# Veterinary Protozoology

# VETERINARY PROTOZOOLOGY

by

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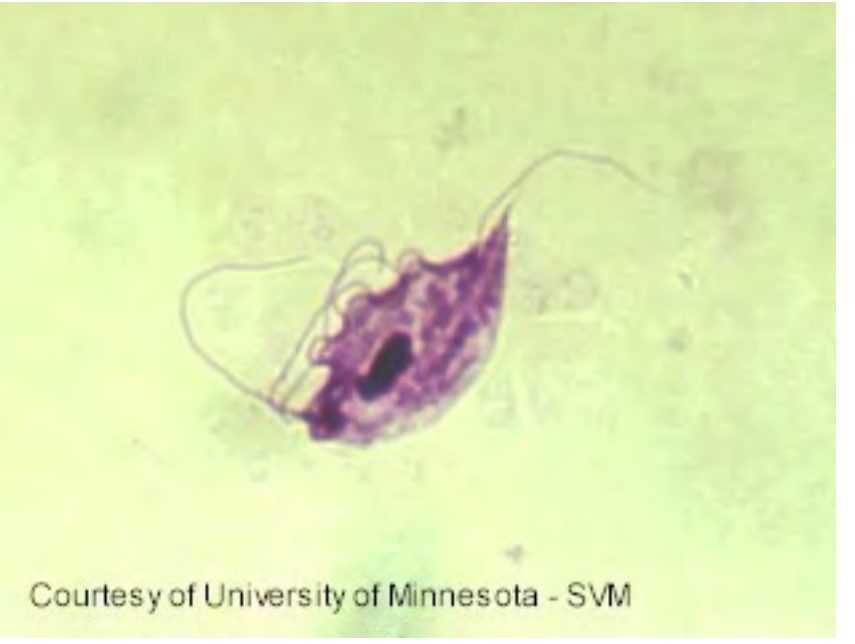


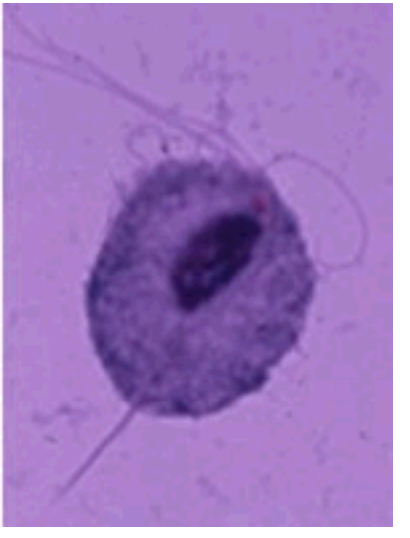

Tritrichomonas foetus

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# PREFACE

At the present time no textbook which deals exclusively with the protozoan diseases of animals of veterinary importance in North America has been published.

Several very useful reference books have been written on the morphology and life histories of protozoan diseases of animals but those which are of importance to the veterinarian have not been readily available.

During the past several years of teaching protozoology and parasitology the writers have felt the need of a handbook for veterinary students, practicing veterinarians and agricultural students

The present book is an attempt to fulfill this need. The volume gives a brief description of the protozoan parasites of domestic animals and the diseases they cause. It is intended as a practical treatise with emphasis on morphology, life histories, pathology, diagnosis, treatment and control of the parasitic protozoa which occur in the domestic animals of North America. It may also be helpful and useful to protozoologists, zoologists, biologists, parasitologists and public health workers

This book is an outgrowth of lecture material of courses on Veterinary Protozoology and Veterinary Parasitology which the writers have given during the past few years to students in the College of Agriculture, University of Wisconsin and students in the School of Veterinary Medicine, Michigan State College.

Since our knowledge of certain protozoan diseases of our domestic animals is quite meager, as shown throughout this text, it is hoped that calling attention to these scientific gaps will stimulate investigators to pursue research further in order to obtain the solution to some of these perplexing, but important problems.

The writers have collaborated and conferred with each other on all phases of this book for the purpose of obtaining adequate unification of the subject matter. Both authors assume responsibility for all of the chapters presented

In a book of this kind errors will appear despite every effort to eliminate them. Consequently, the writers welcome any helpful suggestions or constructive criticisms. Calling errors to our attention will prevent their appearance in future editions.

The order followed for the Protozoa is systematic according to phylogenetic relationships. The arrangement of the hosts has followed more or less the order given by Benbrook (List of Parasites of Domesticated Animals in North America, 1945, Burgess Publishing Co.) and Dikmans (Check List of the Internal and External Animal Parasites of Domestic Animals in North America, 1945, Amer. Jour. Vet. Res. 6: 211-241)

The writers have leaned heavily on the publications of others in order to make the work as complete as possible. Textbooks, reference books and periodicals were consulted freely. Only the more important American references were cited as it is realized that the undergraduate student does not have the time or the inclination to translate foreign languages.

## ACKNOWLEDGEMENTS

The writers wish to thank Dr. Ward Giltner, School of Veterinary Medicine, Michigan State College, East Lansing and Dr C. A. Brandly, Department of Veterinary Science, University of Wisconsin, Madison, for their friendly encouragement in this work at all times. Thanks are also due to Miss Elizabeth F. Skolaski, who typed the manuscript, Mr. Everett Schiller, who drew the plates, and Mrs. Philip A. Hawkins for checking proofs.

## CHAPTER I

## GENERAL INTRODUCTION

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## GENERAL INTRODUCTION

Protozoa are one-celled animals which may occur singly or in colonies. Each protozoan is a unit capable of performing all of the physiological functions of life which in the higher animals are performed by specialized cells.

As a rule, protozoa are regarded as the lowest or first phylum of the animal kingdom. The science of protozoology includes the study of both free-living and parasitic protozoa. Veterinary protozoology concerns those unicellular animals which are either symbionts, commensals or parasites of domestic animals. Parasitic protozoa have adapted themselves to an extreme mode of livlihood at the expense of the host.

These single-celled animals are all grouped in the Phylum Protozoa which is divided into two subphyla, the Plasmodroma and the Ciliophora. The subphylum Plasmodroma contains three classes which include many protozoa of veterinary interest, the Mastigophora or Flagellata, the Sarcodina or Rhizopoda, and the Sporozoa, while the subphylum Ciliophora or Infusoria contains two classes, the Ciliata and Suctoria.

MASTIGOPHORA: Whip-like appendages or flagella serve as organs of locomotion. Reproduction is by longitudinal fission.

SARCODINA. Pseudopodia provide means for locomotion and procurement of food for these organisms which are not protected by a pellicle or cell wall except in the cyst forms. Reproduction is by fission or cyst formation.

SPOROZOA. This entire class is completely parasitic There are no definite structures for locomotion. Reproduction is by schizogony (spore formation)

CILIATA. Short filaments, called cilia provide the organism with locomotion. The cilia are present throughout life Reproduction is mainly by conjugation, asexual reproduction by binary fission occurs.

SUCTORIA: Locomotion is provided by cilia which are present only during the early stages of development. Reproduction is either by conjugation or binary fission.

The following key will help differentiate the various classes of protozoa:

### KEY TO CLASSES OF PROTOZOA

Trophozoite with flagellum ..... Class Mastigophora

Trophozoite with pseudopodium ..... Class Sarcodina

Without organelles of locomotion: producing spores, all parasitic ..... Class Sporozoa

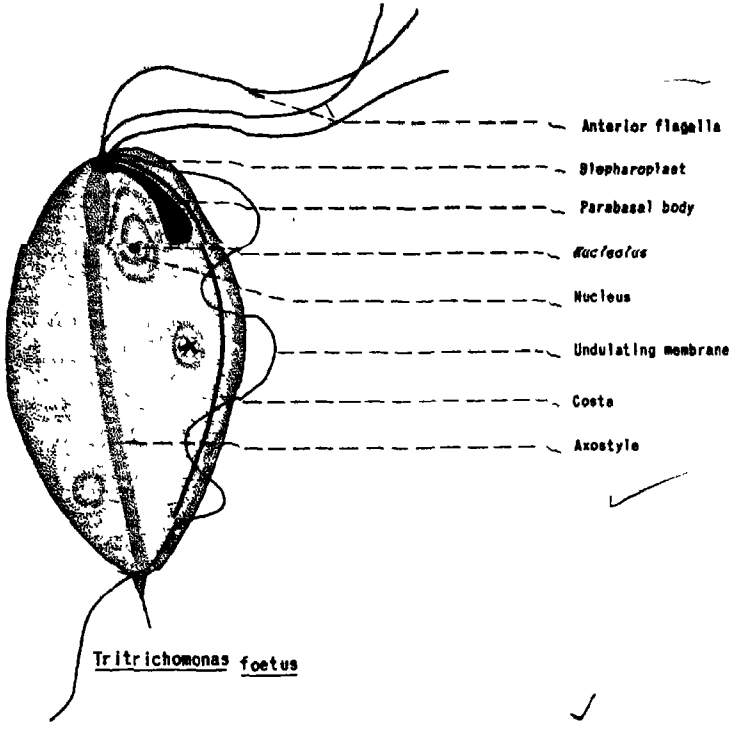
Cilia present throughout trophic life ..... Class Ciliata

Adult with tentacles; cilia only while young ..... Class Suctoria

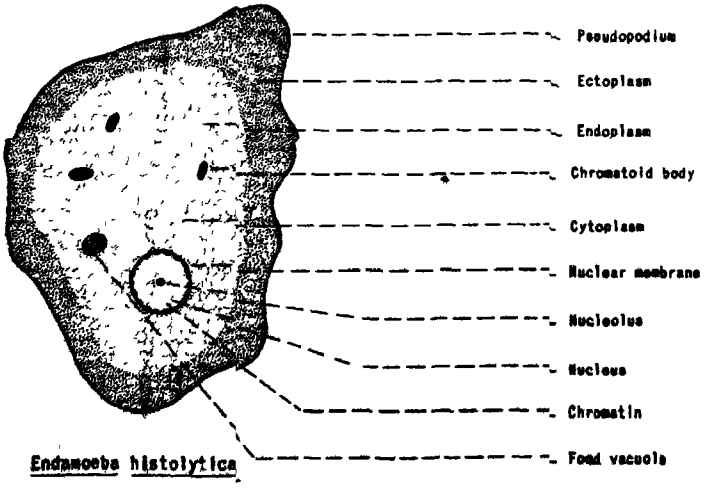
### MORPHOLOGY

A protozoan consists of a single-cell which possesses a nucleus or nuclei which is surrounded by cytoplasm. The cytoplasm appears to be granular and is divided into an outer portion, the ectoplasm, and an inner portion, the endoplasm.

PLATE I




Tritrichomonas foetus




Entamoeba histolytica

The ectoplasm is associated with movement, excretion, respiration, ingestion of food and cyst formation Flagella, pseudopodia, or cilia which are the organelles of locomotion are simply prolongations of the ectoplasm. Food particles may be ingested at any point in the cytoplasm Depending upon the type of protozoan, there may be a peristome which is a definite opening for the collection of food. This may lead to a cavity known as a cytostome or mouth The cytostome may connect with a tube, the cytopharynx where the food material passes directly into the endoplasm. The cytoplasm may contain highly specialized skeletal structures to maintain the shape of certain protozoa.

The endoplasm is vitally concerned with nutrition and reproduction. It contains the nucleus, food vacuoles, contractile vacuoles, chromatoidal bodies and other cellular inclusions. The contractile vacuoles have the main functions of regulating osmotic pressure, digestion and elimination of waste products. The contractile vacuoles usually expand and contract at regular intervals These are not to be confused with food vacuoles which contain various types of ingested material.

The nucleus is important for its role in maintaining and reproducing life. The nuclear membrane separates the nucleus from the cytoplasm. The nucleus is filled with nuclear fluid and chromatin More than one nucleus may be present In the nucleus is the deep staining karyosome.

In certain flagellates other complex structures are known The flagellum usually terminates internally in a basal granule which sometimes is connected by a larger structure called a blepharoplast Occasionally the blepharoplast is further connected by a fibril known as the rhizoplast. Near the cytostome there is frequently found a structure called a parabasal body It may vary from a single line to curved, or spiral-like body

A flexible structure varying from a filament to rod-like form termed an axostyle is found in some of the flagellates Ciliates are equipped with a neuromotor apparatus. Delicate membranes on the periphery of various protozoa are called the undulating membrane. In certain ciliates the cilia are replaced by cirri A cirrus is a number of fused cilia Other cilia may develop into tentacles as found in the Suctoria.

## PHYSIOLOGY

Protozoa may obtain nourishment by ingestion of solid particles or absorption of liquid food through the cell wall The types of nutrition of parasitic protozoa include holozoic (capture of food, ingestion, digestion, assimilation, and elimination) and saprozoic (nourishment by diffusion through the body surface)

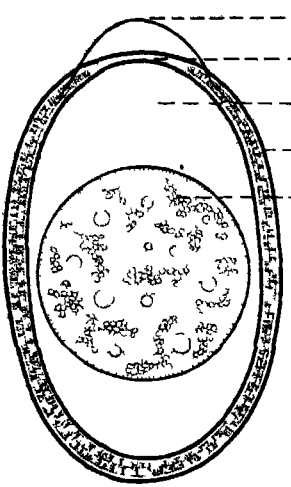
There is apparently no definite structure associated with the respiration of protozoa. Protozoa may take in oxygen and expel carbon dioxide directly, or by the action of enzymes The majority of the parasitic protozoa are anaerobic since free oxygen is rarely found in animal tissues. There is no doubt that some of the parasitic protozoa secrete or liberate proteolytic enzymes, and toxic materials which may account for clinical symptoms

Under certain conditions some protozoa in order to protect themselves to outside environments form cysts The vegetative form (trophozoite) secretes a resistant wall, undergoes nuclear division and stores reserve food. Cysts are associated with the transmission of various protozoan disease through contamination of food and water.

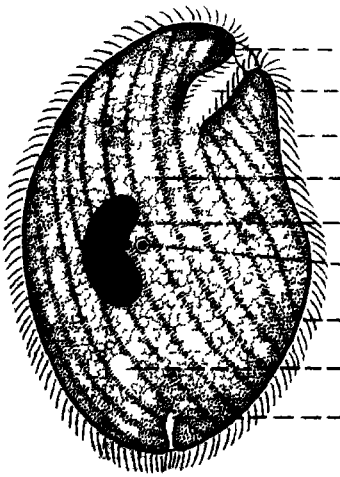
Reproduction in the parasitic protozoa may be asexual or sexual. The most common asexual method is by simple fission. This is a division of the body through the center of the long axis into daughter individuals.

The Sporozoa reproduce by either method. Asexual development is called schizogony, in which the nucleus continues to divide without immediately forming new cells. Sexual reproduction in the Sporozoa is called sporogony.

PLATE 11



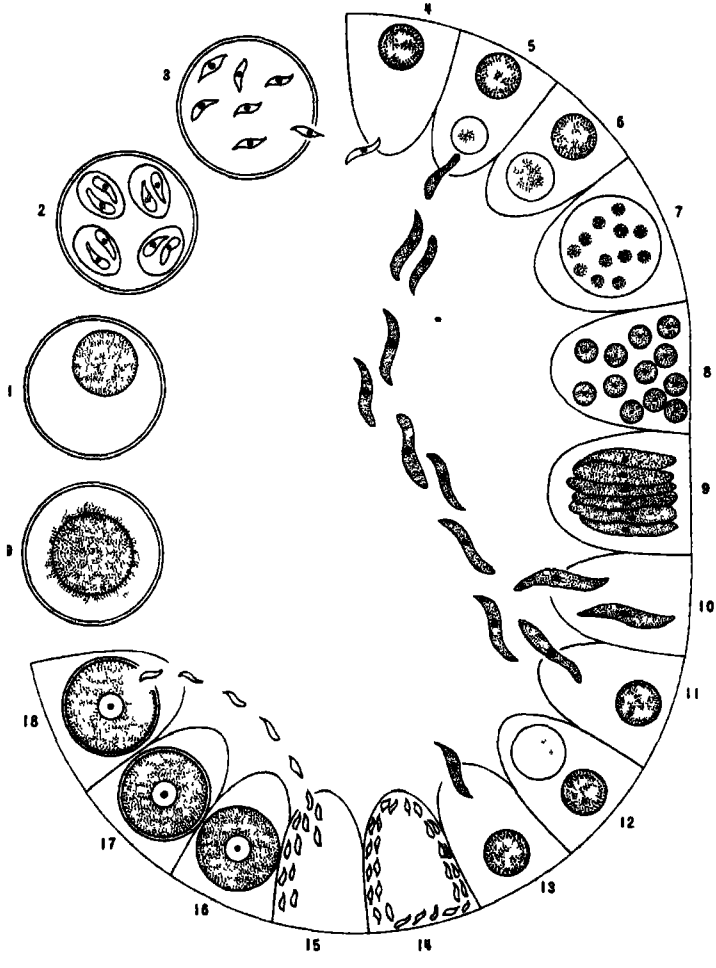

Eimeria intricata




Balantidium coli

PLATE III

COCCIDIA LIFE CYCLE (DIAGRAMMATIC)




(1) Oocyst (2) sporulated oocyst (3) liberation of sporozoites (4) sporozoites entering epithelial cells (5-11) schizogony: formation of schizonts and merozoites (12) sporogony: formation of macrogametocyte (13-15) sporogony: formation of microgametocytes (16-17) development of macrogametocyte (18) fertilization (19) formation of oocyst.

Sexual reproduction in protozoa other than Sporozoa where conjugation takes place, the sexual union is called syngamy. Isogamy is the union of cells of similar size and structure, anisogamy between dissimilar cells. The cells are called micro- and macrogametocytes whose union produces a zygote.

## HISTORY OF VETERINARY PROTOZOOLOGY

Veterinary protozoology is one of the youngest of the biological sciences. Since the majority of protozoa cannot be observed with the naked eye, they were unknown prior to the development of the microscope. Antony van Leeuwenhoek (1632-1723), the Dutch lens grinder, has been commonly called the father of protozoology. He made more than 400 microscopes, one with a magnification of 270

Goldfuss (1817) first coined the term Protozoa. Ehrenberg (1828, 1830, 1838) summarized the various genera of Protozoa, many of which still stand as they were so well described. Dujardin (1841) published a monograph on protozoa and suggested the term Rhizopoda. Various students proposed classes for the different protozoa such as Ciliata (Perty, 1852), Flagellata (Cohn, 1853), Mastigophora (Diesing, 1865), and others.

Work on the free-living forms went on at a much faster pace than on the parasitic forms. Apparently Leeuwenhoek (1674) according to Dobell was the first to observe a parasitic protozoan. He probably saw what is now known as Eimeria stiedae from the gall bladder of a rabbit. Parasitic protozoa were not observed after Leeuwenhoek's time until 1828 when Dufour described gregarines from the intestines of beetles In 1838 Hake rediscovered the oocysts of Eimeria stiedae. Balantidium coli was described in 1857 by Malmsten. Eimer (1870) made an extensive study of Coccidia occurring in different animals.

Leuckart (1879) proposed the name Sporozoa Laveran (1880) discovered the malarial parasites in the blood of man One of the most important landmarks in veterinary protozoology was the first demonstration by Smith and Kilborne (1893) that arthropods transmitted a protozoan disease, Texas cattle fever, caused by Babesia bigemina. Ross (1898) first showed that the female mosquito transmitted bird malaria. Trypanosomes in horses were discovered about 1895 Later, tsetse fly transmission of trypanosomes were brought to light. Since 1900 our knowledge of these one-celled animals in connection with veterinary problems has increased tremendously.

## TYPES OF PROTOZOAN PARASITES

The term parasite may be used in a broad or restricted manner Animal parasitism may include any association in which one species of animal depends upon another. In a narrow sense, parasitism usually means damage by the parasite to the host animal

Symbiosis strictly speaking implies the permanent association of two different organisms which are dependent on each other so that without the other they cannot exist. The most classical example of symbiosis is that of the protozoan fauna in the intestines of the termite. These protozoa change the wood material which the termites eat into a digestable form. Without this help the termite would die.

Commensalism is an association whereby the animal parasite (commensal) is benefited but the host is neither benefited or harmed. An example of this type of association would include certain species of protozoa in the rumen of cattle.

The term mutualism is occasionally used in parasitology. This denotes an association whereby both the host and the parasite derive mutual benefit from each other.

Parasitism is the condition when the parasite benefits and the host is harmed. The term parasite should be used to indicate a plant or animal that lives upon or within a host and maintains itself at the expense of the host

The host is the animal which harbors the parasite. The host which harbors the adult stages of the parasite (parasite in the sexual stage) is known as a definitive host (primary) The host which harbors the larval stages of the parasite (parasite in the asexual stage) is called the intermediate host (secondary) This may be further divided into first, second, or third intermediate host The term reservoir host is utilized to denote a host harboring parasites which may be transferred to man or domestic animals Aberrant parasite - a parasite which gains entrance into organs of the host's body where it does not ordinarily live. Pseudoparasites are objects which may be mistaken for parasites. Infection - establishment of a parasite within a host - infestation - refers to the presence of external parasites

## HOST SPECIFICITY

Parasites as a rule show different degrees of host specificity Certain parasites are found only in one species or related species of hosts Only a few parasites have a wide range of hosts This ability to live in certain hosts is a variable factor which depends upon the susceptibility of the host and the aggressive power of the parasite Some parasites of sheep live in cattle, others do not Certain parasites which live in the donkey or zebra may not survive in the horse Many wild animals harbor parasites which may be transferred to related domestic animals Other parasites such as the coccidia demonstrate a very strict host specificity, the Eimeria of cattle are found only in cattle or closely related species

If parasites enter a host accidentally it usually dies within a short time. Some forms wander all through the host's body before they die Parasites also have a certain amount of organ specificity. For example, Tritrichomonas foetus is found only in reproductive tract of cattle Practically all organs of the host's body are subject to various forms of parasites

## PATHOLOGY PRODUCED BY PROTOZOAN PARASITES

The pathological lesions produced by protozoan parasites in the host depends upon the species, the severity of the infection and the location of the parasite The different parasites vary in their behavior to produce damage to the host In most cases the parasites cause trouble by their efforts to obtain food.

Parasites may affect their hosts in the following ways:

1. Utilize food and other materials necessary for the host.
2. Mechanical obstruction by the accumulation of large numbers of parasites. This may include the blocking of the intestinal tract, obstruction of bile ducts, or plugging of lymphatics
3. Loss of blood or lymph fluid from the host.
4. Actual destruction of host tissue
5. By secretion of toxic products liberated by the parasites which may be detrimental to the host such as the various hemolysins, histolysins and anticoagulants.
6. Producing nodule formation and perforation of various organs.

7. Certain parasites may interfere with the calcium and phosphorous metabolism of the host.

8. Mechanical breakdown of capillaries producing hemorrhage (coccidia, malaria).

9. Some protozoan parasites may bring about a change in the normal blood picture (anemia, leukocytosis, lymphocytosis, eosinophilia).

10. Practically all of the processes of pathology can be demonstrated by the action of various parasites on the host. Hosts infected with parasites may show no symptoms or only slight disturbances, show marked symptoms of parasitism or end fatally. The special pathology of each protozoan parasite will be discussed in the following chapters under their appropriate headings.

## SPREAD OF PARASITES

Since animals live under conditions which cannot be compared with that of man, the spread of parasitic infections can occur in many ways. Animals are not adapted to "modern plumbing facilities" and must void their feces or urine on pastures or in the stables where they are housed. Infective stages of parasites may be spread by wind, water, insects, earthworms, mammals and birds

The host becomes infected in several ways. The majority of protozoan parasites inhabit the intestinal tract, consequently, the infective stages may be present in food or water. The intermediate hosts may inoculate the host such as, mosquitoes carrying the infective stages of malaria. Other protozoan parasites may be spread venereally such as, Tritrichomonas foetus which lives in the reproductive tract of cattle. An unusual mode of transmission of parasites is the prenatal infection by the mother to the offspring.

When proper moisture and humidity are present, the next important factor in the development of certain protozoan parasites is temperature. They cannot grow except at suitable temperatures. Temperature also governs the rate at which development takes place. Cold temperatures usually retard growth, warm temperatures accelerate it.

It is the general opinion that parasitic diseases are more prevalent and cause more economic loss to livestock in the warm, humid Southern and Southeastern states than any other large region in the United States. Concise information on this point is lacking. More investigations are needed on the entire subject of weather and its relation to protozoan infections of livestock before conclusions can be drawn

## CLASSIFICATION

At the present time animal parasites are classified according to the International Code of Zoological Nomenclature. Each protozoan parasite has a proper phylum, class, order, family, genus and species to which it belongs. In many branches of science further divisions are made by utilizing suborder, superfamily, subfamily or subspecies. All names are in Latin and binomial for species.

A brief classification of the Protozoa as far as Orders, is given here to give a comprehensive view of the Phylum and its range of scope in the animal kingdom. The genera which are of veterinary importance are marked with an asterisk (*).

## PROTOZOA OF VETERINARY IMPORTANCE

### PHYLUM PROTOZOA GOLDFUSS, 1817

SUBPHYLUM PLASMODROMA Doflein, 1901
CLASS MASTIGOPHORA Diesing, 1865
SUBCLASS PHYTOMASTIGINA Doflein
ORDER CHRYSOMONADINA Stein (fresh, salt water)
ORDER CRYPTOMONADINA Stein (fresh water)
ORDER PHYTOMONADENA Blochmann (fresh, salt water)
ORDER EUGLENOIDINA Blochmann (fresh, salt water)
ORDER CHLOROMONADINA Klebs (fresh, salt water)
ORDER DINOFLAGELLATA Butschli (marine, fresh water)
SUBCLASS ZOOMASTIGINA Doflein
ORDER RHIZOMASTIGINA Butschli
*Genus Histomonas Tyzzer
ORDER PROTOMONADINA Blochmann
*Genus Trypanosoma Gruby
Genus Oikomonas Kent
Genus Bodo Ehrenberg
Genus Monas Muller
ORDER POLYMASTIGINA Blochmann
Genus Enteromonas Fonseca
Genus Chilomastix Alexeieff
Genus Callimastix Weissenberg
*Genus Hexamita Dujardin
*Genus Giardia Kunstler
ORDER TRICHOMONADIDA Kirby
*Genus Trichomonas Donne
*Genus Tritrichomonas Kofoid
*Genus Pentatrichomonas Mesnil
Genus Monocercomonas Grassi
ORDER HYPERMASTIGINA Grassi (parasitic in arthropods)
CLASS SARCODINA Butschli, 1880
SUBCLASS RHIZOPODA Siebold
ORDER PROTEOMYXA Lankester (fresh water, algae)
ORDER AMOEBINA Ehrenberg
Genus Vahlkampfia Chatton and Lalung-Bonnaire
*Genus Endamoeba Leidy
*Genus Endolimax Kuenen and Swellengrebel
*Genus Iodamoeba Dobell
ORDER TESTACEA Schultze (fresh, salt water)

ORDER FORAMINIFERA d'Orbigny (marine)

SUBCLASS ACTINOPODA Calkins

ORDER HELIOZOA Haeckel (fresh, salt water)

ORDER RADIOLARIA Muller (marine)

CLASS SPOROZOA Leuckart, 1879

SUBCLASS TELOSPORIDIA Schaudinn

ORDER GREGARINIDA Lankester (parasitic in arthropods and annelids)

ORDER COCCIDIA Leuckart

*Genus Eimeria Schneider

*Genus Isospora Schneider

ORDER HAEMOSPORIDIA Danilewsky

Genus Plasmodium Marchiafava and Celli

*Genus Haemoproteus Kruse

*Genus Leucocytozoon Danilewsky

*Genus Babesia Starcovici

*Genus Theileria Bettencourt, Franca and Borges

*Genus Toxoplasma Nicolle and Manceaux

SUBCLASS ACNIDOSPORIDIA Cepede

ORDER SARCOSPORDIA Balbiani

*Genus Sarcocystis Lankester

ORDER HAPLOSPORIDIA Caullery and Mesnil (parasites of invertebrates and lower vertebrates)

SUBCLASS CNIDOSPORIDIA Doflein

ORDER MYXOSPORIDIA Butschli (parasites of fishes)

ORDER ACTINOMYXIDIA Stolc (parasites of annelids)

ORDER MICROSPORIDIA Balbiani (parasites of arthropods and fishes)

ORDER HELICOSPORIDIA Kudo (parasites of arthropods)

SUBPHYLUM CILIOPHORA Doflein, 1901

CLASS CILIATA Perty, 1852

SUBCLASS PROTOCILIATA Metcalf (parasites of frogs, reptiles, fish)

SUBCLASS EUCILIATA Metcalf

*ORDER HOLOTRICHA Stein

Genus Butschlia Schuberg (stomach of cattle)

Genus Blepharoprosthium Bundle (colon of horse)

Genus Didesmis Fiorentini (colon of horse)

Genus Blepharosphaera Bundle (colon of horse)

Genus Blepharoconus Gassovsky (colon of horse)

Genus Bundleia Cunha and Muniz (colon of horse)

Genus Polymorphus Dogiel (colon of horse)

Genus Holophryoides Gassovsky (colon of horse)

Genus Blepharozoum Gassovsky (cecum of horse)

Genus Prorodeopsis Gassovsky (colon of horse)

Genus Paraisotrichopsis Gassovsky (cecum of horse)
Genus Sulcoarcus Hsiung (feces of mule)
Genus Alloiozona Hsiung (colon of horse)
Genus Ampullacula Hsiung (colon of horse)
Genus Paraisotricha Fiorentini (colon of horse)
Genus Isotricha Stein (stomach of cattle, sheep)
Genus Dasytricha Schuberg (stomach of cattle)
Genus Charon Jameson (colon of horse, stomach of cattle)
Genus Blepharocorys Bundle (*colon of horse, stomach of cattle*)
*ORDER SPIROTRICHA Butschli
*Genus Balantidium Claparede and Lachmann
Genus Entodinium Stein (cattle, sheep)
Genus Eodinium Kofoid and MacLennan (cattle, sheep)
Genus Diplodinium Schuberg (cattle)
Genus Eremoplastron Kofoid and MacLennan (cattle, sheep)
Genus Eudiplodinium Dogiel (cattle)
Genus Diploplastron Kofoid and MacLennan (cattle, sheep, goat)
Genus Metadinium Awerinzew and Mutafowa (cattle, sheep, goat)
Genus Polyplastron Dogiel (cattle, sheep)
Genus Elytroplastron Kofoid and MacLennan (cattle)
Genus Ostracodinium Dogiel (cattle, sheep)
Genus Buxtonella Jameson (cecum of cattle)
Genus Enoploplastron Kofoid and MacLennan (cattle)
Genus Epidinium Crawley (cattle, sheep)
Genus Cycloposthium Bundle (colon of horse)
Genus Spirodinium Fiorentini (colon of horse)
Genus Triadinium Fiorentini (colon of horse)
Genus Tetratoxum Gassovsky (colon of horse)
Genus Trypalmaria Gassovsky (colon of horse)
Genus Cochliatoxum Gassovsky (colon of horse)
Genus Ditoxum Gassovsky (colon of horse)
ORDER CHONOTRICHA (attached to crustaceans)
ORDER PERITRICHA (free living, attached to plants, aquatic animals)
CLASS SUCTORIA Claparede and Lachmann, 1858
Genus Allantosoma Gassovsky (colon of horse)

## CHAPTER II

## PROTOZOA OF THE HORSE

## Chapter II

## PROTOZOA OF THE HORSE

### CLASS. MASTIGOPHORA
### ORDER: PROTOMONADINA
### Family Monadidae

Oikomonas equi Hsiung, 1930

Hsiung (1930) reported the occurrence of a small flagellate with a single flagellum in the cecum of 8 horses in Iowa. The organism is small being 3 5 to 7 microns in length and 3 to 5 5 microns wide. The flagellum is about 20 microns long They are colorless and the cytoplasm is filled with minute dark staining granules. A single spherical nucleus with a large central karyosome is located at the anterior end This flagellate is probably a harmless commensal and is not known to produce any disease in horses

**TREATMENT** Unnecessary

Family Trypanosomidae

Trypanosoma equiperdum Doflein, 1901

**SYNONYMS** T. rougeti Laveran and Mesnil, 1901, Trypanozoon equiperdum (Luke, 1906), Castellanello equiperdum (Chalmers, 1918)

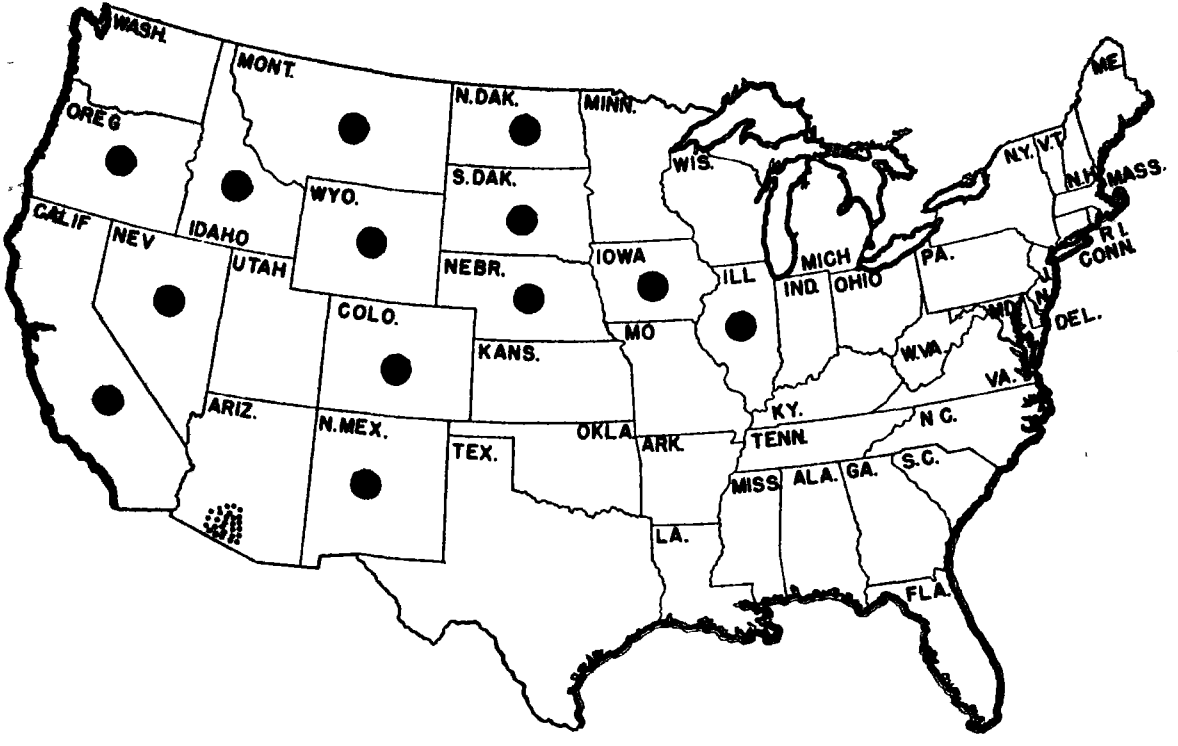
**DISEASE** el dourine, maladie du coit, covering disease, equine syphilis, genital glanders, breeding paralysis, chancrous epizootic, epizootic paraplegia It is known as dourine in the United States

**HISTORY.** Dourine was first diagnosed in the United States by Williams in 1886 in Illinois The actual diagnosis was based on clinical symptoms The infection was traced to a stallion imported from France The disease was eradicated from the state in 1888 by quick, rigid control measures However, some animals had been shipped to other parts of the United States In 1892 the disease was reported in Nebraska. The Bureau of Animal Industry put on control measures and dourine was apparently stamped out Dourine was diagnosed in 1899 and 1901 in South Dakota and in Iowa about 1911 After the development of the complement-fixation test for diagnosis, several cases were diagnosed in Montana (1912), North Dakota, South Dakota, Arizona, New Mexico, Wyoming and Nebraska Since 1912 dourine has been eradicated from Nebraska, North Dakota, South Dakota, Wyoming, Montana, Iowa and New Mexico It is now limited to a small range in Nevada, Oregon and Arizona on certain Indian reservations. The condition is being closely controlled by the Bureau of Animal Industry According to the 1946 report of the Chief of the Bureau of Animal Industry the only center of infection is in Arizona on the Papago Indian Reservation.

**MORPHOLOGY** T equiperdum is a monomorphic trypanosome which always possesses a single flagellum It ranges in size from 25 to 28 microns and from 1 to 2 microns in width

**TRANSMISSION** Dourine is chiefly a disease of breeding animals. The organisms can be transmitted under laboratory conditions to dogs, rabbits, rats, mice, monkeys, sheep, goats and other susceptible animals by inoculation with large amounts of infective blood. Mechanical transmission has been demonstrated by means of the stable fly and horse fly

**SYMPTOMS:** The symptoms vary a great deal with individual animals, no two animals having all of the known manifestations The disease effects mainly the sexual organs, but in the later stages shows disturbances of the nervous system.




MAP 1. Dourine in the United States. Large black circles indicate states which have reported and eradicated the disease. In 1946 the only center of infection was in Arizona on the Papago Indian Reservation which is designated by the dotted area.

The incubation period ranges from 8 days to about 2 months. In the stallion irritation and swelling appear on the penis. This may extend throughout the reproductive organs, groin, and enlargement of the regional lymph glands. A few days later blisters on the penis occur which rupture and discharge a yellow fluid. These areas become ulcerated and raw. The ulcers heal rapidly leaving permanent white scar tissue. Rarely the urethra is inflamed or the testicles involved.

In the mare, dourine first appears in the genital tract with swelling and inflammation. A yellowish discharge may occur. Vesicles appear which rupture and ulcerate. Scar tissue forms very rapidly

Degeneration of the nervous system may not occur for months or years Any apparent recovery is not permanent. Occasionally, a mare may abort although many animals are foaled by infected mares The nervous symptoms include staggering, swaying and little control over the hind legs. The animal may become emaciated and gaunt Paralysis of the hind quarters eventually sets in. Sometimes paralysis of the foreleg, face, ear, nostril or lip may be noticed Characteristic swellings or plaques may spread over the back and belly. These areas are round, flat and about 1 to 2 inches in diameter which contain a blood-like fluid. The temperature remains normal. After paralysis of the hind quarters occur, the animal goes down and dies from nervous exhaustion

DIAGNOSIS Since this is the only trypanosome disease of importance in domestic animals in the United States the complement-fixation test is of great value as a diagnostic aid. This test is sensitive enough to diagnose accurately the latent cases of dourine. Complement-fixation tests on dourine will be run on the sera of suspected horses by the Bureau of Animal Industry.

TREATMENT At the present time there is no known specific therapeutic treatment for dourine. Research on immunization has been negative

CONTROL Recovery from an infection of T equiperdum is uncertain. The mortality rate ranges from 50 to 70 per cent Since the disease occurs in restricted areas in the United States, the best procedure is eradication. The most effective way is the prompt destruction of all infected animals

The Bureau of Animal Industry (Pathological Division) maintains a diagnostic laboratory in Washington, D C for conducting the complement-fixation test for dourine. The Bureau also cooperates with veterinarians and livestock sanitary authorities for field work wherever the parasite exists. The animals are held in strict quarantine and the positive animals are destroyed. On Indian reservations, the entire expense is paid by the federal government

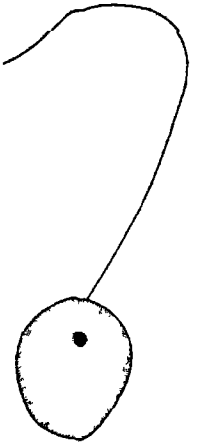
ORDER. POLYMASTIGINA

Family: Callimastigidae

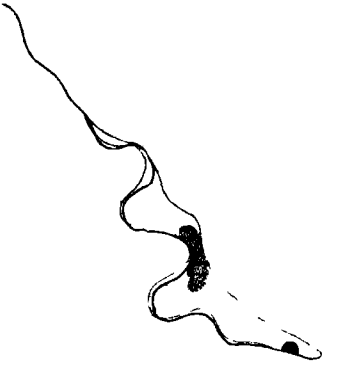
Callimastix equi Hsiung, 1929

This is a small kidney-shaped flagellate which inhabits the cecum and colon of the horse. Hsiung (1930) found this parasite in nine out of 46 horses examined. It is approximately 12 to 18 microns long and 7 to 10 microns wide. At the hilus 12 to 15 flagella which are 25 to 30 microns long originate. The organism is nonpathogenic.

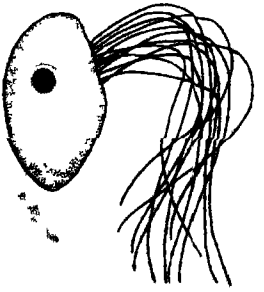
TREATMENT: Unnecessary.



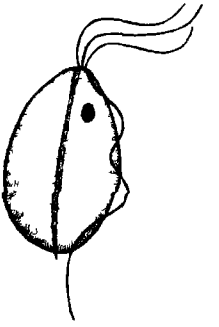
Oikmonas equi



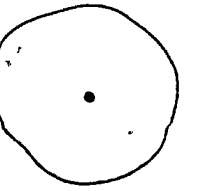
Trypanosoma equiperdum



Callimastix equi



Tritrichomonas equi



Endamoeba gedoelsti

ORDER: TRICHOMONADIDA
Family: Trichomonadidae

Tritrichomonas equi (Fantham, 1921)

This parasite was observed by Hsiung (1930) from the colon, cecum and feces of several horses in Iowa This trichomonad possesses three anterior flagella, an undulating membrane and a slender axostyle. The organism is quite small with a length of 4 to 6.5 microns and 3 to 5 microns wide. It has no pathological significance.

TREATMENT: Unnecessary.

CLASS SARCODINA
ORDER: AMOEBINA
Family: Endamoebidae

Endamoeba gedoelsti (Gedoelst, 1911)

Only one species of amoeba has been reported from the horse in the United States. It is apparently nonpathogenic and inhabits the cecum and colon The organism is known only in the trophozoite form which ranges in size from 6.5 to 12.5 microns long by 6 to 11 microns wide. Nourishment is obtained from the intestinal contents by ingesting bacteria. The nucleus is similar to that of E coli found in man. The karyosome is eccentric in position The only report of this parasite from horses in North America was made by Hsiung (1930).

TREATMENT: Unnecessary

CLASS. SPOROZOA

No coccidia have been reported from North American horses.

CLASS: CILIATA

Approximately 50 species of ciliated intestinal protozoa, comprising over 25 genera are commonly found in the cecum and colon of equines. From the evidence at hand, these ciliates are apparently nonpathogenic and probably do not produce any manifestations of disease. Hsiung (1930) believes they are harmless commensals and are in no way a significant help to the horse. Perhaps some of these protozoa may help break down indigestible food material into a readily assimilated form. Practically every horse harbors some species of intestinal ciliates. The method of transmission from horse to horse is not yet fully understood. It is assumed that horses pick up the organisms by swallowing them in their food and water. Becker and Hsiung (1929) attempted to infect the rumen of a goat with the infusoria from the colic contents and fecal material from horses with negative results. This indicated that the infusoria of the horse is specific and will not live or survive in the stomach of ruminants. The excellent monographic work of Hsiung (1930) give the following species of ciliates occurring in the cecum, colon and feces of North American horses.

ORDER: HOLOTRICHA

Family: Butschliidae

1. Didesmis ovalis Fiorentini, 1890.
2. D. quadrata Fiorentini, 1890.
3. D. spiralis Hsiung, 1929.
4. Blepharoprosthium pireum Bundle, 1895.
5. Blepharosphaera intestinalis Bundle, 1930.
6. B. ellipsoidalis Hsiung, 1930.
7. Blepharoconus cervicalis Hsiung, 1930.
8. B. benbrooki Hsiung, 1930.
9. Bundleia postciliata (Bundle, 1895).
10. Alloiozona trizona Hsiung, 1930.
11. Polymorpha ampulla Dogiel, 1929.

