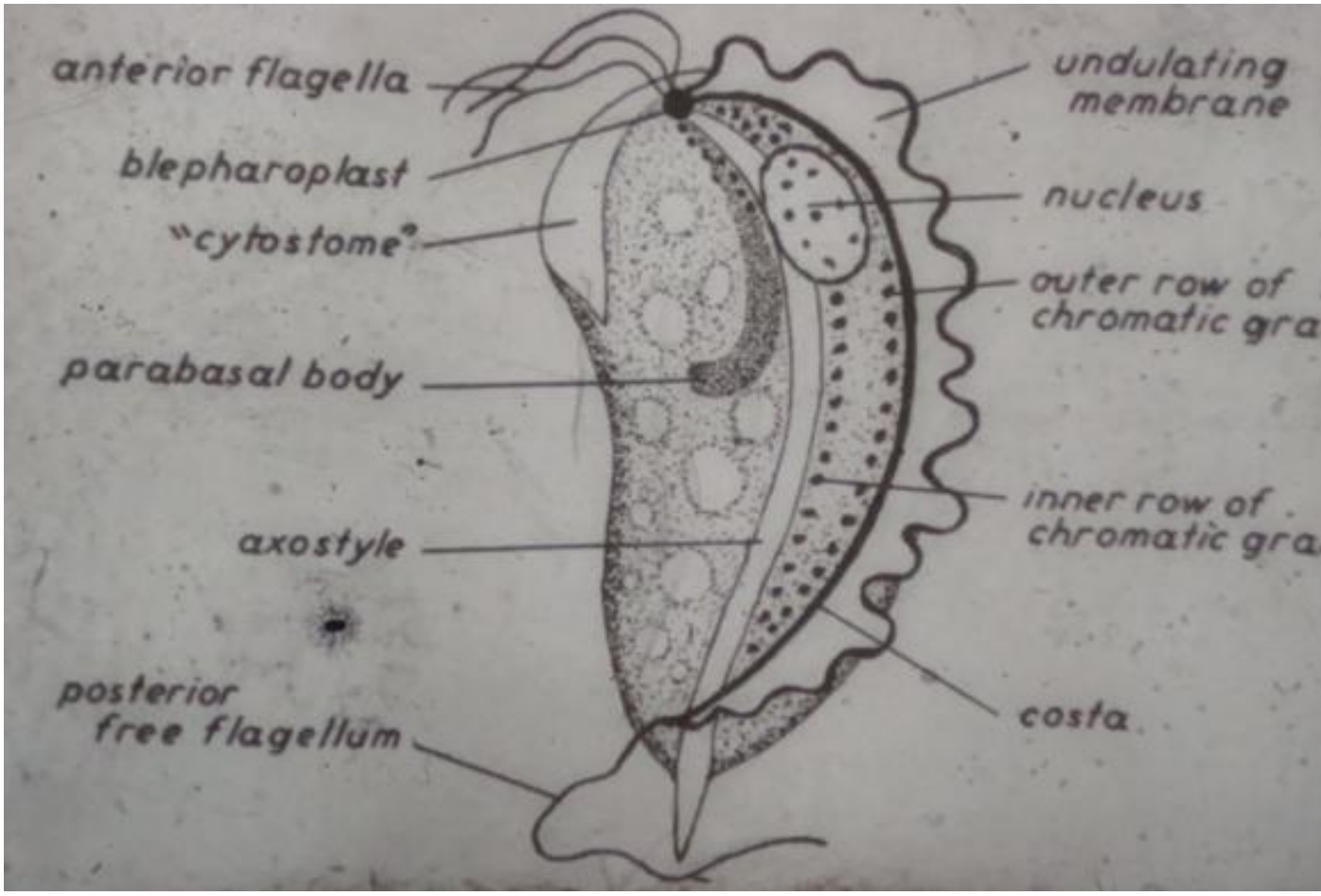
"Trichomonad"- a general term used for a number os species parasitizing the tubular organs of a wide range of hosts including man, cattle, chicken and rats.

T. vaginalis- very common in (man) male and female urogenital tract causing vaginitis and urethritis

T. tenax[T.buccalis]- Human mouth- associated with infections of the gums, caries, pyorrhoea

T. foetus- Cattle. Transmitted venereally from infected bulls to heifers. Attacks vagina and uterus causing abortion. Females are self-curing but males are apparently infected for life.

Trichomonas spp.



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