Family: Paraisotrichidae

12. Paraisotricha colpoidea Fiorentini, 1890.
13. P. beckeri Hsiung, 1930.
14. P. minuta Hsiung, 1930.

Family: Blepharocoridae

15. Blepharocorys uncinata (Fiorentini, 1890).
16. B. balbata (Fiorentini, 1890)
17. B. jubata Bundle, 1895.
18. B. corvigula Gassovsky, 1919.
19. B. angusta Gassovsky, 1919.
20. B. cardio-nucleata Hsiung, 1930.
21. Charon equi Hsiung, 1930

ORDER: SPIROTRICHA

Family: Cyclogosthiidae

22. Cyclogosthium bipalmatum (Fiorentini, 1890).
23. C. dentiferum Gassovsky, 1919.
24. C. edentatum Strelkow, 1928.
25. C. scutigerum Strelkow, 1928.
26. C. affinel Strelkow, 1928.
27. C. corrugatum Hsiung, 1930.
28. Spirodinium equi Fiorentini, 1890.
29. Triadinium caudatum Fiorentini, 1890.
30. T. gallee Gassovsky, 1919.
31. T. minimum Gassovsky, 1919.
32. Tetratorum unifasciculatum (Fiorentini, 1890).
33. T. excavatum Hsiung, 1930.
34. T. parvum Hsiung, 1930.
35. Tripalmaria dogieli Gassovsky, 1919.
36. Cochliatoxum periachtum Gassovsky, 1919.
37. Ditoxum funimuciporum Gassovksy, 1919.

TREATMENT: Unnecessary.

CLASS: SUCTORIA
Family: Acinetidae

Only three species of Suctoria have been recorded from the cecum and colon of North American horses, all by Hsiung, (1930). They are probably harmless commensals and are not known to produce any digestive disturbances. The three species are as follows:

1. Allantosoma intestinalis Gassovsky, 1919.
2. A. dicorniger Hsiung, 1928.
3. A. brevicorniger Hsiung, 1928.

TREATMENT: Unnecessary.

## ORGANISMS OF UNCERTAIN ZOOLOGICAL CLASSIFICATION

### Sarcocystis bertrami Doflein, 1901

Under the present zoological classification the order Sarcosporidia contains only one genus, Sarcocystis Many species are found widely distributed among the vertebrates. Since the excellent work by Spindler et al (1945, 1946, 1947) on the form found in swine, the classification is uncertain as evidence now shows that this parasite may be a fungus.

The parasite invades the striated muscles, particularly those of the skeletal body of the horse. They form long tubular sacs of spores (Miescher's tubes). The mature tube has two layers and an interior net of trabeculae The spores are banana or sickle-shaped and are about 10 to 15 microns long When liberated these spores are quite motile.

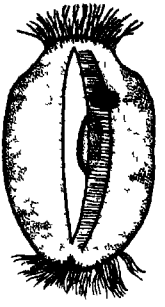
For more details on the life cycle of Sarcocystis see Chapter V concerning the protozoa of swine Spindler et al (1945, 1946, 1947) working with this form gives in detail the life cycle and latest research on its classification. Since the form from swine is the only one studied, the species recorded from the horse must await confirmation by experimental studies.

This parasite apparently causes no serious damage to the horse. It produces a true toxin, sarcocystin, which in small amounts is fatal to rabbits. The tissues surrounding the tubes show some fibrosis and leucocytic infiltration. The infection has been seldom reported from horses, Dikmans (1945) lists it only from Ohio, Wilson and McDonald (1938) reported it from Virginia

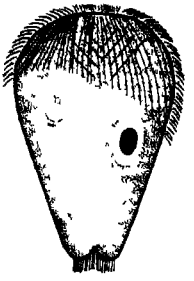
TREATMENT Unnecessary

PLATE V

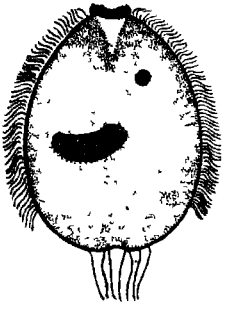
CILIATES AND SUCTORIA FROM THE HORSE



Didesmis quadrata



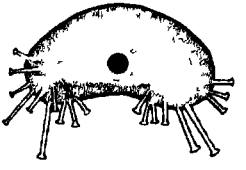
Blepharoconus hemiciliatus



Blepharosphaera ellipsoidalis

Cycloposthium endentatum

Spirodinium equi



Allantosoma intestinalis

## CHAPTER III

## PROTOZOA OF CATTLE

## Chapter III

## PROTOZOA OF CATTLE

CLASS: MASTIGOPHORA
ORDER: PROTOMONADINA
Family: Trypanosomatidae

Trypanosoma theileri Laveran, 1902

SYNONYM: T. americanum, Crawley, 1901, T. rutherfordi, Hadwen, 1912.

This trypanosome is a large flagellate measuring 60 to 70 microns in length and 4 to 5 microns in width. It is further characterized by well-developed myonemes. The organism does not become numerous in the blood of adult cattle and are apparently nonpathogenic Biting flies may act as vectors. The organism can be transmitted from infected adult cattle to calves by large inoculations of trypanosome positive blood. This trypanosome has been reported from Louisiana, Texas and Maryland. The organisms can often be demonstrated culturally by mixing one part of aseptically collected blood with two parts of veal infusion broth. The tube is incubated about a week at room temperature. White colonies appear which contain trypanosomes

TREATMENT: Unnecessary.

ORDER: PROTOMONADINA
Family: Monadidae

Monas communis (Liebetanz, 1910)

Becker and Talbott (1927) reported this organism from the rumen of a few cows in Iowa. It is spherical and about 4 microns in diameter. A primary flagellum arises from a basal granule. During locomotion this flagellum is directed backwards. The secondary flagellum is much shorter. It is nonpathogenic.

TREATMENT: Unnecessary.

ORDER: POLYMASTIGINA
Family: Callimastigidae

Callimastix frontalis Braune, 1913

This organism has also been reported by Becker and Talbott (1927) from the rumen of cows in Iowa. It has a clear disc shaped area in the anterior end, on the margin of which are 12 basal granules that give rise to 12 flagella. They are about 30 microns long. The body of the flagellate is from 12 to 14 microns in diameter. The 12 flagella appear to move as a single unit. It is apparently nonpathogenic.

TREATMENT: Unnecessary.

Family: Hexamitidae

Giardia bovis Fantham, 1921

Very little is known concerning the pathogenicity of Giardia in cattle. Becker and Frye (1927) found this species in the feces of Iowa cattle. The organism is 12 to 14 microns long and 9 to 10 microns wide.

TREATMENT: Unnecessary.

ORDER: TRICHOMONADIDA

Family: Monocercomonadidae

Monocercomonas ruminantium (Braune, 1913)

SYNONYM: Eutrichomastix ruminantium.

This flagellate has been observed by Becker and Talbott (1927) from sheep. Morgan and Noland (1943) and Morgan (1944) recorded a similar organism from the sheath of bulls. The organism is about 8 to 10 by 12 to 14 microns. There are 3 anterior flagella and one trailing flagellum.

TREATMENT: Unnecessary.

Family: Trichomonadidae

Tritrichomonas ruminantium (Braune, 1913)

This organism has been observed in North American cattle by several workers. Becker and Frye (1927) and Becker and Talbott (1927) recorded this flagellate from Iowa cattle as did Morgan and Noland (1943). It is 8 to 10 microns long, with 3 anterior flagella slightly longer than the body. An axostyle is present. The undulating membrane is very shallow. A survey was made to determine the prevalence of intestinal trichomonads in cattle was reported by the Chief of the Bureau of Animal Industry (1938) Of 17 cattle examined all were found to harbor this organism. Trichomonads were also isolated from the rumen, reticulum, cecum and colon of a bull that was slaughtered. According to Dikmans (1942) T. ruminantium has been found in association with cases of diarrhea in cattle, but, so far as known, does not bear any relationship to this condition

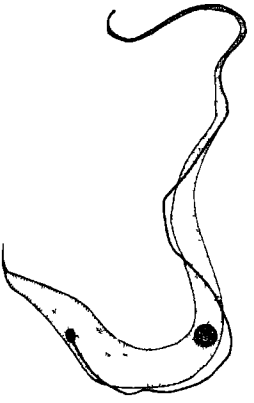
TREATMENT: Unnecessary

Tritrichomonas foetus (Riedmuller, 1928)

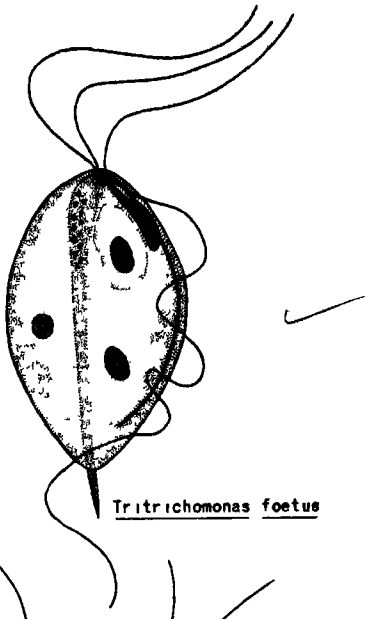
SYNONYMS: Trichomonas bovis Riedmuller, 1930, T. genitalis Witte, 1933, T. vaginalis bovis Feiling, 1935.

DISEASE: Bovine trichomoniasis, bovine genital trichomoniasis.

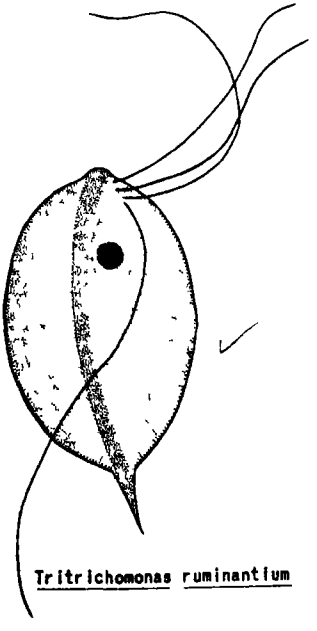
Tritrichomonas foetus has been shown by numerous investigators to produce reproductive disturbances in cattle. The organism which is now known as T. foetus was first observed by Kunstler (1888) in France. Mazzanti (1900) has been given credit for discovering T. foetus in Italy although Kunstler's work antedates that of Mazzanti by 12 years. These two reports went unrecognized until about 1925 because of Bang's discovery in 1897 of the organism causing contagious abortion (Bang's disease).



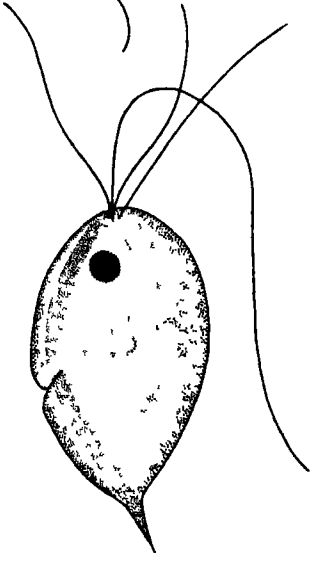
Trypanosoma theileri



Tritrichomonas foetus



Tritrichomonas ruminantium



Monocercomonas ruminantium

Drescher (1925) reported that in 1924 Hopfengartner had found trichomonads in the abomasum of an aborted fetus. Riedmuller (1928) reported his classical work on T. foetus which stimulated active research on this subject.

The geographical distribution of the disease is probably world-wide, or, at least, in any part of the world where cattle are located. Trichomoniasis has been reported throughout the United States.

**MORPHOLOGY:** The organism ranges from 10 to 25 microns in length and 3 to 15 microns in width. It is pear-shaped with 3 anterior flagella and an undulating membrane. An axostyle is present.

**SYMPTOMS AND LESIONS:** As a rule trichomoniasis may produce several clinical manifestations. Symptoms may vary from little or slight disturbance of the genital tract to those of severe trichomoniasis. All of the factors involved in the various consequences of the disease are not known.

The invasion of T. foetus into the uterus may produce a low grade vaginitis and endometritis accompanied by uterine, cervical, or vaginal catarrh. Repeated breedings, purulent discharges and delayer conceptions are characteristics of this type of trichomoniasis.

Conception may occur, but due to the prevailing uterine infection abortion may follow. The abortions may take place any time during the gestation period but usually occur between 1 to 16 weeks after the last breeding. Early abortion is one of the definite characteristics of trichomoniasis. Late abortions are comparatively rare, evidenced by only 6 reports in the literature of abortions occurring after 6 months gestation.

Abortions may be of two types. One type is characterized by the destruction of the placental attachments resulting in the removal of the fetus with the fetal membranes intact. As a rule, cows showing this type of abortion recover and will conceive later on. In the second type of abortion the fetal membranes are retained in the uterus, which usually results in a chronic catarrhal or purulent endometritis. Animals with this type of trichomoniasis are usually permanently sterile because of the destruction of the uterine mucosa.

Under certain conditions which are unknown, there may be an early death of the fetus in utero with retention of the corpus luteum and the cervical seal of pregnancy. The fetus is not expelled but becomes macerated while uterine secretions continue to accumulate. The presence of the cervical seal prevents the escape of exudate although there may be a slow escape of the uterine fluid when the animal is recumbent or when there is too much pressure created by the accumulation of fluid. There is usually a lack of heat during pyometra.

The fluid is thin, greyish white, with a few clumps of leucocytes and almost odorless. The reaction is usually alkaline. The fluid may contain millions of trichomonads although in long standing cases the organisms may die.

Under certain conditions a normal gestation and calving may result in spite of the infection. This is a rare occurrence; however, trichomonads have been isolated throughout the gestation period though the cases resulted in normal parturition.

It has been definitely demonstrated by numerous workers that T. foetus is transmitted in nature by coitus from the bull to the cow or vice versa. Bovine trichomoniasis is a true venereal disease of cattle. The disease can also be spread by artificial insemination by using semen from infected bulls.

Practically all investigators have agreed that the primary site of T. foetus is the bovine uterus. The original site is the vagina when the organism is transmitted by the bull. The trichomonads may invade the uterus through the cervix or remain in the vagina. Under different conditions, some known, many unknown, various manifestations take place

in the uterus. In the case of a pregnancy which is terminated by an abortion, trichomonads may be found in the amniotic and allantoic fluids.

Fetal membranes and fetuses may also be invaded by trichomonads. They have been demonstrated in the various organs and fluids of aborted fetuses. The most common location in aborted fetuses is the stomach or abomasum.

The work of Hammond and Bartlett (1943) and Morgan (1946) has shown that the preputial cavity is the most common and preferential site for T. foetus in the bull. Occasionally, the organisms may invade the upper reproductive tract such as the epididymis, seminal vesicles and testicles Bulls as a rule when they contract the disease are permanently infected.

DIAGNOSIS: A positive diagnosis of bovine trichomoniasis depends upon the demonstration of living, motile T foetus in the genital exudate of infected animals or in the tissues or fluids from infected fetuses If there is a heavy infection, the organisms can usually be seen in direct microscopic examination of this material. If the results are negative it is necessary to prepare cultures and examine for trichomonads after a brief period of incubation. Both methods require the use of a microscope and identification of the causative protozoan.

Materials for diagnosis which can be collected and examined include washings from the vagina and uterus, aborted fetuses, samples of fetal stomach fluid, fetal oral fluid, fetal membranes, uterine pus, pieces of placenta, amniotic and allantoic fluid, washings from the sheath, seminal fluid and semen.

Recently Hammond and Bartlett (1943) reported that the diagnosis of bovine trichomoniasis could be improved if microscopic examination of the sample is made after 1 to 3 hours of sedimentation. They further reported (1945) that a higher percentage of positive samples were taken from the vagina between the 19th and 20th day after exposure and that the trichomonads were most numerous on the 14th to 18th day. Samples should be collected from cows during this time

In collecting samples from the bull a pipette or cotton swab is inserted into the prepuce. The animal should be secured in a stall and restrained by an assistant using a sideline to reduce exertion to a minimum A glass speculum is inserted in the preputial cavity and the swab introduced and moved in a circular motion. The posterior portion of the penis and prepuce should be well swabbed Care must be taken not to contaminate the sample with fecal material Fecal material contains several species of coprozoic protozoa which may confuse the diagnosis by error of identification of these organisms.

The identification of T. foetus is not a simple matter. The examination of diagnostic material has yielded a wide variety of protozoa which may be erroneously identified as T. foetus. Some of the organisms include T. ruminantium, Monocercomonas ruminantium Bodo foetus, B. glissans, Spiromonas angusta, Cercomonas crassicauda, Polytoma uvella, Monas obliqua and Lembus pusillus.

GENERAL REMARKS: The complement-fixation test for the diagnosis of trichomoniasis has not been satisfactory for routine practice because of non-specific reactions. Kerr and Robertson (1941) have utilized the agglutination test with some degree of success. Positive tests indicates a herd infection. Kerr (1943) claimed that the agglutination test give an over-all result of 60 per cent. About 80 per cent may be found in the clinical cases such as endometritis and pyometra. Morgan (1943) could not use this test on a practical basis. Critical tests on over 72 infected cows and bulls demonstrated that the test was no better than the routine microscopic examinations.

Kerr (1944) reported on an intradermal test for the diagnosis of trichomoniasis. An extract of T. foetus called "tricin" was injected into the skin of bovines. A reading was usually made within 30 to 60 minutes. The reaction appeared in 10 minutes, reached its peak within 30 minutes and disappeared in about 6 hours. A shallow plaque was the characteristic action, more obvious to the eye than by measurement. Kerr tested 592 cows

passing through an abattoir and obtained 50 positive skin reactors. Trichomonads were recovered from 11 on direct examination. Thirteen bulls were positive and 8 were found to harbor trichomonads. In another group of 65 bulls tested, 21 or 23 per cent were positive for the skin test. Trichomonads were found in only 3 animals by direct smears. Cows with an active infection such as a pyometra, did not respond to the test.

Svec (1944) isolated a polysaccharide from T. foetus by the Fuller formamide method. This extract gave no specific precipitation reaction with the sera of normal cattle or with cattle injected with living or formalinized T. foetus. Four other fractions isolated from T. foetus also gave poor results. Morgan (1946 and unpublished data) injected these fractions intracutaneously into normal and trichomonad positive cows and bulls with negative results.

Kerr and Robertson (1945) reported on the appearance of serological varieties among strains of T. foetus. Until 1944 all strains of T. foetus examined by Kerr had shown similar serological reactions. However, two strains have proved to be aberrant as regards to agglutinability. Variation in strains was suggested earlier by Morgan.

At the present time practical methods have not been devised to utilize our knowledge of immunity in cows recovering from trichomoniasis. The mechanisms involved with trichomonad immunity are not well understood. Hammond and Bartlett (1945) observed a trichomonad being phagocytized by a leucocyte. The fluctuation in the number of trichomonads in the vagina may in part be ascribed to this defensive leucocytic action.

TREATMENT AND CONTROL. It has been established experimentally and in the field that T. foetus infection is self-curing in most females but the bull remains permanently infected.

At the present time there is no recognized, proven specific therapeutic treatment for trichomoniasis in either cows or bulls. Cows that have aborted early during the gestation period should be given sexual rest for at least three months. If the abortion is complete or a low grade vaginal or uterine infection present cows sometimes recover spontaneously.

The spread of trichomonas can be prevented by slaughtering infected bulls, giving breeding rest to infected cows and using artificial insemination with a clean bull.

Encouraging results on the experimental treatment of trichomoniasis in bulls has been reported by Bartlett (1946).

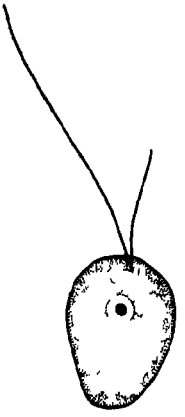
Eight bulls infected with T. foetus, two of which were reinfected (total of 10 infections) received treatment with potassium iodide orally and/or sodium iodide, by intravenous injections. Six infections were cured and four were not Experimental iodide treatments show considerable promise as chemotherapeutic agents against T. foetus infection in bulls.

CLASS: SARCODINA
ORDER. AMOEBINA
Family: Amoebidae

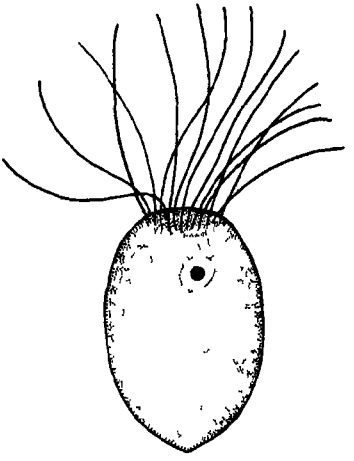
Vahlkampfia lobospinosa (Craig, 1912)

Only two species of amoeba have been reported from the rumen of North American cattle. They are apparently nonpathogenic. V. lobospinosa, a free-living amoeba, was found by Becker and Talbott (1927) in the rumen of one cow. Whether they were ingested recently or had established a foci as an accidental parasite is not known. The uninucleate forms measured 10 to 18 microns, the multinucleate forms 18 to 24 microns. Cysts with one or two nuclei measured 7 to 11 microns.

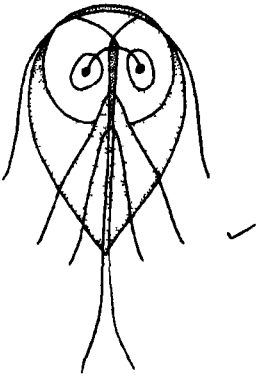
TREATMENT: Unnecessary.



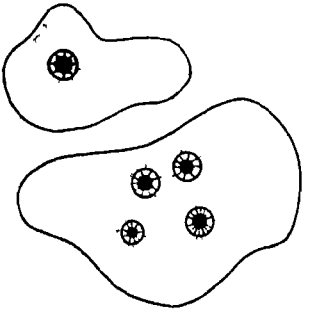
Monas communis



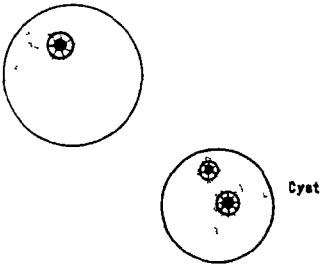
Callimastix frontalis



Giardia bovis



Vahlkampfia lobospinosa




Endamoeba bovis

Family: Endamoebidae

Endamoeba bovis (Liebetanz, 1910)

Becker and Talbott (1927) reported this amoeba from the rumen of several cattle. It appeared similar to E. histolytica. Cysts were not observed. The trophozoites measured about 20 microns. Nothing is known of its pathogenicity.

TREATMENT: Unnecessary.

KEY TO OOCYSTS OF EIMERIA FOUND IN CATTLE*

1. Relatively small and colorless, without perceptible micropyle ..... 2

   Relatively large and tinted; micropyle a gap in wall at one end of oocyst ..... 6

2. Typical oocysts subspherical or spherical ..... 3

   Typical oocysts pyriform, ellipsoidal or cylindrical ..... 4

3. Relatively small, 9 to 13 microns long; pallid, thin-walled, delicate; 96 to 120 hours required for complete sporulation ..... E. subspherica

   Larger; 15 to 22 microns long; more conspicuous and crystalline in appearance; 48 to 72 hours required for complete sporulation ..... E. zurnii

4. Typical oocysts distinctly pyriform, but varying from subellipsoidal to subcylindrical; 13 to 24 microns long, pallid, thin-walled, delicate, peculiar parachute-shaped caps at ends of sporocysts preceding sporozoite formation, 92 to 120 hours required for complete sporulation ..... E. alabamensis

   Typical oocysts regularly ellipsoidal or cylindrical, more conspicuous and less fragile than oocysts of E. alabamensis ..... 5

5. Majority of specimens regularly ellipsoidal, varying from spherical to nearly cylindrical; 12 to 27 microns long; 48 to 72 hours required for complete sporulation ..... E. ellipsoidalis

   Majority of specimens regularly cylindrical, varying from ellipsoidal to narrowly cylindrical, 16 to 27 microns long; 48 hours or less required for complete sporulation ..... E. cylindrica

6. Typical oocysts regularly ellipsoidal, with some slightly tapered or nearly cylindrical forms in range of variation; 25 to 37 microns long; wall homogeneous, delicately yellowish-brown; 72 to 96 hours required for complete sporulation ..... E. canadensis

*Adapted from Christensen (1941).

Typical oocysts ovoidal, 34 to 42 microns long; 24 to 29 microns wide; 144 hours required for sporulation ..... E. brasiliensis

Typical oocysts ovoidal, 31 to 54 microns long and 22 to 34 microns wide; 36 to 72 hours required for sporulation ..... E. ildefonsoi

Typical oocysts tapered toward micropylar end ..... 7

7. Wall distinctly thickened and yellowish-brown to dark brown in color, these being the most intensely tinted oocysts noted from bovines; 33 to 41 microns long; pyriform; 96 to 168 hours required for complete sporulation ..... E. bukidnonensis

Typical oocysts ovoidal, 37 to 45 microns long and 26 to 30 microns in width, 120 to 168 hours required for sporulation, oocyst wall speckled ..... E. wyomingensis

Wall not distinctly thickened or so intensely tinted, 48 to 72 hours required for complete sporulation ..... 8

8. Typical oocysts stoutly egg-shaped or ovoidal, 23 to 34 microns long, almost colorless to pale greenish- or yellowish-brown, wall smooth and homogeneous in all specimens ..... E. bovis

Typical oocysts elongated ovoidal, 32 to 46 microns long, pale to distinctly yellowish-brown, wall typically homogeneous, but variants occur having numerous, small, rounded mammillations in the wall ..... E auburnensis

CLASS. SPOROZOA
ORDER. COCCIDIA
Family: Eimeriidae

## Bovine Coccidiosis

Coccidia are tissue invading parasites, primarily of epithelial cells. Bovine coccidiosis is an infectious disease of cattle which produces bloody scours or red diarrhea. The disease is of great economic importance in the United States.

There are at least 12 valid species of bovine coccidia. All of the species belong to the genus Eimeria which infect cattle. Species differentiation is based on the following characters: (1) size, shape, color of the oocysts, (2) nature of the sporocysts, (3) sporulation time, (4) and physiological characters. The basic characteristics of the species found in North American cattle are listed below.

### Eimeria alabamensis Christensen, 1941

Oocysts range between 13 to 24 microns in length by 11 to 16 in transdiameter. The shape is typically pyriform with variations to subellipsoidal and subcylindrical forms. There is no micropyle. The wall is thin; the oocyst appears colorless, under oil immersion they are grayish-lavender to pale brownish-yellow tint. Sporulation time is between 96 and 120 hours. Sporulated oocysts contained 4 elongated, tapered sporocysts, each having two indistinct sporozoites.

Eimeria suburnensis Christensen and Porter, 1939

Oocysts range between 32 to 45 microns long by 20 to 25 microns wide. The shape is typically elongated ovoidal varying between subellipsoidal to tapering. Micropyle resembles an operculum. The walls are typically smooth but varying in structure from the transparent type to a rare semi-transparent heavily mammillated type. Sporulation time is between 48 to 72 hours.

Eimeria bovis (Zublin, 1908)

SYNONYM: Eimeria smithi Yakimov and Galouzo, 1927.

The oocysts measure between 23 to 34 microns long to 17 to 23 microns wide. The shape is typically stout ovoidal, blunted across the narrow end. Variations are considerable with subellipsoidal, asymmetrical and elongated forms. Micropyle appears as a gap in the cell wall. Under low-power the color is pale, cloudy, greenish to yellowish brown. The time of sporulation is about 48 to 72 hours.

Eimeria brasiliensis Torres and Ramos, 1939

Oocysts are about 34 to 42 by 24 to 29 microns. They are ovoidal with a micropyle. The wall is smooth and green yellow in color. Sporulation time is about 6 days. A residual body is present.

Eimeria ildefonsoi Torres and Ramos, 1939

Oocysts measure 31 to 54 microns long and 22 to 34 microns wide They are egg shaped The wall is smooth and brown in color. A micropyle is present Sporulation occurs between 36 to 72 hours. A residual body is also present

Eimeria bukidnonensis Tubangui, 1931

Oocysts are about 33 to 41 microns long and 24 to 28 microns in diameter They are distinctly pyriform with little variation in shape. The wall is about 2 microns thick and is greater than the other species of coccidia from cattle. The oocysts are brown to yellow-brown in color. A micropyle is present. Sporulation time ranges from 4 to 7 days.

Eimeria canadensis Bruce, 1921

SYNONYM. Eimeria zurnabadensis Yakimov, 1931.

The oocysts are 28 to 37 microns long by 20 to 27 microns wide The shape is typically ellipsoidal, varying from nearly cylindrical to stoutly ellipsoidal. The micropyle is inconspicuous. Oocysts are brown to pale brown in color. Sporulation requires about 72 to 96 hours.

Eimeria cylindrica Wilson, 1931

Oocysts of this species measure 16 to 27 microns long by 12 to 15 microns in diameter. The shape is typically cylindrical but may vary from ellipsoidal to narrow cylinders. The micropyle is imperceptible. The oocysts appear colorless to slightly tinted. Sporulation time is about 48 hours.

Eimeria ellipsoidalis Becker and Frye, 1929

Oocysts are from 12 to 27 microns long by 10 to 18 microns wide. The shape is predominantly ellipsoidal with some spherical to subspherical forms. Micropyle imperceptible. Under low power the oocysts are colorless but under oil immersion the wall has a pale lavender to yellowish tint. Sporulation requires from 48 to 72 hours.

Eimeria subspherica Christensen, 1941

The small oocysts of this species measures 9 to 13 microns long by 8 to 12 microns in diameter. The shape is typically subspherical varying from spherical to bluntly ellipsoidal. No micropyle is visible. The oocysts appear colorless to a faint yellow tint and fragile. The time of sporulation is approximately 96 to 120 hours.

Eimeria wyomingensis Huizinga and Winger, 1942

The oocysts range from 37 to 45 microns long by 26 to 30 microns in width. The shape is typically ovoidal to elongated ovoidal but occasionally slightly pyriform. The wall is 3 microns thick with a yellowish-brown to greenish-brown color. Micropyle is conspicuous. The sporulation time is 120 to 168 hours.

Eimeria zurnii (Rivolta, 1878)

The oocysts measure from 15 to 22 microns in length by 13 to 18 microns in transdiameter The shape is spherical to bluntly ellipsoidal The micropyle is not visible. The oocysts appear colorless, under oil immersion they have a faint grayish-lavender tint. Sporulation time is about 48 to 72 hours

Bovine coccidiosis was first reported in North America by Smith in (1893). Since that time many outbreaks of bovine coccidiosis has been recorded in the literature. The twelve different species differ widely in their ability to produce clinical coccidiosis. The relative pathogenicity, localization in the tissues and the various intracellular stages in the epithelial cells of the bovine has not been studied on a comparative basis.

Wilson (1931), Becker (1934) and Christensen (1941) summarized in detail the information on the coccidia from domestic cattle As time goes on the veterinarian must learn to recognize and diagnose this disease as its presence has been overlooked although it is of almost universal distribution in the United States. The three species most frequently involved in clinical coccidiosis are E. zurnii, E. bovis and E. ellipsoidalis.

LIFE CYCLE The life cycle of a typical coccidium from cattle involved only one host. Oocysts after leaving the cow by way of the feces sporulate under favorable conditions to the infective stage (sporulated oocyst) from 2 to 7 days). The sporulated oocyst contains four sporocysts which contains 2 sporozoites each for a total of 8. The sporulated oocyst is ingested with food or water The sporozoites are liberated and each enters an epithelial cell of the intestinal mucosa Each sporozoite in turn becomes a trophozoite and finally enlarges to a spherical schizont which fills the cell. The nucleus of the schizont divides continually and each daughter nucleus forms a small merozoite. When the epithelial cells rupture the motile merozoites enter new epithelial cells and the asexual cycle is repeated several times. This process is known as schizogony.

After schizogony occurs there appears a generation of merozoites which develop into male and female gametocytes to commance the sexual phase of the cycle In the epithelial cells the male gametocyte divides into a number of microgametes which are liberated. These penetrate the micropyle of the large oval gamete which has developed from the female gametocyte (macrogametocytes). The resulting zygote produced by this fission secretes a wall and becomes an oocyst. These oocysts are passed out in the feces where the nucleus of the oocysts divides into daughter nuclei which form sporocysts and are called sporulated oocysts. This infective stage is ingested by the cow to complete the cycle.

According to Hammond et al (1946) the asexual stage of E. bovis differs from the usual Eimeria by its location in the center of a villus, in its large size, the number of merozoites produces by a single schizont and in the occurrence of a single generation of schizonts. The sexual stages are limited to the cecum and colon.

PATHOLOGY: The damage done by coccidiosis in cattle results directly or indirectly from the tremendous multiplication of coccidia in the epithelial linings of the small intestine, cecum, colon or rectum according to Boughton (1942). Gross lesions include:

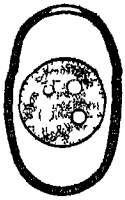
# PLATE VIII

## COCCIDIA FROM CATTLE

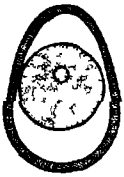
Eimeria subspherica

Eimeria zurnii

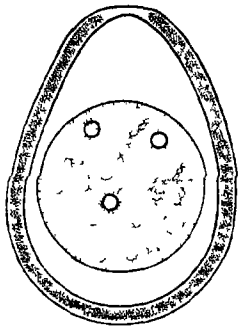
Eimeria ellipsoidalis



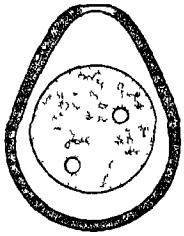
Eimeria cylindrica



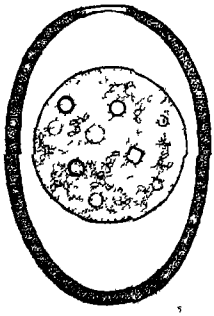
Eimeria alabamensis



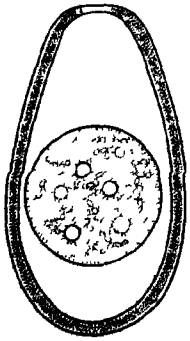
Eimeria bukidnonensis



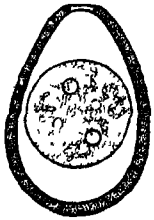
Eimeria bovis



Eimeria canadensis



Eimeria auburnensis



Eimeria wyomingensis

(1) loss of surface epithelium, (2) thickening of the mucous membrane, (3) hemorrhages, which may be petechial or diffuse and (4) catarrhal enteritis. Destruction of the intestinal glands may also occur.

SYMPTOMS: The main symptoms associated with bovine coccidiosis are bloody diarrhea (stringy masses of mucus and clotted blood), anemia, weakness and emaciation. Severe straining accompanies defecation. Secondary bacterial infections may complicate the picture. Pneumonia often develops. If an animal does not die within a week or 10 days it may be expected to recover Calves often have a rather severe non-fatal coccidiosis which do not show the spectacular symptoms of bloody diarrhea.

Symptoms usually occur about 2 weeks after swallowing the sporulated oocysts First symptoms include the observation of the feces being streaked with blood. The diarrhea becomes more severe the following day with frequent spurting discharges of bloody fluid, clots of blood and feces. This may continue for 3 or 4 days. The hind quarters become soiled from the discharge. There is a definite loss of appetite

TRANSMISSION. Many healthy, adult cattle harbor coccidia without showing any signs of the disease. The number of oocysts discharged may be very high. These animals are known as carriers. Carriers become immune to repeated attacks although they become a constant and continual source of oocysts.

Bovine coccidiosis is transmitted by means of infected oocysts which must be of bovine origin. These oocysts are quite resistant. Since calves are most susceptible they become infected by overcrowding and eating or drinking fecal contaminated feed and water.

DIAGNOSIS *Diagnosis depends upon finding the characteristic oocysts in the feces* by microscopic examination Identification can best be made by observations on freshly discharged oocysts The asexual stage of E bovis has relatively large schizonts which are visible to the naked eye These are most abundant in the small intestine where they are embedded in the villi just under the intact epithelium .According to Boughton (1942) this macroscopic lesion is of value in the diagnosis of bovine coccidiosis caused by E bovis

Oocysts may not be present in the feces at the beginning of an attack Two or three days later the organisms may be present in large numbers

CONTROL· The control and prevention of clinical bovine coccidiosis are based on the knowledge that immune animals (coccidiosis carriers) are universally present and that continual excessive exposure to coccidia is particularly dangerous to young stock. Sanitary precautions and management practice must be designed to prevent the young animals from swallowing large numbers of infective sporulated oocysts

Young calves should never be introduced directly into a group of calves at various ages if they are confined to close quarters According to Boughton (1942) calves are bound to pick up a few stray oocysts and develop non-clinical infections These animals will pass large numbers of oocysts. Other calves will ingest more oocysts and clinical cases may occur

Daily removal of manure and contaminated bedding is good practice and important. Isolation of young stock into individual stalls is helpful. Feed should not be allowed to come in contact with feces.

If it is impossible to isolate animals or keep the pens cleaned daily, segregation of calves by age into separate pens according to age groups will help. The age groups to keep in mind are calves under 3 weeks old, 3 to 6 weeks, 6 weeks to 3 months and 3 months or older. Sanitary measures should be practised at all times.

Animals fed outside on wet ground around feed sheds, watering troughs or haystacks are also a problem. All puddles, wet spots should be filled and properly drained. Manure should be removed often. Rotation of feeding places is of value.

Oocysts are highly resistant and can remain infective in fecal material for several months.

**TREATMENT:** Many drugs have been used for the treatment of bovine coccidiosis. Mild cases usually recover without treatment. Severe cases may end fatally. No specific treatments are known. The usual course is to give protective materials such as, mineral oil and milk containing astringents or intestinal antiseptics. Claims for cures of coccidiosis have been usually the result of spontaneous recovery under good sanitary conditions. Boughton (1943) reported favorable results with sulfaguanidine in the case of E. bovis infections. The drug was given daily at the rate of 0.1 gram per kilogram of body weight for a three week period beginning two days after inoculation or in 5 gram doses for an 8 day period beginning 13 days after inoculation.

**GENERAL REMARKS:** At one time it was believed that coccidia were non-specific in so far as the host was concerned. Experimental work has shown that a high degree of host specificity exists with the various species. Animals acquiring coccidiosis usually develop some degree of immunity

## ORDER: HAEMOSPORIDIA
### Family. Babesiidae

### Babesia bigemina (Smith and Kilborne, 1893)

**SYNONYM:** Piroplasma bigeminum.

**DISEASE:** Piroplasmosis, babesiasis, babesiosis

**COMMON NAMES:** Cattle tick fever, tick fever, bovine malaria, red water, black water, southern cattle fever, splenic or splenetic fever, hemaglobinuria, Texas fever

**HISTORY:** Cattle tick fever is a specific infectious disease of the blood of cattle caused by Babesia bigemina which are transmitted to the animals by the cattle tick (Boophilus annulatus).

It is not known when or where cattle tick fever made its first appearance in the United States. It was probably introduced into Mexico during the Spanish colonization. The disease was noted in the United States about 1800 When southern cattle were associated with native cattle the disease was spread It was also noted this disease occurred in the summer and subsided in the fall after the first frost. Enormous losses would occur when cattle from Texas were driven to other states. The disease became so widely spread that the Federal Government established control measures on all areas where the disease was known to exist.

In 1883 a survey was made to locate the northern limits of the disease. In July 1889 the first national quarantine order was established. This was done while Dr. Salmon was chief of the Bureau of Animal Industry.

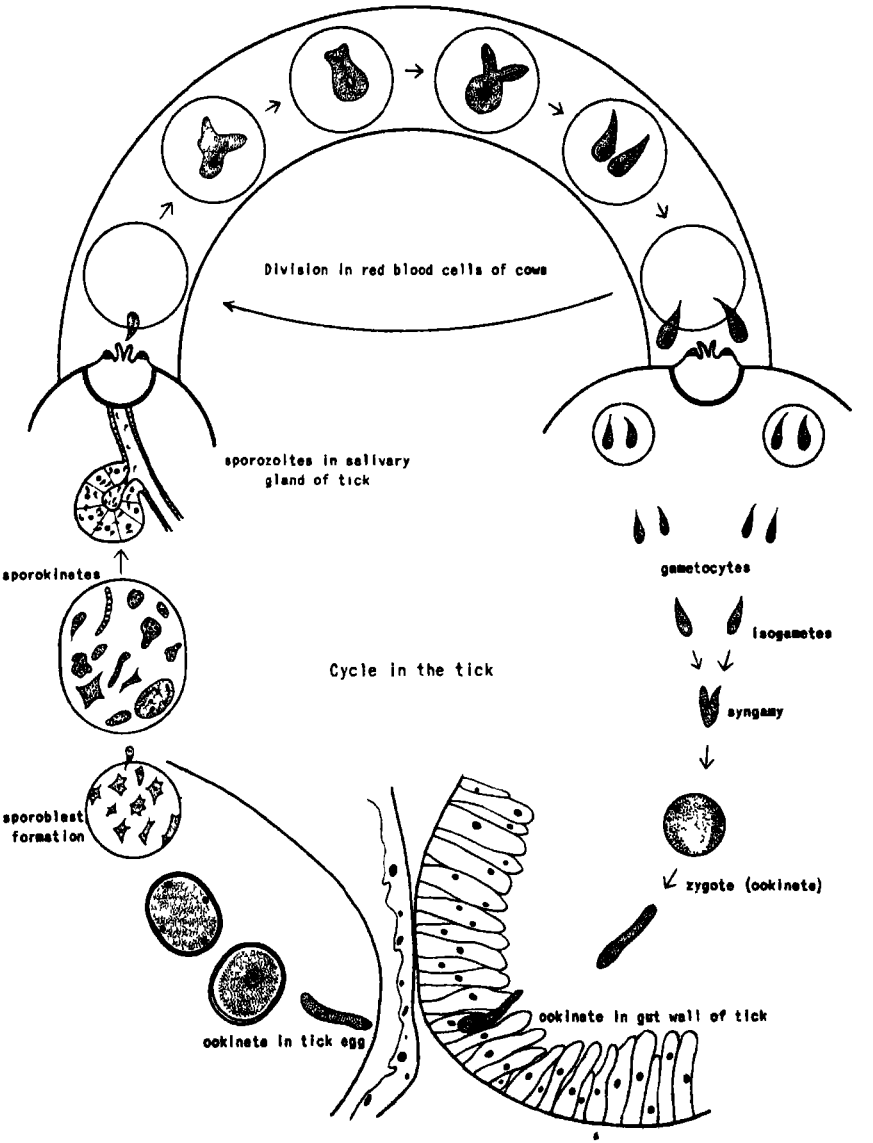
During this time investigators in the Bureau of Animal Industry were working on the etiology of the disease. Smith and Kilborne (1893) found that cattle tick infestation was necessary for the transmission of the organism. This was the first time proof was evident that an arthropod could transmit protozoan diseases.

**MORPHOLOGY:** The organisms are located in the red blood cells of cattle. They may assume various shapes but their most characteristic form is round, oval to pear-shaped. The pear-shaped parasites are usually found singly or in pairs. These organisms measure from 2 to 4 microns long and 2 microns wide.

**LIFE CYCLE:** According to Dennis (1932) sexual reproduction followed by sporozoite formation occurs in the tick (Boophilus annulatus). When the tick feeds on an infected cow, the blood parasites in the lumen of the tick gut form isogametes which are 5 to 6 microns

PLATE IX

LIFE CYCLE OF BABESIA BIGEMINA



long. Through further multiplation (isogamy) motile club shaped ookinetes 7 to 12 microns long are formed. These pass through the gut wall of the tick and penetrates the reproductive organs. The ova of the tick are invaded by the ookinetes which round up and develop into sporonts about 7 to 12 microns in diameter. These grow into multinucleated (4 to 32 nuclei) forms known as sporokinetes and are up to 15 microns long. The sporokinetes migrate throughout the tissues of the developing tick. Many of these forms develop in the salivary gland cells. Sporokinetes develop into sporozites before or just after the ticks hatch out. Cattle become infected by the bit of infected ticks. The organisms pass from the ticks into the blood stream of susceptible cows. The disease can be transmitted by dehorning or by injections of infected blood into susceptible animals.

LIFE CYCLE OF THE TICK: The female ticks when fully engorged with blood drops to the ground and lays eggs. She is capable of laying over 5000 eggs. After egg laying the female tick dies. The eggs incubate after a time as short as 18 to 20 days in the summer or as long as 200 days if they are laid in the fall or winter seasons. The eggs hatch out into six legged larvae called seed ticks. After a few days the young ticks climb on plants and vegetation. They attach to cattle as they brush against the plants. The parasite dies of starvation if unable to find its host.

The tick crawls over the cow to the area of the dewlap or inside the thighs and flanks. After two weeks the ticks molt into the nymph form with eight legs. Later they molt again and become sexually mature. At this stage the males and females can be identified. The male is brightly colored and is smaller than the female. After mating the female sucks blood, engorges and falls to the ground to lay eggs and completes the cycle.

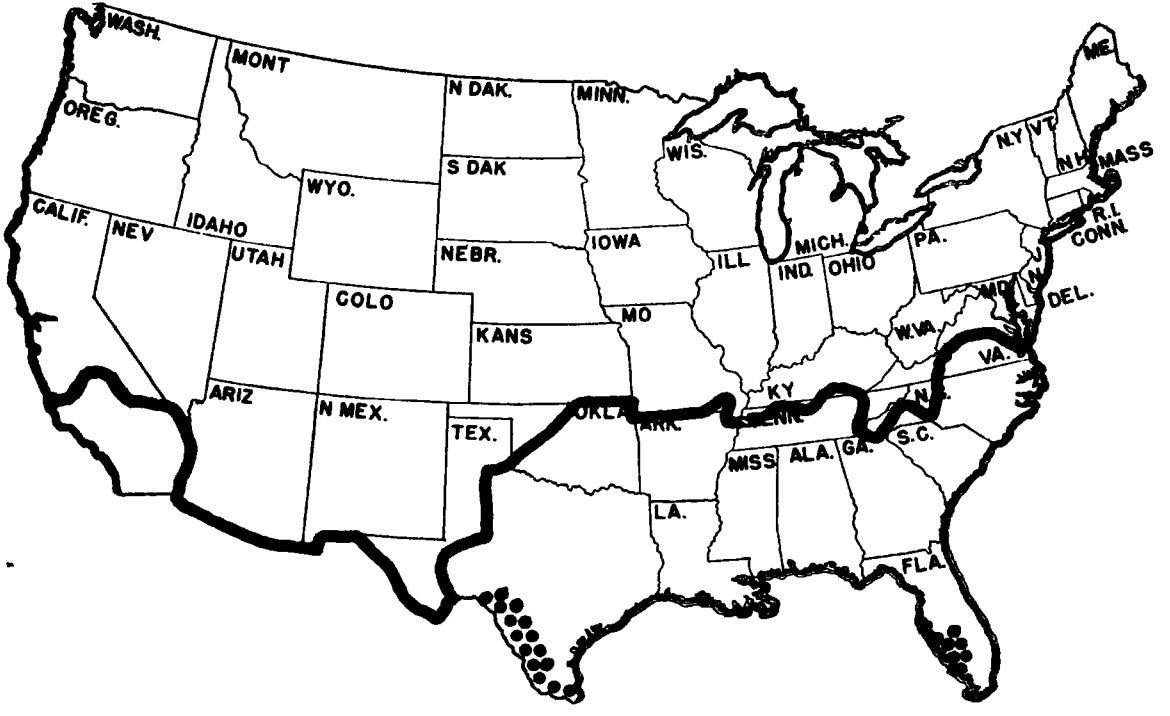
SYMPTOMS: Tick fever is primarily a disease of mature animals, cattle under one year of age rarely show symptoms. The incubation period varies from one to two weeks. In acute cases the onset is sudden with the animal showing depression, lack of appetite and a temperature of 105° to 107° F. Fever may continue for two or three days. The mucous membranes are icteric, constipation is usually followed by diarrhea. Hemoglobinuria is usually present, the urine varying from light red to black depending on the number of red cells destroyed. Blood clots slowly and is light colored, the cell count may drop to 1 or 2 million. Death occurs in about one week, with a mortality rate of about 90 per cent. From two to six weeks after recovery from an acute attack, a relapse may occur in a mild chronic form.

The symptoms of the chronic form is similar to the acute type but usually there is no hemoglobinuria. The course is irregular, the temperature is 103° to 104° F. The mortality rate is low, but the disease may extend over weeks or months.

PATHOLOGY: On post-mortem examination the spleen is found to be enlarged from two to four times normal size. The blood is thin and watery like. The liver is enlarged with a yellowish mahogany-brown appearance with some fatty degeneration. Hemorrhages are frequent on the walls of the heart, subcutaneous tissue and the mucosa of the urinary bladder.

CONTROL: The control and eradication of this disease is by dipping the cattle to kill the ticks. This is fully explained by Kilenberger and Chapin (1940).

There are two varieties of cattle fever ticks in the United States which can spread tick fever, the North American fever tick Boophilus annulatus which formerly infected the majority of the quarantined areas and the tropical variety of the tick, Boophilus annulatus var. microplus found in Florida and the gulf coast of Texas. Due to the tick eradication campaign by the Federal Government which started in 1906, the cattle fever tick and tick fever has been practically eliminated from the United States. Only a small portion of southern Florida and southeastern Texas were considered tick infested areas in 1941. The disease has been practically cleaned out although over 14 states were considered infested areas at one time. Pasture rotation for 8 to 10 months in which all animals are removed from an infested area will cause the ticks to die of starvation.




MAP 2. Black line indicates the northern boundary of Boophilus annulatus (cattle tick) at the beginning of the tick eradication program in 1906. The dotted areas in Texas and Florida are the present quarantined areas. (From Yearbook of Agriculture, 1942.)

**TREATMENT:** Therapeutic agents were not used in the United States for the treatment of this disease. The injection of trypan blue intravenously in a dose of 100 cc of a one per cent solution in physiological saline has been recommended. Acaprin (Bayer) is also effective. It is administered subcutaneously. Trypaflavine (acriflavine (0.5 to 1 gram)) 1:1000 per intravenous dose has been reported to have a curative action on cattle fever.

Artificially, immunity can be established by the inoculation of young cattle with infected blood which produces a mild case of the disease. This is not successful in animals over one year of age.

**GENERAL REMARKS:** The small endemic foci of this disease in the United States apparently is kept alive by deer reservoir hosts of the tick. There is always a potential danger of this disease re-entering the Southern United States from Mexico.

## ORGANISMS OF UNCERTAIN ZOOLOGICAL CLASSIFICATION

### Anaplasma marginale Theiler, 1910

**DISEASE:** Anaplasmosis.

**HISTORY:** This disease has been reported from many countries. Anaplasmosis has been known and studied in the United States for over twenty years. It is believed that the disease was introduced into this country by carrier animals from the Tropics. Smith and Kilborne (1893) first described this parasite as an observation with their study of Texas fever. The disease has gained a foothold in this country and has been reported from 21 states: Alabama, Georgia, Idaho, Illinois, Iowa, Kansas, Louisiana, Maryland, Mississippi, Missouri, Montana, Nevada, New Mexico, North Carolina, Ohio, Oklahoma, Oregon, South Carolina, Texas, Virginia and Wyoming.

**MORPHOLOGY:** The so-called anaplasma body is a spherical granule which stains bright red with Wright's stain. These granules occur on the margins or red blood cells, usually about 2 or 3 per cent. On rare cases 6 or more parasites may occupy a cell. There has been much discussion as to the nature of these structures

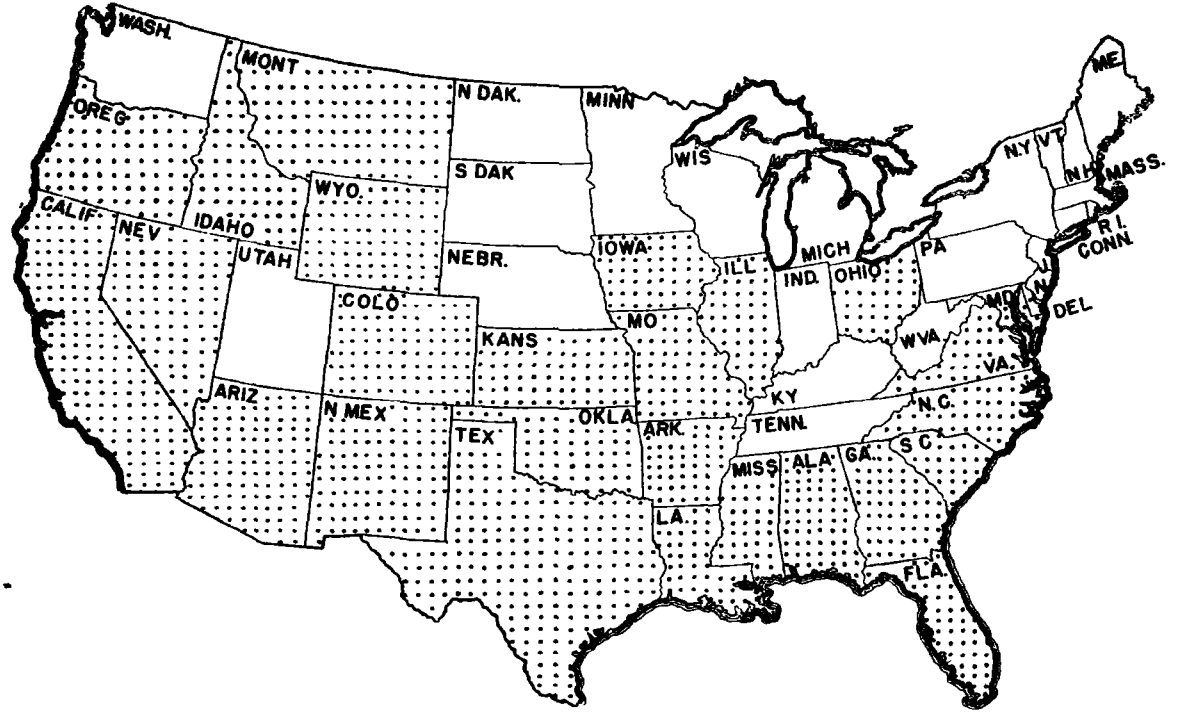
Two types of anaplasma can be recognized, those which are spherical in outline with smooth contours and measure 0.2 to 0.5 microns on diameter (smooth) and those which are roughly spherical, ovoidal, or cuboidal and measure about 0.6 to 0.9 microns (rough). Jolly bodies and basophilic stippling can be distinguished from the anaplasma by their staining reaction.

These granules vary in size from 0.1 to 0.8 microns. Sometimes two or three of these bodies occur close together to give a dumb-bell appearance. Attempts to cultivate the parasite and to reproduce the disease from cultures inoculated into cows have failed.

During the height of infection more than 50 per cent of the red blood cells may be infected. If the animal recovers the number of cells containing the marginal bodies decreases to a point where they cannot be found on microscopical examination. The blood of recovered cows is infectious for the life of the animal and they become carriers. The infection may be transmitted from the dam to unborn calf which in turn becomes a carrier.

**LIFE CYCLE:** The life cycle of this anaplasmata is unknown. The method of multiplication is not understood nor whether these bodies are actually protozoa.

Multiplication of these bodies has been thought to take place by simple binary fission. Lotze and Yiengst (1942) and Lotze (1946) believed that each anaplasma divides in some manner during the process of multiple fission to form 8 small spherical bodies. Lotze and Yiengst (1942) also presented an excellent summary of the morphology of this organism. They concluded that the anaplasma is a parasite which invades the red blood cell, eventually undergoes multiple division to form 8 small spherical bodies in each




MAP 3. Anaplasmosis in the United States. Shaded areas indicate states which have reported the disease.

anaplasma. The disappearance of the large irregular shaped bodies from the blood stream suggest that they are in some way responsible for the erythrocytes which are harboring the parasites. The infected red blood cells apparently do not remain in the blood stream for more than 3 or 4 days.

The occurrence of extra erythrocyte bodies during the anaplasma phase and the attachment of this structure to mature red blood cells apparently is part of the life cycle during the stage when the organism invades the cell.

**TRANSMISSION:** Since this disease resembles tick fever in many respects investigators have studied various insects and ticks with regard to its transmission. Several investigators have demonstrated that many types of insects and ticks are capable of spreading this disease. According to Stiles (1942, 1946) the following arthropod vectors have been found to transmit anaplasmosis: Argas persicus (chicken tick), Boophilus annulatus (cattle fever tick), B. decoloratus (Blue fever tick), B. microplus, Dermacentor albipictus (moose tick), D. andersoni (Rocky mountain spotted fever tick), D. occidentalis (Pacific coast tick), D. variabilis (common wood or dog tick), Hyalomma lusitanicum, H. algytium, Ixodes ricinus (castor-bean tick), I. scapularis (common shoulder tick), Ornithodorus lahorensis, Rhipicephalus appendiculatus, R. bursa, R. evertsi, R. sanguineus (brown dog tick), R simus (black-pitted tick). Several species of horseflies can also transmit the disease. Tabanus sulcifrons, T. abactor, T. venustus, T equalis, T. erythraeus, T. americanus, T. oklahomensis, T. fumipennes. Stomoxys calcitrans (stable fly) can also transmit the disease. Certain mosquitoes are also capable of transmitting anaplasmosis, Psorophora columbiae, P. ciliata and Aedes aegypti

In the transmission by horseflies Lotze and Yiengst (1942) showed that Tabanus sulcifrons after feeding on an infected bull, transmitted anaplasmosis to 3 normal cows. They became infected within 24 to 28 days. Howell et al (1941) showed conclusively that mosquitoes transmitted the disease. Only a few bites of these arthropods are required if the infecting animal is in the acute stage of the disease. Sanborn et al (1938) showed that the disease was transmitted through both female and male ticks and later Howell et al (1944) reported hereditary transmission.

An important means of transmission is through the use of contaminated surgical instruments which have not been disinfected. Outbreaks have been reported in herds after dehorning operations, bleeding of animals, castration, slitting of ears or other surgical operations. Rees (1930) demonstrated that contaminated hypodermic needles can transmit anaplasmosis. The disease can be transmitted by 0.025 cc of infected blood. Moe et al (1940) transmitted the disease by tipping (removing a few inches of the horn) the horns of cattle. In areas where anaplasmosis is known to exist, veterinarians should be extremely careful to disinfect their instruments. Hilts (1928) noted anaplasmosis in cattle following dehorning.

**SYMPTOMS:** In the early stages of acute anaplasmosis a fever of 103° to 107° F usually occurs. As the disease progresses the fever may become normal or subnormal before the animal dies Breathing is accelerated and heavy. Exhaustion, lack of rumination, loss of appetite are other general symptoms. The skin and all visible membranes become yellow and anemic. Depraved appetite occurs. Animals may walk with a stiff, unsteady gait.

Urination is frequent but the urine is normal in color. Occasionally the animals are constipated with dark, blood colored feces covered with mucus. Muscular tremors of the neck, shoulder and flank have been reported. Enlarged glands, rough coat and edema around the eyes may occur. As a rule, young animals are quite resistant. In acute cases death may occur within 24 hours after the first symptoms. The average fatal case lingers about 2 or 3 days. In the chronic cases the animals live longer, show anemia with the red cell count less than 1 million per cmm. Hemoglobin may be less than 10 per cent. Recovery takes place very slowly. Convalescence requires many days or weeks before the animal is back to normal. In late pregnancy the animal may abort.

The mortality rate varies widely from 30 to 50 per cent of the animals infected. Animals which recover from anaplasmosis remain carriers for life and are resistant to subsequent infections.

PATHOLOGY: On post-mortem, the lymph glands are slightly enlarged and edematous. The heart is enlarged and covered with petechial hemorrhages. The blood is thin, watery, clots in the large blood vessels appear as long ropey masses. The lungs are anemic with some emphysema. The liver is saturated with bile and enlarged. The spleen is enlarged with soft pulp. The entire vicera shows some yellow discoloration

DIAGNOSIS: Diagnosis depends upon finding the marginal bodies in the red blood cells upon microscopic examination. As the disease progresses various cellular changes associated with anemia occur such as polychromatophilia or basophilia. Microscopical examination of carrier cows is of no practical value. Boynton and Woods (1935) reported a non-specific test which appears to have some value in the diagnosis of anaplasmosis. Blood is drawn and allowed to clot. A small amount of clear serum is obtained Two drops are added to 2 cc of distilled water The sera of normal animals does not become cloudy in water whereas animals infected with anaplasmosis the sera becomes cloudy immediately. After standing overnight a white precipitate deposits on the bottom of the tube The test depends upon the precipitation of euglobin.

TREATMENT: Sick animals should be kept in the shade and given plenty of green feed and water. Animals should be handled gently If the animals are constipated a mild saline purge is indicated. Boynton et al (1937) recommended an intravenous injection of 1000 cc of a 5 per cent dextrose in distilled water to which has been added enough sodium cacodylate to make a dosage of 30 grains of cacodylate per 100 pounds of animal weight This drug stimulates red blood cell formation

So far as known, no chemical compound has been effective in destroying the parasite. However, any medicinal treatment which investigators have shown to be of some value in aiding recovery should be tried According to Koger (1941) large quantities of citrated blood (2000 to 4000 cc) from healthy cows injected intravenously appeared to reduce mortality Observations have been noted that animals which drink water throughout the course of the disease usually recover

CONTROL. To prevent the spread by mechanical means, all instruments should be sterile before use A sufficient number of bleeding needles should be provided Used needles or surgical instruments can be washed in cold water to remove the blood and sterilized by boiling several minutes in water containing 2 per cent washing soda Other disinfectants which can be used are 2 per cent solution of cresol, formalin or 70 per cent alcohol. Segregation of known carriers from normal animals is good practice Control or eradication of ticks and other arthropod vectors by spraying and dipping should be done.

GENERAL REMARKS This disease of cattle is not transmissible to man Many animals such as elk, sheep, goat, deer, antelope and camel are susceptible to anaplasmosis. Since the malady is primarily spread by arthropod vectors the disease is usually reported in the summer and fall

## Bartonella bovis Donatien and Lestoquard, 1934

SYNONYM. B sergenti Adler and Ellenbogen, 1934.

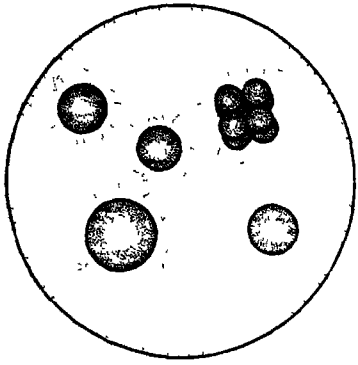
DISEASE: Bartonellosis.

Lotze and Yiengst (1942) reported the presence of "Bartonella-like" structures in the red blood cells of American cattle infected with anaplasmosis. In a later report Lotze and Bowman (1942) noted these structures in cattle free from Anaplasma marginale. The height of the Bartonella infection occurred in cattle 2 to 4 days before anaplasmas were first observed. In some cases Anaplasma and Bartonella were found in the same erythrocyte. Bartonella was detected in an anaplasmosis-free calf soon after splenectomy. The importance of this parasite in cattle is not clearly understood. Under certain conditions this organism may produce some pathology. The parasite resemble the bacilliform and coccoiform structures found in Bartonella of rats.

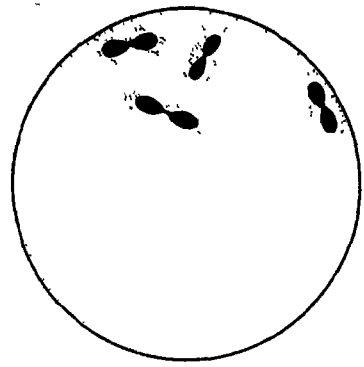
TREATMENT: Unknown.

PLATE X

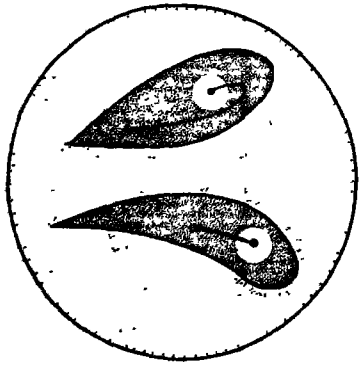
## BLOOD PROTOZOA FROM CATTLE



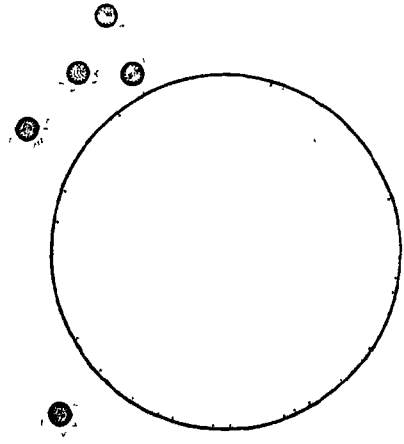
Anaplasma marginale



Bartonella bovis



Babesia bigemina



Eperythrozoon wenyoni

Eperythrozoon wenyoni Adler and Ellenbogen, 1934

DISEASE: Eperythrozoonosis.

Lotze and Yiengst (1941) first reported Eperythrozoon wenyoni in the United States from the blood of a cow experimentally infected with Anaplasma marginale. It appears that anaplasmosis and eperythrozoonosis are possibly two distinct types of infections. The organisms stained with Giemsa are pinkish purple in color, and about 0.3 to 1.5 microns in diameter. They are loosely attached to the red blood cells. A typical parasite is shaped like a delicate ring, but various forms are recorded including ovoid, comma, rod, dumb-bell forms Multiplication apparently takes place by budding or fission This disease may be of importance if in association with other blood diseases although it may be of little importance when occurring alone More information is required to show whether these structures are concerned in producing a specific disease.

TREATMENT: Unnecessary

Sarcocystis sp.

This parasite has been reported sporadically from cattle Since the excellent work of Spindler (1946, 1947) investigation of Sarcocystis in swine; the true classification of the parasite is uncertain. New evidence brought to light indicates this parasite may be a fungus.

For more details see Chapter V on the protozoa of swine. The parasite apparently does not cause any serious trouble in cattle. Wilson and McDonald (1938) examined the heart muscle of 35 cows Cysts were found in 30 These cysts were 74 to 252 microns long and in sections cut 4 microns thick there was a range of 3 to 18 cysts to 1/8 square inch of tissue No cysts were found in the heart muscle of 29 calves 6 to 8 weeks old. The writer (B.B M.) observed a cow condemned by meat inspectors in a local packing plant because of this parasite

TREATMENT: Unnecessary.

## CLASS: CILIATA

About 30 or more species of ciliates from the rumen of cattle have been recorded from North American cattle Becker and Talbott (1927) in an excellent monograph of the protozoan fauna of the rumen and reticulum of American cattle gave a good review of these ciliates Further work on the protozoa of the intestine of Mexican cattle was conducted by Chavarria (1933) Species recorded from North American cattle are listed below.

### ORDER: HOLOTRICHA

Family. Isotrichidae

1. Isotricha prostomata Stein, 1859.
2. I. intestinalis Stein, 1859
3. Dasytricha ruminantium Schulberg, 1888

Family: Butschliidae

4. Butschlia parva Schulberg, 1888.
5. B. neglecta Schulberg, 1888.
6. B. lanceolata Fiorentini, 1890.

ORDER: SPIROTRICHA
Family: Ophryoscolecidae

7. Entodinium bursa Stein, 1858.
8. E. minimum Schulberg, 1888.
9. E. caudatum Stein, 1858.
10. E. bicarinatum Cunha, 1914.
11. E. furca Cunha, 1914.
12. E rostratum Fiorentini, 1889.
13. E. dentatum Stein, 1859.
14. Diplodinium magii Fiorentini, 1889.
15. D. bursa Fiorentini, 1899.
16. D. medium (Awerinzew and Mutafowa, 1914).
17. D. ecaudatum var. ecaudatum Fiorentini, 1889.
18. D. ecaudatum var. bicaudatum Sharp, 1914.
19. D. ecaudatum var. tricaudatum Sharp, 1914.
20. D. ecaudatum var. quadricaudatum Sharp, 1914.
21. D. ecaudatum var. catanei Fiorentini, 1889.
22. D. eberleini Cunha, 1913.
23. D. dentatum Fiorentini, 1889.
24. D. denticulatum Fiorentini, 1889.
25. D. minimum Becker and Talbott, 1927.
26. D. clevelandi Becker and Talbott, 1927.
27. D. helseri Becker and Talbott, 1927.
28. D. neglectum Dogiel, 1927.
29. D. multivesciculatum, Dogiel, 1927.
30. Ophryoscolex inermis Stein, 1858.
31. O. purkynjei Stein, 1858.
32. O. caudatus Eberstein, 1895.
33 Elytroplastron hegneri Becker and Talbott, 1927.

The role of the protozoa in the rumen of cattle is not clearly understood. Several views have been suggested, some which merit further investigations. These organisms may aid in digestion, help digest cellulose, serve as a protein diet to the cow or act as commensals.

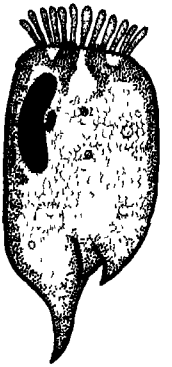
Recent interesting experiments by Hungate (1943) indicated that Diplodinium digested cellulose and could be considered a symbiont. Cellulose digestion in the rumen by protozoa and the problem of protozoan relationships in the ruminant were reviewed by Hastings (1944) and Hungate (1946).

**GENERAL REMARKS:** According to Becker and Hsiung (1929) ciliates are acquired from one animal to another by mouth contamination. To become infected through food an animal must eat food contaminated with infected saliva. Infusoria of the stomach of the cow, goat and sheep show no host-specificity within these three hosts.

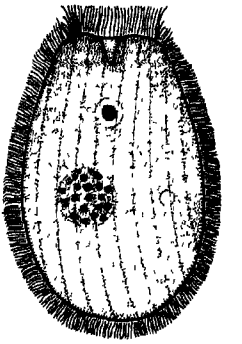
**TREATMENT:** Unnecessary.

PLATE XI

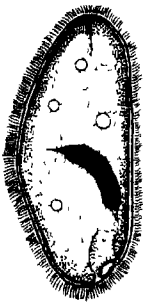
CILIATES FROM THE RUMEN OF CATTLE



Entodinium caudatum

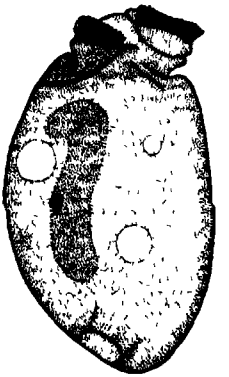


Butschlia parva

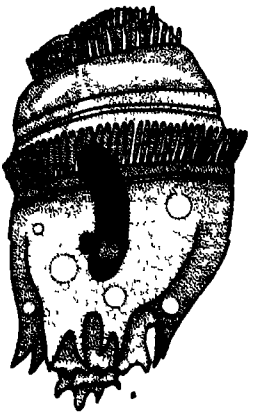


Isotricha prostoma

Entodinium minimum



Diplodinium medium



Ophryoscolex caudatus

ORDER: HOLOTRICHA
Family: Pycnothricidae

Burtonella sulcata Jameson, 1926

Becker and Hsiung (1929) described cysts and trophozoites of this organism from the feces of calves at Iowa. Rees (1930) reported this protozoan from the cecum of 8 out of 32 head of cattle slaughtered at Louisiana. This ciliate probably has no economic importance. It is about 162 microns long and 116 microns wide. The entire surface is covered with fine cilia. Cysts have been found in the feces of infected cows.

TREATMENT: Unnecessary.

## CHAPTER IV

## PROTOZOA OF SHEEP AND GOATS

## Chapter IV

## PROTOZOA OF SHEEP AND GOATS

CLASS: MASTIGOPHORA
ORDER: PROTOMONADINA
Family: Bodonidae

### Retortamonas ovis (Hegner and Schumaker, 1928)

SYNONYM: Embadomonas ovis Hegner and Schumaker, 1928.

Hegner and Schumaker (1928) described this species from trophozoites and cysts which appeared in cultures made from feces of Maryland sheep The organism is pear-shaped and averages 5.2 microns long by 3.37 microns wide There are two flagella of equal length which are approximately as long as the body. It is apparently nonpathogenic

TREATMENT: Unnecessary.

CLASS. SPOROZOA
ORDER. COCCIDIA
Family: Eimeriidae

### Ovine Coccidiosis

Approximately 9 species of Eimeria have been described from sheep and goats in North America. The morphology of the various species are listed below

### Eimeria arloingi Marotel, 1905

This species shows the greatest range in size of any of the species of coccidia in sheep. It measures 17 to 42 microns in length and 13 to 27 microns in width (average 27 microns in length and 18 microns in width) The oocyst is ellipsoidal and possesses a well developed micropyle and polar cap. However, the polar cap may be lost in some specimens. The cyst wall is characterized by the presence of a single black contour line between the oocyst membrane and the inner surface of the wall. The wall is transparent and varies in color from a faint to distinct yellowish-brown color Sporulation time is two to three days and the spores measure 13 by 6 microns

### Eimeria ah-sa-ta Honess, 1942

This species has been described from the Rocky mountain bighorn sheep (Ovis c. canadensis) and domestic sheep in Wyoming. It is difficult to differentiate from E. arloingi and it is doubtful if it is a distinct species They measure from 29.46 by 33.51 microns in length and 21.58 by 24 9 microns in width. The oocyst has a faint pink color.

### Eimeria crandallis Honess, 1942

This species has also been described from the bighorn sheep in Wyoming, although Hawkins (unpublished data) has observed a very similar species in domestic sheep in Michigan. The oocysts measure 17.5 by 23.24 microns in length and 17.5 to 21.58 microns in width. The polar cap varies from being almost imperceptible to 1.66 microns in height. The spores average 9.53 microns by 6.36 microns. The oocyst may vary from being colorless to a faint pink and the cyst wall has a greenish tinge. The oocyst wall presents a double contoured appearance. This oocyst would seem to present the same relationship to E. arloingi that E. parva bears to E. nina-kohl-yakimovi. Hawkins found the peak of infection with this oocyst in Michigan occure at a different time than does E. arloingi.

Eimeria faurei Moussu and Marotel, 1901

Oocysts measure 25 to 33 microns in length and 18 to 24 microns in width and average 28.9 microns in length and 21 microns in width. This species is shaped characteristically like a hen's egg with a distinct micropyle at the narrow end. There is no polar cap. The cyst walls are transparent and brownish yellow in color. The cyst wall is not double contoured as in E. parva. The inside of the cysts may sometimes be a delicate salmon pink or colorless. The sporulation time is 24 to 48 hours.

Eimeria granulosa Christensen, 1938

These oocysts measure 22 to 35 microns in length and 17 to 25 microns in width averaging 29.4 microns in length and 20.9 microns in width. The oocyst is urn-shaped or pyriform, with the cap on the broad end. The micropyle is 3 to 5 microns in diameter and is covered with a well developed polar cap. The cyst wall is transparent and a pale yellowish brown to brownish yellow in color. The oocyst wall is not double contoured. The sporulation time is 72 to 120 hours.

Eimeria nina-kohl-yakimovi Yakimov and Rastegaeva, 1930

Oocysts measure 20 to 28 microns in length and 15 to 22 microns in width and average 23.1 microns in length and 18.3 microns in width. The shape of the oocyst is ellipsoidal to ovoidal in shape, and usually possesses an imperceptible micropyle. No polar cap is present. The cyst wall is thin, transparent, usually colorless but sometimes a light brownish yellow. The cyst wall is not double contoured. The oocyst may sometimes appear to be a delicate salmon pink color. The sporulation time is 24 to 48 hours.

Eimeria pallida Christensen, 1938

Oocysts measure 12 to 20 microns in length and 8 to 15 microns in width and average 14.2 microns in length and 10 microns in width The shape is ellipsoidal and there is a barely perceptible micropyle. There is no polar cap. The cyst wall is not double contoured, and is a pale yellow to yellowish green in color giving the cysts a pallid appearance. The sporulation time is 24 hours.

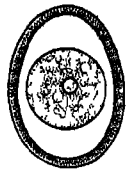
Eimeria parva Kotlan, Mocsy and Vajda, 1929

Oocysts measure 12 to 22 microns in length and 10 to 18 microns in width (average 16.5 microns in length and 14.1 microns in width) The shape is ellipsoidal to subspherical and possess no perceptible micropyle There is no polar cap The cyst wall is transparent, faintly yellow to yellowish green, and demarcated on each side by a heavy black refraction line giving it a double contoured appearance The sporulation time is 24 to 48 hours

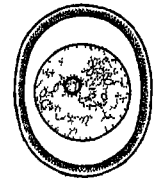
The most important species of these parasites is not known at the present time, therefore, further discussion on these parasites must of necessity be of the group as a whole, regardless of the pathogenicity of individual parasites It is of interest to note that the most common species are Eimeria arloingi, E. parva and E. faurei. This does not mean they are the most pathogenic.

LIFE CYCLE: The life histories of these parasites are unknown individually, however, it is apparent they closely resemble those of other animals.

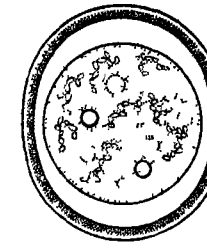
SYMPTOMS: Coccidiosis is most important as a feed lot disease, although outbreaks may occur under other conditions. The disease makes its appearance from 12 days to 3 weeks after the arrival of the lambs in the feed lot Depression, loss of appetite and scouring are the first indications of the infection. The diarrhea will continue for several days or more and results in weakness in the lambs, which will rapidly lose flesh and some will die. During this period large numbers of oocysts are present in the feces, and they may be found quite readily even without concentration. Death losses occur during the period of severe scouring and usually stop thereafter. In most lambs the scour-



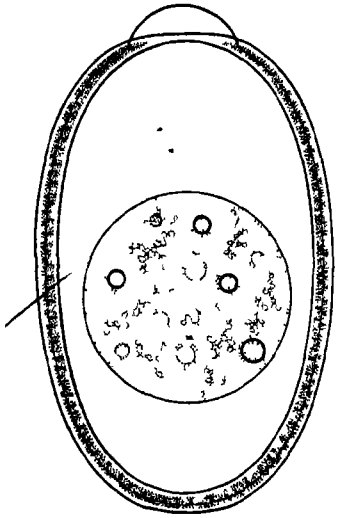
Eimeria pallida



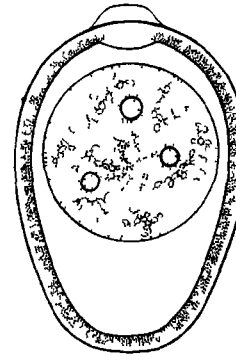
Eimeria parva



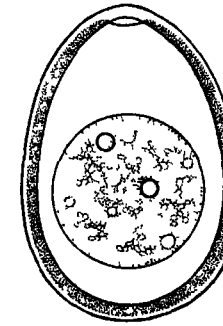
Eimeria nina-kohl-yakimovi



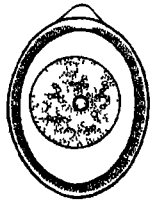
Eimeria intricata



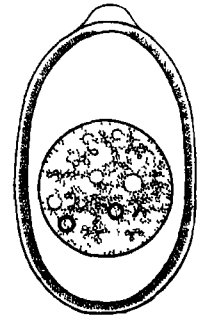
Eimeria granulosa



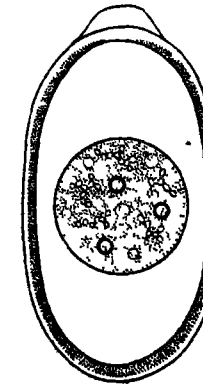
Eimeria faurei



Eimeria crandallis



Eimeria arloingi



Eimeria ah-sa-ta

ing will persist for not more than two weeks although it will last longer in a few animals. Mortality varies considerably in different outbreaks, however, death losses above 4 to 5 per cent are very uncommon

**PATHOLOGY:** Anatomical changes occur principally in the small intestine. The middle portion of the small intestine is usually the most heavily parasitized. White or yellowish white spots may be seen from the serosa or mucosa throughout the affected area. These spots vary in size from 0.5 mm. to 6.0 mm. in diameter. These areas are filled with macrogametocytes and oocysts, and the relative severity of the infection may be judged from the number of them present. A marked enteritis is present. The contents of the small intestine are semi-fluid, slimy and usually yellow or brown in color or bloody. Large numbers of oocysts may be found in scrapings In acute cases a severe enteritis is apt to be more common in the large intestine.

**DIAGNOSIS:** Diagnosis in feed lot lambs may be made from the appearance of symptoms 2 to 3 weeks after their arrival in the feed lot and by the presence of large numbers of oocysts in unconcentrated fecal samples

**TREATMENT:** As in the case of coccidiosis in other animals there is no satisfactory treatment for this disease in lambs Most sulfonamides which have been tried have little affect in controlling an outbreak once it has started. Spindler (1939) and Christensen (1940) have shown that a mixture of copper and ferric sulphate will decrease the numbers of oocysts, but it is not known whether it has any affects in a clinical outbreak. The feeding of 2 per cent sulfur in the grain mixture has proven useful in the prevention of the disease.

Christensen (1944) found that commercial flour sulfur in portions ranging from 0.5 to 3 0 per cent of the total ration effectively prevented the development of clinical coccidiosis in 800 lambs. Foster et al (1941) administered sulfaguanidine in 2 gram amounts per lamb daily for approximately one month and prevented completely the acquirement of natural coccidiosis in 5 lambs Administration of the drug in 1 gram daily doses per lamb inhibited the initial discharge of oocysts for at least 4 weeks No toxic symptoms of the drug was noted.

**CONTROL:** Coccidiosis in lambs is a disease which is brought into the feed lot with the lambs, it is not carried over in the lot from one season to the next. In normal farm outbreaks the coccidia are always present The conditions which favor the development of the disease is overcrowding, as in the feed-lot, and the presence of sufficient moisture for the oocysts to become infective. Providing a sufficiently large acreage, a light ration during the first month of feeding and controlled feeding to maintain moisture at a minimum will do much to prevent clinical outbreaks. It has been shown by Christensen (1940) that silage is an ideal material for the development of infective oocysts. After the first month in the feed lot most of the danger of coccidiosis is over, due to the development of immunity in the lambs

**GENERAL REMARKS:** Coccidiosis in the feed lot develops after two to three weeks. Studies in Colorado have shown there is a rapid increase in the number of oocysts passed by lambs in the first three weeks, whether they develop clinical symptoms or not. The numbers of oocysts drop rapidly after the third week regardless of the conditions, and there is no further danger of the disease or the number continues to increase and clinical symptoms develop. The sporulation and viability of oocysts of E. arloingi was studied in detail by Christensen (1939).

### KEY TO OOCYSTS OF EIMERIA IN SHEEP*

1. Polar cap absent ..... 2

   Polar cap present ..... 5

2. Ellipsoidal to subspherical; transparent, colorless, oocyst 12 to 22 microns long ..... 3

   Ellipsoidal or egg-shaped, transparent, delicate salmon pink to pale brownish-yellow, oocysts 20 to 33 microns long ..... 4

3. Ellipsoidal; pallid, with single dark refraction line marking inner surface of wall ..... E. pallida

   Ellipsoidal to subspherical, clear-cut, with two dark refraction lines, one on each side of wall ..... E. parva

4. Ellipsoidal, stout, micropyle inconspicuous or imperceptible ..... E. nina-kohl-yakimovi

   Egg-shaped, micropyle conspicuous at narrow end ..... E faurei

5. Wall opaque, thick, transversely striated, dark brown, oocysts 39 to 53 microns long ..... E. intricata

   Wall transparent, thin, pale yellowish-brown, oocysts 17 to 42 microns long ..... 6

6. Typically shaped like broad-shouldered urn, or pyriform, cap a soft, gelatinous flat to convex truncated cone situated upon the broad end of the oocyst, early sporont densely granular ..... E granulosa

   Typically ellipsoidal, cap a tough, firm, rounded cone or crescent, early sporont not densely granular, oocyst 27 by 18 microns ..... E arloingi

   Oocyst 31 by 23 microns ..... E. ah-sa-ta

   Oocyst 21 by 18 microns ..... E. crandallis

### CLASS CILIATA

The same species of ciliates which occur in the rumen of cattle also occur in sheep. The exact role of these ciliates is not known, but they are probably harmless commensals. Certain species may aid digestion by breaking down cellulose. Further research is necessary on this point. The species are listed in Chapter III in connection with the protozoa of cattle.

**TREATMENT:** Unnecessary.

*Adapted from Christensen (1938)

ORDER: SPIROTRICHA
Family: Bursariidae

Balantidium sp.

Hegner (1924) reported finding Balantidium sp. in the intestines of Maryland sheep. The vegetative stage measured 45 microns long and 33 microns wide (average). This form is smaller than the species found in man or pigs but has a similar morphology. A funnel-shaped peristome is located at one side near the anterior end; the excretory pore is posterior. The macronucleus is large and kidney-beaned shaped while the micronucleus is small and adjacent to the macronucleus. This ciliate is probably not pathogenic in sheep.

TREATMENT: Unnecessary.

ORGANISMS OF UNCERTAIN ZOOLOGICAL CLASSIFICATION

Globidium gilruthi (Chatton, 1910)

This parasite was reported from sheep for the first time in the United States by Marsh and Tunnicliff (1941). The organism was associated with enteritis and severe diarrhea. G. gilruthi is usually found in the walls of the abomasum and intestines.

The parasite appears as spherical cysts from 200 to 500 microns in diameter. The cyst has a rather thick wall and contains numerous sickle-shaped spores approximately 1.5 by 10 microns. The cysts can be observed with the naked eye as minute, opalescent nodules under the mucosa. The cysts rupture into the lumen of the intestine and causes hemorrhages. In heavy infections, symptoms may be produced, such as severe diarrhea, bloody feces, loss of appetite and emaciation.

LIFE CYCLE: Unknown

DIAGNOSIS: Diagnosis can only be made on post-mortem observations

TREATMENT. Unknown.

CONTROL: Unknown.

GENERAL REMARKS: This parasite is closely related to the sarcosporidia. It may be a fungus. The organism has been reported only from Montana and Wyoming.

Toxoplasma sp

Only one case of toxoplasmosis has been reported in sheep Olafson and Monlux (1942) reported on a case of toxoplasma encephalomyelitis in a sheep from New York The animal showed nervous symptoms for several days. There was marked dyspnea with some nasal discharge.

At autopsy no gross lesions were present. Examination of the brain showed encephalomyelitis with slight meningitis. The cervical and thoracic regions of the spinal cord showed severe lesions. These consisted of marked monocytic perivascular infiltration. Cyst-like structures with typical morphology of Toxoplasma were present in the inflamed areas. Occasionally the elongated organisms were arranged in a row at the periphery of the cyst with the long axis of the parasites pointing toward the center. The parasites appear as round, oval or elongated forms. They measure from 1.5 to 2.5 microns in width and from 2.5 to 3.5 microns in length. A single, eccentrically placed oval nucleus is about 1/4 to 1/3 the size of the parasite. The cytoplasm contains a few granules.

LIFE CYCLE: Unknown.

DIAGNOSIS: Diagnosis can be made only upon post-mortem examination and histological sections of the tissues.

TREATMENT: Unknown.

CONTROL: Unknown.

GENERAL REMARKS: Much more needs to be learned about this parasite in domestic animals. The disease is apparently fatal in animals.

Eperythrozoon sp.

This blood parasite has been reported on one occasion from sheep in Louisiana. It apparently has no veterinary importance in North America. E wenyoni in cattle has been discussed in Chapter III.

TREATMENT: Unknown.

Sarcocystis tenella Railliet, 1886

With the publications of Spindler et al (1945, 1946, 1947) the classification of Sarcosporidia is uncertain as new evidence now indicates that Sarcocystis may be a fungus.

The organisms invades the striated muscles of sheep including the skeletal muscles, diaphragm and heart. They form Miescher's tubes, slender, elongated sacs which are found between the muscle fibers. This tube is lined with trabeculae and contains many spores. These spores are somewhat crescent shaped and range from 5 to 15 microns in length. When these tubules are dissected out and ruptured the spores become motile.

Two recent publications by Scott (1943) has summarized the world literature on S. tenella in sheep In Wyoming the infection may be present in 50 to 100 per cent of the animals. Since the infection is of a generalized nature the effects are somewhat insidious and the condition is not readily diagnosed. Scott (1943) has seen lambs less than one year of age with 5,387 sarcocysts per com. and in good condition. He is of the opinion that sheep picking up the infection year after year will finally die because of muscle destruction, emaciation and anemia

For more details of the life cycle of Sarcocystis the reader is referred to Chapter V concerning the protozoa of swine Spindler et al (1945, 1946, 1947) studied the life cycle in pigs. This work awaits further confirmation in other animals.

TREATMENT· Unknown and probably unnecessary.

Addendum on Globidium

Alicata (1930) gave a brief report on Globidium in sheep. Prior to 1930, the occurrence of Globidium in the abomasum of American sheep had not been reported. Seven out of 78 sheep which originated in Indiana were slaughtered and found to be infected. They appeared as small whitish nodules about 1 mm in diameter located in the mucous lining of the abomasum. The cyst inside the nodule is elliptical in shape and measured 450 to 700 microns in length and 325 to 465 microns in width. The contents of the cysts consists of crescent shaped sporozoites, 5.5 to 7.5 microns long and 1.5 to 2 microns wide. Later, other lots of sheep were slaughtered, 11 out of 101 sheep from West Virginia and 6 out of 72 sheep from Idaho were found to be infected.

## CHAPTER V

## PROTOZOA OF SWINE

## Chapter V

## PROTOZOA OF SWINE

CLASS: MASTIGOPHORA
ORDER: TRICHOMONADIDA
Family: Trichomonadidae

### Tritrichomonas suis (Gruby and Delafond, 1843)

This parasite was observed by Frye and Meleney (1932) from cultures of the feces of swine. From 63 specimens inoculated into culture media, 47 or 75 per cent were positive for trichomonads. This organism has three anterior flagella, an undulating membrane and a slender axostyle. It is quite small measuring from 8 to 10 microns in length. Kessel (1928) also observed this parasite. It apparently has no pathological significance.

TREATMENT: Unnecessary.

### Trichomonas sp.

Hegner (1938) reported on the finding of trichomonads in the facial lesions of a pig. The animal had a swelling on the right side of the upper jaw. There was a necrosis of the underlying tissue extending to the facial bones. Bacteriological examination revealed Pseudomonas pyocyaneus and Actinomyces. The organisms measured 7 to 15 microns in length and 4 to 7 microns wide. The possibility was suggested that the organisms may have inhabited the mouth and invaded the necrotic tissue.

ORDER: POLYMASTIGINA
Family: Chilomastigidae

### Chilomastix mesnili (Wenyon, 1910)

MORPHOLOGY: This flagellate is asymmetrical and pear-shaped in the trophozoite stage. It is rounded at the anterior end and tapers posteriorly. It measures about 10 to 24 microns in length. The body has a spiral-like twist. There are 3 anterior flagella, a cystostomal flagellum which resembles an undulating membrane and one trailing flagellum. The oval to lemon-shaped cysts are bluish-green in color. They average about 8 microns in diameter.

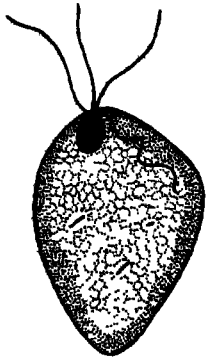
LIFE CYCLE. The trophozoites live in the mucosa of the large intestine and multiply by binary fission. The vegetative form becomes a cyst and leaves the host in the feces. When the encysted form is swallowed excystation takes place in the intestine and one trophozoite emerges from each cyst.

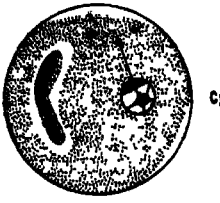
PATHOLOGY: Apparently this organism is nonpathogenic.

DIAGNOSIS: Diagnosis is made by direct microscopic examination of the feces.

TREATMENT: Unnecessary.

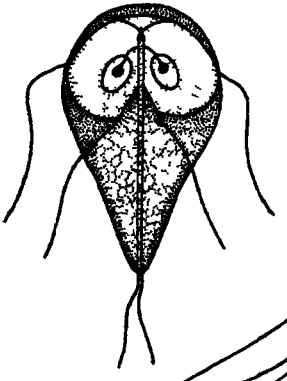
GENERAL REMARKS: Frye and Meleney (1932) found this parasite in three of 127 pigs examined for an incidence of 2.4 per cent. Kessel (1928) also observed this flagellate. This parasite occurs primarily in man.

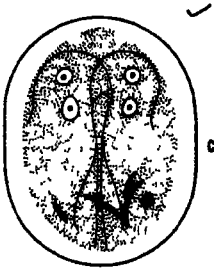




Cyst

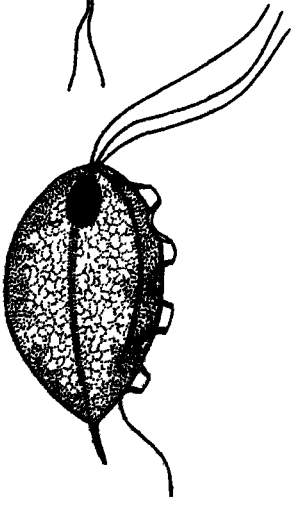
Chilomastix mesnili

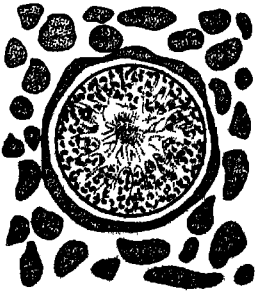




Cyst

Giardia lamblia





Sarcocystis sp.

Family: Hexamitidae

Giardia lamblia Stiles, 1915

DISEASE: Giardiasis, lambliasis.

MORPHOLOGY: The vegetative form is a bilaterally symmetrical, pear-shaped organism. The anterior end is broad, rounded and tapers posteriorly. It measures from 12 to 15 microns in length. The dorsal surface is convex while suction disc occupies the flat ventral surface. There are two nuclei, two axostyles and four pairs of flagella The cysts are oval bodies about 9 to 12 microns long and contain 2 to 4 nuclei.

LIFE CYCLE: The trophozoites reproduce by longitudinal fission. The cysts are swallowed and excystation takes place in the small intestine. New trophozoites are liberated from the cysts and migrate to the large intestine. Man is the natural host.

PATHOLOGY: Probably nonpathogenic in domestic animals.

DIAGNOSIS. Giardial infections may be determined by finding cysts or trophozoites in the feces.

GENERAL REMARKS: This flagellate is primarily a parasite of the small intestine of man. Frye and Melaney (1932) first reported it from a pig in Tennessee. Its distribution is not known.

TREATMENT: Unnecessary.

CLASS: SARCODINA
ORDER AMOEBINA
Family: Endamoebidae

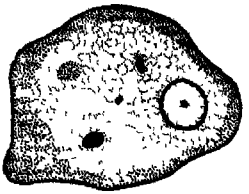
Endamoeba histolytica (Schaudinn, 1903)

DISEASE: Amebiasis, amebic dysentery

MORPHOLOGY. The trophozoite or vegetative form ranges from 15 to 40 microns in diameter. They are greenish yellow in color, the nucleus is indistinct. The protoplasm appears homogeneous except for small diffuse granules. Ectoplasmic pseudopodia move about rapidly. The trophozoites may contain several red blood cells. The precystic amoeba are colorless, spherical cells devoid of food vacuoles and the pseudopodia are sluggish in movement. The cyst form is quite characteristic. They are round, hyaline-like bodies and measure from 5 to 20 microns in diameter. Usually a pair of dark staining refractive bars are present. Mature cysts contain four nuclei.

LIFE CYCLE. The cysts are ingested with food and water. A quadinucleate amoeba emerges and nuclear division forms eight amoebulae. These migrate to the large intestine and develop into mature trophozoites. The active trophozoites divide by binary fission. The trophozoites under certain conditions become precystic and later secrete a cyst wall about themselves, and pass out of the body. By rapid divisions the cysts finally contain four nuclei.

PATHOLOGY: The amoeba first multiply rapidly in the intestinal lumen and in about 2 to 8 days produce ulcers in the mucosa. Histological changes include thrombosis in the capillaries, necrosis and cellular infiltration. Diarrhea is a common symptom. Secondary invaders may set up pyogenic infections in the intestinal ulcers. Animals which recover may harbor a low grade infection and become carriers. There are apparently no immune responses to this disease.

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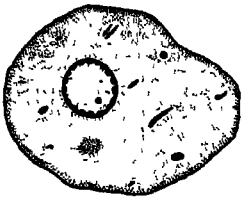
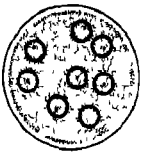
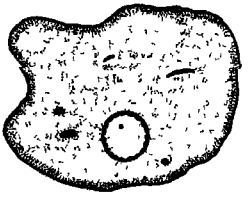
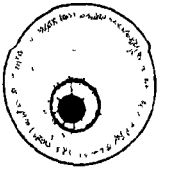
Entamoeba histolytica



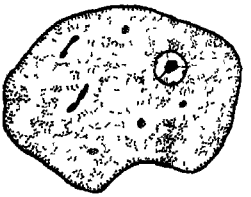



Endamoeba coli ✓

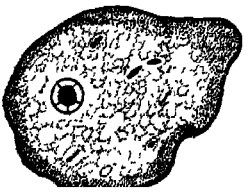


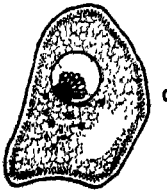



Endamoeba polecki




Endolimax nana ✓






Iodamoeba butschlii

DIAGNOSIS: A positive diagnosis of amebiasis depends upon the identification of the amoeba in the feces. It must be differentiated from similar appearing forms.

TREATMENT: No treatment is known for pigs. Some of the remedies suggested for human therapy might be tried.

CONTROL: Amebiasis is primarily a disease of man. Animals become infected by human carriers, careless in their personal habits. Pigs may be a potential source of danger as a reservoir host.

GENERAL REMARKS: Kessel (1928) was able to infect 7 out of 14 pigs with E. histolytica from human sources. Cysts were recovered from the infected pigs at frequent intervals. Frye and Meleney (1932) examined 127 pigs for protozoa parasites and found one two-months old pig infected with E. histolytica. The owner of the farm was also infected. Generally this parasite is rather uncommon in swine. The distribution in domestic animals is not known. It is primarily a disease of man and domestic animals become infected when sanitary facilities are lacking

Endamoeba coli (Grassi, 1879)

DISEASE: This amoeba is primarily a nonpathogenic commensal which ordinarily lives in the large intestine of man

MORPHOLOGY: The trophozoites measure about 20 to 30 microns in diameter The pseudopodia are blunt and granular The nucleus is distinct with a ring of refractive granules The precysts are 15 to 45 microns in diameter, while the spherical cysts are 12 to 22 microns. The mature cysts contain eight nuclei.

LIFE CYCLE: The life cycle is similar to that of E. histolytica After ingestion of the cysts in the food or water the trophozoite forms are liberated in the small intestine. Active trophozoites migrate to the large intestine and multiply by binary fission. Under certain conditions they encyst and are passed out in the feces

DIAGNOSIS: This amoeba should be differentiated from E. histolytica by microscopic examination of the feces

TREATMENT: Unnecessary.

CONTROL: Similar to that of E. histolytica

GENERAL REMARKS: Theobald Smith (1910) reported an amoeba similar to E. coli from pigs suffering from hog cholera. Kessel (1928) also found this parasite in swine. It is of very little economic or veterinary importance Kessel (1928) reported on the experimental infection of hogs with E. coli. In a survey of protozoa in domestic swine Alicata (1932) found one out of 35 infected with an amoeba indistinguishable from E. histolytica.

Endamoeba polecki Prowazek, 1912

SYNONYM: E. suis Hartmann 1913.

MORPHOLOGY: This is a uninucleated amoeba which varies in shape from spherical (5 to 12 microns) to elongated forms (15 microns). It resembles the precystic stage of E. histolytica.

LIFE CYCLE: Unknown but probably similar to E. histolytica or E. coli.

PATHOLOGY: Apparently nonpathogenic.

TREATMENT: Unnecessary.

GENERAL REMARKS: On the examination of 127 pigs Frye and Meleney (1932) found 80 pigs or 63 per cent infected with E. poleoki. It has no economic or veterinary importance. Alicata (1932) found 15 of 35 pigs infected with this parasite.

Endolimax nana (Wenyon and O'Connor, 1917)

MORPHOLOGY: The small vegetative or trophozoite form measures from 6 to 15 microns. The pseudopodia are broad and blunt. The nucleus is indistinct in the living animal but when stained it shows a large eccentric karyosome. The precystic amoeba is a spherical body about 5 to 14 microns in diameter. The cysts are oval, thin walled and measure from 8 to 10 microns. Mature cysts have four nuclei, usually clustered at one end.

LIFE CYCLE: Similar to E. coli. The trophozoites multiply by binary fission, the cysts by nuclear division. The mature cysts probably produces only four new amoeba after excystation.

PATHOLOGY: Nonpathogenic in the large intestine.

DIAGNOSIS: This amoeba is identified by microscopic examination of the feces. The small size, with oval four-nucleated cysts are characteristic.

TREATMENT: Unnecessary.

GENERAL REMARKS: Frye and Meleney (1932) found 7 out of 127 pigs infected with this amoeba (5.5 per cent). From 35 pigs examined Alicata (1932) found only one pig infected with cysts indistinguishable from E nana.

Iodamoeba butschlii (Prowazek, 1912)

SYNONYM: I. suis, O'Connor, 1920.

MORPHOLOGY: This amoeba in the trophozoite stage measures from 5 to 20 microns. The short pseudopodia move about very slowly. The stained nucleus contains a characteristic karyosome. The precystic amoeba is about the same size as the trophozoite. It forms a single nucleated cyst about 5 to 14 microns in diameter.

LIFE CYCLE: The life cycle is not completely known. It probably is similar to other cyst forming amoeba. The trophozoites multiply by binary fission. A single amoeba emerges from the cyst.

PATHOLOGY: Nonpathogenic in the large intestine.

DIAGNOSIS: This amoeba is readily identified upon microscopical examination of the feces.

TREATMENT: Unnecessary.

GENERAL REMARKS: From 127 pigs examined in Tennessee 31 or 24 per cent were positive for Iodomeba probably I. butschlii according to Frye and Meleney (1932). Alicata (1932) found 9 cases out of 35 infected with cysts indistinguishable from I. butschlii.

CLASS: SPOROZOA
ORDER: COCCIDIA
Family: Eimeriidae

Porcine Coccidiosis

There are at least five species of the genus Eimeria and one species of Isospora which infect swine in North America. These Sporozoa invade the epithelial cells of the alimentary canal, mainly the large intestine. The different species found in swine are g[illegible] on following page.

Eimeria perminuta Henry, 1931

Oocysts are small and range from 11 to 16 microns in length by 9 to 12 microns wide. The shape varies from ovoidal to spherical. The wall is rough and yellow in color. Sporulation requires about 11 days.

Eimeria spinosa Henry, 1931

Oocysts range between 16 to 22 microns long and 12 to 16 microns wide. The shape is typically ellipsoidal. The walls are yellow in color and studded with spines about 1.0 micron apart. Sporulation time is about 12 days.

Eimeria scabra Henry, 1931

The oocysts measure between 22 to 36 microns long and 16 to 25 microns wide. The wall is 1.0 to 2.0 microns thick, with a rough surface and brown color. The shape varies from ellipsoidal to slightly ovoidal. Sporulation requires from 9 to 12 days

Eimeria debliecki Douwes, 1921

SYNONYM: Eimeria suis Noller, 1921.

The oocysts are variable in size ranging from 12 to 30 microns long by 12 to 20 microns wide. Their shape is primarily ovoidal. The wall is smooth, colorless to brownish. A micropyle is not present. Sporulation requires from 7 to 9 days

Eimeria scrofae Galli-Valerio, 1935

Only a brief description is available for this coccidium. The oocysts are cylindrical and measures 24 microns long by 15 microns wide A micropyle is present.

Biester and Murray (1929) were the first to study extensively porcine coccidiosis in in the United States Becker (1934) summarized the literature on coccidiosis of pigs. Although the disease has been overlooked in the past, coccidiosis should be kept in mind when making a diagnosis. The two most pathogenic species appear to be E. debliecki and E scabra.

LIFE CYCLE: Oocysts are passed out in the feces and sporulate under favorable conditions to the infective stage (sporulated oocysts) from 7 to 12 days. The infective oocyst contains four sporocysts each which carry 2 sporozoites for a total of 8 and are swallowed with the food or water The sporozoites are liberated and each enters an epithelial cell of the large intestine. Each sporozoite becomes an active trophozoite and enlarges to a spherical schizont which fills the cell. The schizonts in turn form merozoites. When the cells rupture the motile merozoites enter new epithelial cells and the asexual cycle is continued for several generations

After several generations, the merozoites develop into male and female gametocytes to start the sexual cycle The microgametocytes fertilize the macrogametocytes which form zygotes or oocysts.

PATHOLOGY: Biester and Murray (1929) found that on experimental infections in pigs by feeding heavy doses of sporulated oocysted produced severe diarrhea, emaciation, constipation and death. Pigs which did not die were pot-bellied In many cases the walls of the large intestine became thickened. Bloody diarrhea was never observed. Heavy infections may cause a loss of the surface epithelium of the large intestine. Alicata and Willett (1946) found that experimentally infected pigs had a loss of appetite, diarrhea lasting from 7 to 11 days and made no weight gains Heavy infections may also interfere with digestion and absorption of food and water. Some animals recovered spontaneously while others remained permanently weak and unthrifty.

**TRANSMISSION:** Biester and Schwarte (1932) found that complete immunity was produced in pigs by feeding oocysts daily for a period of 100 days or more. Partial immunity was noted with light infections. Adult pigs may become carriers and shed oocysts in their feces continually in small numbers Eimeria from pigs are not infective to other animals and the reverse is also true. The disease is transmitted by ingestion of infective oocysts of porcine origin.

**DIAGNOSIS:** Diagnosis depends upon finding the oocysts by microscopical examination. Freshly discharged feces should be collected. Oocysts may not be present in the feces at the first symptoms. Two or three days later the organisms may be present in large numbers.

**CONTROL.** The control or prevention of clinical porcine coccidiosis must be considered from the standpoint of immune (carrier) animals and overcrowding Sanitary precautions should be made to prevent young pigs from eating large numbers of infective oocysts. Daily removal of manure and contaminated bedding is good sanitary practice. Isolation of young stock is helpful. All feed should be given in troughs to prevent contamination with the feces.

**TREATMENT:** Many drugs have been tried for curing porcine coccidiosis with little effect. Most remedies act as an astringent on the intestinal mucosa Some cases cure spontaneously. No specific drugs are known Biester and Murray (1933) obtained negative results with collodial iodine. Alicata and Willett (1946) inhibited the production of oocysts and prevented the symptoms of coccidiosis caused by E. debliecki and E scabra with sulfaguanidine. The treated pigs received the drug in their feed at the rate of 1 gram per 10 pounds of body weight. In the prophylactic treatment the drug was given two days before experimental infection and continued for 7 to 10 days In the curative dose the drug was administered for three days starting with the second day of oocyst discharge

**GENERAL REMARKS:** Swanson and Kates (1940) reported on a case of coccidiosis in a litter of 9 pigs.

Avery (1942) found that the oocysts of E debliecki and E scabra could survive and become infective after 15 months in soil Infected plots were tested at intervals of 4 to 8 weeks by maintaining for a period of 3 days a pig that had been raised coccidia free Infections acquired from unshaded plots were lighter than those acquired from shaded areas. Temperatures of the surface soil of the two plots ranged from - 40° C to -4.5° C. Unsporulated oocysts of E. debliecki and E. scabra cultured in tap water were exposed to (1) a temperature just above freezing 6 to 8° C., (2) alternate freezing and thawing at 0.5 to -3° C., (3) continuous freezing -2 to -7° C. After 26 days the various cultures were examined and the percentage sporulation ascertained Control cultures showed 93 per cent sporulation while none of the three types of cultures had sporulated. The cultures were then maintained at room temperature for 14 days and re-examined. The sporulation of the three groups was 88 per cent, 67 per cent and 55 per cent, respectively

## Isospora suis Biester, 1934

**MORPHOLOGY:** The oocysts in fresh swine feces are subspherical in shape and measure 22 microns long by 19 microns wide. The double wall of the oocyst is yellow to brown in color. No micropyle is visible.

**LIFE CYCLE:** Oocysts after being expelled in the feces sporulate under favorable conditions in about 4 days. The sporulated oocyst contains two ellipsoidal sporocysts which harbor 4 sporozoites each. The remaining portion of the cycle is similar to Eimeria. From the time of ingestion to the time of the first oocysts (prepatent period) requires from 6 to 8 days.

**PATHOLOGY:** Experimentally infected pigs showed a diarrhea on the sixth and seventh day. Diarrhea is followed by constipation. The small intestine is the primary site of the infection. Histological observation reveals a subepithelial and interstitial inflammation.

Typical surface desquamation is common accompanied with a hemorrhagic condition of the intestine. The disease apparently is not fatal but produces an alteration and destruction of the intestine to retard growth.

**DIAGNOSIS:** Diagnosis depends upon finding the characteristic oocysts in the feces. Further examination of sporulated material for specific identification will aid in making a positive diagnosis.

**TREATMENT:** Unknown.

**CONTROL:** Sanitation as given for Eimeria.

**GENERAL REMARKS.** This species of coccidia has been reported only from Iowa but probably is more widespread in its distribution.

### KEY TO OOCYSTS OF EIMERIA FOUND IN SWINE

| | | |
|---|---|---|
| 1 | Oocyst with micropyle | 2 |
| | Oocyst without micropyle | 3 |
| 2. | Oocyst 24 microns long and 15 microns wide, cylindrical | E. scrofae |
| 3. | Oocyst with smooth wall | 4 |
| | Oocyst with rough wall | 5 |
| 4 | Oocyst 12 to 30 microns long by 12 to 20 microns wide, oval in shape | E. debliecki |
| 5 | Oocyst with prominent spines, 16 to 20 microns long by 12 to 16 microns wide, ellipsoidal, oocyst brown | E. spinosa |
| 6 | Oocyst 11 to 16 microns long by 9 to 12 microns wide, yellow oocyst wall | E. perminuta |
| | Oocyst 22 to 36 microns long by 16 to 25 microns wide, yellow oocyst wall | E. scabra |

### ORGANISMS OF UNCERTAIN ZOOLOGICAL CLASSIFICATION

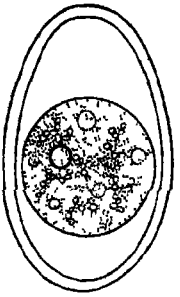
Sarcocystis miescheriana Kuhn, 1865

Parasitic forms grouped in the genus Sarcocystis are usually found in the striated muscles of mammals, birds and reptiles. The elongated forms are known as Miescher's sacs which are divided into compartments. The compartments contain crescent or banana-shaped spores known as Rainey's corpuscles. Until recently these parasites have been classified with the Protozoa.

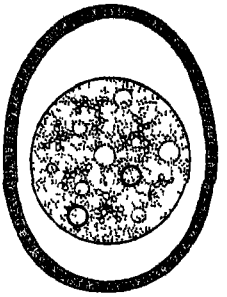
Due to the excellent work of Spindler and Zimmerman (1945), Spindler et al (1946) and Spindler (1947) it appears that Sarcocystis belongs to the fungi instead of the Protozoa.

PLATE XV

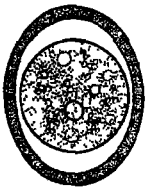
COCCIDIA OF SWINE



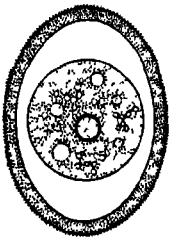
Eimeria debliecki



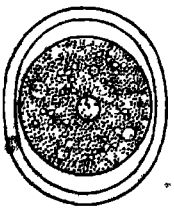
Eimeria scabra



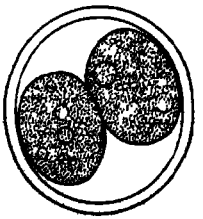
Eimeria perminuta



Eimeria spinosa



Isospora suis



Isospora suis
(Undergoing sporulation)

Spindler and Zimmerman (1945) removed aseptically from the muscles of swine several Miescher's sacs, ruptured and inoculated them in sterile dextrose cultures and incubated at 37° C. for 24 hours and subsequently at room temperature. After two weeks the spores budded off minute coccoid bodies which developed by budding, septate mycelium with vertical hyphae bearing spores. This fungus has been tentatively identified as Aspergillus sp. Twenty-five of 50 pigs injected or fed material harvested from cultures, after 4 to 6 months, harbored typical sarcocysts in their muscles. The control animals were negative.

Pigs, rats and mice fed sarcocysts passed yeast-like bodies in their urine or feces. These bodies, when cultured produced a fungus like the original material cultured from sarcocysts. Mice showed these bodies in the kidneys, rats and mice contained clumps of fungi attached to the walls of the ileum and cecum

In a later investigation Spindler et al (1946) fed pork infected with sarcocycsts to pigs, dogs, cats, rats, mice and chickens. They eliminated with their feces and/or urine a stage that is infective to swine if after ingesting such feces or urine. Infection was accomplished by feeding feces and urine eliminated subsequent to 14 days after the infected pork was eaten. Pigs fed infected muscle did not acquire the infection unless they ingested their own feces.

Symptoms observed in swine after eating infected muscle included vomiting, diarrhea, lack of appetite and temporary posterior paralysis. These same symptoms were noticed in pigs fed infective urine and feces. Infections showing 40 or more sarcocysts per gram of diaphragm tissue showed unthriftiness and stiffness of the muscles Lesions found at autopsy were enlargement of the kidneys and hyperemia of the stomach and intestinal mucosa.

Further evidence that Sarcocystis is really a fungus was shown by Spindler (1947). Histological sections of Miescher's sacs revealed that the sacs contained a network of jointed, hypha-like structures. These structures gave staining reactions characteristic of fungi. Rainey's corpuscles appeared to be exogenous growths on the jointed hypha-like structures. From this work it is concluded that the sarcosporidia found in swine is a fungus. The species name has not yet been determined.

Alicata (1932) in a study of an incidence of protozoa in 180 California hogs fed garbage revealed 135 infected with Sarcocystis. This may account for pigs becoming infected by feeding on garbage containing infected flesh

**TREATMENT:** Unnecessary.

**CLASS: CILIATA**
**ORDER: SPIROTRICHA**
Family. Bursariidae

## Balantidium coli (Malmsten, 1857)

**SYNONYM:** B. suis, McDonald, 1922.

**DISEASE:** Balantidiasis, balantidiosis.

**MORPHOLOGY:** The trophozoite measures from 30 to 150 microns in length to 25 to 120 microns wide. It is ovoid in shape and greyish-green in color. The entire body is covered with a delicate pellicle which bears spiral longitudinal rows of cilia which arise from granules in the ectoplasm. At the anterior end there is a narrow triangular peristome which leads to the cytostome and the cytopharynx. At the posterior end is an indistinct excretory pore called a cytopyge. There is a kidney-shaped macronucleus and a small spherical micronucleus. Cyst formation is apparently for protection rather than reproduction. The trophozoite secretes a wall and rounds up in a quiescent cyst stage. They measure about 45 to 65 microns in diameter

**LIFE CYCLE:** The trophozoite reproduces by transverse binary fission. Conjugation has been reported. The trophozoites live in the mucosa of the large intestine. Under certain conditions cysts are formed and become numerous in the rectum. When ingested the cyst wall dissolves and the trophozoite invades the mucosa and multiplies in the tissues.

**PATHOLOGY:** This parasite produces a mild colitis with diarrhea in man but apparently is not very pathogenic to swine. When large numbers of balantidia are found it usually is associated with other dysentery-like diseases.

**DIAGNOSIS:** Diagnosis depends upon the accurate identification of motile trophozoites or cysts found in the feces.

**TREATMENT:** Unnecessary.

**PREVENTION AND CONTROL:** B. coli is a common parasite of swine, incidences of 68 to 75 per cent have been reported. Since the pig is the chief source of human infections it becomes a public health problem. Adequate disposal of pig manure is essential. The use of hog manure for fertilizing truck crops should be discouraged. Individuals working in slaughter-houses or persons coming in contact with swine should be instructed in matters of personal hygiene. Contamination of water supplies may be reduced if sanitary precautions are taken for the animals.

**GENERAL REMARKS:** This organism is the most common parasite of pigs since nearly all pigs harbor a few organisms in the large intestine. Alicata (1932) found B. coli from 12 out of 35 pigs. Ray (1937) is of the opinion that B. coli is a definite pathogen in swine. More work is needed to confirm this observation

## CHAPTER VI

## PROTOZOA OF THE DOG AND CAT

## Chapter VI

## PROTOZOA OF THE DOG AND CAT

**CLASS: MASTIGOPHORA**
**ORDER: POLYMASTIGINA**
**Family: Hexamitidae**

### Giardia canis (Hegner, 1922)

**MORPHOLOGY:** This form is characterized by being bilaterally symmetrical, possessing two nuclei and is rounded anteriorly and pointed posteriorly. There is a dorsal convex surface and a flattened ventral surface, although there is ventral sucking disc with raised margins covering most of this area. There are eight flagella, four arising from the margin of the sucking disc, two from the posterior end of body, and two from about the median notch of the sucking disc. The trophozoite measure 11.9 to 17.0 microns in length by 7.6 to 10.2 microns in width, averaging 13.8 microns long and 8.5 microns wide. Characteristic ovoidal cysts are produced which measure 9.4 to 12.7 microns long and 6.8 to 8 9 microns in width.

**LIFE CYCLE.** The life cycle of this parasite is direct, with the cysts transmitting the infection from one host to another. The parasite localizes in the duodenum and jejunum.

**GENERAL REMARKS:** The importance of this parasite is not known at the present time, however, it is probable that G. canis may be the cause of some of the diarrheas of undetermined etiology in the dog. Catcott (1946) has found the organism in 17.7 per cent of a series of dogs examined in Ohio, and in one third of these cases there were symptoms of enteritis characterized by diarrhea. It has been shown by several investigators that diet will effect infections with this parasite as well as other intestinal flagellates. A high carbohydrate diet is much more favorable for the development of G. canis than is a high protein diet.

**TREATMENT:** Treatment has not been studied to any extent in the control of this infection in dogs, however, it would be expected that atebrin may successfully control the infection, as has been shown in man.

### Giardia felis Hegner, 1925

**MORPHOLOGY:** This species is very similar to G canis in the dog. Further study may prove it to be synonomous with G cati Deschiens, 1925. Giardia felis is morphologically similar to the species found in dogs The trophozoites measure 10.5 to 17.5 microns in length by 5.25 to 8.75 microns in width averaging 12 66 microns long and 6.6 microns wide. The cysts are 10.5 microns long and 7 35 microns wide.

The importance of this organism is unknown. Further study must substantiate the specificity of this form from that of the dog as well as its pathogenicity in the cat

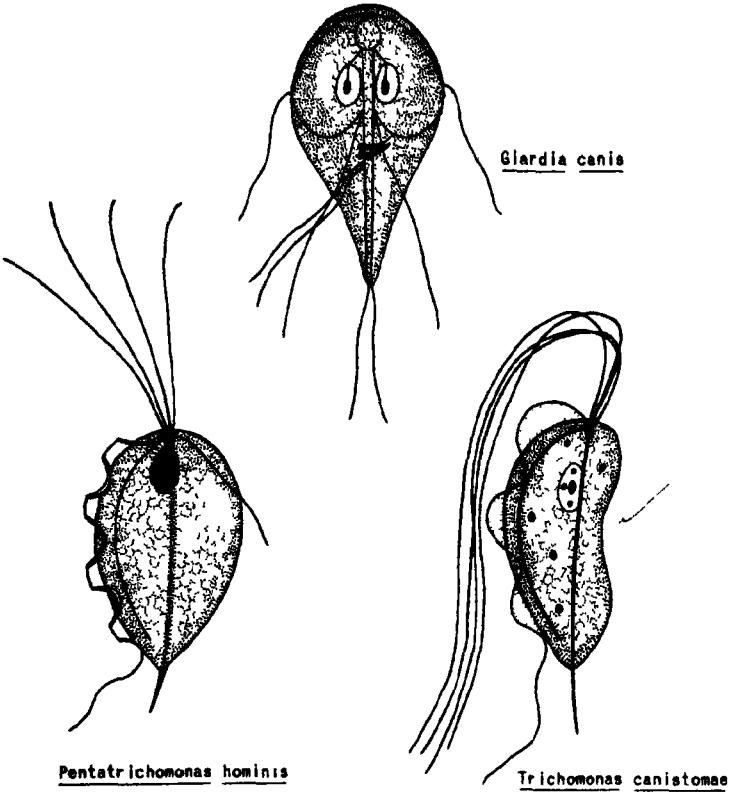
**TREATMENT:** Unknown.

**ORDER: TRICHOMONADIDA**
**Family: Trichomonadidae**

### Pentatrichomonas hominis (Davaine, 1860)

**SYNONYMS:** Trichomonas hominis (Davaine, 1860), Trichomonas felis Cunha and Muniz, 1922, Trichomonas parva Alexieff, 1911, Pentatrichomonas canis auri Chatterjee, Harendranath and Mitra, 1929, Trichomonas ardin delteili Derrieu and Raynaud, 1914.

PLATE XVI


Giardia canis
Pentatrichomonas hominis
Trichomonas canistomae
Toxoplasma sp.
Babesia canis

Little is known about the intestinal trichomonads which occur in dogs and cats in the United States. Bruce (1941) reported a case of intestinal trichomoniasis in a puppy from Kansas. The animal was positive for distemper inclusion bodies.

Recent evidence, both experimental and morphological, suggests that the intestinal trichomonad from man (P. hominis) is similar to the forms found in monkeys, cats, dogs, foxes, rats and squirrels. Kessel (1928) described experimental infections of kittens with trichomonads from man, monkey and rat. Simic (1932) transferred P. hominis from man to cats and dogs; from cats and dogs to man, and again from man, cats and dogs to dogs. Brumpt (1936) concluded the trichomonads from the cat, dog and man were similar. Simic (1933) demonstrated that T. parva of rats may be transferred readily to cat, dog and man and concluded that T. parva was identical to P. hominis. This was confirmed by Hegner and Eskridge (1935, 1937).

All of these forms possess from 3 to 5 anterior flagella with 5 predominating. The difficulties in ascertaining the exact number of flagella has been discussed by Kirby (1943) and Wenrich (1944). More study is needed on the trichomonad relationships in cats and dogs.

TREATMENT: Unknown.

Trichomonas canistomae Hegner and Ratcliffe, 1927

This species has been recorded from the mouth of dogs in Maryland. It possesses four anterior flagella, one trailing flagellum which passes along the margin of the undulating membrane. The body is pyriform. The average length is 9 microns and the width is 3.4 microns No cysts are known for this species

TREATMENT: Unknown, probably unnecessary.

Trichomonas felistomae Hegner and Ratcliffe, 1927

This species has been observed in the mouth of cats in Maryland. It is very similar to T canistomae The average length is 8.3 microns and the width is 3.3 microns.

Further study is required concerning the specificity of these two species as well as their pathogenicity. At the present time they are considered to be harmless.

TREATMENT. Unknown, probably unnecessary.

CLASS SARCODINA
ORDER: AMOEBINA
Family: Endamoebidae

Endamoeba histolytica (Schaudinn, 1903)

This parasite has been found in the dog in several parts of the United States in natural infections Both the dog and cat have been used in experimental infections. However, it is probably an uncommon parasite of the dog in this country and does not occur naturally in the cat.

The trophozoites of E. histolytica are usually 20 to 30 microns in diameter, but either smaller or larger forms may occur. Pseudopodia are formed with a characteristic explosive violence when freshly passed in the stool. The cytoplasm of the pseudopodia are clear when first formed, but the granular cytoplasm rapidly flows into them. Red blood cells may often be found in the cytoplasm, a characteristic not found in certain other amoeba. The motility of this form is only seen in the fresh stool, after being expelled from the body for a short time the amoebae become sluggish and difficult to distinguish from nonpathogenic forms. Cysts are passed in more chronic cases and are usually

found in the formed stool. They vary in diameter from 5 to 20 microns, and are usually oval in shape. Four nuclei are present in the mature cyst. There is a small central karyosome, which has a fine ring of chromatin granules on the inner surface of the nuclear membrane. Chromatoidal bodies may be present.

Cysts of this parasite will not be detected by the usual methods of concentration with sodium chloride, sugar or sodium nitrate.

In man, this parasite produces amebiasis which frequently has serious consequences.

TREATMENT: No treatment is known for dogs. Some of the remedies suggested for human therapy might be tried.

CLASS: SPOROZOA
ORDER: HAEMOSPORIDIA
Family: Babesiidae

Babesia canis (Piana and Galli-Valeria, 1895)

SYNONYMS: Piroplasma canis.

DISEASE: Canine piroplasmosis, canine babesiasis.

MORPHOLOGY: B. canis is typically a pear shaped organism, 4.5 to 5 microns in length, pointed at one end and round and bulbous at the other. The nucleus in stained films is a deeply appearing granule located near the pointed end, and extending from it to the rounded end is a fine string of granules. There is generally a vacuole in the cytoplasm. Although the organism is typically pear shaped it may be observed in a number of forms depending largely on its state of reproduction. In many instances multiple infections of from four to 16 parasites may be seen in a single cell.

LIFE CYCLE: The life cycle of B. canis in the United States has not been carefully studied, largely due to its uncommon occurrence. However, it is probable that B. canis is similar to the form described from cattle. It is suspected that the ticks Rhipicephalus sanguineus and Dermacentor variabilis act as vectors. The cycle in the tick has been studied by Christophers (1907) in India.

SYMPTOMS: In acute cases of canine piroplasmosis there is a high fever, progressive and marked anemia, a history of tick infestation, jaundice and hemoglobinuria usually terminating fatally. B. canis may usually be easily demonstrated in blood smears from such animals. However, this is not the type of infection which has been encountered in the United States. Here the symptoms are often vague, but over a period of weeks there is observed a general weakness, anemia, slight icterus of the sclera, intermittant fever, enlargement of the spleen and often a cough. Ticks may usually be found on such animals. Those cases terminating fatally develop a jaundice and hemoglobinuria. It is difficult to demonstrate any parasites in blood smears taken from these chronic cases. Red blood cells are reduced and leucocytes are greatly increased in number.

DIAGNOSIS: The only certain method of making a diagnosis in many cases is the injection of blood from the suspect into a susceptible dog, preferably an adult dog. (Splenectomy of the test dog will help to demonstrate the organisms in the blood stream.)

PATHOLOGY: Post-mortem examination of dogs which have died will show jaundice of the internal organs. The spleen and liver are enlarged and the kidneys are swollen and congested. The urinary bladder is distended and may contain greenish or red colored urine.

TREATMENT: Trypan blue has been used successfully for the treatment of canine piroplasmosis, and more recently trypaflavine (gonacrine) and Acapring (Bayer). Trypan blue is administered as a 1 to 2 per cent solution. The dye is triturated in a mortar, made up

to the desired percentage, filtered and autoclaved at 120° C for 20 minutes. An intravenous injection of 5 to 6 cc. for an average sized dog is recommended. Treatment may be repeated in 24 hours if necessary. The mucous membranes and skin will take on a blue color after treatment, but this will gradually disappear. Trypaflavin is administered at the rate of 0.3 cc. of a 2 per cent solution per pound of body weight.

CONTROL: The status of canine piroplasmosis at the present time makes control difficult since the vectors are unknown. However, any procedure to reduce the tick and blood sucking arthropod population will undoubtedly serve to lower the incidence of the disease. R. sanguinsus (brown dog tick) has been incriminated as a vector.

GENERAL REMARKS: Canine piroplasmosis is not widely recognized in the United States, having been reported from Florida by Eaton (1934) and Sanders (1937) and possibly from Texas (Merenda, 1939).

## ORDER: COCCIDIA

### Family: Eimeriidae

#### Genus Isospora Schneider, 1881

The genus Isospora is characterized by possessing, after sporulation, two spores, each containing four sporozoites. This is in distinction to the genus Eimeria which possesses four spores, each with two sporozoites.

#### Isospora bigemina (Stiles, 1891)

SYNONYMS: Coccidium bigeminum Stiles 1891.

DISEASE: Canine coccidiosis.

The oocysts are spherical to ovoidal in shape with no indication of a micropyle. They occur in two size ranges, which may represent two species, but at the present time are considered to be one. It is of interest to note that unsporulated oocysts are only passed during the acute stages of the infection, sporulated oocysts or single spores are passed at other times. The small race measures 10 to 14 microns in length and 7.5 to 9.6 microns in width. The spores are 7.5 to 9 microns in length and 5 to 7 microns in width. The large race measures 18 to 20 microns in length and 14 to 16 microns in width. Intermediate forms between the small and large races occur. In a single infection it is common to have either the small or large race, but not both. In the chronic forms in which the sporulated oocyst is passed, the oocyst wall appears very delicate and is closely wrapped about the spores.

LIFE CYCLE: In the chronic form of the disease the sporulated oocyst is passed in the feces and is apparently immediately infective to other animals. In the acute stages, however, only the unsporulated oocysts are passed and these require approximately 96 hours outside the body of the host to become infective. Infection occurs by the ingestion of the sporulated oocyst by the dog, cat, fox, mink and probably other closely related carnivores. The sporozoites are liberated and penetrate the tissues of the small intestine. Further development may take place in either the epithelium or sub-epithelial tissues of the villi, however, further clarification of this cycle in the dog is required. It is probable that the early stages are found in the epithelium, as Wenyon and Sheather (1925) found these cells in the small intestine filled with parasites during the early acute stages of the disease, but found none in the sub-epithelial tissues. The schizonts which occur in the epithelial tissues are smaller than those found later, as are the sexual stages. The unsporulated oocysts are liberated in the feces six to seven days after infection. Further asexual development and the production of sporulated oocysts occurs in the sub-epithelial tissues.

TREATMENT: Unknown.

Isospora felis Wenyon, 1923

SYNONYMS: Diplospora bigemina Wasielewski, 1904.

The oocysts of this species are oval or egg-shaped and measure 39 to 48 microns in length and 26 to 37 microns in width, with most of them measuring about 45 by 33 microns. The oocyst is passed in the feces in the unsporulated form.

LIFE CYCLE: After the oocyst is passed in the feces of infected dogs, cats and other closely related carnivores approximately 96 hours is required for sporulation and the development of infective oocysts. The spores measure 20 to 27 microns in length by 18 to 21 microns in width. Infection occurs by the ingestion of the sporulated oocyst. The liberated sporozoites penetrate the epithelial cells in the small intestine and to some extent the cecum. Subepithelial forms do not occur. The schizonts, macrogametocytes and microgametocytes retain an elongate gregariniform character to a rather late stage in their growth. The oocysts when they are formed in the tissues do not have the characteristic resistant wall, this developing after passing into the lumen.

TREATMENT: Unknown.

Isospora rivolta (Grassi, 1879)

The oocyst of this species is egg shaped, much like that of I. felis, but smaller, measuring 20 to 25 microns in length and 15 to 20 microns in width. At the pointed end a micropyle may sometimes be seen. The oocysts are usually unsporulated when passed in the feces, but occasionally sporulated forms occur, thus, apparently being intermediate between I. bigemina and I. felis

LIFE CYCLE: When the oocysts are passed they are usually unsporulated and thus not infective. Sporulation requires approximately 96 hours. The spores measure about 16 microns in length by 10 microns in width. Infection takes place by the ingestion of the sporulated oocyst. Development of the organism takes place in the epithelium of the small intestine and is generally similar to I. felis, although in this species occasional development in the sub-epithelium may occur.

Canine and feline coccidiosis

SYMPTOMS: At the present time the symptomatology of coccidiosis in the dog and cat cannot be described by species, but is considered generally similar in each form. The predominent symptoms of coccidiosis in severe cases are a bloody diarrhea, rapid progressive emaciation, anemia and general weakness. Symptoms usually occur on the fifth to sixth day after infection and are introduced by a catarrhal diarrhea which becomes bloody in about two days. Oocysts will usually be found in the feces five to six days after the infection. The animal is depressed, weak, loss of appetite and there may be a rise in temperature. Muscular tremors of the posterior limbs may be observed. Coccidiosis may occasionally be confused with distemper. If the animal recovers the bloody diarrhea gives way to mucous passages after two to four days, and most symptoms disappear entirely in a week to ten days after symptoms were initiated. It should be noted that many dogs pass oocysts but are apparently in perfect health. These carrier animals apparently do not suffer themselves but are of importance in the transmission of the disease to other dogs. Animals which recover from coccidiosis are relatively resistant to further infection.

PATHOLOGY: The changes occurring in coccidiosis are primarily confined to the small intestine. A hemorrhagic enteritis, frequently with ulceration occurs throughout the small intestine in severe infection. These lesions are most severe in the lower ileum, less so in the jejunum and least in the duodenum. The hemorrhage varies from petechia in light infections to diffuse in acute infections. The mucosa is thickened and there may be widespread desquamation of the epithelium. In recovered cases, and in those which have undergone repeated infections, there is an excessive amount of connective tissue.

DIAGNOSIS: Diagnosis is dependent upon recovery of oocysts in the feces. However, a diagnosis of clinical coccidiosis must also take into consideration the symptoms of the infected animal. Many dogs will harbor coccidia without being visibly affected.

TREATMENT: There is no treatment for coccidiosis in the dog or cat. Since the disease will usually run its course and end rather rapidly in either death or recovery, various drugs have frequently been given credit for the recovery. Careful nursing, supplemented with injections of dextrose and blood transfusions are useful. There are no sulfonamides which have been carefully tested, although it is possible that sulfadiazine or sulfamerazine may have some value if used very early in the course of the disease.

CONTROL: The prevention of coccidiosis in dogs and cats is very difficult. Due to the fact they are frequently allowed to run freely, the acquisition of infection becomes entirely a matter of chance. Kennels should be kept clean in order to reduce the intake of oocysts to a low level.

GENERAL REMARKS: Coccidiosis in the dog has been shown to be quite common in the United States. Hall and Wigdor (1918) reported an incidence of 7.5 per cent in Detroit, Michigan, Lee (1934) found 13.8 per cent in Iowa, Gassner (1940) found 79 per cent in Colorado and Catcott (1946) found 10.6 per cent in Ohio It is of interest to note that in Gassner's report the incidence of the three species was as follows: I. felis 6 per cent, I.rivolta 20 per cent and I. bigemina 74 per cent with multiple infections occurring in some dogs. Catcott's report on the same species was 3.5 per cent, 4.45 per cent and 2.6 per cent, respectively.

Coccidiosis in the dog, as in other species of animals, is a self limiting infection. Infections which penetrate deeply into the mucosa such as I. bigemina seem to produce a greater resistance to reinfection than do the other superficial infections of I. felis or I. rivolta. However, infection with all three species will produce a resistance against reinfection.

### Eimeria canis Wenyon, 1923

This parasite is apparently rare in the dog and has only been seen in the oocyst stage. The oocyst measures 18 to 45 microns in length by 11 to 28 microns in width. It is ovoidal in shape and has a fairly thick wall, often covered with a rough membrane which tends to separate in fragments from the cyst wall proper. A small micropyle is present and the oocyst may present a pink color. Three to four days is required for sporulation. Skidmore and McGrath (1933) reported the occurrence of this parasite in Nebraska dogs.

TREATMENT Unknown.

### Eimeria felina Nieschulz, 1924

This species has been described from the cat and only the oocyst has been reported. It has not been reported in the United States. Its main difference from E. canis was the lack of the rough outer membrane and they were colorless. However, Skidmore and McGrath (1933) described colorless oocysts of E. canis in Nebraska.

TREATMENT: Unknown.

## ORGANISMS OF UNCERTAIN ZOOLOGICAL CLASSIFICATION

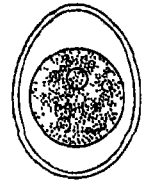
### Toxoplasma sp.

Olafson and Monlux (1942) reported the first cases of toxoplasmosis in dogs in the United States (New York). Symptoms varied but in general there is gradual emaciation with enlargement of the lymph nodes. Dyspnea was a common symptom. Bloody diarrhea

# PLATE XVII

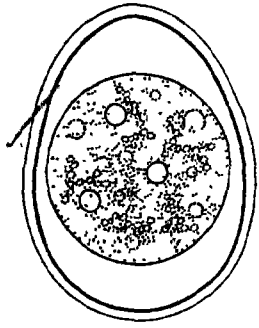
## COCCIDIA OF CARNIVORES

Isospora bigemina

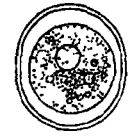


Isospora rivolta

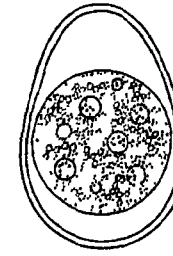
Isospora felis



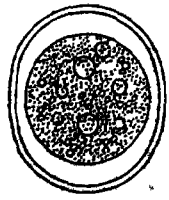
Isospora laidlawi



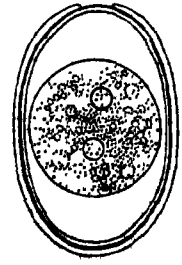
Isospora canivelocis



Eimeria mustelae



Eimeria yison



Eimeria canis

Eimeria felina

occasionally occurs. The enlarged mesenteric lymph nodes are inflamed with edema and hemorrhage of the adjacent tissues. Nodules occur in the lungs and intestines. Necrotic areas of the liver and spleen may occur. Wherever the organisms are found there is some eosinophilic infiltration. The disease, is usually fatal in dogs.

In fresh preparations of the various tissues (lungs, liver, muscle, brain, kidney) the organism appears as an elongated structure with pointed ends Stained films show oval or pear shaped bodies. These bodies average from 2 to 4 microns in size. Motility and flagella have not been observed in fresh material. In tissues these organisms may be found singly or in groups inside a cyst-like sac. The method of reproduction is by some means of longitudinal division and is thought to occur intracellular. When the host cell dies the parasites are released.

Only one case has been described from cats in the United States. According to Olafson and Monlux (1942) the symptoms and lesions were similar to those found in dogs. However, the diagnosis was based on the lesions and demonstration of typical parasites. The pathogenicity was not proved. There was a marked proliferation of epithelial cells in the lungs.

TREATMENT: Unknown

## CHAPTER VII

## PROTOZOA OF POULTRY

## Chapter VII

## PROTOZOA OF POULTRY

CLASS: MASTIGOPHORA
ORDER: RHIZOMASTIGINA
Family: Mastigamoebidae

### Histomonas meleagridis (Smith, 1895)

SYNONYM: Amoeba meleagridis Smith, 1895.

DISEASE: Blackhead, infectious enterohepatitis, histomoniasis.

HOSTS: Chicken, turkey, pea fowl, ruffed grouse, Hungarian partridge, quail, pigeon, ducks, geese.

MORPHOLOGY: This parasite of chickens, turkeys and other birds is a pleomorphic flagellate and exists in different forms The organism will be described under the various stages used by Tyzzer (1919) although these are not entirely satisfactory.

The first form is the invasive stage and is found in early lesions of the disease, rarely in older lesions. It appears to be entirely amoeboid in nature, 8 to 17 microns long, although some organisms as long as 30 microns have been noted. It is found between the cells and is never intracellular in nature. The cytoplasm is basophilic and consists of a clear ectoplasm and a finely granular endoplasm. There is a small extranuclear body present although its location is variable. Ingested particles may be found in vacuoles in the cytoplasm, but bacteria are never observed in this stage.

The second form of development is the vegetative stage. This is found in slightly older lesions and is usually larger than the invasive stage described above. It is 15 to 21 microns in length and about 12 microns in width. They are present in great numbers and are associated with distension and disruption of the tissues. It is believed from the rounded form that active amoeboid movements have ceased, or are not of common occurrence. The cytoplasm is still basophilic, clear and transparent, but the inclusions which were found in the earlier stage have disappeared.

The third form is the resistant stage and is found in the oldest lesions of the disease. These forms are small, varying in size from 4 to 11 microns in diameter. The cytoplasm is acidophilic in contrast to the basophilic cytoplasm of the previous two stages, and is filled with small granules or globules which give it a rather coarse appearance. This stage is eventually enclosed in definite spaces due to a tissue reaction of the host, or are taken up by phagocytic cells. In these spaces they may appear dissociated from one another. At times the organisms are spherical in shape, or compressed in masses with their shape modified with contact by other flagellates. The number of organisms in such masses is not constant, but varies from two to several dozen. No organism is found at this stage of the disease which approaches that of the previous two stages. After being enclosed in the tissue spaces the parasite becomes surrounded by a transparent thick layer which appears almost cyst-like in nature. Although these forms are described as resistant stages, this is a misnomer. Even though a cyst-like membrane surrounds the parasite, it is still very susceptible to environmental conditions and can live only a very short time outside the body.

In addition to the tissue stage the parasite may develop flagellated stages in the lumen of the cecum or in culture media. There is usually only one very short stumpy flagellum in these forms, although as many as four have been observed. In the cecal contents the living organism exhibits a characteristic type of motility. It possesses a pulsating, rhythmic, intracytoplasmic type of movement or they may exhibit a more amoeboid type of locomotion with the formation of pseudopodia. The movement of this organism is very similar in many ways to trichomonads, however, close observation will reveal that an undulating membrane or axostyle is not present. This stage may be present in large num-

bers in the lumen of the cecum. In many other instances they are very difficult to find.

Wenrich (1943) made comparative studies of histomonads from the ceca of pheasants and chickens. Measurements of 400 organisms from pheasants gave a range from 9 to 23 microns in diameter with an average of 13.9 microns. Measurements from 400 organisms from chickens gave a range from 5 to 18 microns in diameter with an average of 7.86 microns. The nucleus of the pheasant histomonads is regularly near the periphery and associated with the flagellar apparatus. Four flagella are regularly attached to the blepharoplast. Food objects are ingested by histomonads. The polymorphism of Histomonas with its wide range in size from 5 to 30 microns in diameter, its variation in flagellar apparatus, often no flagella, with one or more up to four is quite striking.

LIFE HISTORY. Many details are still unknown in the life cycle of Histomonas meleagridis, particularly the transmission of the organism under natural conditions. Infection occurs either by the ingestion of the parasite in the droppings of infected birds, or from soil containing the infected eggs of the cecal worm Heterakis gallinae. There can be no doubt that ingestion of the organism from infected droppings plays a major role in the spread of the infection, particularly in epizootic outbreaks of the disease. In order for birds to become infected they must ingest the organisms shortly after they have been passed out of the body of the host. This is due to the fact that the organisms are very susceptible to environmental conditions, and die very rapidly. Most of the parasites will die in a few hours after leaving the host and practically all will be dead in 24 hours. Experimentally it is difficult to infect birds by the oral route, but this is probably due to the fact that relatively small numbers of organisms are given and they are only administered once, in contrast to continued infection under farm conditions. In epizootics where large numbers of birds are involved it seems apparent this direct transmission through the ingestion of infected droppings is one of the main methods of passage.

The eggs of the cecal worm, Heterakis gallinae, may become infected with the etiological agent of blackhead, and when these embryonated eggs are ingested infection will result. The egg must be embryonated and contain infective larvae, otherwise the eggs would pass through the digestive tract without the liberation of the infective agent. It is interesting to note that although Histomonas meleagridis has never been seen in the nematode egg the circumstantial evidence for its presence is so strong that it is an accepted fact. However, the organism has been observed intracellularly in the epithelium of the gut of the cecal worm in acute cases of the disease. Most carry over on farms is undoubtedly in the eggs of this worm. This is due to the fact these eggs, like ascarid eggs, are extremely resistant and capable of living for long periods under adverse environmental conditions. McKay and Morehouse (1947) in a series of 54 experiments involving 313 turkey poults receiving embryonated H. gallinae eggs, 251 (80 per cent) died of blackhead.

A third method of transmission which may play a role under natural conditions is the mechanical transmission by means of arthropods. This method is probably not of great importance. If arthropods, flies in particular, play a major role in the transmission of the disease more cases of sporadic blackhead would be noted rather than infections of entire flocks.

Although not a different method of transmission it should be noted that the most serious cases of blackhead develop in turkeys when they are closely associated with chickens, or are allowed to run on ground that has previously harbored chickens. Practically all chickens harbor the cecal worm, and apparently large numbers also are infected with H. meleagridis. Although not suffering clinical symptoms of the disease chickens may act as carriers. Thus, most soil from poultry yards will contain embryonated eggs of the cecal worm which may harbor the etiological agent of blackhead. We cannot emphasize to strongly the dangers of maintaining chickens and turkeys in close proximity, or of running turkeys on land that has had chickens.

Histomonas meleagridis first localizes in the cecum. The chronological course of development is not well understood, but probably follows very closely the sequence of morphological stages discussed in the previous section. The organism may be demonstrated

in the droppings of infected birds in from one to five days following infection, and will be continued to be discharged for a week more or less, after which time their appearance is sporadic due to the obstructive lesions which develop in the cecum. The liver is usually affected in most cases, and the parasite is probably carried to this organ in the blood stream.

SYMPTOMS: This infection is characteristically a disease of turkeys under 12 months of age or about the time the combs and wattles are developing. Although it is often thought of as a disease of young birds, older birds are equally susceptible, farmers often losing large numbers of birds about the time they are ready for market. In other cases breeding flocks occasionally suffer losses from the disease. Thus, the disease may occur at any age, but there can be no doubt that the mortality in older birds is never as high as in the younger age groups. The incubation period usually varies from one to two weeks. It should be remembered that birds may be passing organisms in their droppings previous to the development of symptoms and are spreaders of the disease.

The first evidences of the disease are droopiness or the appearance of sulfur colored droppings, although the latter are not constantly present, particularly in younger birds. Blackhead, the common name of this disease, is a misnomer, since it is not a symptom characteristic of this disease alone. In fact the head does not become darkened more frequently in this disease than in other types of infections. In acute infections death may occur very rapidly, with practically no other evidence of disease. The mortality rate may be very high, approaching 100 per cent in severe outbreaks, and undoubtedly averaging 50 per cent unless it is kept under control. These high death losses only occur in birds in the younger age groups, in older birds the death loss rarely exceeds 25 per cent.

The disease may occur in chickens with symptoms closely resembling those found in the turkey although this is usually rare Many chickens harbor the parasite, but show no symptoms of the infection. In most cases gross evidences of the disease are not present.

PATHOLOGY. The principal changes occurring in blackhead are in the liver and cecum. Occasionally turkeys may be found with only lesions in the cecum, but these are usually the early infections. Lesions of the liver occasionally occur alone, but these have probably spread from a lesion in the cecum which has healed.

One or both ceca may be involved. The earliest lesions in the ceca are small, raised pin-point ulcers in which the organisms may be readily demonstrated. Later these become much enlarged and thickened with yellowish patches from 10 to 15 mm in size scattered along the surface of the serosa. Occasionally these ulcers may perforate the cecal wall and result either in a fatal peritonitis or adhesions of the ceca to other organs in the abdominal cavity. When the cecum is opened it may be filled with a tough leathery, yellowish white material On removing it is found to be tightly adherent to the mucosa, so when pulled out a rough denuded area is left. On gross sections this plug is found to have been deposited in concentric layers and is often hollow in the center. The ceca are frequently enlarged.

The liver is involved in practically all fatal cases. The surface is covered with spherical, saucer-shaped depressed areas up to a centimeter or more in diameter. The early lesions are a mottled red in color, later becoming muddy yellow and finally a dirty green. If the liver is sectioned grossly it will be found that these lesions extend throughout the liver parenchyma.

The various stages of the parasite may be found in the types of lesions described in the earlier section on morphology. In turkey poults the infection is accompanied by very little tissue reaction, whereas, in the chicken it is accompanied by an intense infiltration with polymorphonuclear leucocytes. In that portion of the lesion which has been infected longest there are large numbers of giant cells which destroy many of the parasites.

Repair is marked by a general disappearance of H. meleagridis from the tissues. A line of demarcation develops between the living tissue and the mingled necrotic tissue and exudate. In the cecum these materials are incorporated in the cecal plug in the lumen. The diseased tissue is replaced by actively growing blood vessels and connective tissue and the entire reaction is lymphoid in nature. In turkeys which recover from the infection extensive scarring of the cecal mucosa and liver results. In young turkey poults the entire mucosa may be destroyed and the lumen obliterated. In older birds where the lesions may not be as severe the mucosa and the liver may regenerate. In chickens the repair may be so perfect the ceca and liver of recovered birds cannot be differentiated from the normal.

Levine (1947) reported an interesting case of histomoniasis in a turkey showing kidney lesions. The kidneys contained numerous, white, circular lesions about one mm in diameter. On histological examination the parasites were found to be distributed singly and in large masses in the necrotic areas.

DIAGNOSIS: The disease may be diagnosed from the characteristic pathological picture in the cecum and liver. It is not necessary to demonstrate the presence of the parasite.

CONTROL: The foundations for the successful present day practices of turkey production were laid down in the work of Cooper Curtice in 1906 at the Rhode Island Experiment Station. He noted that greatly improved results occurred when turkeys were reared without any contact with chickens. He succeeded in maintaining over 60 per cent of his turkeys when they were reared away from chickens in contrast to the 17 per cent reared in close proximity to chickens. Since this time much evidence has been accumulated to show that it is impossible over a period of time to raise turkeys in the presence of chickens, or to raise turkeys with the older turkeys, which like chickens may act as carriers of the disease.

The prevention of blackhead today consists of good management, particularly a rigid program which prevents all contact between turkey poults and older turkeys or chickens. The most successful program of rearing turkeys consists of artificial hatching, brooding and rearing the birds in confinement or practicing a definite program of rotation. A movable brooder house suitable for chicks makes an ideal building for brooding turkey poults on small farms. Water fountains and feeders should be placed on wire to avoid heavy contamination about these areas. Where large numbers of turkeys are reared a wire floor is very satisfactory. It is easily cleaned and prevents the birds from coming in contact with their droppings. Many breeders are successful in using a sun porch with a hardware cloth floor which may be attached to a conventional brooder house, or one with a wire floor. Pens must be cleaned at regular intervals, preferably daily, if they are to give satisfactory results. Roosts should be put in the brooder houses when the poults reach 3 or 4 months of age. When the poults are six to eight weeks of age they are moved to a range that has not been used for several years by any adult turkeys or chickens.

Billings (1928) described a four pen system for the raising of turkeys that has been very successful. The underlying principal is to keep the young poults on clean fresh ground at all times. These pens or ranges need not be large, as turkeys do not require a large area over which to roam if the ground is clean. The house should be placed in the middle of a plot and by dividing this into four yards the poults are given access to a new yard each month. There should of course be a door cut into each corner of the house so that the birds will have access to their yards at the proper time.

Another system has been used successfully consists of having a raised wire platform at one of the yards on which the birds are fed, watered and near their roosts. It has been observed the great majority of the droppings will be deposited around the feeders, waterers and roosts. In this method the droppings will pass through the wire and out of contact with the birds. Instead of having a yard which is rotated at regular intervals, the yard is covered with a coarse gravel or cobblestones on which the birds are kept from three months of age until they are marketed. Some producers raise large numbers of birds with this system with no losses from blackhead.

**TREATMENT:** There is no drug known at the present time which is successful in the treatment of blackhead. Arsenicals such as Mapharsen and Tryparsamide have been tried but the results have not been conclusive. Recently Bolin, Goldsby and Eveleth (1947) have reported that di-isobutyl phenoxy ethoxy ethyl di-methyl benzyl ammonium chloride (Formula 144) is effective in the control of blackhead in a flock after a diagnosis has been made, as well as a prophylactic before infection has occurred. From the limited evidence presented in support of these claims further studies on this compound are indicated.

Phenothiazine has been widely heralded as an agent effective in the control of the disease. It should be borne in mind this drug has no effect against H. meleagridis, although it is very effective in the removal of cecal worms, Heterakis gallinae, and thus over a period of years might have some prophylactic value.

**GENERAL REMARKS:** In addition to occurring in the chicken and turkey Histomonas meleagridis has also been found in the pea fowl, ruffed grouse, Hungarian partridge, quail, pigeon, ducks and geese. The severity of the infection of the disease in these different hosts varies although in some cases typical blackhead occurs.

Tyzzer (1934) reported a loss of virulence in the course of long cultivation of this parasite and immunization of turkeys could take place by infecting susceptible birds with these avirulent strains. However, further cultivation of the organisms may in addition to the loss of virulence result in a loss of immunizing power.

DeVolt (1943) described a new improved medium for the cultivation of H. meleagridis. The formula of the medium is given in the appendix.

Durant (1930) and Delaplane and Stuart (1933) have shown that blackhead can be prevented by surgical abligation of the ceca or by the use of aluminum clamps. There is a considerable surgical mortality with this method and it probably cannot be used as a practical application for the control of this disease. It does show that the ceca are the points of entry for the infection.

## ORDER. PROTOMONADINA
### Family: Bodonidae

### Pleuromonas jaculans (Perty, 1852)

Uribe (1921) reported this flagellate in large numbers in the cecal contents of young chickens which had been fed Heterakis material. He believed this free-living protozoan, common in stagnant water, could become adapted to entozoic life. It can also multiply in media outside of the animal body.

The flagellate is 5 to 12 microns long and about 5 microns wide. It is colorless and somewhat kidney-shaped. There are two flagella, one short anterior flagellum and one long trailing flagellum two to three times the body length. Apparently 4 to 8 organisms emerge from one spherical cyst. It probably has no pathological significance.

**TREATMENT:** Unnecessary.

## ORDER: TRICHOMONADIDA
### Family: Trichomonadidae

### Pentatrichomonas gallinarum (Martin and Robertson, 1911)

**HOSTS:** Turkey, chicken, guinea-fowl.

**MORPHOLOGY:** This trichomonad is one of the most common flagellates occurring in the cecum of the turkey and to a lesser extent in the chicken. Cross infection is possible between

these birds. The organism is usually pear-shaped, although the form is variable. When pear-shaped it measures 6 to 8 microns in width and 9 to 12 microns in length. There are five anterior flagella, and one trailing flagellum which is free posteriorly. The latter arises from the anterior blepharoplast complex and passes posteriorly as the marginal filament of the undulating membrane. Anteriorly there are two blepharoplasts. The nucleus is near the anterior end of the body and has chromatin distributed on the nuclear membrane. The parabasal body lies at the base of the undulating membrane and appears as a row of granules. The cytostome appears as a slight depression at the anterior end of the body and is usually very difficult to see. The axostyle is slender and projects from the posterior margin of the body at about the same point that the trailing flagellum leaves the body. No cysts are formed.

SYMPTOMS: Hadley and Amison (1911) and Jowett (1911) held that infectious enterohepatitis was caused by trichomonads, but this work was discredited for a number of years by the work of Tyzzer (1919, etc). Recently this theory has been revived, although not conclusively demonstrated, by Allen (1936, 1940). She considers there are two types of the disease, one produced by Histomonas meleagridis and a second produced by P. gallinarum.

Two types of trichomoniasis have been described by Allen and Olson (1942), namely; acute and chronic. Acute trichomoniasis affects younger birds and may or may not be severe enough to produce death. The disease is characterized by a liquid pale yellow cecal diarrhea, roughened feathers, loss of appetite and resultant loss in weight. The mortality ranges from 0 to 44 per cent.

Chronic cases are characterized by an intermittant diarrhea ranging in color from a pale to deep yellow to orange, and in advanced cases the droppings contain large quantities of urates. There is a loss of appetite, loss of weight and later a subnormal temperature. The chronic phase, which consists of survivors of the acute, develops slowly and only a few birds in a flock develop fatal cases at any one time.

PATHOLOGY: The pathology as described by Allen and her co-workers very closely resembles that of blackhead but may be differentiated. The cecal lesions are essentially the same, however, the liver lesions make the differentiation possible. They are usually smaller, have irregular outlines and are raised or level with the surface of the liver in contrast to the depressed lesions of histomoniasis. Mixed infections containing both Histomonas and Pentatrichomonas are large, depressed in the centers and granular and elevated at the borders.

GENERAL REMARKS: The role of these organisms in blackhead still remains to be determined. In addition a blastomycete has been described by Knigk (1935) in Germany as a cause of this disease. Further investigations concerning the etiology of this disease are required Hawkins and Dunlap (unpublished data) were unable to produce any lesions or evidence of disease in turkey poults infected with this organism, although large numbers remained present over a period of several months until the turkeys were sacrificed.

Gabls and Hillaert (1947) have succeeded in cultivating P. gallinarum in the chorio-allantoic fluid of chick embryos. The organisms persisted until hatching of the chicks and could be recovered from the small intestine.

TREATMENT: Olson and Allen (1940) reported on the treatment of cecal and liver trichomoniasis in turkeys by fever therapy. Birds were placed in a thermostatically controlled cabinet for periods of one to two hours. The internal body temperature of the birds were raised from 2 to 6 degree above the normal of 106.5° F. by maintaining an air temperature within the incubator of 104° F. at a relative humidity of 60 to 70 per cent. Usually three treatments at intervals of every other day were sufficient to arrest the disease. Advanced cases required 6 treatments. Twenty of 24 birds recovered from the disease by this treatment. Whether this method is practical remains to be seen.

## Trichomonas gallinae (Rivolta, 1878)

**SYNONYMS:** Trichomonas columbae (Rivolta and Delprato, 1880), Trichomonas hepaticum (Rivolta, 1878), Trichomonas diversa Volkmar, 1930, Trichomonas halli Yakimoff, 1934.

**DISEASE:** Avian trichomoniasis.

**HOSTS:** Pigeon, turkey, chicken, doves, hawks, etc.

**MORPHOLOGY:** This organism has been found in natural infections in the upper digestive tract of pigeons, turkeys, chickens, ring doves, mourning doves, sparrow hawks, falcons and Java sparrows. *In addition it has been possible to infect a number of other birds.* Trichomonas gallinae is roughly pear-shaped and measures 2.3 to 8.5 microns in width and 6.2 to 18 9 microns in length (average 10.5 microns in length by 5.2 microns in width). There are four anterior flagella The marginal filament of the undulating membrane extends down one side to a point two thirds to three-quarters the total length of the body. It does not terminate as a trailing flagellum. There is a blepharoplast at the anterior end from which arises the flagella, axostyle, costa and parabasal complex. The nucleus is situated in the anterior part of the body, it is oval in shape and possesses one or sometimes two karyosomes. The parabasal body is located in the anterior third of the body and is associated with the parabasal fibril which extends almost to the posterior end of the body. The axostyle passes through the long axis of the body and protrudes for a short distance posteriorly The costa is a conspicuous fibril extending from the blepharoplast along the base of the undulating membrane to the posterior tip of the marginal filament. A cytostome is present, but is usually difficult to demonstrate

**TRANSMISSION.** The exact requirements for the transmission of this organism in chickens and turkeys are not known, but in pigeons it is passed directly from the carrier mother to the squab during the feeding process. Cauthen (1936) reported that day old chicks fed a normal mash diet failed to become infected with T. gallinae, whereas, if fed a mixed whole grain cracked corn ration infection took place although no lesions were observed. Levine and Brandly (1940) found that the susceptibility of chicks to this parasite varied with the source of the chicks. While most chicks were very resistant to infection, one lot from a different source was relatively susceptible and lesions were produced in a number of them. Stabler and Engley (1946) infected trichomonad free squabs with a strain of this protozoan with and without bacteria, which had been isolated from a falcon which died of the infection. The bacteria isolated from the falcon produced no demonstrable lesions, whereas the bacteria free culture of T. gallinae killed 15 to 17 squabs which were infected.

**SYMPTOMS:** There are no symptoms pathognomonic of this condition. The disease in chickens and turkeys is sporadic in character, however, it is usual in these birds to have pathological lesions produced with the presence of the parasite. However, in the pigeon 80 to 90 per cent of the adults harbor the organism and never show any symptoms of the disease. In pathogenic strains of this organism the infection will run a rapid course and result in the death of the bird about 10 days after infection. Birds which die are usually in poor flesh and before death stand huddled with the feathers ruffled and when forced to walk will frequently topple over. Almost invariably there is an excessive accumulation of greenish fluid in the mouth which on examination will contain large numbers of trichomonads.

**PATHOLOGY:** The pathology associated with this disease varies with the different hosts. Since the pigeon is probably the normal type of host the lesions in this bird will be described first. Stabler and Engley (1946) noted the earliest visible lesions appeared in the mouth cavities. These are most common in the soft palate, although the pharyngeal regions may also be severely affected. Though less involved than the mouth cavities, the esophagus, crop and proventriculus may also be involved. The digestive tract from the proventriculus posteriorly is not affected by this organism. In the body cavities lesions in the liver, lungs, small intestine (on the serous surface), pancreas and heart are sometimes noted. Apparently all of the lesions in these organs spread from original infections of the liver. This area is almost as severely affected as the mouth cavities.

The first sign of infection in the pigeon is a small yellowish area on the oral mucosa which appear 3 to 14 days after infection. These generally increase in size and number and frequently reach such proportions that the esophagus and trachea are completely blocked. The tissues in the roof of the mouth are frequently invaded producing large caseous masses and occasionally include the bones in the floor of the skull. The early lesions in the crop and esophagus are small white nodules whose contents are semi-caseous and may be expressed on pressure. Later, circumscribed necrotic areas yellowish grey in color appear, which are firmly attached to the mucosa, may occlude the lumen of the crop and esophagus. The gizzard and the small and large intestines are never seen to bear lesions internally. Lesions of the liver are quite common and are small solid caseous areas similar to those seen in the oral cavities. These range from a few small foci to others which may represent 90 per cent of the entire liver. The other lesions noted in the body cavity appear to be merely contact extensions from the liver.

In the chicken and turkey the main pathology occurs in the crop with less frequent involvement of the lower esophagus and proventriculus. Lesions in the oral cavity and liver are rare in these birds.

DIAGNOSIS: The condition can usually be diagnosed in the pigeon by opening the mouth and observing the oral lesions. It is confirmed by a microscopic demonstration of the trichomonads.

TREATMENT: There is no effective treatment for this disease at the present time. Hinshaw (1943) has ascribed some success to the use of a 1:2000 solution of copper sulfate in place of the drinking water. This solution should be kept before the birds for two or three days and then repeated after a few days if improvement is not noted. All other sources of water should be removed during such treatment.

Recent experiments by the United States Department of Agriculture indicated that an aqueous solution of copper sulphate at a ratio of 35 milligrams in 100 cc of water (1.4 grams per gallon) substituted for drinking water cured the disease in pigeons. At this dilution the organisms in pigeons with young were cured within 76 to 180 days. A concentration of 100 milligrams per 100 cc of water (4.2 grams per gallon) freed nonbreeding pigeons of trichomonads of the upper tract in a period of 20 days.

CONTROL: As noted above the transmission of this disease in chickens and turkeys is not well understood, therefore, no concrete proposals can be given for its control. As it seems to be primarily associated with insanitary surroundings, measures should be taken toward the correction of feeding, watering, housing and general management of the flock. As soon as the disease is noted the affected birds should be segregated, and the rest of the flock provided with clean dry facilities. In the case of pigeons infection is impossible to prevent with our present knowledge due to the parental method of feeding the young birds. Thus, the squabs, due to the regurgitational method of feeding, are infected at their first meal, if the parent birds are infected.

GENERAL REMARKS: Stabler (1947) has demonstrated strains of T. gallinae which differs greatly in virulence. He found different strains of this organism which produced no visible pathology to those which killed four out of five birds. Infection with less virulent strains of this organism will confer some protection against the damage produced by the virulent strains.

### Trichomonas anseri Hegner, 1929

HOST: Geese.

This species occurs in the ceca of the goose and has been transferred successfully to chicks. The body is oval in shape and averages 7.9 microns in length and 4.7 microns in width. There are four anterior flagella which arise from two anterior blepharoplasts. A marginal filament passes posteriorly bordering the undulating membrane and terminates as a trailing flagellum near the posterior end of the body. The axostyle is broad, hyaline and protrudes considerably at the posterior end. A large cytostome is present. It is apparently nonpathogenic.

TREATMENT: Unnecessary.

Tritrichomonas eberthi (Martin and Robertson, 1911)

**HOST:** Chicken.

This is the second trichomonad which has been described from the cecum of the chicken. It is carrot shaped, measuring 4 to 6 microns in width and about 9 microns in length. There are three anterior flagella and one marginal filament bordering the undulating membrane. The filament terminates in a trailing flagellum. The pathogenic significance of this species in fowl is not known.

**TREATMENT:** Unknown, probably unnecessary.

Family: Monocercomonadidae

Monocercomonas gallinarum (Martin and Robertson, 1911)

**SYNONYM:** Eutrichomastix gallinarum.

**HOST:** Chicken.

This flagellate was first described by Martin and Robertson from the ceca of chickens. It has been observed on several occasions by Morgan (unpublished data) from Wisconsin chickens. The body is pear-shaped, and measures from 5 to 8 microns long and 3 to 4 microns wide. There are three anterior flagella and a longer trailing flagellum. It is apparently nonpathogenic.

**TREATMENT:** Unnecessary.

ORDER: POLYMASTIGINA
Family. Cochlosomidae

Cochlosoma rostratum Kimura, 1934

**HOSTS:** Ducks, turkeys.

Kimura (1934) described this species in the intestines of Muscovy and white Pekin ducks in California. The organism measures 6 to 10 microns long and 3.9 to 6.7 microns wide. It is oval with the broad end anterior. There are six flagella, four posterolateral and two trailing. A sucking disc and an axostyle are also present. Living specimens move in a constant circle in a small radius. The parasite was more abundant on ranch raised birds than in market birds.

McNeil and Hinshaw (1942) reported this flagellate from turkey poults in California. It was found throughout the intestinal tract and always in association with Hexamita or in combination with Hexamita and Salmonella. The significance of this protozoan in turkeys or ducks has not been determined.

**TREATMENT:** Unknown, probably unnecessary.

Cochlosoma anatis Kotlan, 1923

**HOST:** Ducks.

The species was described from the droppings and intestinal mucus of ducks by Kotlan (1923). Travis (1938) observed the organism in various species of wild ducks and tame mallards. The organism has a sharply outlined depression at the anterior end of one side, similar to the sucking of disc of Giardia. It measures from 6 to 7 microns wide and 10 to 12 microns in length. There are 6 flagella arising from the anterior end, all of which are directed posteriorly adjacent to the surface of the body. Two axial fibrils, which seem to be similar to an axostyle, arise from a granule near the blepharoplast and pass through the body to the posterior end. Oval cysts with four or more nuclei are formed. The pathogenic significance of this species is unknown.

**TREATMENT:** Unknown and probably unnecessary.

Family: Chilomastigidae

Chilomastix gallinarum Martin and Robertson, 1911

**HOST:** Chicken.

This protozoan is commonly found in poultry. It possesses a pyriform body and measures from 11 to 13 microns in length and 5 to 6 microns in width. The cytostome has the shape of a large pouch and extends from the anterior end backwards to almost the middle of the body. The margin of the cytostome is supported by fibrils. There are three anterior flagella and a fourth flagellum which vibrates in the cytostome along the border as a small undulating membrane. Cysts are formed which are 7 to 8.5 microns in length and 4 5 to 5 5 microns in width. It has no pathological significance.

**TREATMENT.** Unnecessary.

Family. Hexamitidae

Hexamita meleagridis McNeil, Hinshaw and Kofoid, 1941

**DISEASE:** Hexamitiasis, infectious catarrhal enteritis.

**HOST·** Turkey.

**MORPHOLOGY:** In the acute stages of this disease large numbers of the parasites may be found in the small intestine, and in fewer numbers in the cecum, and in older birds in the bursa of Fabricius The movement of the living organism is very rapid and can usually be readily differentiated from the jerky turning type of motility of trichomonads. There is also a tendency for these organisms to cling to bits of tissue with their flagella. The organism measures 6 to 12 microns in length and 2 to 5 microns in width averaging 3 microns wide and 9 microns long.

The organism is bilaterally symmetrical. The two nuclei in stained specimens are distinct and possess karyosomes about two thirds of the diameter of the nucleus. Anterior to the nuclei are two large blepharoplasts from which arise four anterior and two anterio-lateral flagella. The anterior flagella usually curve back posteriorly Just posterior to the two large blepharoplasts are two smaller ones from which arise the two caudal flagella, which pass posteriorly in the cytoplasm and emerge near the posterior end of the body. The posterior flagella are quite long sometimes being two to three times the length of the body of the protozoan. These are the flagella which appear to attach themselves to pieces of tissue and in these cases the living organisms will appear to swing back and forth like a pendulum. Cysts are not produced by this parasite.

**LIFE HISTORY:** The life cycle of this organism has not been completely worked out, but it is direct, with transmission occurring through contaminated food and water. The adult turkey is the most important source of infection, more than one third of the birds remain infected after recovery from the disease. Chickens have never been found to be infected. In addition to the turkey, similar organisms have been found to occur naturally in the California valley quail, Gambel's quail and chukar partridges. These forms are probably not the same species as occurs in the turkey, although appearing morphologically identical. Cross infection took place with some forms but no disease was produced in the turkey.

**SYMPTOMS:** Turkey poults 1 to 9 weeks of age are most susceptible to this infection. The presence of other diseases increases the susceptibility of the poults. If there are several age groups on a farm those from the first hatch show only slight or no ill effects from the infection. However, as the number of organisms increase on the premises birds from later hatches have been found to suffer severely. There are no specific symptoms associated with this disease. The birds seem to require more heat than normal, the gait is stilted, and the feathers appear ruffled. There is a foamy watery diarrhea, but the cecal droppings do not appear to be changed. Most of the birds will continue to eat and

in some instances will even consume more food than is normal, but due to impaired digestion they will lose weight very rapidly.

In the later stages of the disease the poults become listless, sit under the hovers and finally die. In experimental infections the course of the disease is very rapid with symptoms appearing 4 to 7 days after infection with a heavy inoculum. Mortality in such infections will usually start a day after the first appearance of symptoms and the peak in death losses is reached 7 to 10 days later. Heavy losses in turkey poults do not occur in birds over 8 weeks of age unless there is some other factor which lowers the resistance of the birds. Mortalities as high as 70 and 80 per cent have been recorded in natural outbreaks. Large numbers of stunted individuals result from the infection with Hexamita meleagridis.

**PATHOLOGY:** Infected birds are in very poor condition due to their lack of ability to properly utilize their food. In the upper intestinal tract there is a catarrhal enteritis and a marked lack of tone. The contents of this area vary from a thick mucus type of exudate to a thin watery foamy material The walls are often thin with localized bulbous areas filled with watery material. None of the pathological changes are pathognomonic of the disease.

**DIAGNOSIS:** A positive diagnosis of hexamitiasis can only be made with the demonstration of the organism. Care must be taken not to confuse Hexamita meleagridis with other protozoa which may occur in the small intestine, although if it is present other forms are usually absent. However, trichomonads and Coohlosome have been found in the turkey.

**CONTROL:** No general procedures can be given to prevent infections with this parasite as they will vary in different cases. Hinshaw and McNeil (1940) have given certain recommendations by means of which they have been able to prevent the infection. They are as follows: (1) Separate units and caretakers for the breeding flock and the young poults, (2) separate equipment for each age group, (3) use of cement floors and wire pens, (4) feeders and watering equipment arranged so that the attendant does not need to enter the pen, (5) if the poults have undergone an attack of pullorum disease avoid changes in brooding until they are 12 to 16 weeks of age and (6) seal all breeding birds 2 weeks before any poults are hatched is advised wherever possible.

**TREATMENT:** At the present time there is known treatment for this infection. All remedies either in the feed or drinking water should be avoided. Nursing and good management are of the greatest importance in preventing severe losses from the disease.

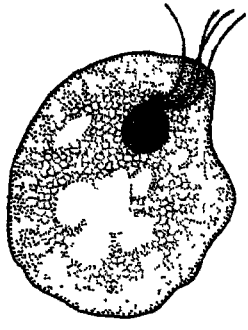
**GENERAL REMARKS** This disease was first reported from California where the etiological agent was discovered by Hinshaw and his co-workers. Since then it has been reported in the mid-west and on the east coast Consequently, it may be assumed that the disease is fairly widely distributed throughout the United States.

### Hexamita columbae (Noller and Buttgereit, 1923)

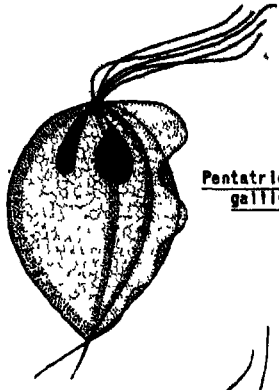
**HOST.** Pigeon.

McNeil and Hinshaw (1941) reported on the first cases of Hexamita from pigeons in the United States. Most of the birds examined showed a catarrhal enteritis. The organisms were recovered in scrapings from the jejunum, ileum and rectum. Turkeys could not be infected with H. columbae. It is not likely that Hexamita meleagridis occurs in pigeons, since pigeons were examined and found free of Hexamita, whereas, Hexamita meleagridis was abundant in turkeys on the same farm.

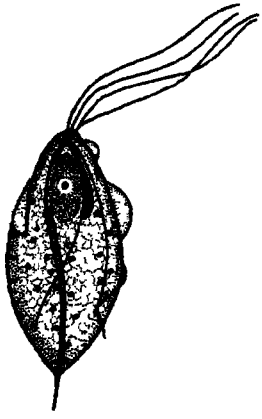
**TREATMENT:** Unknown.



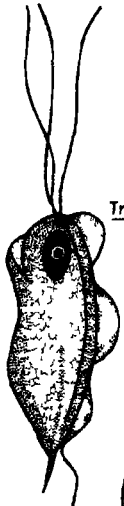
Histomonas meleagridis



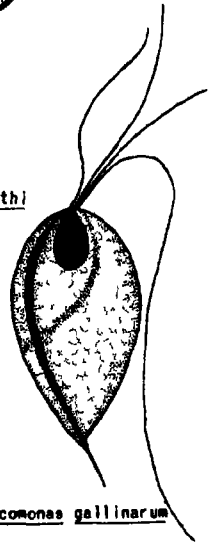
Pentatrichomonas gallinarum



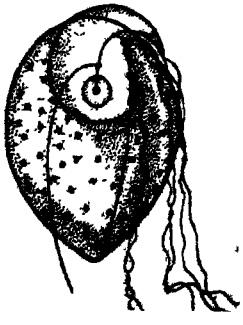
Trichomonas gallinae

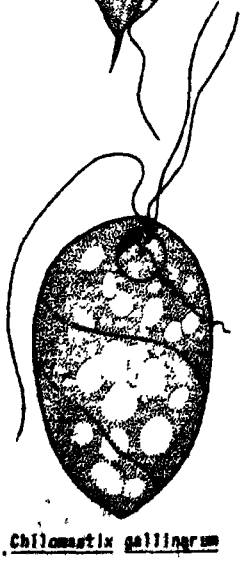


Tritrichomonas eberthi

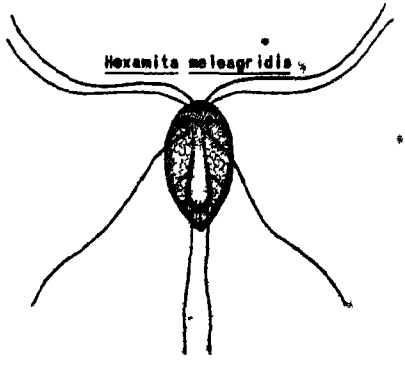


Monocercomonas gallinarum





Chilomastix gallinarum



Hexamita meleagridis

Hexamita sp.

**HOSTS:** California Valley quail, Gambel's quail, chukar partridge.

McNeil, Platt and Hinshaw (1939) reported on Hexamita occurring in quail and partridge from California. The species in quail very closely resembles the one found in pigeons and turkeys. The species from turkeys has been transmitted to quail but attempts to transmit the quail form to turkey poults and chicks failed. The species in chukar partridge can be transmitted to turkeys.

Many of the birds showed ulcerative enteritis, but whether Hexamita have any etiological relationship to this condition is not known The distribution of Hexamita in the intestinal tract of quail include the ileum, jejunum, duodenum, bursa of Fabricius and cecum. The form in quail measured 5 8 microns in length by 2.3 microns in width.

**TREATMENT:** Unknown

**CLASS:** SARCODINA
**ORDER:** AMOEBINA
Family Endamoebidae

Endamoeba gallinarum (Tyzzer, 1920)

**HOSTS:** Chicken, turkey.

Tyzzer (1920) first described this species from the cecal excrement of young turkeys and chickens. The trophozoites measured from 9 to 25 microns in diameter, average 16 to 18 microns. These forms are quite active at room temperature. The nucleus measures from 3 to 5 microns in diameter. The endosome is centrally located.

The cysts contain 8 nuclei when mature although immature quadrinucleate forms are often observed. The cysts are spherical and measure 12 to 15 microns in diamter.

This amoeba has not been reported to be pathogenic in poultry.

**TREATMENT:** Unnecessary

Endamoeba anatis (Fantham, 1924)

**HOST:** Duck.

*Fantham (1924) originally described this species from the feces of a duck which had* died of an acute enteritis. The morphology of the amoeba is similar to E. histolytica. Further study should be made on this organism.

**TREATMENT:** Unknown.

Endolimax numidae (Hegner, 1929)

**HOST:** Guinea fowl.

Hegner (1929) described this small amoeba from the guinea fowl The average size is 4.2 by 3.4 microns. The mature cyst contains 4 nuclei. It is apparently nonpathogenic.

**TREATMENT:** Unnecessary.

Endolimax gregariniformis (Tyzzer, 1920)

**SYNONYMS:** Pygolimax gregariniformis Tyzzer, 1920; Endolimax janisae Hegner, 1926.

**HOSTS:** Turkey, chicken.

This amoeba was described by Tyzzer (1920) from the ceca of turkeys. The vegetative forms are oval in shape and measure 4 to 13 microns in size, average measurements were 8.75 by 5.5 microns. Their movement is slow at room temperatures. The ectoplasm is not differentiated from the endoplasm. The cytoplasm contains various numbers of food vacuoles harboring bacteria and other types of food The nucleus is from 1.5 to 2.0 microns in diamter; a karyosome is centrally located. Cysts have not been completely described.

Tyzzer (1920) was able to transmit E. gregariniformis to young chicks by feeding small amount of feces of infected adult birds. Large numbers of trophozoites were observed 4 days after ingestion.

**TREATMENT.** Unnecessary.

**CLASS: SPOROZOA**
**ORDER. COCCIDIA**
Family. Eimeriidae

Eimeria tenella Railliet and Lucet, 1891

**SYNONYM:** Eimeria avium (Rivolta, 1878).

**DISEASE:** Coccidiosis of chickens, cecal coccidiosis.

**HOST.** Chicken.

**MORPHOLOGY:** The oocysts of E tenella are passed unsporulated in the feces of infected chickens. No authenticated report of birds passing sporulated oocysts has been recorded They are of a broad, ovoid shape with no great difference in the width of the two ends. As in most species of coccidia there is a considerable variation in the size of the oocysts, they measure 19.5 microns to 26 microns in length, and from 16.5 microns to 22.8 microns in breadth with an average of 19.0 microns wide, and 22.6 microns long. The cytoplasmic mass of the oocyst is slightly irregular when discharged and is separated from the cyst wall by a relatively large clear space The cyst wall is quite smooth and there is no apparent micropyle at the smaller end. Within the cyst wall at the anterior end there is a small bright refractile granule.

When the oocyst is kept at room temperatures with sufficient oxygen and moisture it will sporulate in about 48 hours. The sporulated oocyst contains four spores, each containing two sporozoites. The spores are bluntly ovoid in shape and measure about 7 microns wide and 11 microns long. At the smaller end of the spore is a small globular plug filling an opening in the spore wall and projecting slightly outward. The sporozoites are small sausage shaped forms, two in each spore, showing a globular mass of hyaline material near one end, and near it, a space devoid of any granules which is thought to be the nucleus. A residual mass is present.

**LIFE CYCLE:** Bionomic of the oocyst: A number of studies have been carried out on the bionomics of the oocysts of E. tenella, but to the present time most of them have been carried out under laboratory conditions and thus it is difficult to determine the resistance of these oocysts to many environmental conditions. Ellis (1938) found that eggs in a commercial incubator at a humidity of 47 per cent and a temperature of 39° C. which had been smeared with viable oocysts of E. tenella carried only dead oocysts at the end of the period of incubation. This has been confirmed by most workers. The temperature at which the oocysts are killed will depend on the length of time the oocysts are exposed.

Chang (1937) has determined the time for a 50 per cent kill of oocysts and has observed a linear relationship between temperature and time.

Fish (1932) has observed a thermal death time of 10 minutes of sporulated and unsporulated oocysts at a temperature of 53° to 54° C. These relatively low temperatures which kill coccidial oocysts should not encourage the use of heat in their destruction in the poultry house. Boiling water used in cleaning floors and other permanent fixtures would be so rapidly cooled that in most instances these temperatures would never be reached. The flame-gun and blow-lamp which is advertised to kill coccidial oocysts is extremely inefficient as Horton-Smith and Taylor (1939) have shown that it may require as long as 16 hours to kill the oocysts in a chicken house 20 by 12 feet.

Patterson (1933) has shown that continuous, or intermittent freezing of oocysts in soil has no lethal effect for at least 12 weeks, although one of us has noted that freezing and thawing of the exposed oocysts every two days for two weeks results in a 100 per cent mortality. Patterson also observed that oocysts of E. tenella in feces were killed in four weeks by drying.

It has long been known that the oocysts of the coccidia are extremely resistant to the commonly used disinfectants, yet many poultrymen want some compound that may be sprinkled about the poultry house to kill oocysts. It cannot be emphasized too strongly that no chemical exists which will have any effect in the presence of an excess of organic matter such as straw, feed and feces. The studies of disinfectants made on compounds cited below are tests carried out on exposed oocysts, not oocysts protected by organic material. For any practical value a compound should kill the oocyst very rapidly. Anderson and Mallman (1943) have shown an interesting relationship between the type of solvent used in iodine compounds and also the marked efficiency of iodine suspensoid They point out that iodine suspensoid (collodial iodine) is the only compound they tested which possessed marked powers of penetrability, a characteristic so necessary for the destruction of oocysts.

Fish (1932) has shown that ultraviolet light has a marked lethal effect on oocysts Three-fourths of a zinc sulphide unit (Clark) at a distance of 22 inches killed unsporulated oocysts, whereas 2 zinc sulphide units (Clark) were necessary to kill sporulated oocysts. The greater resistance of sporulated oocysts would be expected due to the extra protection of the sporocyst wall.

Studies of the survival of the oocysts of E tenella are not numerous and most have not been carried out under natural conditions These studies have consisted either of exposing samples of artificially infected soil for varying periods of time under different environmental conditions, or of taking naturally infected soil and feeding relatively large quantities to susceptible chickens. Warner (1933) found that soil naturally infected with oocysts in July was still infective for birds in 197 days, but not in 217. However, in soil exposed in December, infective stages were still present in 49 days but not 81. Delaplane and Stuart (1933) in Rhode Island found that soil naturally infected with oocysts of Eimeria sp was still infective after four months on a sunlight exposed plot, 9 months on a swampy plot and 15 to 18 months on a wooded range.

Not a great deal is known about the resistance of the oocysts of E. tenella when ingested by various insects, although undoubtedly, they play some part, probably a minor one, in the transmission of cecal coccidiosis. Baker (1933) has noted that flies which had been caught in poultry yards were capable, when fed to coccidia free chicks, of initiating an infection with this parasite in a few instances. None of the infections which were produced were serious in nature. Delaplane and Stuart (1933) have demonstrated both sporulated and unsporulated oocysts in the gut of larvae of the common house fly, but when these larvae were allowed to pupate and develop into mature flies, and fed to coccidia free birds were not capable of producing infection. Metelkin (1935) has shown that oocysts will remain viable in various species of flies up to 24 hours and in the discharges of these flies until they have dried.

DEVELOPMENT OF THE OOCYST: If the oocysts are kept at temperatures of 20° to 25° C. evidences of sporulation will be noted in a short time. It has been noted when the oocyst is discharged in the droppings the cytoplasmic mass is frequently of a slightly irregular shape. At room temperatures, and with adequate moisture these irregularities become accentuated. Within 24 hours the single cytoplasmic mass divides into four cells, or sporoblasts, which soon assume a smooth oval shape. About each sporoblast is formed a cyst wall, or sporocyst. The cytoplasm inside the cyst will then divide into two sporozoites. Thus at the end of 48 hours each sporulated, infective oocyst contains four spores, each enclosed in a sporocyst, and each spore contains two sporozoites, or eight sporozoites in the sporulated oocyst. The sporulated oocyst is the only infective stage of the coccidium, the unsporulated oocyst being incapable of producing infection.

DEVELOPMENT IN THE HOST: Infection in the chicken takes place under natural conditions through the ingestion of the sporulated oocyst and the liberation of the contained sporozoites in the small intestine. Pratt (1937) has observed that the spores may be liberated in the crop, as well as further down the intestinal tract. He has also described the liberation of sporozoites in the crop. Instead of the sporozoites being liberated from the spore, and the sporozoite escaping from the oocyst, Pratt noted that the spores were liberated by a fracturing of the oocyst wall.

Goodrich (1944) has made this same observation on the method of liberation of the spore from the oocyst, and liberation of some of the spores in the crop, but she did not note the presence of any sporozoites in the crop. She also noted this liberation of spores could be accomplished in vitro, which has been confirmed by one of us.

Goodrich noted that the presence of trypsin is necessary for the liberation of the sporozoites, and this could be accomplished in vitro by the addition of a 0.5 per cent trypsin to the sporulated oocysts. In feeding sporulated oocysts to chicks she has found that one hour after infection there were many intact oocysts in the crop and gizzard. One and three quarters hours after infection there were many broken or empty oocysts in the duodenum. Three hours after infection practically all the oocysts were in the rectum and most of them were empty.

Levine (1942) failed to obtain infection of chickens with E. tenella or other species of coccidia of this host when the pancreatic ducts were ligated. Herrick and Edgar (1942) have found that in birds from which feed has been withheld from the previous evening until 7 to 8 o'clock the following morning the sporozoites reached the cecum in about one hour. Whereas, in birds that had feed before them continuously, two and one-half to four hours were required before the sporozoites or oocysts reached the ceca.

The liberated sporozoites average about 12.8 microns in length, and in general are crescentric in outline, tapering to a point anteriorly and bluntly rounded posteriorly. They are sluggishly motile when kept at the body temperature of the chicken.

The descriptions of the various stages in the tissues were made by Tyzzer (1929). It seems that all of the sporozoites do not penetrate the epithelium of the cecum at the same time, and this will result in a lag in the development of the future generations in the tissues. The sporozoites penetrate the epithelial cells in the fundi of the cecal glands. This initiates those asexual stages of development designated as Generation I. The sporozoite in the epithelial cell is known as a trophozoite and is characterized by the presence of a small globule of eosinophilic material. With the growth of the Generation I trophozoite the nucleus begins to divide. As the parasite undergoes schizogony it is known as a schizont. The invaded epithelial cell increases greatly in size and there is hypertrophy of the cell nucleus. Finally, the cytoplasm of the Generation I schizont divides about the numerous nuclei and the enclosed merozoites are liberated by a rupture of the host cell either while it is in the intact epithelial layer, or after it is squeezed out into the lumen of the gland. It has been estimated that about 900 Generation I merozoites are formed. The merozoites of the first generation are very small, thick fusiform bodies, 2 to 4 microns in length and 1 to 1.5 microns in width. They are liberated into the lumen of the cecum 2-1/2 to 3 days after the host has been infected.

After their liberation into the lumen of the cecum the Generation I merozoites soon penetrate other epithelial cells in the same area in which they were liberated. They penetrate the epithelium and do not commonly penetrate below the nuclei of the epithelial cells. Then the trophozoite which develops, initiates a second asexual generation, or Generation II.

The penetration of the epithelial cells by the first generation merozoites initiates an immediate response on the part of the host cell, which undergoes marked changes. As in the previous generation the cell begins soon to increase in size, but in this generation the cells no longer form a continuous layer with the epithelium. Next the cells seem to become independent units and migrate into the subepithelial tissues, and infiltrate the surrounding tissues so that all of the tissues between the glands become filled with the developing stages of the parasites. The unparasitized epithelium grows together over the parasitized cells.

The schizonts which develop from the Generation II trophozoites are much larger than those of the preceding generation. The nuclei of the schizonts appear to be in vesicles without any limiting nuclear membrane. The schizonts are quite large, averaging 31 microns by 21 microns, although some may be as large as 40 microns by 54 microns. The merozoites are apparently formed by an almost simultaneous segmentation of the cytoplasm about the nuclei, rather than by a process of budding which has been described in some forms. It has been estimated that about 200 to 350 Generation II merozoites are formed from the segmentation of the cytoplasm. The merozoites are much longer and relatively more slender than those produced by the first generation, the average size being 2 microns by 16 microns. The mature merozoites are found packed together in the submucosa, and are covered, in the case of a single infection, by the unparasitized epithelium.

As Generation II reaches maturity the first signs of hemorrhage into the ceca are noted. This occurs during the fourth day after infection, and about 24 hours prior to the liberation of the merozoites from the parasitized cells. This has been described as being due to a widespread leakage, rather than a localized extravasation. Copious hemorrhages may be noted in the droppings of birds five days after infection. At this time the parasitized epithelial cells in the lamina propria, by virtue of their number and size, rupture the intact epithelium covering them and liberate the merozoites into the lumen of the cecum. This results in widespread sloughing of the mucosa and, consequently, considerable hemorrhage. The nucleus is found slightly posterior to the middle of the body, and the chromatin is contained in the ill-defined nuclear vesicle which also occurred in the earlier schizont. There is usually a very small granule at or near the sharp posterior extremity. Their movement is similar to that of the sporozoite.

Due to the enormous numbers of merozoites produced many will be passed out of the body of the host, rather than penetrate new epithelial cells. If it may be assumed that on the ingestion of a sporulated oocyst the eight sporozoites succeed in penetrating epithelial cells, that each sporozoite results in the production of 900 Generation I merozoites and each first generation merozoite results in the production of 350 Generation II merozoites, then 2,440,000 (8 x 350 x 900) second generation merozoites result from one oocyst.

The second generation merozoites that succeed in penetrating an intact epithelial cell will usually develop into sexual forms, although a small number may initiate a third asexual generation. The Generation II merozoite usually passes beneath the nucleus after penetrating the new cell. The Generation III schizont which develops average 7.6 by 9 microns and remains in the epithelial layer. Only small numbers of merozoites are produced, varying from four to thirty. They are also smaller than those of the second generation, averaging 1 by 6.8 microns. The schizonts of the third asexual generation are often difficult to find owing to their small numbers. How many more generations may be produced of asexual forms is not known. On liberation, the merozoites of this generation will probably initiate a sexual cycle.

The sexual stages found in the epithelial layer of the third generation predominate and are similar to those described in many other species of coccidia. The early stages of development cannot be differentiated, but as growth proceeds it is evident there are

two types being produced. The microgametocyte (or male gametocyte for convenience) undergoes numerous divisions of the nucleus, resulting finally in the formation of a number of very small, biflagellated microgametes. These are found in groups varying in size from 5.5 microns to 18 microns in diameter.

The macrogametocyte ( or female gametocyte) increases in size but does not undergo a division of the nucleus. As the macrogametocyte develops it is very noticeable by the presence of numerous haematoxylinophilic granules scattered throughout the cytoplasm. As they reach maturity the granules arrange themselves about the periphery of the organism. These granules are the precursors of the cyst wall. As the cyst is formed the granules slowly disappear. While the cyst wall is developing the macrogamete assumes the form of an oocyst, but until after fertilization occurs there is no shrinking of the cytoplasm away from the wall. Fertilization of this species has not been observed It is not known whether this occurs while the macrogamete is still in the epithelial cell, or after liberation into the lumen of the cecum.

After fertilization the oocysts are discharged in the droppings of the host seven days after infection. In some instances the macrogamete, or oocyst fails to be released from the host cell and becomes encapsulated. These forms may be found not only retained in the epithelial cell, but may even be found beneath the muscularis mucosa, probably carried there by giant cells. Herrick, Ott and Holmes (1936) have demonstrated viable oocysts in the ceca of birds as long as 7-1/2 months after a single infection.

COURSE OF THE INFECTION: The prepatent period is seven days. However, the time required to detect the parasite by routine laboratory techniques might also be the time required for the passage of either the first or second generation merozoites, since they could be detected by microscopic examination of the feces. This time is greater than the incubation period, e.i., the symptoms of hemorrhage occur first.

The patent period varies in E. tenella infections, but is generally considered to be self limiting. Fish (1931) found that oocysts were not recovered in droppings of infected birds after 17 days. Tyzzer, Theiler and Jones (1932) recorded 19 days as the longest period of oocyst discharge Thus, the greatest numbers of oocysts are discharged in a very short time, the few remaining either being trapped in the tissues or in a cecal plug and only irregularly released. Under natural conditions it is to be expected that birds will be infected repeatedly and thus may pass oocysts for a much longer period. Levine (1940) found oocysts of E. tenella in 9 of 30 birds examined which showed no symptoms of infection.

Johnson (1927) has pointed out that the severity of coccidiosis is chiefly dependent on the number of sporulated oocysts which the susceptible bird receives. It is possible to infect chickens with one sporulated oocyst of E. tenella. An infection of one oocyst will produce no recognizable symptoms, nor will dosages of slightly larger numbers. Although the actual numbers of oocysts required to produce symptoms or death vary, as reported by different investigators, the numbers are fairly consistant considering the possibilities of variation in strains of coccidia which are isolated in different parts of the country.

Jankiewicz and Scofield (1934) reported that a dosage of up to 150 sporulated oocysts produce neither symptoms nor mortality; dosages from 150 to 500 produce slight hemorrhage, but no mortality; 1000 to 3000 sporulated oocysts a fairly heavy degree of hemorrhage and a light mortality, 3000 to 5000 produce marked hemorrhage and a moderate mortality; while over 5000 oocysts produced a severe hemorrhage and a higher mortality.

The course of E. tenella infections is very constant. The entire cycle almost occurring like clockwork, and is not disturbed by environmental factors. The blood first occurs in the fourth day after infection, marked hemorrhage on the fifth and sixth days and the appearance of oocysts is noted on the seventh day.

SYMPTOMS: The symptoms of the disease are closely related with the course of the infection. There are no symptoms until the second generation schizonts begin to greatly enlarge and cause widespread leakage of blood into the ceca four days after infection.

At this time the birds will appear to be listless. On the fifth and sixth days the infected birds are inactive, consume small quantities of feed and pass large quantities of blood in the droppings. The passage of blood is associated with the breaking out of Generation II merozoites, and widespread sloughing of the cecal mucosa. Since the birds consume less feed, they may drink two or three times more water than uninfected birds. Approximately 90 per cent of the mortality occurs in the first week following infection. If the birds do not die at this time recovery may be assumed. After the seventh day small amounts of blood are still being passed in the droppings

PATHOLOGY: The lesions associated with infections of Eimeria tenella occur primarily in the ceca, and have been well described by Tyzzer (1929), Tyzzer, Theiler and Jones (1932) and Mayhew (1937). The dilated part of the cecum is primarily involved. If birds are killed on the fourth day after infection hemorrhage is found throughout the cecal mucosa. On the fifth day the cecum will be filled with large quantities of usually unclotted, or only partially clotted blood. At this time the feathers and skin about the vent are smeared with blood. By the sixth day after infection the cecum is grossly dilated with clotted blood. By the seventh day cecal cores may be found. If the cecum is opened the core is tightly adherent to the mucosa After the seventh day, the core is free in the lumen.

Approximately seven days after infection the cecum turns from a reddish color, to a mottled reddish or milky white due to the presence of numbers of oocysts. The cecal wall is greatly thickened. The core or clot is now a whitish or yellowish color. Occasionally these cores are passed intact in the droppings of the infected bird. In most instances they are broken up in small pieces and are not grossly recognized. After infection the cecum returns to approximately normal size and gross appearance, although it may be slightly larger and thicker. Unusual gross changes occur occasionally due to the retention of large cores, rupture of the cecum or adhesions.

When the Generation II schizonts are developing in the lamina propria this area is infiltrated with eosinophiles. At this time a marked congestion is noted and the wall of the cecum is thickened due to the engorgement of the vessels with blood. In areas where the protozoa are most numerous the epithelial layer may be torn apart and some of the organisms, blood and tissue cells allowed to escape into the lumen. On the fifth day the Generation II merozoites begin to rupture and is associated with extensive sloughing of the epithelium. However, as regeneration of the cecal epithelium occurs the core loosens and is free in the lumen.

With light infections the regeneration of the epithelium is complete In more severe cases recovery is associated with a slow and often incomplete regeneration of the mucosa. A marked inflammatory reaction occurs with extensive infiltration with lymphoid and plasma cells, some giant cells and an increase in connective tissue. In regions of severe hemorrhage the epithelium is not replaced between the glands and the mucosa consequently remains exposed to the contents of the cecum. Numerous cysts may occasionally be seen in the cecal mucosa due to the constriction of the glands through inflammatory processes. Oocysts may be found in these pinched off glands, both in the epithelium and lumen. Sometimes oocysts occur in giant cells beneath the muscularis mucosa.

There may be severe after effects resulting from outbreaks of cecal coccidiosis. Birds which have been infected with E. tenella, are incapable of utilizing their feed as efficiently as birds which have not been infected. Mayhew (1932) showed that at least 10 to 12 weeks pass after an infection before infected birds again equal or are comparable in weight to uninfected birds. Mayhew (1934) also demonstrated that chicks which had been infected at the age of six to eight weeks laid 19.25 per cent fewer eggs. They did not start to lay until six or seven weeks after the uninfected birds. Davidson, Thompson and Moore (1936) studied positive and negative groups as determined by the presence of oocysts in the droppings. They found groups passing oocysts showed a 121 per cent higher mortality; the negative group on hen-day basis egg production was 15.2 per cent higher, while it averaged 0.44 pounds heavier than the positive group. This study was carried on over an 11 month period.

## BIOLOGY

**IMMUNITY:** There is a marked immunity to infections with E. tenella. Numerous experiments have been performed trying to infect other birds and animals with this organism, and despite a few reports of successful transfer the known host is the chicken. There is also a marked organ specificity for the ceca. The parasite does not occur in the small intestine, although it may occasionally produce lesions in the large intestine and extreme lower portion of the small intestine as reported by Tyzzer (1929). Herrick (1936) joined one cecum to various levels of the small intestine and left the other in its normal location. This resulted in producing one physiologically abnormal cecum, while the other was normal. In those birds in which the cecum was united, either to the midpoint of the duodenal loop or posterior to this point, infection occurred in both ceca. When the cecum was united just posterior to the gizzard, infection did not take place, probably due to the absence of pancreatic enzymes.

Another example of natural immunity to cecal coccidiosis is age resistance. It has frequently been stated that chickens of any age are equally susceptible to E. tenella infections. Chickens of all ages may be infected with this parasite, but the infection will not be equally severe. Tyzzer (1932) showed that in infections of chickens up to 70 days of age there was no difference of resistance in the various groups.

Herrick, Ott and Holmes (1936) showed in a group of birds varying from one-half to two months of age the mortality of 123 birds was 62.6 per cent. In the group of three to fifteen months given from two to five times as many oocysts the mortality was only 3.5 per cent. However, there was still a marked drop in the numbers of red blood cells, the decrease varying from 29.0 per cent to 46.8 per cent in the older groups compared to a decrease of 45.8 per cent to 60 5 per cent in the younger groups suffering the heavier mortality.

Johnson (1927) first carried out careful studies on the immunization of chickens to coccidia, and although he did use mixed cultures he was able to produce with regularity a high degree of resistance. He showed that daily inoculations of approximately 2,000 sporulated oocysts for a period of fifteen days would produce an immunity, which in two instances lasted for six and one-half months after the final infection. This work showed that the lack of coccidiosis on some farms is the continuous ingestion of small numbers of oocysts which were capable of protecting the host against a heavy infection. Herrick (1935) has found that chickens immunized to E tenella and kept free from infection for a period of one year were still resistant as evidence by no reduction in the numbers of red blood cells or decrease in egg production.

Jankiewicz (1942) devised an arbitrary scale for rating the percentage of immunity elicited by single infections of several graduated doses of sporulated oocysts of E. tenella. He has noted that an infection with 50 sporulated oocysts elicited an average immunity rating of 10 per cent, 250 oocysts 30 per cent, 500 oocysts 50 per cent, 1000 to 3000 oocysts 70 per cent and 6000 to 100,000 oocysts 90 per cent. Tyzzer (1929) has shown that severe infections with this species will excite a prompt and well marked immunity, but that single light infections with a few oocysts were not sufficient to protect. Farr (1943) administered 1000 sporulated oocysts of E. tenella daily for 15 days to chickens and produced a strong immunity as manifested by the lack of hemorrhage or mortality on the test dose. A similiar resistance was produced if 15,000 sporulated oocysts were administered in doses of 1000, 3000 and 9000 at five day intervals. In these experiments in which 15,000 oocysts were used for the immunization the chickens were capable of maintaining this resistance for a period of at least 14 months.

Little work has been done on the value of using attenuated oocysts for immunizing chickens against cecal coccidiosis. The administration of killed oocysts will not produce any resistance against future infection. Jankiewicz and Scofield (1934) have found that heating either sporulated or unsporulated oocysts at 48° C. for 20 minutes and after sporulation a series of immunizing doses produced successful immunization. It appears that these oocysts were attentuated. Warler (1941) and Albanese and Smetana (1937) have demonstrated similar properties in x-ray treated oocysts. Tyzzer (1929) failed to demonstrate any passive immunity.

There is little evidence that chickens have inherited resistance to infections with this parasite. Herrick (1934) found that the first generation of birds raised from resistant parents were 100 per cent more resistant to infections than were birds from unselected stock. Mayhew (1934) presented similiar evidence. Rosenberg (1941) obtained two groups of White Leghorns of markedly different susceptibility to cecal coccidiosis. He also noted that Barred Plymouth Rocks and Jersey White Giants suffered a significantly higher mortality than did White Leghorns, New Hampshires or Rhode Island Reds.

EFFECTS OF DIET: The role of diet in influencing the course of infections with E. tenella is not understood at the present time. In this country widespread interest has been evidenced in the role of the diet in coccidiosis since Beach (1917) suggested that milk products were of value in the control of this disease. In the discussion of this subject it is well to bear in mind as Mayhew (1934) has pointed out the meaning of the words control and treatment. Unfortunately, these terms have been used almost interchangeably in discussions of the effect of diet on this disease. Therefore, in this work control will be used in the sense of giving a preparation or a ration before birds are infected, in order that infection may be controlled or prevented, whereas, treatment is the administration after infection.

Beach and Davis (1925) reported on various methods of feeding milk products to chickens and the resultant course in the control of coccidiosis. They were able to demonstrate favorable results with 40 per cent dry skimmed milk, although the mortality in birds given a mixture containing 20 per cent lactose was not as great as those on plain mash. They attributed this success to an increased acidity of the cecum. Beach (1925) used a porcelain spot plate and demonstrated that the pH range of the cecal contents changed from 6.0 to 7.4 in normal chickens and from 4.4 to 5.6 by feeding various milk preparations, and concluded that the acid environment was unfavorable for the development of coccidia. Numerous investigators have shown wide variations in the pH of the cecal contents, but it seems extremely doubtful if this is a factor influencing the effect of milk products in coccidiosis

Becker and Wilke (1938) carried out studies on the effects of various milk preparations in the prevention of cecal coccidiosis. If dried buttermilk was incorporated in ordinary rations at a level of 10 to 40 per cent the severity of the infections was increased However, a modified California ration consisting of yellow corn meal 30 parts, ground oats 20 parts, wheat bran 10 parts, dried buttermilk 10 parts and cod liver oil one part, did not increase the severity of the infection nor result in any great decrease in mortality. Although milk products in some of their rations did increase the severity of the infection as measured by mortality, there is a possibility of some other substance in the ration which influences unfavorably the course of the infection. Becker and Waters (1938, 1939) have shown that various combinations of dried skim milk in the ration predisposed birds unfavorably to attacks with E. tenella. The use of milk products in controlling cecal coccidiosis is still unsettled.

Mayhew (1934) gave birds a 40 per cent buttermilk ration after they were visibly sick and using weight as a criteria of efficiency, concluded there were no beneficial results from the treatment.

Jones (1934) could find no correlation between the amounts of hemorrhage and mortality in birds on various levels of vitamin A and protein, although Allen (1932) had presented data which indicated a benefit from high proteins and vitamin A.

Murphy, Hunter and Knandel (1938) observed in a natural outbreak of coccidiosis that birds which were maintained on a low level of cod liver oil suffered much more severely from the infection than those on higher levels. However, most of the severe effects noted in their work were undoubtedly due to the low levels of vitamins A and D. Baldwin, Wiswell and Jankiewicz (1941) have noted that when chicks were protected by the anti-hemorrhagic factor, vitamin K, the mortality in protected birds was only 10 per cent compared to 70 per cent in unprotected birds. However, Hawkins (1945) has shown that neither vitamin K nor 2-methyl-naphthoquinone are of any value in decreasing the severity of cecal coccidiosis.

Holmes, Herrick and Ott (1937) have found that an increase in the per cent of calcium in the ration in the form oyster shell increased the mortality as compared to infected birds on a basal ration.

MISCELLANEOUS: Smith and Herrick (1944) have published the only study on the respiration of oocysts of E. tenella, although Hawkins (1943) briefly described the respiration of this species through the process of sporulation. The former workers have found that the respiration of parasitized tissues is greater than that of normal cecal tissues. They found that the oxygen consumption of oocysts for the first 12 hours was quite uniform, varying from 11.2 to 16.6 cmm. of oxygen per million oocysts per hour, however, at 12 hours the rate increased to 35.2 cmm. per hour. This was explained on the basis that no visible change is seen in the oocysts until they are about 12 hours old at which time evidences of division are seen. This was in marked contrast to the consumption of the sporulated oocysts which varied from 0.16 to 1.30 cmm.

These observations could be explained by the work of Edgar, Herrick and Fraser (1944) on the glycogen content of oocysts. They noted that the unsporulated oocysts stored glycogen which practically disappeared after sporulation occurred. Only a very small amount of glycogen was noted in the central portion of the liberated sporozoite.

Pratt (1940) found that the effect of cecal coccidiosis raised the blood sugar and similar increases occurred after withdrawing large quantities of blood from normal chickens The increase in blood sugar in E. tenella infections was not only noted in birds which were being fed continuously, but in those fasted for 19 hours. Pratt (1941) reported that liver glycogen was considerably higher during the fifth and seventh days after infection, but about normal on the sixth day and also noted a low muscle glycogen content on the sixth day after infection. However, both liver and muscle glycogen vary within wide limits.

Warler (1941) in addition to noting the increase in blood sugar found there was an increase in blood chlorides on the sixth and seventh day of an infection with cecal coccidiosis. Warler (1941) also found that by feeding concentrated physiological saline, starting on the fourth day after infection, that the weight gains were markedly greater and the mortality lower, in those birds given the salt solution The feeding of the saline solution also resulted in a decrease in blood sugar.

DIAGNOSIS: A diagnosis of cecal coccidiosis may be made from the appearance of the bloody droppings, but should be confirmed at autopsy. It should be borne in mind that oocysts a are not passed until the seventh day following infection, and thus most of the symptoms and mortality occur before this time.

CONTROL: Cecal coccidiosis is a filth borne disease and can only be controlled by sanitary procedures. Whatever method of control used, however, will only produce relative freedom from the disease, as absolute freedom probably will never be attained. Furthermore, subclinical infections are advantageous due to the immunity resulting from exposure, and the consequent protection of the birds. Despite the fact that light infections may be welcome it cannot be too strongly emphasized that only a maximum of cleanliness and sanitation will attain this result without serious damage.

Herrick and Edgar (1942) and Edgar and Herrick (1944) have carried out studies on the influence of feeding habits on the severity of cecal coccidiosis. They noted that birds that have access to feed at all times are more resistant to coccidiosis, that is, birds on range, 3 times as many died when they were not fed from the previous evening, until 8 a.m. the following morning, in contrast to chickens which had feed presented to them continuously. The reason for this is not known but in birds with an empty crop oocysts and sporozoites were found in the ceca 1 hour after infection, whereas, in birds with full crops, 2-1/2 to 4 hours passed before they reached the ceca. It would seem, therefore, that larger numbers of sporozoites may be able to reach the ceca in birds that are off feed.

In connection with control of coccidiosis a word should be said of the role of arthropods in the spread of infection. Baker (1933) has found that flies caught in poultry yards and fed to parasite-free chicks were capable in some instances of producing light

infections. Metelkin (1935) tested several species of flies and found that oocysts remained unaltered in their digestive tract and were viable for 24 hours. Delaplane and Stuart (1935) noted that both sporulated and unsporulated oocysts could be found in the gut of larvae of Musca domestica, but when these were allowed to pupate and metamorphose into adult flies, no infection resulted on feeding them to parasite-free chickens. Thus, mechanical arthropod transmission is definitely a possibility, it is relatively unimportant in comparison to infection by picking up sporulated oocysts in the feed water or on the litter.

The first point in control is to bear in mind the distribution of the oocysts in the poultry yard or house. Andrews and Tsuchiya (1931) have found that the heaviest accumulations of oocysts occur under the perches, under the drinking fountains, in sediment from the drinking fountain and under the brooder. In addition, they observed fairly large numbers of oocysts in the soil of chicken runs, from scraping near the feeders and in manure piles and litter outside the houses. Thus all of these areas should require particular attention. First, all equipment could be thoroughly clean when put into use with the first batch of chicks. Second, place feeders and waterers and perches on hardware-cloth covered frames so that droppings in these areas will not come in contact with the birds. Droppings must be cleaned out under the frames at regular intervals, otherwise the frames are useless. Third, provide feeders and waterers which as much as possible prevent birds from scratching or defecating on them. And fourth, see that the birds are living in a clean environment by regularly changing the litter or covering the floor with fresh litter. If an outbreak has occurred, it is necessary that some measures be taken to prevent further infection or reinfection. If possible, destroy immediately all infected birds. The visibly unaffected birds may then be removed to clean quarters, or to other quarters temporarily, while the house is cleaned. Cleaning should consist of dry cleaning. Since the presence of moisture produces favorable conditions for sporulation, moisture should be avoided. As no disinfectant is practical for farm use, dessication should be depended on to destroy the oocysts. Horton-Smith, Taylor and Turtle (1940) described ammonia fumigation as successfully destroying oocysts in a poultry house in a very short time. They recommended 3 oz./10 cubic feet of house.

As has already been pointed out, the fire-gun is useless Since 48 hours are required for the sporulation of the oocysts of this species, two practices are available to prevent their ingestion. First, the entire litter may be removed every day and clean litter put in. Or second, instead of removing the litter, more litter may be placed on the old after it has been stirred up to insure aeration. This latter deep litter practice has been successfully used in many parts of this country, and seems to effectively bury the oocysts and prevent their ingestion in large numbers Warmth and plenty of food and water should be provided sick birds.

Herrick and Holmes (1936) demonstrated that 5 to 10 per cent sulfur in the ration would prevent coccidiosis if it was given 4 days before the birds were infected. Holmes, Deobald and Herrick (1938), however, showed that unless certain precautions were taken birds on a sulfur ration would develop rickets Rickets did not develop in those birds fed 2 to 5 per cent of sulfur in the ration, if they had direct access to sunlight, had 2 per cent liver oil added to the ration or were irradiated 15 minutes daily. However, in those birds that were fed these levels of sulfur (no rickets occurred) growth was poor. In addition to the adverse effects of sulfur on growth, Herrick and Holmes (1939) demonstrated that sulfur at a level of 5 per cent in the ration had an adverse effect on egg production. Levine (1941) noted that sulfur may result in rickets, dermatitis, cloacitis, poor feathering and retarded weight gains on prolonged feeding. Goff and Upp (1940) studied the effects of the fineness of grind of the sulfur on its efficiency and noted that of the four grades tested, all prevented mortality, although they stated that the finer grinds seemed to be more effective.

Goff (1942) found that birds are highly susceptible to cecal coccidiosis 24 hours after sulfur is withdrawn. If deemed necessary to feed sulfur, despite the possible damaging effects on the bird, the chemical should not be fed at a level higher than 2 per cent. Some birds may contract the infection at this level but the mortality will be low. Sulfur given after infection is of no value.

Levine (1941) found that sulfaguanidine at a level of 1 per cent in the ration markedly reduced the severity of cecal coccidiosis when given before infection, but had no value after infection. Allen and Farr (1942) used 1/2 per cent sulfaguanidine in the ration, and not only found that it gave marked protection, but that it permitted the birds to acquire a light infection and thus resistance to infection after the sulfaguanidine was withdrawn. They concluded that 1/2 per cent sulfaguanidine in the ration was high enough to protect birds from severe outbreaks of cecal coccidiosis, and yet to allow them to acquire a resistance to the infection. No toxic reactions are noted in birds when kept on this level of sulfaguanidine.

Sulfaguanidine at a level of 0.5 per cent in the feed given intermittently for three days and removing it for three or four days has produced satisfactory results in the prevention of the disease. Some birds will succumb to the disease with this intermittent method of medication, but the low cost makes its use advisable over the continuous feeding. This compound has some value if given when the first birds in the flock show evidences of the disease, however, here it is not as effective as some of the other sulfonamides discussed under treatment.

Flowers of sulfur is similar to sulfaguanidine in being able to prevent cecal coccidiosis if given before infection. It should probably not be given at a level higher than 2 per cent. Although some birds may contract the infection at this level, the mortality resulting will be low and the detrimental effects of the compound will not be as noticeable. Sulfur at higher levels will result in rickets, and other evidences of its toxicity have been noted.

The status of the diet in relation to the control of cecal coccidiosis is not well understood at the present time. However, widespread interest in the role of the diet has been evidenced since Beach (1917) proposed that milk products were of value in the control of the disease. It is possible that milk products, 10 to 40 per cent of dried skim milk or dried buttermilk, may be of some value before infection has taken place. However, there can be no doubt that these products are detrimental after infection has occurred.

Herrick and Edgar (1942) have noted that birds which have access to feed at all times are more resistant to coccidiosis than are birds which are fed intermittently and infected with empty crops.

Herrick, Holmes and DiGiusti (1942) tested some organic sulfur compounds and found that loral thiocyanate and tetraethylthiuram monosulfide in single doses prevented the development of cecal coccidiosis, but that continuous feeding of these compounds not only failed to prevent the infection, but was toxic to the chickens. Harwood and Guthrie (1943) have found that triethanolamine hydrochloride, and mixtures of micronized wettable sulfur with urea were apparently of value as a prophylactic, but failed when given 4 days after infection. Morehouse and Mayfield (1944) have also shown that some of the aryl arsonic acids in very small amounts in the drinking water are of value in preventing infection, but again are of no value after infection occurs. Harwood, Stunz and Wolfgang (1947) found that 5-nitro furfural semi-carbizone was effective when given before infection and up to two days after infection.

Swales (1947) has developed a new method for the control of cecal coccidiosis which combines immunization and therapeutic control of the disease. He recommended that special pens be maintained in which susceptible birds are placed where it is known they will become infected. Three to four days after being placed in this pen they are treated with sulfamerazine or sulfamethazine, preferably the sodium salts. This will prevent the development of symptoms and mortality, or reduce it to a neglible amount. The infection has been established long enough for the immunization of the birds, and thus give future protection. This method should be resorted to only in special cases and only under competent supervision.

**TREATMENT:** After the symptoms of cecal coccidiosis have become obvious there is no treatment for the individual bird. However, as the flock is usually regarded as a unit some of the newer drugs may be of value. If these drugs are to be of value they must be used at

the earliest possible moment. This requires that every bird which dies or is sick must have a diagnosis, and the very first signs of blood droppings must be recognized. Many diagnoses are made in the flock when the majority of the birds are sick or dying. At this time the peak of infection has been reached and regardless of the treatment given recovery will ensue. The most important point that should be emphasized in the treatment of cecal coccidiosis, is that most remedies recommended are of absolutely no value.

Sulfamethazine and sulfamerazine at a level of 0.5 per cent in the mash or preferably the sodium salts at a level of 0.2 per cent in the drinking water are effective. Hawkins (1945) and Delaplane et al (1947) found that sulfaquinoxaline is also effective. The latter investigators showed sulfaquinoxaline at levels of 0.0125 to 0.033 per cent in the mash is effective, however, levels of 0.05 per cent are toxic if fed constantly, although this latter dosage is satisfactory when given intermittently, i.e., fed four days and ommitted four days.

The so-called "milk flush" is not to be recommended for birds exhibiting symptoms of coccidiosis. Repeated trials have shown that milk products given after infection has increased rather than alleviate the severity of the disease.

Horton-Smith and Taylor (1942, 1943, 1945) have demonstrated that sulfamethazine and sulfadiazine are of value in the treatment of cecal coccidiosis up through 4 days after infection. These compounds were given in the mash and as saturated solutions substituted for the drinking water. In artificially induced coccidiosis they showed that these compounds were efficacious and also the treated birds developed a strong immunity to reinfection.

Hawkins (1943, 1944) and Hawkins and Kline (1945) demonstrated that concentrations of sulfamethazine in the mash of 0.4 to 1.0 per cent were effective for treatment, but sulfamethazine levels of 5 0 to 6.0 mg. per cent by the fifth day after infection are necessary to prevent loss. They noted that sulfamethazine interferred with normal weight gains in uninfected birds, but weight losses in treated infected birds were slight compared to losses in untreated infected chickens. Swales (1944), Hawkins (1944) and Hawkins and Rausch (1946) have shown that sodium sulfamerazine in the drinking water is as efficacious as either sulfadiazine or sulfamerazine and slightly less toxic. Further, it has the advantage of being given in the water which is consumed in larger quantities by infected birds, whereas, the feed consumption and thus drug consumption is greatly lowered in infected chickens. Sodium sulfamerazine is very effective in concentrations of 0.2 per cent in the drinking water. Ripsom (1944) and Ripsom and Herrick (1945) have shown that this group of sulfonamides will prevent the development of the sexual stages. As to the use of these drugs little can be said, but it would seem advisable that birds be given the drug as soon as a diagnosis is made, but they should not be maintained on the treatment for more than 3 days.

Hardcastle and Foster (1944) demonstrated that 2 per cent borax in the mash protected birds when given for 24 hours, not later than 72 hours, after infection, providing that it is given in the 72 to 96 hour period. Three-tenths per cent in the water was apparently as effective and not as toxic. At the present time this drug cannot be used because of its marked toxicity.

Finally, to return to the use of milk flush for treatment, there is ample evidence that when milk products are used after infection they increase rather than alleviate the severity of the infection.

## Eimeria necatrix, Johnson, 1930

**SYNONYM:** Eimeria avium.

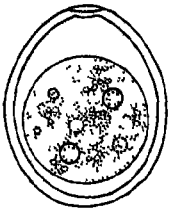
**DISEASE:** Chronic intestinal coccidiosis of chickens.

**HOST:** Chicken.

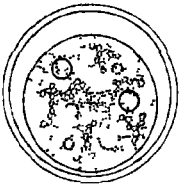
PLATE XIX

COCCIDIA OF POULTRY

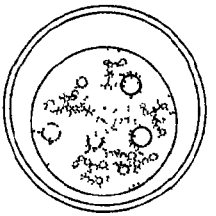
Eimeria mitis



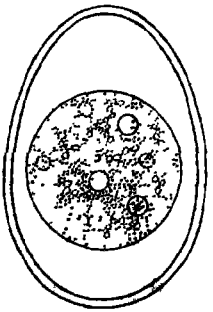
Eimeria acervulina



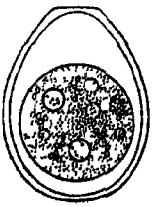
Eimeria necatrix



Eimeria tenella



Eimeria maxima



Eimeria truncata

MORPHOLOGY: The oocysts of this species are smaller than those of Eimeria tenella and are passed in the feces unsporulated. The size of the oocysts is 13.2 to 22.7 microns in length and 11.3 to 14.2 microns in breadth and averages 14.2 microns wide and 16.7 microns long. It overlaps the measurements of E. tenella as well as those of some of the other species in the chicken. Consequently, size is an unreliable criterion for differentiation. The oocysts are broadly oval in shape and the cytoplasm more nearly fills the cyst than is the case with E. tenella and seems to be more irregular in outline. The cyst wall is quite smooth and there is no apparent micropyle. The small refractile granule seen in the anterior end of E. tenella does not seem to be present in this species until a short time before cleavage of the cytoplasm occurs. Sporulation requires 48 hours at room temperatures in the presence of adequate moisture and oxygen. The spores are elongated and occupy a larger space in the oocyst than is the case in E. tenella. Otherwise, the oocyst is similar to the previously described species

LIFE CYCLE Although only a limited amount of work has been done on this species, the bionomics and development of the oocyst is similar to that of E. tenella. The description of the life cycle of this species is due largely to the work of Johnson (1930) and Tyzzer, Theiler and Jones (1932). Infection occurs by the ingestion of sporulated oocysts and the liberation of the contained sporozoites, although Levine (1940) infected birds by placing merozoites in the crop and intestine. The liberated sporozoites average 8.6 microns in length. There is a large amount of hyaline eosinophilic material and the nucleus lying anterior to this mass is near the pointed anterior end. After liberation from the oocyst, the sporozoites very rapidly penetrate the epithelial cells of the glands of the small intestine. They may be found in epithelial cells in as short a time as an hour after infection.

The development of the Generation I schizonts and merozoites after the penetration of the sporozoites is almost identical with that in E. tenella, except that the latter occurs in the cecum and E necatrix in the small intestine. The first generation merozoites are liberated into the lumen of the glands 2-1/2 to 3 days after infection. They do not show any great tendency to a disseminate, but penetrate adjacent epithelial cells. A characteristic of this species is the development of very large generation schizonts in the sub-epithelium Johnson (1930) has found these schizonts ranging in size from 32.96 microns to 39.14 microns by 53 56 microns to 65 92 microns, averaging 38.07 microns by 51.73 microns. The schizont divides into a slightly larger number of merozoites than is the case with E tenella. The merozoites of Generation II are also smaller than the latter species. The size ranges from 1.5 microns to 7 9 microns by 2.0 microns to 11.25 microns and average 1.8 microns by 8.66 microns They are relatively thicker and present a more stubby appearance. The nucleus is located posteriorly and shows a variable number of granules in the cytoplasm The Generation II merozoites begin to be liberated in the lumen during the fifth day after infection, but occur in greatest numbers on the sixth, seventh and eighth days.

In E. necatrix infections there is from the beginning a marked lack of uniformity in development. At the time the Generation I merozoites are being formed at the end of 2-1/2 to 3 days many earlier stages of development also occur, some even remaining in the sporozoite stage without any appreciable increase in size. While such forms do not appear in large numbers Tyzzer, Theiler and Jones (1932) reported the presence of one unchanged sporozoite in an epithelial cell 5 days after infection. They also noted that birds continued discharging Generation II merozoites up to the twenty-third day after infection. In another bird they found what were apparently immature Generation II schizonts at the end of 18 days, stages comparable to that attained by most schizonts in 4 days.

The development of the third generation, whether asexual or sexual, occurs in the cecum rather than the small intestine. Five days after infection Generation II merozoites may be seen penetrating the epithelium of the cecum. The surface epithelium is heavily infected, but some will penetrate the glandular epithelium. Some of these merozoites will develop into gametocytes, but the greater proportion will initiate a third asexual generation. The number of asexual generations occurring in the cecum is not known, but it seems that more than one develops. The schizonts occurring in the cecum are small compared to those in the small intestine, and cause no marked reaction in the host cell which is merely

stretched into a sac with the growth of the parasite. In some areas large numbers of schizonts may be found in the epithelium, sometimes 3 or 4 in a single cell. In the case of several schizonts in a single cell they are in consequence quite small, and result in the production of correspondingly small merozoites. However, with single schizonts the merozoites produced may equal in size those of the second generation. These schizonts in the cecum never produce a large number of merozoites, some may have only 6 or 8 while others may have as many as 16.

The gametocytes are similar to those produced by E. tenella but in contrast to this species large numbers of them are never found at one time. Oocysts are first passed in the droppings seven days after infection. This species very closely resembles E. tenella, except for the occurrence of the first two asexual generations in the small intestine. However, even those forms in the small intestine closely resemble E. tenella.

COURSE OF THE INFECTION: The prepatent period of E. necatrix is 7 days, if it is assumed this period represents the time from infection to the discharge of oocysts. As in cecal coccidiosis the time required for the passage of oocysts is longer than the incubation period of the disease. Severe hemorrhages may occur on the fifth and sixth days after infection. The length of the patent period in the case of experimental birds in which the infection is prevented is very similar to that of E. tenella; oocysts of E. necatrix having been found in the droppings up to 19 days after infection by Tyzzer, Theiler and Jones (1932).

E. necatrix infections are often referred to as chronic coccidiosis, however, this does not mean the infection itself will persist for a long period of time. As will be pointed out in discussing the histopathology of this infection the chronicity is due not to persistence of infection but to scar formation in the small intestine. Although the infection does not persist for long periods, it is present in rather a high percentage of sub-clinical cases. According to Levine (1940), who examined 39 chickens which showed no symptoms of coccidiosis, 15 were infected with E. necatrix.

IMMUNITY: E. necatrix like E. tenella has only been found in the chicken and there have been no successful attempts to transfer it to other hosts. There is also marked organ specificity. The first two asexual generations occur in the small intestine and succeeding generations, both asexual and sexual occur in the cecum. In contrast to the age resistance to E. tenella which develops at about 3 months, Tyzzer, Theiler and Jones (1932) have shown that younger birds are more resistant to infection with E. necatrix than are older birds. They noted that the mortality in birds 8 days, 35 days, 52 days and 73 days was 14.3 per cent, 87.5 per cent, 100 per cent and 100 per cent, respectively. They believed this inverse age resistance was due to a lack of physical and physiolgical maturity of the small intestine.

Tyzzer, Theiler and Jones (1932) have shown that in infections with E. necatrix an immunity will develop to reinfection the same as in E. tenella. Either a single heavy infection or repeated light infections will result in a marked protection. They also made interesting histological examinations of immune birds, finding that sporozoites were in the epithelial cells of both immune and nonimmune birds an hour after infection. In the nonimmune birds the sporozoite on entering the epithelial cell indents the nucleus and stimulates an increase in volume of both the nucleus and cell cytoplasm. At the end of 24 hours in nonimmune birds the parasitized cell has increased in size and at 66 hours numerous Generation I schizonts are found. However, in immune birds, although the entrance of the sporozoite dents the cell nucleus, it apparently is not capable of stimulating any increase in size of the host cells. On the contrary, the sporozoite in the immune bird appears to be shrunken, often showing no structure apart from the eosin staining granule. The cytoplasm of the invaded host cell shows evidences of serious injury and is often characterized by the presence of only a few shreds of granules.

PATHOLOGY: Gross evidences of infection as described by Tyzzer, Theiler and Jones (1932) with this parasite are noticed on the fourth day after infection and are distributed throughout the small intestine, mostly concentrated in the middle third. These early changes are visible as small white opacities seen through the serous, but not through the

mucous surface. These represent the developing second generation schizonts which are deep in the mucosa. Five days after infection the white spots have increased in size. The lesions seldom exceed a millimeter in diameter, but they may coalesce and become much larger. At this time there is a varying degree of distension of the gut, particularly through the middle third where it may be greatly swollen. The wall from the serous surface is a dull red and on close examination petechial hemorrhages may be seen spreading from the white opacities representing the second generation merozoites. There is a marked loss of contractility of the gut wall which becomes friable and often appears gangrenous.

It will be found on opening the small intestine that the areas of greatest involvement may be filled with either clotted or unclotted blood. The wall is greatly thickened and a dull red, indicating approaching venous stasis. No evidence of infection in the ceca may be noted grossly. However, due to stasis of the contents in the small intestine, the ceca are contracted on a mass of markedly dehydrated material. Gross emaciation is found in seriously affected birds

The primary damage produced by this parasite is associated with the development of the second generation schizonts and merozoites in the sub-epithelial tissues. These lesions are strikingly similar to those produced by E tenella. While the changes are taking place in the migrating epithelial cells, acute inflammatory processes are developing. Granular leucocytes begin to accumulate with the first invasion of glands by the sporozoite, however, the degree of infiltration varies. Some areas are extensively infiltrated while others contain scarcely any leucocytes Hemorrhage into the lumen of the gland may be first noted at the end of the third day and increases in the course of the infection The hemorrhage as in E tenella is apparently not due to defects in the blood vessels, at first, but to widespread leakage. With discharge of the Generation II merozoites and the occurrence of widespread hemorrhage associated with the sloughing of the epithelium, the processes of repair commence.

By the end of the sixth day a network of fibrin is found in destroyed areas and intermingled with it numbers of mononuclear cells A few days later connective tissue cells are found in the fibrin mass The destroyed areas rapidly become filled with connective tissue. Thus, the chronicity characterized by this infection is not associated necessarily with a persistence of infection, but with extensive scar formation which consequently influences absorption.

The after effects of E. necatrix infections are much more severe than those associated with E tenella, the reason being that large areas of the entire small intestine are destroyed so the absorption of nutrients will never again take place It has been our experience that birds suffering from infections with this parasite are worthless to keep and try to develop into producing birds In contrast to the statement of Becker (1934) this species is not too important because it is uncommon, we have found it a common cause of losses in poultry flocks.

CONTROL AND TREATMENT: The control and treatment of E. necatrix infections is essentially the same as that for E. tenella. Sanitation and cleanliness are necessary to prevent serious infections and compounds such as sulfaguanidine and sulfur may be used to prevent infection. It is probable as Levine (1941) has shown that slightly higher concentrations of sulfaguanidine are necessary. He believed that 1-1/2 per cent is necessary to prevent infection, although lower concentrations as used for E. tenella would undoubtedly be effective under field conditions. Limited trials by Hawkins (unpublished data) on the use of sulfamethazine as a therapeutic agent after blood began to be passed in the droppings, showed the drug would prevent losses, but birds failed to develop satisfactorily on recovery.

A recent report by Delaplane et al (1947) showed that sulfaquinoxaline is also of value in this infection.

## Eimeria brunetti Levine, 1942

**HOST:** Chicken.

**MORPHOLOGY:** The oocysts of this species are passed in the feces unsporulated. The oocyst of E. brunetti measure 20.7 microns to 30.3 microns in length and 18.1 microns to 24.2 microns in width and average 21.7 microns wide and 26.8 microns in length. With the exception of E. maxima this is the largest of the oocysts described from chickens. The oocysts of this species are oval and, according to Levine (1942), can be differentiated from those of E. maxima by its smaller size, thinner walls, more curved walls and more rounded ends. Sporulation of the oocysts requires 24 to 48 hours at 30° C. A refractile body appears in the sporulated oocyst but its position varies considerably. It is most often found at the narrow end but may be found among the spores. Some oocysts may contain two or three of these bodies.

The oocysts of this species are first discharged in the feces 5 days after infection. The stages in the tissues have not been studied.

It would seem, as Levine pointed out, the differentiation of a new species solely on the basis of oocyst size was unreliable However, by making use of immunological and pathological criteria, similar to those used in characterizing the species of Leishmania, he has undoubtedly established the validity of this species By immunizing chickens against E. hagani, E. mitis, E. praecox and E. maxima it is still possible to infect them with E. brunetti. Eimeria tenella and E necatrix would be ruled out by pathology and the time required for the oocysts to appear in the droppings. Furthermore, a chicken immunized against E. brunetti is susceptible to infection with any of the other species, but not E. brunetti. Furthermore, E. brunetti reaches its peak of oocyst production between 9 a.m. and 3 p.m. in contrast to E. hagani which had its peak between 3 p.m. and 9 p.m., as does E. mitis, E. praecox and E. maxima, although the peaks in some are not as sharp as in others.

**PATHOLOGY:** The location of the parasite and the lesions produced are different from those of any other species of coccidium in the chicken. The organism localizes throughout the lower half of the small intestine, rectum, ceca and cloaca, in very heavy infection even the upper intestine may harbor organisms Light infections produce no visible gross lesions Moderate infections result in the thickening of the gut wall and pink or blood-tinged catarrhal exudate. The droppings may become quite fluid and blood-tinged. These changes occur 4 to 5 days after infection and persist for several days

Hemorrhagic ladder-like streaks are found on the surface of the mucosa of lower intestine and rectum. Severe infections result in a necrotic enteritis which may even involve the entire intestinal mucosa, with the sloughed mucosa in the lumen sometimes appearing like cottage cheese A dry necrotic membrane may form the lining of the intestine, usually patchy rather than continuous. Circumscribed white patches may be seen through the serosa and in some instances perforation of the gut wall with resulting peritonitis occurs. Although the cecum may also be the site of severe lesions, these are restricted to the tubular portion. No gross lesions other than a reddening of the mucosa occur in the dilated portion, although oocysts may be found in scrapings throughout the cecal mucosa.

**CONTROL:** The control measures are essentially the same as those described in infections with E. tenella, except that according to Levine (1945) sulfur has no effect in preventing infection.

**TREATMENT:** Unknown.

## Eimeria acervulina Tyzzer, 1929

**HOST:** Chicken.

**MORPHOLOGY:** The unsporulated oocysts of this species are egg-shaped and measure 13.7 microns to 16.3 microns breadth, by 17.7 microns to 20.2 microns in length and average 14.5

microns in breadth by 19.5 microns in length. There is a thinning of the oocyst at the narrow end with a slight elevation at the margin. The oocysts develop very rapidly after being passed from the body, sporulation being complete in 20 hours.

SYMPTOMS: The outstanding characteristic of this species is its tendency to develop superficially in both the asexual and sexual stages in the upper half of the small intestine. Oocysts are discharged in enormous numbers 4 days after infection. Although this infection is often referred to as a cause of chronic coccidiosis, it is extremely doubtful if birds infected with this parasite encountered more than a slight set back. Levine (1940) in an examination of 39 birds with no symptoms of infection found that 53 per cent were infected with E. acervulina or E. mitis (the two were not differentiated).

Dickinson (1941) studied the effects of variable dosages of E. acervulina oocysts on chickens and found that massive doses only produced a temporary drop in weight and a complete temporary cessation of egg production. However, in a short time weights and egg production were almost back to normal. During the period of 4 to 9 days after infection he noted the birds were noticeably droopy, passed less feces of a slimy mucoid character and ate much less food. However, he stated that even the administration of 500,000 sporulated oocysts of E. acervulina daily for 50 days did not produce a permanent detrimental effect.

PATHOLOGY: No marked pathological changes occur in E. acervulina infections comparable to those seen in the previously described species. The lesions produced by this species may be recognized by a thickening of the intestine and by white streaks usually running transversely across the mucosa of the duodenum, although they may go as far as the middle of the small intestine. These streaks consist of accumulations of schizonts and oocysts which may be observed in large numbers from these areas. A catarrhal exudate may be present, but hemorrhage is only rarely observed as would be expected from the superficial position of the parasites.

CONTROL: The control may be carried out in the same manner as in E. tenella except that Dickinson and Scofield (1939) have demonstrated that sulfur is of no value in preventing infection.

TREATMENT: Unknown.

### Eimeria maxima Tyzzer, 1929

HOST: Chicken.

MORPHOLOGY: The oocysts of this species are the largest coccidia of the domestic fowl. They measure 16.5 microns to 29 8 microns in width and 21 5 microns to 42.5 microns in length and average 22.6 microns wide and 29.3 microns long. The cyst wall is often slightly roughened, egg-shaped and has a yellowish appearance. After being discharged in the droppings, the oocysts sporulate in 48 hours.

LIFE CYCLE: The parasite may be distributed throughout the small intestine, but is most commonly in the middle third. The asexual stages occur superficially next to the nucleus of the epithelial cell and the sexual stages which are quite large in contrast to the asexual, are found deep in the epithelial cell. Due to the size of the sexual stages the epithelial cells are much enlarged and may be displaced into the reticular base of the epithelial layer. Oocysts are discharged at the end of 6 days. Elimination of oocysts will occur for a few days after which they will disappear and the birds are refractory to reinfection.

PATHOLOGY: This species may be responsible for a blood-tinged catarrhal enteritis. The condition cannot be diagnosed from gross lesions, but by finding the large oocysts in scrapings of the mucosa a differentiation may be made. The exact status of this species as regards pathogenicity is not known, but it is probably in the same position as E. acervulina. Levine (1940) observed this species in 28 per cent of 39 birds.

CONTROL: The control of this species is the same as described for E. tenella, except that sulfur will not prevent infection.

TREATMENT: Unknown.

Eimeria hagani Levine, 1938

HOST: Chicken.

This is one of the three nonpathogenic species of Eimeria occurring in the chicken. The oocysts are oval and measure 14.3 microns to 19.5 microns in breadth and 15.8 microns to 20.9 microns in length, averaging 17.6 microns wide and 19.1 microns long. Sporulation requires 24 to 48 hours. The development of this parasite in the tissues has not been followed. Oocysts are discharged in the droppings 7 days after infection. Grossly the infection may be characterized by pin-point like hemorrhages in the anterior half of the small intestine, but these lesions are not characteristic enough to permit recognition of the disease. The species was determined, as was E. brunetti, by both its position and by immunological studies. The control of this species is the same as for E. tenella except that sulfur is of no value for prophylaxis.

TREATMENT: Unknown, probably unnecessary.

Eimeria mitis Tyzzer, 1929

HOST: Chicken.

This is the second of the nonpathogenic species occurring in the chicken. The oocysts are nearly spherical in shape and measure 13.0 microns by 17.0 microns wide and 14.3 microns to 19.6 microns long, averaging 15.5 microns wide and 16.2 microns long. Sporulation occurs in 48 hours at room temperature This species is found in greatest numbers in the upper portion of the small intestine and may develop in the cytoplasm of the cell below the nucleus. Oocysts are discharged 5 days after infection. No gross lesions are produced by the parasite and it apparently produced no damage to the host. Levine (1940) found that 53 per cent of 39 birds were infected with E mitis or E. acervulina (the two were not differentiated). The infection may be controlled as outlined under E tenella except that sulfur is of no value as a prophylactic.

TREATMENT: Unknown.

Eimeria praecox Johnson, 1930

HOST: Chicken.

This is the third of the nonpathogenic species of Eimeria occurring in the chicken. The oocyst is oval and measures 15 7 microns to 19.8 microns wide and 19.8 microns to 24.7 microns long, averaging 17.1 microns wide and 21.3 microns long. Sporulation occurs in 48 hours. The organism is found in the upper third of the small intestine with the various stages developing superficially to the epithelial cell nucleus. No gross lesions of infection are evidence. Levine (1940) in an examination of 39 sub-clinical cases found this species in 33 per cent of the birds. The infection may be controlled as described for E. tenella except that sulfur is of no value for prophylaxis.

TREATMENT: Unknown.

Eimera meleagridis Tyzzer, 1927

HOST: Turkey:

The oocysts of this species are ellipsoidal in shape and measure 19.14 microns to 29.7 microns long and 14.52 to 23.1 microns wide, averaging 17.33 microns wide and 23.79 microns long. The oocysts require about 24 hours for sporulation. At this time several

globular inclusions may be seen in the cyst. Oocysts are found in the droppings 5 days after infection. The development of this species according to Tyzzer (1929) seems to be confined to the cecum since the oocysts were confined to this region, although in young poults he has found that the lower half of the small intestine and the greater part of the large intestine may be involved.

The stages occurring in the tissues have not been completely worked out, but the parasites occur only in the surface epithelium, rather than the glandular epithelium. The parasitized cell show no marked reaction to infection, although it is gradually distended and eventually destroyed. The schizonts are large and in contrast to the other species of Eimeria. Tyzzer (1929) could not infect chickens, pheasants or quail with this species. It is relatively nonpathogenic in turkeys.

Steward (1947) dosed two young chickens with 300,000 and 260,000 sporulated oocysts of E. meleagridis from turkeys and a third was kept uninfected as a control. The chickens became infected and passed oocysts which resembled those from the turkey. Turkeys were subsequently infected with oocysts obtained from the chickens. Slight variations were noted on the measurements of the oocysts of E. meleagridis in chickens. E. acervulina from chickens was not infective to a coccidia-free turkey chick.

TREATMENT: Unknown, probably unnecessary

Eimeria meleagrimitis Tyzzer, 1929

HOST: Turkey

This species has relatively broader, more ovoid oocysts than E. meleagridis and measure 16.5 microns to 20.46 microns and 13.2 microns to 17 23 microns wide and averages 15.28 microns wide and 18.12 microns long Tyzzer (1929) pointed out this species agrees very closely in morphology and in location in the intestinal tract with E. mitis of the chicken, although chickens could not be infected. After infection oocysts are discharged in the droppings in 6 days, a day longer than is required for E mitis

The parasites are distributed throughout the small intestine, occurring generally in the epithelium of the villi Most are found deep in the epithelial cells below the nucleus, although some are superficial to the nucleus. No subepithelial forms have been found. The schizonts measure from 6 to 9.5 microns long by 5.8 to 7.7 microns broad and usually produce about sixteen short, thick merozoites.

There is little evidence as to the pathogenicity of this species, although it seems that E. meleagrimitis may occasionally produce a serious infection particularly in turkey poults about two weeks old. No characteristic symptoms are evidenced. Infected birds are listless, with drooping wings, ruffled feathers and a lightish-brown diarrhea or abnormally chalky discharges. At post-mortem a catarrhal enteritis in the lower half of the small intestine may be noted and in severe cases the intestine may be filled with a whitish-grey semi-gelatinous pus containing numerous oocysts. This exudate is found adherent to the surface of the intestinal wall and leaves the area denuded when scraped off. A definite diagnosis can only be made by demonstrating the oocysts under the microscope.

The status of coccidiosis in turkeys is not known, but it is not the problem which is present in chickens. However, further investigations could profitably be carried out with these species.

TREATMENT: Unknown

## Eimeria truncata Railliet and Lucet, 1890

**HOST:** Goose.

**DISEASE:** Renal coccidiosis.

Like coccidiosis in turkeys, little is known of these parasites in the goose. Eimeria truncata, although apparently an uncommon parasite of the goose in the United States, is extremely interesting because of its habitat. It is found in the epithelium of the uriniferous tubules of the kidney the only host among the domesticated birds or animals which suffer from renal coccidiosis. The oocysts are truncate at the anterior end and according to McNutt (1929) measure 13 to 15 microns wide and 20 to 27 microns long. The sporulation time is reported to be 5 days and a residual body is found in the sporulated oocysts.

McNutt (1929) has described an outbreak in Iowa, the first case of its kind reported in the United States. Losses were confined to goslings, with the old females in this case carrying the infection for at least two years. One year an 87 per cent loss resulted in 100 goslings, the second year a 17 per cent loss in 90. Losses occurred in the young geese from three weeks to three months of age and the younger the bird the more fatal the disease. The course of the infection is rapid, infected birds becoming weak, emaciated and dying in one to three days. Ducks or chickens raised with the geese were not affected. The kidneys are greatly enlarged, very light in color, showing on the surface and throughout the kidney substance small nodules, streaks and lines, which on gross examination could not be distinguished from retained urates. The entire kidney is affected.

Microscopic examination showed that the coccidia destroyed the cells of the tubules both directly and by swelling and pressure affect against the uninfected adjacent cells. About 20 per cent of the tubules were affected. They were so engorged with urates and coccidia they appeared to be five to ten times the diameter of uninfected tubules. Allen (1933) found E. truncata in the intestines and kidneys of 4 to 6 weeks old goslings in Washington, D. C Sections from infected birds revealed that the coccidia had invaded the intestinal mucosa.

**TREATMENT.** Unknown.

## Eimeria phasiani Tyzzer, 1929

**HOST.** Pheasant.

The oocysts of this species are quite elongate and ellipsoidal and measure 13.2 microns to 17.82 microns in width and 19.8 microns to 26.4 microns in length and average 15.89 microns in width and 23.04 microns in length. One or more polar inclusions occur. Sporulation takes 24 hours and a development period of five days is required from the time of infection until oocysts are discharged in the droppings.

The reaction of invaded cells, which are in the lower small intestine and ceca in greatest numbers, are peculiar to this species. The parasite passes beneath the nucleus of the epithelial cell, causing a marked enlargement. The cell becomes ballooned in the portion containing the nucleus and parasite (portion next to the lumen), while the distal portion assumes the appearance of a stalk. The swelling of the cell would indicate that it is eliminated on the completion of the parasitic development without leaving any serious defect in the intestinal epithelium.

This species is not infective for chickens, turkey or quail. There is apparently some immunity against this species as it occurs fairly commonly in young pheasants, but only to a slight extent in mature birds.

**TREATMENT:** Unknown.

## Eimeria dispersa Tyzzer, 1929

**HOST:** Quail.

The oocysts of this species in the quail are ovidal in shape and measure 17.16 microns to 26.4 microns in length to 15.44 microns to 22.44 microns in width and averages 18.84 microns wide and 22.75 microns long. Tyzzer (1929) noted slight variation from this size in different hosts and strains. The oocysts of E. dispersa differ from any others found in gallinaceous birds by the absence of any well-defined polar inclusions in the oocyst. Sporulation takes place in 24 hours, although in some strains up to 72 hours may be required, with very large spores being produced. On infection oocysts are discharged in the feces four days later. The time varies. In the turkey and pheasant five days are required for the passage of oocysts. The heaviest infection occurs in the first part of the small intestine, with the parasites occurring in the epithelium of the villi rather than that of the glands.

This species occurs naturally in the quail or "Bob-white" (Colinus virginianus virginianus). Cross infection with this species has been obtained in the turkey and slight infections occurred in the chicken. E. dispersa of quail origin has not been transmitted to pheasants (Phasianus colchicus torquatus), but E. dispersa of pheasant origin has been transmitted to quail. In quail this parasite exhibits poor immunizing power, as evidenced by the fact that it is extremely difficult to rid birds of infection.

The pathogenicity of this species is not understood; however, it is probable that any losses it may produce are in young birds. Older birds appear relatively resistant.

**TREATMENT:** Unknown.

## Eimeria labbeana Pinto, 1928

**HOST:** Pigeon.

The oocysts are elliptical or round and light yellowish brown in color. No micropyle is visible. The oocysts measure from 15 to 26 microns in length and 14 to 24 microns in width.

The sporozoites after penetrating the epithelial cells of the intestine develop into trophzoites. During the process of schizogony 15 to 20 sickle-shaped merozoites are formed. This requires about three days

The actual pathology produced by this parasite has not been studied in detail.

**TREATMENT:** Unknown

## Eimeria angusta Allen, 1934

**HOST:** Grouse.

This coccidium has been reported from a few occasions from the ceca of grouse. The oocysts are elliptical and measure about 16 to 17 by 27 to 33 microns in size. Little is known concerning its pathogenicity

**TREATMENT:** Unknown.

## Eimeria bonasae Allen, 1934

**HOST:** Grouse.

Oocysts are spherical and about 21 microns in diameter. They occur in the small intestine. Its pathogenicity is unknown.

**TREATMENT:** Unknown.

Tyzzeria perniciosa Allen, 1936

**HOST:** Ducks.

The oocysts of this species are elliptical and measure 9 to 10.8 microns wide and 10 to 13.3 microns long. There is no visible operculum or micropyle. They differ from the sporulated oocysts of Eimeria by lacking spores, but having the 8 sporozoites lying free inside a thick oocyst wall. Sporulation requires about 24 hours and results in the production of 8 sporozoites measuring about 10 microns long and 3.5 microns wide. A large residual mass is present.

The parasites develop in the small intestine with at least 3 generations of schizonts occurring, since schizogony continues long after oocysts are first formed. Oocysts are first observed in the droppings six days after infection Experimental infections resulted in a high mortality in young ducks. No characteristic symptoms occur.

Macroscopically, inflammation and hemorrhage is noted in the small intestine, especially in the upper half, and small rounded, white spots may be seen from the serosal surface. In severe cases the lumen of the intestine may be filled with blood, and a cheesy exudate may be present, with the epithelial layer of the mucosa sloughing off; sometimes sections of it being intact and can be lifted out as a tube. The lesions resemble those of E. necatrix in chickens. Microscopically, it is noted that the parasite penetrates both the mucosa and sub-mucosa to the muscle layers, which is much deeper than observed in most species of coccidia. In heavy infections the majority of the cells are infected, with their subsequent destruction

**GENERAL REMARKS:** Although coccidia are undoubtedly of common occurrence in ducks and losses due to this parasite has been described, very little can be said concerning the species concerned.

**TREATMENT:** Unknown.

Cryptosporidium sp

This group of coccidia usually occur in mice Tyzzer (1912) reported Cryptosporidium in the intestinal epithelium of chickens However, the specific identity of these forms has not yet been made. The mature oocysts contain four sporozoites

**LIFE CYCLE:** Unknown.

**TREATMENT:** Unknown.

**ORDER: HAEMOSPORIDIA**
Family: Plasmodiidae

Plasmodium Marchiafava and Celli, 1885

The malarial parasites are not of any economic importance in poultry in the United States. However, malaria is one of the most widespread and serious protozoan diseases of man. The disease is transmitted in nature by anopheline mosquitoes.

Enormous amount of investigations have been carried out with avian malaria as a test organism for the development of anti-malarials for human therapy. Bird malaria is transmitted by culicine mosquitoes with the exception of one species. Most of the research has been conducted with canaries. The species of malaria used include Plasmodium cathemerium, P. lophurae, P. praecox and P. gallinaceum.

Plasmodium gallinaceum, normally occurring in chickens in Ceylon, does not occur in the United States, however, it is being maintained in certain laboratories where studies

are being made because of its close relationship to human malaria. It is highly pathogenic for chickens. Complete information can be found on bird malaria in the excellent monograph by Hewitt (1940).

Family: Haemoproteidae

Haemoproteus columbae Kruse, 1890

HOST: Pigeon.

DISEASE: Pigeon malaria.

MORPHOLOGY: Only the morphology of the stages seen in the peripheral blood (gametocytes) will be described in this section. All stages of growth of the gametocytes are seen in normal sized red blood cells in the peripheral circulation of pigeons. The mature gametocytes are elongated, sausage shaped bodies partly or in some instances completely encircling the nucleus of the red cell. The nucleus of the red blood cell is usually laterally displaced by the organism. There are a number of pigment granules scattered throughout the cytoplasm of the parasite.

The microgametocyte has hyaline cytoplasm staining a pale blue and a nucleus which is diffuse and stains pink in dried films with the Romanowsky stains The pigment granules in the microgametocyte are frequently aggregated into several groups. The macrogametocyte has a denser cytoplasm than the male and stains a darker pink or red in comparison to the microgametocyte and a distinct karyosome may sometimes be seen The pigment granules are uniformly distributed throughout the cytoplasm.

LIFE HISTORY: Further development of the gametocytes requires their removal from the blood stream of the pigeon by a hippoboscid fly, Pseudolynchia maura. Microgametes are formed in the stomach of the fly by the process of exflagellation. Fertilization occurs and the zygote develops into an elongate motile ookinete. These penetrate the gut wall and form oocysts on its outer surface. The oocysts reach maturity 10 to 12 days after the fly is infected, whereupon they burst and liberate large numbers of sporozoites into the body cavity These sporozoites migrate to all parts of the body, particularly the salivary glands which are engorged with them The cycle in the hippoboscid fly is very similar to that of the true malarial parasite in the mosquito.

Infection of the pigeon occurs by the introduction of the sporozoites by the bite of the fly. They enter the blood stream and penetrate the endothelial cells lining the blood vessels, particularly in the lungs, liver and spleen where they undergo schizogony. The sporozoite after entering the host cell, rounds up and undergoes growth accompanied by division of the nucleus. The cytoplasm of this schizont breaks up into 15 or more cytomeres, each with a single nucleus. The nucleus of each cytomere now undergoes repeated division until the cytoplasm of the host cell, greatly hypertrophied, becomes filled with a number of multinucleate bodies Within each of these bodies is formed the merozoites. The schizonts may be seen with fully formed merozoites which will show the old cytomere structure (Huff, 1942). These mature schizonts with their contained merozoites are tortuous, and may send out branches along the capillaries so they may be bifurcate, triradiate or even multiradiate. These stages occur about four weeks after infection.

At this time the schizonts will break down and liberate the merozoites which enter red blood cells; gametocytes occur 28 to 30 days after the infection. Sometimes as many as a dozen merozoites will penetrate a red blood cell. However, it is only rarely that more than one mature gametocyte is seen in a cell, indicating that either cells with multiple infections die or some of the parasites leave the cells. These soon become elongate and appear as mature gametocytes.

SYMPTOMS: The effects of this parasite on the host vary from infections which appear to be benign with no symptoms to those which are fatal. There are no symptoms pathognomonic of this disease. Usually, there are no outward symptoms, the only indication of infection

being the presence of gametocytes in the red blood cells. In mild infections the birds are restless and off feed for several days. This disease is either thrown off or becomes chronic. In the latter instance the birds are anemic, generally weakened, and usually die following exposure to unseasonal climate conditions or after the breeding season.

In some birds acute cases may develop in the late spring or early summer, either as the result of a relapse from a previous infection or from a new infection. The birds lose flesh rapidly, are unable to fly, refuse food, become droopy and may die. A high percentage of the red blood cells in such cases are parasitized.

**PATHOLOGY:** There are no lesions characteristic of this infection except the presence of gametocytes in the red blood cells. The liver and spleen may be swollen and blackened. Small areas of focal necrosis may occasionally be seen of the liver. Variable pigment deposits are seen in the lungs, bone marrow and testes.

**TREATMENT:** There is no known treatment.

**CONTROL:** This infection will be controlled only be eradication of the louse fly on pigeons. The simplest way is to clean the nests and floors thoroughly every 3 weeks in order to remove the pupae. The pupal stage lasts about 25 days. The pupae are smooth and round and drop to the bottom of the nest boxes or floor, where they may roll around freely Pyrethrum powders, derris or cube or DDT will effectively destroy the flies on the pigeons. Cold weather will usually kill the flies in lofts in the northern parts of the United States

**GENERAL REMARKS:** Coatney (1933) has followed the course of the infection in pigeons particularly as regards the occurrence and character of the relapse He has found that relapses occur with extreme irregularity and could not be artificially induced. Birds could recover from an infection and still be susceptible to reinfection

### Haemoproteus lophortyx O'Roke, 1930

**HOST:** Quail.

This species has been reported from the California Valley quail and its life history has been studied by O'Roke (1930). It is transmitted by the hippoboscid fly Lynchia hirsuta The organism has been found extensively in wild quail in California and in breeding flocks on game farms.

**TREATMENT** Unknown

### Haemoproteus sp.

**HOST:** Turkey.

Morehouse (1945) reported on the presence of Haemoproteus in a turkey poult from Texas. He believed this to be a new species, but since it was found only in a single bird no specific name was presented The poult had an anemic appearance. It was assumed this was associated with the Haemoproteus infection.

**TREATMENT:** Unknown.

### Leucocytozoon simondi Mathis and Leger, 1910

**SYNONYM:** Leucocytozoon anatis Wickware, 1915.

**HOSTS:** Ducks.

**MORPHOLOGY:** Only the structure of the attenuated gametocytes will be discussed in this section, not the round forms found in the blood stream a few days earlier. The attenuated mature gametocytes are seen in the blood stream about 10 days after infection. The mature microgametocyte occurs in greatly enlarged host cells. The parasite lies parallel to the

elongated nucleus of the host cell, the latter averaging about 45 microns in length. The male gametocyte inside the cell is 3.1 to 4.2 microns wide and 14.7 to 18.9 microns long. The cytoplasm stains a pale blue, with Giemsa or Wright stain and contains numerous small pigment granules. The nucleus is diffuse, 3 to 4 microns in diameter, oval in shape and pale pink in color. The macrogametocytes are slightly larger as is the parasitized cell. The host cell averages 55 microns in length and 10 to 15 microns in width, the spindle-shaped parasite within the cell is 3.2 to 4.4 microns in width and 14.5 to 22 microns in length. The cytoplasm stains a much darker blue than in the male gametocyte, and it also contains numerous pigment granules. The nucleus is central, spherical and about 3 microns in diameter. It is a compact nucleus staining a dark red color and has a distinct karyosome.

LIFE HISTORY: The best descriptions of the life history of this parasite are those of O'Roke (1934) and Huff (1942). The earliest stages are the schizonts. They are found in macrophages or to some extent extracellularly in the liver, spleen and occasionally in the bone marrow. The smallest forms are ovoid bodies lying within vacuoles of the infected host cell and show a separation of densely staining material into two parts. The breaking up of these areas of darker staining material is apparently continuous. The small forms in the liver are very similar to those occurring in macrophages. The large forms fill the cytoplasm of infected hepatic cells. In each hepatic schizont there is a multiplication of the denser masses which are called cytomeres. When the cytomeres fill the entire cell they break into smaller bodies (merozoites).

In the mature hepatic schizonts the merozoites are arranged haphazardly without any trace of the earlier cytomeres Megaloschizonts develop in the heart, spleen and occasionally in the liver and intestines. The smallest form of this stage is very similar to the hepatic schizont. The parasite soon fills the cytoplasm of the host cell which may be a macrophage. The host cell nucleus continues to increase in size until it is many times its original volume. Cytomeres form in the megaloschizonts and their development closely resembles those seen in the hepatic schizonts. Several hundred individual cytomeres will be found in one cell, and measure from 3 to 5 microns in width and 5 to 9 microns in length The final stage in the schizogony of the megaloschizont, which may reach a size of 60 to 105 microns, is represented by the formation of a large number of merozoites. These are first separated into islands corresponding to the original cytomeres and then become arranged haphazardly.

It is not yet clear whether the gametocytes result from the merozoites of the hepatic schizonts, although it is probable they originate from the merozoites of the megaloschizonts. At the time these merozoites are being liberated, the blood stream contain many young stages of the gametocytes. The earliest gametocytes have been seen in myelocytes and late polychromatophil erythroblasts from the myeloid series, in lymphocytes, monocytes and macrophages of the non-granular series. The only cells in which Huff (1942) was able to find closely spaced stages of growth were the lymphocytes. The great distortion suffered by the host cell during the growth of the gametocytes has been a source of constant confusion to investigators. This has led not only to a varied classification of the infected host cells, but some workers question whether the host cell is actually present. Huff (1942) presented evidence that points rather conclusively to the lymphocyte and monocyte as being the normal host cell. He believed that errors in the past have been due to mistakes in diagnosing cell types after they have been altered beyond recognition, although he recognized the possibility that different species may infect different types of cells.

The earliest gametocytes are first seen in the circulating blood seven days after infection. They are oval in shape, 1 to 2 microns in diameter and may occur anywhere in the cytoplasm of the host cell. As it increases in size the nucleus of the host cell is pushed to one side and appears as a crescent-shaped, marginal band staining a dark purplish color. The gametocyte increases in size and remains spherical until it is 9 to 10 microns in diameter. The attentuated gametocytes are seen in the blood stream of infected birds about 10 days after infection.

Martin (1932) presented the view that attentuated gametocytes are probably degenerated forms, and only the rounded forms undergo gametogenesis. He has observed, in vitro, the formation of micro- and macrogametes only from the rounded forms. He also observed the process of fertilization and the development of the ookinete. These stages were similar to those which have been observed in the intermediate host. Whether or not these attenuated forms are actually degenerate organisms, has not been determined.

Further development of the sexual stages of L. simondi require that the gametocytes be removed from the blood stream by the black fly. Simulium venustum, S. nigroparvum, S. occidentale serve as vectors of this organism. After the blood has been removed by the fly, the female gametocytes round up and result in the formation of the macrogamete which is about 8 microns in diameter. At the same time the male gametocytes round up and produce 4 to 8 microgametes. These are very slender and 23 to 24 microns in length. They become actively motile organisms in the gut of the fly. Soon after their formation fertilization takes place with the formation of the zygote. Five hours after the black flies have fed on infected birds the zygote elongates and becomes an ookinete. The latter is a vermiform shaped organism averaging 33.3 microns in length and 4.6 microns wide. The ookinete penetrates the stomach wall of the fly and results in the production of spherical oocysts on the outer wall. The exact time required for the development of the oocyst is not known. Repeated division within the oocysts results in the formation of numerous elongated sporozoites. The sporozoites are later liberated from the oocysts and find their way to the lumen of the salivary glands, where they may be discharged into a susceptible host during the feeding activities of the fly.

At the present time only simuliid flies have been incriminated as vectors of this disease in ducks. However, it seems possible that other biting or blood sucking arthropods may play a role in its transmission. It should be noted this organism cannot be transmitted from infected to susceptible ducks by the inoculation of infected blood which contains only the gametocyte stage. It is necessary to transmit the schizogonous forms, which are not found in the blood stream and hence would not take place under natural conditions. Thus, simple mechanical transmission of this disease from host to host is not possible.

**SYMPTOMS:** The outstanding characteristic of this disease is the suddenness with which it strikes. A flock of ducklings may appear to be active and well in the morning, by mid-afternoon or evening act sickly and refuse food and the next morning they may be dead. In very sick birds there is rapid and labored breathing due to the mechanical obstruction of the capillaries in the lungs by the parasites. There is a brief period of nervous excitement just preceding death. In young birds from 10 days to a few weeks old these acute symptoms are the most constant. Chronic adult carriers, aside from being thin, show no symptoms peculiar to this infection. Adult ducks contracting the infection for the first time do not exhibit the characteristic acute symptoms of ducklings. They are listless and if they are wild ducks they will appear tame and show little interest in what is occurring about them. Symptoms appear more gradually and it is unusual for an adult to die in less than 4 days after the disease has been noted. However, death is not common in the older birds.

If a duckling recovers from an acute attack and becomes a chronic carrier of the parasite it may either be stunted and fail to grow. Some birds become to all outward appearances normal. Recovery, however, depends upon the mildness of the initial attack, but all recovered birds remain carriers. In adult birds that suffer relapses no characteristic symptoms are noted.

Mortality varies from 0 to 100 per cent in various sections of the country and in different outbreaks. O'Roke (1934) over a three year period observed losses of 35 per cent, 85 per cent and 57 per cent of the young ducks under his observation. In adults the losses were very low.

**PATHOLOGY AND DIAGNOSIS:** The pathology produced by L. simondi is not characteristic. There is a marked hypertrophy of the liver, enlargement and blackening of the spleen and anemic blood which has lost its power to clot. A diagnosis must be confirmed by the demonstration of the parasites in the blood stream.

**TREATMENT:** At the present time there is no treatment which is effective against this disease. The known antimalarials and sulfonamides have not been shown to have value.

**CONTROL:** Control at the present time rests primarily in the protection of ducks from the attacks of the black flies, and the removal of the carrier ducks. The former may be carried out either by rearing the birds in areas free of black flies or in screening the ducks completely against the attacks of these flies. It should be borne in mind that screening is very difficult unless one wishes to use a very expensive fine mesh, wire screen. Ordinary window screening with 16 mesh to the inch is ineffective against black flies. A mesh of 32 to 36 mesh to the inch is required to prevent their entrance. If wire of this mesh is used the cost would be prohibitive, therefore, a good grade of cheesecloth has been recommended as being satisfactory for one summers use.

## Leucocytozoon smithi (Laveran and Lucet, 1905)

**HOSTS:** Turkey

**MORPHOLOGY:** Both micro- and macrogametocytes occur in the peripheral blood of infected turkeys about 9 days after infection The early forms are rounded, but these later become attenuated as in L. simondi In wet smear preparations the mature gametocyte stages which are much elongated with the host cell averaging 58.2 microns in length and 5.4 microns in width. In stained preparations the parasitized cells containing the microgametocytes measure 44.75 microns long and 13 75 microns wide The microgametocyte measures 22.0 by 6 25 microns and the macrogametocyte 20 25 by 6.25 microns.

The nucleus of the host cell frequently appears as a lateral bar either on one or both sides of the gametocyte, in the latter case the parasite occurs between the two bars

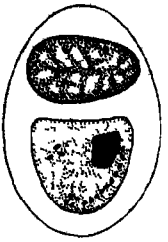
**LIFE HISTORY:** The life history of this species is not completely understood. Simulium nigroparvum, S venustum and S. occidentale have been incriminated as vectors of this organism. S. slossonae has been shown by Underhill (1944) to feed on turkeys and it is assumed this species may also transmit the leucocytozoa. Savage and Isa (1945) reported a severe outbreak in Canada in the absence of any simuliid flies, but reported numerous stable flies and mosquitoes to be present It seems probable the development of this species in the turkey resembles that of L. simondi in the duck. However, Johnson, Underhill, Cox and Threlkeld (1938) and Johnson (1942) have been unable to demonstrate any schizogonous development The stages in the fly resemble those seen in L. simondi.

Simuliid flies are found principally along stream and rivers, with the swifter flowing waters being more suitable than the sluggish The eggs are deposited on rocks and sticks in the water The larvae and pupae are usually found attached to objects one to several inches below the surface of the water. The adult fly on emerging from the pupal skin arises to the surface of the water and takes flight at once. The adult flies are blood suckers and will feed on turkeys from late spring to early fall

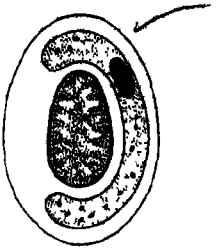
The feeding flies attach themselves to the head and neck of the birds, and are not easily disturbed until they have finished feeding. Usually the blood meal is completed in 2 or 3 minutes. The flies will apparently feed at any time during the day, but prefer warm, quiet humid weather, and will not feed if there is a strong wind. They are most active just preceding or following a rain with the temperature about 80° F. There is little feeding at temperatures below 70° F. or above 90° F It is of interest to note that black flies will migrate considerable distances from their breeding places, species having been recovered feeding on turkeys 5 to 6 miles from running water.

**SYMPTOMS:** Young turkeys up to 12 weeks of age are chiefly affected. Visible symptoms are only noted for 2 or 3 days after which the birds have died or recovered. The onset of symptoms is sudden. The poults lose their interest in food, appear droopy and only move about with difficulty. In the later stages of the disease any excitement will frequently result in the death of the birds. As in the similar infection in the duck recovery results in the birds becoming carriers. Many of these recovered carrier birds develop a chronic form of the disease. The males pay little attention to the females and rarely

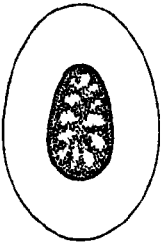
PLATE XX



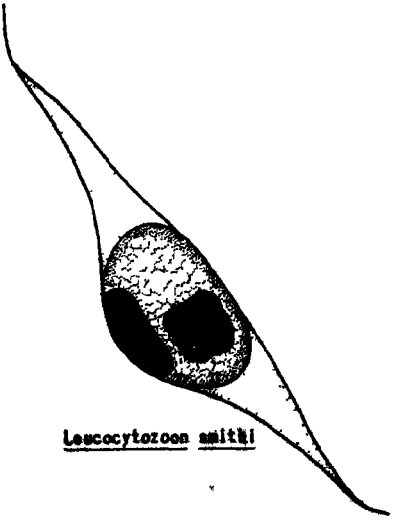
Plasmodium sp.



Haemoproteus columbae



Normal chicken erythrocyte



Leucocytozoon smithi

Tetrahymena geleii

strut Moist tracheal rales are frequently present and they make repeated attempts to clear their throats Mortality varies from practically no losses up to as high as 90 per cent.

PATHOLOGY: The spleen and liver are swollen and enlarged. There may be an enteritis throughout the intestinal tract. There are no pathognomonic lesions of the disease.

TREATMENT AND CONTROL: Same as for Leucocytozoon simondi.

GENERAL REMARKS. At the present time this disease is probably prevalent throughout the United States and Canada It has been reported on both the Atlantic and Pacific Coast, along the gulf states and in Canada. It is probably overlooked in many instances, due to the lack of blood examinations.

Leucocytozoon bonasae Clarke, 1935

HOST. Ruffed grouse.

Clarke (1935) described this parasite from grouse in Canada and showed that it was associated with a cyclic mortality in the grouse population. Later, Clarke (1938) reported on the relationship between schizonts and gametocytes in connection with seasons and the age of the host.

TREATMENT: Unknown.

CLASS. CILIATA
ORDER: HOLOTRICHA
Family Frontoniidae

Tetrahymena gelsii Furgason, 1940

This ciliate has been recovered from the digestive tract, infraorbital sinuses and serous material under the eyelids of chickens by Knight and McDougle (1944) in Missouri. The organism is pyriform in shape, 40 to 60 microns long and 15 to 30 microns wide. The cytostome is funnel-shaped and about one-tenth of the body length There are 17 to 19 ciliary meridians and three distinct membranelles in the mouth cavity in addition to the undulating membrane.

The significance of this organism in chickens is unknown, as it is a free-living form usually found in stagnant pools In the flock parasitized with this ciliate, the organisms could only be found in those birds showing a vitamin A deficiency.

TREATMENT: Unknown, probably unnecessary

## ORGANISMS OF UNCERTAIN ZOOLOGICAL CLASSIFICATION

### Avian Toxoplasma

Toxoplasma has been found to occur naturally in pigeons and is transmissible to chicks. Wolfson (1941) gave a good review of the subject. The method of transmission is unknown. Wolfson (1942) transmitted a guinea pig strain to young chicks, duck and chick embryos.

Chicken eggs which were inoculated with human Toxoplasma developed macroscopic lesions on the chorio-allantoic membranes in about 5 days. The liver and spleen were sometimes necrotic and contained Toxoplasma. The organism was regularly found in the erythrocytes of infected embryos.

TREATMENT: Unknown.

"Avian Babesiasis"

McNeil and Hinshaw (1944) reported the finding of an intraerythrocytic blood parasite from 10-week-old turkey poults. The organisms varied in diameter from 0.5 to 2 microns. It is located halfway between the nucleus and the edge of the red cell. They are generally round, oval or pear-shaped. Usually one parasite is found within a cell, never more than two. It has been found also in the liver. The blood picture shows some anemia. Post-mortem examination revealed a flabby heart, congestion of the intestine with diarrhea and abnormal clotting of blood. The organism resembles Sauroplasma thomasii.

**TREATMENT:** Unknown.

Sarcocystis rileyi (Stiles, 1893)

According to Erickson (1940) Sarcocystis sp. has been reported from twenty-one species of birds, eleven occurring in the United States. There are very few records of its presence in the domestic fowl.

Hawkins (1943) noted the presence of S. rileyi in chickens from Michigan. Sarcocysts were noted on the external surface in the thigh muscles. The parasite is somewhat more prevalent in waterfowl, ducks in particular.

As mentioned throughout this book, Spindler et al (1945, 1946, 1947) have presented evidence to show that Sarcocystis actually is a fungus. This work has yet to be confirmed.

On esthetic grounds alone, birds or animals infected with Sarcoystis should not be used for human consumption.

Erickson (1940) captured a pintail duck which was in a weakened condition. It was so heavily infected that all of the skeletal muscles and heart were parasitized. The duck was quite emaciated. Of 43 ducks of eight species examined in Minnesota three or 6.97 per cent were infected with S. rileyi.

The sarcocysts in ducks measure from 1 to 6 mm in length to 0.5 mm in width The crescent-shaped spores measure about 12 to 14 microns in length.

**TREATMENT:** Unknown.

## CHAPTER VIII

## PROTOZOA OF FUR BEARERS

## Chapter VIII

## PROTOZOA OF FUR BEARERS

### RABBIT

CLASS: MASTIGOPHORA
ORDER: PROTOMONADINA
Family: Bodonidae

Retortamonas cuniculi (Collier and Boeck, 1926)

SYNONYM. Embadomonas cuniculi Collier and Boeck, 1926.

The trophozoite measures from 7.5 to 13 microns in length and 5.5 to 9.5 microns in width. It is generally ovoid in shape but occasionally it is observed with a tail-like process. The body is slightly compressed. The cytostome is located anteriorly as is the nucleus. There are two flagella, one delicate anterior flagellum which is approximately body length and a posterior flagellum which is thick and one-half the length of the body. The cyst is oval and measures 5 to 6.5 microns by 3.5 to 4 microns and contains a central nucleus. Division of the trophozoite occurs by binary fission. The flagellate is found primarily in the cecum of domestic rabbits. It is not very common being reported once from a total of 50 rabbits examined The organism is apparently nonpathogenic.

TREATMENT: Unnecessary.

Family: Hexamitidae

Giardia duodenalis (Davaine, 1875)

SYNONYM. G. cuniculi (Benson, 1908).

This flagellate has been reported sporadically to produce a serious disease in rabbits. More information is required. According to Hegner (1922) the average size is 15.8 microns long by 9.1 microns wide (range 12.7 to 18.7 by 7.7 to 11). The body is pear-shaped. Two nuclei are present. Two axostyles are also present terminating at the anterior end by a single blepharoplast. The dorsal side is convex, the ventral side concave with a sucking disc in the anterior end. There are 4 pairs of flagella. Cysts forms have been observed, oval in shape and containing 2 to 4 nuclei. This organism is apparently nonpathogenic in rabbits.

TREATMENT: Unnecessary.

ORDER. POLYMASTIGINA
Family: Chilomastigidae

Chilomastix cuniculi Fonseca, 1915

This flagellate has been reported several times from the cecum of domestic rabbits. It is morphologically similar to C. mesnili of man. The trophozoite is from 10 to 15 microns in length with an extreme range in size from 3 to 20 microns. The anterior end is rounded while the posterior end is drawn out to a point. A cytostome is present. The nucleus is located at the anterior end. There are three anterior flagella. The flagellate reproduces by longitudinal fission. It is apparently nonpathogenic.

TREATMENT: Unnecessary.

ORDER: TRICHOMONADIDA
Family: Monocercomonadidae

Monocercomonas cuniculi (Tanabe, 1926)

SYNONYMS: Eutrichomastix cuniculi (Tanabe, 1926); Trichomastix cuniculi Tanabe, 1926.

This flagellate occurs in the cecum of the domestic rabbit. It is pyriform in shape and ranges from 5 to 14 microns in length. The nucleus is ellipsoidal with a large karyosome and is located anteriorly. The slender arostyle is hyaline and projects from the posterior end of the body. There are three anterior flagella and one posterior or trailing flagellum. It is apparently nonpathogenic.

TREATMENT: Unnecessary.

CLASS: SARCODINA
ORDER: AMOEBINA
Family: Endamoebidae

Endamoeba cuniculi (Brug, 1918)

SYNONYM: Entamoeba cuniculi (Brug, 1918)

This amoeba has been observed on several occasions from the intestines of the rabbit. It appears to be similar to E. coli which was discussed in detail in Chapter V. The trophozoites measure from 10 to 20 microns. The cysts are oval and measure from 10 to 20 microns in diameter. Typical cysts contain eight nuclei. It is a harmless commensal in the digestive tract of the rabbit.

TREATMENT: Unnecessary.

CLASS: SPOROZOA
ORDER: COCCIDIA
Family: Eimeriidae

There are at least five valid species of coccidia found in rabbits. The basic characteristics of the species reported from domestic rabbits in the United States are given below.

Eimeria stiedae Lindemann, 1865

Oocysts range from 28 to 40 microns in length by 16 to 25 microns wide. The shape is mainly ovoidal or ellipsoidal and have a yellowish-orange to salmon tint coloration. The micropylar end is flat and the micropyle is quite prominent. The oocyst walls are thin. Sporulation time is between 60 and 70 hours.

This species parasitize the bile ducts. The oocysts collect as circumscribed white nodules on the surface of the liver. They make their way to the intestine through the bile ducts, gall bladder and to the common bile duct.

LIFE CYCLE: The endogenous cycle occurs in the epithelial cells of the bile ducts. Rarely, liver cells are invaded. The species has a definite organ-specificity for the liver. Oocysts first appear in the feces 16 to 17 days after becoming infected. By the 22nd day the schizogony phase has been completed.

In severe infections the liver may become enlarged to constitute 20 per cent of its body weight. This was reported by Jankiewicz (1945). The increase of biliary epithelium

and proliferation of new bile ducts account for this enlargement.

TREATMENT: Jankiewicz (1945) treated 8 rabbits with sulfasuxidine for 13 days by mixing 0.625 grams of the drug in feed and 0.75 grams daily for 19 more days. The rabbits were then given 200,000 oocysts of E. stiedae. As controls, 4 rabbits without treatment were infected and 4 rabbits were untreated and uninoculated. After 18 days the rabbits were autopsied and the livers examined grossly and microscopically. The treated rabbits were free from infection. The drug is moderately toxic when given for a period of 32 days.

GENERAL REMARKS: Kessel and Jankiewicz (1931) examined over 2000 rabbits in California and found only 9 per cent infected with this species. It was not common in rabbits from 5 weeks to 3 months of age.

## Eimeria magna Perard, 1925

Oocysts range between 28 to 40 microns long by 20 to 26 microns in width. It is ovoid in shape and is colored brown or yellowish brown. Their shape is typically ovoid A broad micropyle is distinguishable at the tapering end. The sporulation time is approximately 48 hours.

LIFE CYCLE: The endogenous cycle requires 7 days. The parasite prefers the middle jejunum and all of the ileum. According to Rutherford (1943) 12 hours after feeding sporulated oocysts the sporozoites are starting to enter the epithelial cells. Schizonts appear in about 36 hours. First generation merozoites are noticed after 4 days. The merozoites enter other epithelial cells to form either second generation schizonts or develop into gametes. Microgametocytes were observed after five days, macrogametocytes on the fifth and sixth days.

TREATMENT: Lund (1945) used phthalysulfathiazole at doses averaging 0.20 to 0.25 grains per pound of body weight per day to control E. magna infections. Some success was reported.

GENERAL REMARKS: Kessel and Jankiewicz (1931) found about 19 per cent of the rabbits examined in a survey in California were infected with E. magna.

## Eimeria irresidua Kessel and Jankiewicz 1931

Oocysts measure 38.3 microns long by 25.6 microns wide. A micropyle is present. The shape is ovoidal to ellipsoidal. Time of sporulation requires from 50 to 64 hours.

LIFE CYCLE: This coccidium occurs in the epithelium of the villi from the duodenum to the lower ileum. The heaviest infection according to Rutherford (1943) occurs in the first 18 inches of the intestine. Sporozoites liberated from infective oocysts are active for three days and develop into schizonts. After 6 days mature merozoites are seen. The merozoites are liberated by a gradual necrosis of the villi. They are free to produce the second generation of schizonts. Between the 6th and 8th day macro- and microgametocytes appear. From the 9th to 10th day well developed oocysts are passed in the feces.

TREATMENT: Unknown.

GENERAL REMARKS: The incidence of infection in California according to Kessel and Jankiewicz (1931) was 10 per cent.

## Eimeria media Kessel, 1929

The oocysts measure on an average of about 31.2 microns in length by 18.5 microns in width. A micropyle is present. Sporulation requires 40 hours.

LIFE CYCLE: The endogenous phase of the parasite occurs in the small intestine. Schizonts develop in the epithelial cells between 12 and 24 hours. On the 4th day the

first generation of merozoites appear. The second generation of merozoites appear on the 6th day. Microgametocytes occur on the 5th and 6th day as do the macrogametocytes. Oocysts are passed in the feces 6 days after ingestion of infective oocysts.

GENERAL REMARKS: A survey by Kessel and Jankiewicz (1931) on California rabbits showed a 12 per cent incidence.

Eimeria perforans Leuckart, 1879

The oocysts are colorless or light pink and measure between 24 to 30 microns in length and 14 to 20 microns in width. They are ovoidal or ellipsoidal in shape, a micropyle is not readily distinguishable. It is the smallest of the coccidia which occur in domestic rabbits. The sporulation time varies from 30 to 48 hours.

LIFE CYCLE: The endogenous cycle occurs in the epithelial cells of the duodenum to lower ileum. Sporozoites enter the epithelial cells approximately 12 hours after ingestion of sporulated oocysts. Schizonts are formed within 12 hours and the first generation of merozoites occur in 3 to 4 days. According to Rutherford (1943) the second generation of merozoites appear on the 4th and 5th day as do the micro- and macrogametocytes. Oocysts occur in the feces from 5 to 5½ days after ingestion of infective material. This species is the least pathogenic of the coccidia found in the intestine.

GENERAL REMARKS: The incidence of infection in California rabbits was 30 per cent according to Kessel and Jankiewicz (1931).

Bachman (1930) studied 57 rabbits from a colony free of parasites except for E. perforans. Animals with previous infections were immune to experimental infection to the same species of coccidia. Rabbits immune to E. perforans were not resistant to infection with E. stiedae. Attempts to immunize rabbits either actively or passively failed to protect rabbits against E. perforans.

Eimeria neoleporis Carvalho, 1942

This species occurs in the apical process of the cecum and the ileo-cecal valve of cottontail rabbits in Iowa. Carvalho (1944) was able to infect domestic rabbits with this species.

Coccidiosis in Rabbits

Coccidiosis is very common and is one of the main causes of mortality by a protozoan parasite in our domestic rabbits. Oocysts are passed out with the feces in large numbers and when they contaminate food and water continual reinfection takes place. One of the most common methods of infection is by means of contaminated fur. Rabbits licking and grooming themselves after their fur is soiled by lying on soiled floors or bedding become infected.

SYMPTOMS: Symptoms vary with the number of infective oocysts ingested. This may range from a slight attack with no symptoms, to severe acute attacks resulting in death. Young animals just after weaning are most susceptible. Loss of appetite, diarrhea and rough coat are other signs of the disease. Rabbits dying of intestinal coccidiosis generally have an empty stomach.

PREVENTION AND CONTROL: Good, self cleaning hutches should be used. Contamination with feces is prevented by having floors composed of hardware cloth or perforated sheet iron. Coccidia are difficult to eliminate from wooden or earthen floored hutches. Even self cleaning hutches should be cleaned at least once per month. Feed and water containers should be constructed so that fecal contamination is kept at a minimum.

Since older rabbits become carriers it is helpful to separate the young as soon as possible.

DIAGNOSIS: Diagnosis can be made by association of symptoms and a microscopic examination of the feces for oocysts.

TREATMENT: More information is needed on the treatment of coccidiosis in rabbits.

## ORGANISMS OF UNCERTAIN ZOOLOGICAL CLASSIFICATION

### Toxoplasma sp.

Perrin (1943) reported on a spontaneous Toxoplasma infection in a rabbit. The case was not recognized clinically, but was discovered when tissues were subjected to histological studies. The parasite appeared as crescent-shaped structures about 3.0 by 6.0 microns in size. No gross changes were observed at autopsy. More study is needed on this parasitic disease. It is of little veterinary importance at the present time.

TREATMENT: Unknown.

### Sarcocystis leporum Crawley, 1914

This parasite is quite common in the cottontail rabbit but so far as known has not been reported from domestic rabbits. Erickson (1946) presented a complete review of the parasite including incidence and transmission in cottontails. One domestic rabbit that came in contact with heavily infected cottontails developed a light infection. In view of the work by Spindler et al (1945, 1946, 1947) this organism may be a fungus.

TREATMENT. Unknown.

## FOX

CLASS SPOROZOA
ORDER. COCCIDIA
Family: Eimeriidae

### Coccidiosis in Foxes

The occurrence of oocysts of coccidia in the feces of foxes has been reported from time to time. So far as known, no comprehensive studies have been made on coccidiosis in the fox. A critical study should be made on foxes by which the experiment could be controlled by checking the diet of the fox, by repeated fecal examinations and the location of the asexual stage in the epithelial cells. The last precaution should be rigidly followed as small mammals consumed by the fox may be harboring oocysts and pass through the intestines without being digested.

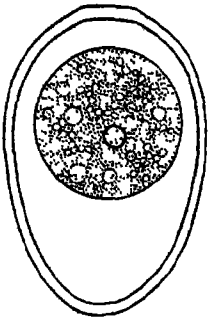
As mentioned in Chapter VI concerning the coccidia of cats and dogs, these coccidia invade the epithelial cells of the small intestine. Apparently there are about four species of coccidia reported from foxes in North America all belonging to the genus Isospora. Just how important coccidiosis in the fox may be from an economic standpoint is not known. The practice of raising foxes on wire has taken care of controlling the disease in most instances. The four species are listed below.

### Isospora canivelocis Weidman, 1915

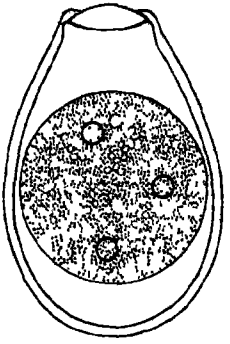
This oocyst was recovered in the feces of two swift foxes (Vulpes velox). Oocyst range between 30 to 40 microns long and 25 to 27 microns wide. The shape is extremely variable from spherical, ovoid or ellipsoidal. Sporulation time is approximately 24 hours.

PLATE XXI

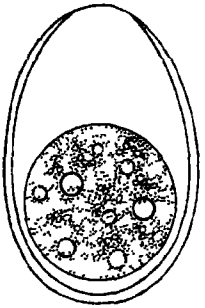
COCCIDIA OF RABBITS



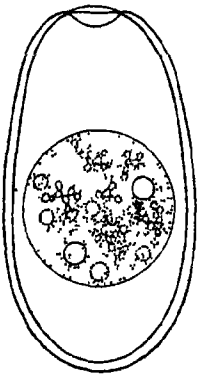
Eimeria irresidua



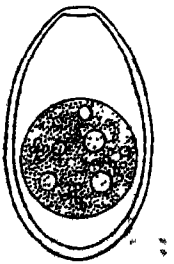
Eimeria magna



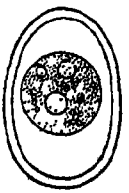
Eimeria stiedae



Eimeria neoleporis



Eimeria media



Eimeria perforans

Weidman found oocysts within ulcers produced in the small intestine and was of the opinion that the organism was pathogenic. It is not known whether this parasite is prevalent in foxes raised on fur farms.

TREATMENT: Unknown.

Isospora bigemina (Stiles, 1891)

This species is quite common in dogs Lee (1934) infected foxes experimentally with I. bigemina of dog origin and also infected dogs and cats with Isospora of fox origin. Oocysts resembling I. bigemina were also observed in naturally infected foxes. Triffitt (1927) described oocysts in foxes from Prince Edward Island, Canada which appear to be this species.

Two types of oocysts are recognized, large and small. Small oocysts measure from 10 to 14 microns in length by 7 to 9 microns in width. Large oocysts 18 to 20 microns by 14 to 16 microns. A micropyle is not present. The shape is broadly oval to spherical. Sporulation usually occurs within 24 hours.

During the acute phases of the disease the epithelial cells are attacked but during the chronic stages the infection is confined to the subepithelial tissues. Also during the chronic stages, the oocysts undergo sporulation in the mucosa, rupture and release sporocysts which are observed in the feces. More investigations are necessary to determine the incidence of this parasite on fur farms and how important it is from an economic and veterinary viewpoint.

TREATMENT Unknown.

Isospora rivolta Grassi, 1879

Oocysts measure 20 to 25 microns in length and 15 to 20 microns in width. They are oval in shape with a micropyle at the tapering end. Oocysts resembling this species have been reported from wild carnivores. This species is probably non-pathogenic. More research is needed on this species. The incidence on fur farms is not known.

TREATMENT: Unknown.

Isospora felis Wenyon, 1923

The oocysts range in length from 39 to 48 microns and from 26 to 37 microns wide. It is a common parasite of the cat. Lee (1934) infected a fox experimentally with I. felis of dog origin. Oocysts were also observed in the feces of foxes which resembled I. felis. These oocysts could be used to infect cats and dogs. The pathogenicity in foxes is not known. More research is needed on this coccidium.

TREATMENT: Unknown.

## MINK

CLASS: SPOROZOA
ORDER: COCCIDIA
Family. Eimeriidae

### Coccidiosis in Mink

Very little information is available on the different species of coccidia which occur in ranch raised mink. The species as reported by Kingscote (1934) and Levine (1947) from mink are listed below.

Eimeria mustelae Iwanoff-Gobzem, 1934

This species was originally described from the weasel (Mustela nivalis) in Europe and reported for the first time from mink in the United States (Illinois) by Levine (1947).

The oocysts are subspherical; 16.1 by 14.4 microns in size. No oocyst micropyle or residual body present. The asexual cycle has not been studied. The pathogenicity of E. mustelae is unknown.

Eimeria vison Kingscote, 1934

SYNONYM: Eimeria mustelae Kingscote, 1934, not Eimeria mustelae Iwanoff-Gobzem.

Kingscote (1934) found this species causing death in mink from enteric coccidiosis. Oocysts are oval to egg-shaped; the wall is double layered, with a yellowish-brown inner layer thicker than the external colorless layer. The wall is approximately 0.75 microns thick. No micropyle is present. The size ranges from 17.0 to 22.1 by 9.0 to 18.0 microns. Average size is about 20.3 microns long and 14.6 microns wide. Time required for sporulation is about 7 days.

Experimental animals (1 mink and 2 ferrets) fed infective oocysts passed nonsporulated oocysts in 6 days and disappeared entirely after 4 days.

Levine (1947) briefly described the oocysts of this species recovered from a dead mink. The oocysts were ellipsoidal and measured 22.8 microns long by 15.4 microns wide. No micropyle was observed.

Isospora bigemina (Stiles, 1891)

This species is discussed more fully in Chapter VI from dogs and cats. It has been reported infrequently from mink. Levine (1947) reported oocysts of this species in a mink from Illinois. The oocyst wall is very thin and stretched by the sporocysts. Oocysts measured 12.4 microns long and 9.0 microns wide. No oocyst micropyle or residual body present. Each sporocyst contains a large residual body.

Isospora laidlawi Hoare, 1927

Oocysts of this species were first described from the ferret by Hoare (1927). It was reported for the first time from mink in the United States by Levine (1947). Oocyst shape is ellipsoidal and measure 34.0 microns long by 26.5 microns wide. No micropyle is visible. Sporulation requires four days.

GENERAL REMARKS: In view of the fact that most ranch raised mink are kept on wire-floored pens or cages, the danger of coccidiosis has been reduced. Mink raised on the ground are more apt to become infected. Young mink may become infected by ingesting sporulated oocysts if kept in nesting boxes too long. The actual problem of coccidiosis in mink has not been studied.

TREATMENT: Unknown.

## CHAPTER IX

## DIAGNOSIS OF PROTOZOAN INFECTIONS

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## DIAGNOSIS OF PROTOZOAN INFECTIONS

### CLINICAL DIAGNOSIS

The clinical manifestations of protozoan diseases in domestic animals as a rule are so general that in the majority of cases diagnosis cannot be based on symptoms alone. The experienced veterinary clinician may recognize certain characteristic symptoms, however, the final diagnosis requires the accurate identification of the protozoan parasite.

Some of the clinical symptoms associated with protozoan diseases include anemia, diarrhea, dysentery, fever, eosinophilia and intestinal disturbances.

### LABORATORY DIAGNOSIS

Accurate laboratory diagnosis requires a suitable knowledge of basic fundamental procedures including the skillful use of the microscope, preparation of material for microscopic examination and practical experience in the recognition of protozoan forms.

### EQUIPMENT NECESSARY FOR THE STUDY OF PARASITIC PROTOZOA

The microscopical study of parasitic protozoa, either in stained or fresh preparations requires certain equipment and chemical reagents. The first essential apparatus needed is a good microscope equipped with 5x and 10x oculars, low, high and oil immersion objective, mechanical stage and condenser. Other equipment includes a microscope lamp, *3 x 1 inch glass slides, cover-glasses, test tubes, dissecting needles, wire loops,* hemocytometer, centrifuge, toothpicks, specimen applicators, pipettes and a dropping bottle of physiological saline. Chemicals for routine laboratory work include alcohol, carbolic acid, iodine solution, Wright's or Giemsa's stain and other reagents.

It is assumed that graduate veterinarians and protozoologists are familiar with the microscope. There are many handbooks and references on microscopy which can be consulted for further information.

The micron is the unit of measurement for microscopical material. One micron is 1/25,000 of an inch or 1/1000 of a millimeter. The apparatus used for measuring this unit is the ocular micrometer which is a scale etched on a glass disc. This disc fits in the eye-piece of the microscope. The ocular micrometer is standardized by the use of a stage micrometer. This is a glass slide which has an etched scale divided into tenths and hundredths of a millimeter. By superimposing the ocular scale with the stage micrometer the value of each division on the ocular micrometer can be determined.

### PSEUDOPARASITES

Many structures or free-living protozoa may occur in diagnostic material. Care must be exercised in the proper identification of protozoa. To the inexperienced diagnostician plant cells or other artifacts may cause some confusion. The greatest source of confusion includes yeast, fungi, pollen grains, starch granules, air bubbles, oil globules, mucus, epithelial cells and other tissue cells.

## DIAGNOSIS OF PROTOZOA FROM THE ALIMENTARY TRACT

### MOUTH

Scrapings should be made from the teeth and gums. Accumulations of tartar should be examined. The only protozoa which occur in the mouth are the various species of trichomonads of dogs, cats and horses. Swabs of the mouth are often made on aborted bovine fetuses in searching for trichomonads.

### FECES

Most of the protozoa which live in the intestinal tract are found in the feces. Feces should be collected as fresh as possible and free from soil, rocks, straw or urine. The material should also be collected in clean, dry, small glass jars or one-half pint waxed paper cups fitted with lids.

Gross examination of the fecal material may give some information concerning the condition of the intestinal tract and the nature of the diet. Some of the characteristics of feces to observe include color, presence of fresh blood indicates hemorrhage of the upper intestine.

Since protozoa are found more frequently after purging than in normal stools, one may administer a saline cathartic. This should be done only if normal stools are negative and intestinal protozoa are still suspected.

Feces may be collected from the rectum by means of a blunt curette, swab, or by the hand enclosed in a rubber glove.

### DIRECT SMEAR

A small amount of feces is placed on a glass slide and with a toothpick is emulsified in one or two drops of warm physiological saline. A cover-glass is placed over the material and the preparation examined under the microscope for protozoa.

A drop of iodine solution may be added to the slide. This kills the motile trophozoites, however, the various protozoa appear somewhat differentiated.

### IODINE SOLUTION

| | |
|---|---|
| Potassium iodide | 4 grams |
| Iodine | 2 grams |
| Distilled water | 100 cc |

### HEIDENHAIN'S IRON HEMATOXYLIN METHOD

1. A film is prepared by smearing a small amount of material on a clean cover glass to form a thin, moist film.

2. Do not let the preparation dry and fix from 10 to 20 minutes in Schaudinn's fluid by floating the cover glass, film side down, in the solution which has been warmed to 37° C. The solution may be made up in sufficient amount to fill a Petri dish. After fixation, the preparation should be turned over with the film side up.

SCHAUDINN'S FLUID

| | |
|---|---|
| Saturated bichloride of mercury | 2 parts |
| Absolute alcohol (or 96% alcohol) | 1 part |

The alcohol is mixed with the saturated solution of bichloride of mercury The 5% glacial acetic acid is added immediately before using

3. The fixative is drained off and the slide covered with 50% alcohol. The process is repeated several times to remove the fixative.

4. Place in 70% Iodine-alcohol for 3 to 5 minutes, 70% alcohol 3 to 5 minutes; 85% alcohol 3 to 5 minutes, 95% alcohol 3 to 5 minutes, 85% alcohol 3 to 5 minutes, 70% alcohol 3 to 5 minutes, 50% alcohol 3 to 5 minutes and 30% alcohol 3 to 5 minutes.

5 The slide is washed in tap water for 10 to 20 minutes and covered with mordant for 15 minutes in incubator at 37°C Good preparations may be obtained by allowing the mordant to act over night in an incubator

MORDANT

| | |
|---|---|
| Ferric ammonium sulphate | 4 grams |
| Distilled water | 100 cc |

6 The mordant is drained off, the slide washed in several changes of tap water 5 to 10 minutes and placed in 0.5% aqueous solution of hematoxylin for the same period as used in the mordant.

HEMATOXYLIN SOLUTION

| | |
|---|---|
| Hematoxylin (any standard hematoxylin) | 0 5 grams |
| Distilled water | 100 cc |

The hematoxylin is dissolved in 5 cc of 95% alcohol and diluted to 100 cc with distilled water It should ripen for at least two days before using

7 The material is differentiated in fresh 2% aqueous solution of ferric ammonium sulphate (iron alum). This is the most critical step in the whole procedure and must be done carefully The preparation is removed with forceps, dipped into the solution and quickly rinsed in a beaker of tap water. The preparation should now be examined under the low power of the microscope to see if sufficient differentiation has taken place If not, the process is repeated until the nucleus stands out blue-black, with greyish cytoplasm.

8. After differentiation, it is washed in several changes of tap water or running water from 20 to 30 minutes.

9. The next step is dehydration in graded alcohols. 30%, 50%, 70%, Iodine-alcohol, 85%, 95% and absolute.

10. After clearing in xylol (oil of thyme or oil of cloves may be used) for 5 minutes, the preparation is mounted in balsam

HEIDENHAIN'S IRON-HEMATOXYLIN METHOD (RAPID METHOD)

1. Smears are made on cover glasses or slides; fixed in methyl-alcohol for about five minutes and dried in the air.

2. After washing in water, it is left in the mordant (4% iron-alum aqueous) for about three hours, preferably in the incubator. Crystals of ferric ammonium sulphate that are pure violet color should be used.

3. After washing thoroughly in tap water the preparation is stained in 1/2% ripened hematoxylin for 1/2 hour. (0.5 gm. of hematoxylin in 10 cc of absolute alcohol and add 90 cc of water. Ripen for 2 or 3 months in sunlight.)

4. The preparation is washed thoroughly in tap water and differentiated in 4% iron-alum until the nuclei are well-defined. This is controlled under the microscope, washing smears in tap water before examination.

5. After the mordant is washed thoroughly in tap water, the slide is dried in the air, mounted with balsam and labeled.

## FLOTATION METHODS

The flotation techniques utilize a difference in specific gravity of various solutions and of the protozoa. The difference in specific gravity causes protozoan cysts, coccidia or trophozoites to rise to the surface while the fecal particles sink to the bottom.

Sugar, zinc sulfate or a saturated solution of sodium nitrate is good for concentrating protozoa for diagnostic purposes. The use of the centrifuge is essential when time is limited.

## SHEARER'S METHOD (BENBROOK MODIFICATION)

To prepare the fecal sample, one or two grams (one tablespoon full) of feces should be removed from the container by a clean spatula or tongue depressor and placed in a clean beaker. Water is added and the material stirred until it appears as a uniform emulsion. This material should be poured through a wire-gauze tea strainer, or cheesecloth which is then discarded, into another beaker. A centrifuge tube is filled about half full of the fecal liquid and an equal amount of the flotation solution is added The tube should be corked in some manner and gently shaken until the solution is thoroughly mixed with the feces. The material is then centrifuged for about 5 minutes at a speed of 1000 to 1500 revolutions per minute. The surface layer of the fluid in the tube is removed to a slide by a beaded glass rod or a wire loop.

The sugar solution is prepared as follows:

| | |
|---|---|
| Granulated table sucrose | 1,280 grams |
| Water | 1,000 cc |
| Phenol solution (preservative) 5% | 50 cc |

A white corn sirup solution may be used instead of the above mixture

| | |
|---|---|
| Sirup | 800 cc |
| Hot Water | 200 cc |
| Melted phenol (1%) | 10 cc |

## KOFOID AND BARBER BRINE-FLOTATION METHOD

1. A fecal sample is thoroughly mixed with about twice its volume of saturated solution of common table salt in a paraffined paste-board cup or a small beaker.

2. A lightly compressed circular disk of No. 1 or No. 0 steel wool about one-eighth to one-quarter inch thick is placed in the cup and pushed to the bottom.

3. The fluid is allowed to stand for about one hour, during which time the protozoa rise to the surface.

4. The surface film is removed with a wire loop about one-half inch in diameter or a glass rod flattened on one end, placed on a slide and examined without a cover glass.

### FAUST'S ZINC SULPHATE METHOD

1. A suspension is made by using one part of specimen with about 10 parts of lukewarm tap water.

2. About 10 cc of the suspension is strained through a layer of wet cheesecloth.

3. The material is centrifuged 45 to 60 seconds at high speed. The supernatant fluid is poured off and 2 or 3 cc of water is added. After breaking up the sediment more water is added to fill the tube. This is repeated (3 or 4 times) until the supernatant fluid is clear.

4. The supernatant fluid is poured off and 3 to 4 cc of zinc sulphate solution of specific gravity of 1.180 (33 per cent solution) is added. The packed sediment is broken up and more zinc sulphate solution is added to within about one-half inch of the rim.

5. The material is centrifuged for 45 to 60 seconds at high speed.

6. With a bacteriological loop or glass rod flattened at one end the material floating in the surface film is removed to a clean slide. One drop of iodine is added and a cover slip placed over the material.

### MEDIA FOR CULTIVATION OF INTESTINAL FLAGELLATES

### BOEK AND DRBOHLAV'S EGG SERUM MEDIUM

Four eggs are broken into a sterile flask containing glass beads. Fifty cc of Locke's solution are then added and the ingredients well shaken. Test tubes are filled with about 2 cc each to produce slants about 1½ inches long upon coagulating by heat. The tubes are overlaid with about 4 to 10 cc of 0.7 per cent saline or Locke's solution, plugged and autoclaved at 15 pounds pressure for 20 minutes. Sterile blood serum (about 1 cc for each tube) may be added aseptically later.

Modifications of this medium are numerous. Ringer's or Locke's solution may be substituted for 0.7 per cent or 0.85 per cent saline. This modification has been reported to grow Trichomonas gallinae and Pentatrichomonas gallinarum.

Allen (1941) added a small amount of liver extract and defibrinated turkey blood to the Locke solution overlay, for P. gallinarum.

### TYZZER'S MEDIUM FOR HISTOMONAS MELEAGRIDIS AND PENTATRICHOMONAS GALLINARUM

| | |
|---|---|
| Agar | 14.0 grams |
| Sodium chloride | 6.0 grams |
| Potassium acid phosphate | 2.7 grams |
| Tap water | 900 cc |

After the above are well mixed by heating in an autoclaved and cooled to 55°C, the beaten white of one egg diluted with a small amount of water is added, and the mixture cooked in the Arnold sterilizer for 45 minutes. It should be well shaken every 15 minutes during this process. After the egg white has settled, the mixture is filtered

through cotton, pH adjusted to 7.2, passed through filter paper, tubed, autoclaved and slanted. The slants are overlaid with physiological saline, horse serum and rice starch. Locke's solution with 5 to 10 per cent horse serum may be substituted. Transfers should be made every 48 hours, and cultures incubated at 40°C. Kay (1946) cultivated P. gallinarum in this medium.

### DEVOLT'S MEDIUM FOR HISTOMONAS MELEAGRIDIS

| | |
|---|---|
| NaCl | 9.0 grams |
| CaCl | 0.2 grams |
| KCl | 0.4 grams |
| $NaHCO_3$ | 0.2 grams |
| Glucose | 2.0 grams |
| Fresh clear turkey serum | 20.0 cc |
| Aqueous N/20 NaOH | 20.0 cc |
| Distilled water | 1000.0 cc |

The medium is prepared by adding together the above ingredients. Chicken serum is equally suitable. Precipitation of the serum proteins by heat in the autoclave is prevented by the NaOH. This adjusts the pH to about 9.0. Ten cc of the medium are placed in culture tubes and plugged with cotton The medium is autoclaved for about 20 minutes.

Rice starch is sterilized in small tubes by dry heat at 160°C. for two hours. One or two milligrams of the starch is added to the medium. Cultures of Histomonas have been propagated by DeVolt for over a year. Transfers are made twice per week, and incubated at 40°C.

### HOGUE'S MEDIUM

The whites of 6 hen's eggs are mixed with 600 cc of 0.7 per cent saline. Glass beads are added and the ingredients thoroughly shaken. The mixture is heated over a water bath for 20 to 30 minutes being constantly agitated and then passed through coarse cheesecloth and filtered. Five cc of the filtrate are placed in tubes, plugged and autoclaved.

The above media can be used by inoculation with feces and incubated at room temperature for 2 or 3 days or 37°C for 24 to 48 hours and then examined for protozoa.

### BARRET AND YARBROUGH'S MEDIUM FOR BALANTIDIUM COLI

| | |
|---|---|
| Inactivated serum | 1 part |
| Sodium chloride solution (0.5%) | 16 parts |

The material is sterilized by filtration and distributed in 8 cc portions in test tubes.

Tubes should be inoculated with 0.1 cc of feces and incubated at 37° C for 24 hours. Examinations should be made in 24 hours and transfers every 24 to 48 hours. Ringer's solution can be substituted for the 0.5 per cent saline.

### SPORULATION OF OOCYSTS

One or two per cent potassium dichromate is mixed with a small amount of feces containing oocysts and placed in a shallow dish. The potassium dichromate prevents putrefaction. This material should be examined microscopically within a few days.

## PRESERVING PROTOZOAN CYSTS IN FECES

Feces are diluted with distilled or tap water to a watery consistency and mixed simultaneously with an equal amount of hot (80° C) 10 per cent formalin into a separate container. After standing a few hours, the supernatant is removed and replaced with 5 per cent formalin. It is then bottled, packed and shipped to a central laboratory. Formalinized protozoan cysts often do not float well in zinc sulfate solution but stain with iodine. Cysts remain well preserved for over a year.

## KNOWLE'S AND COLE'S METHOD OF COUNTING PROTOZOAN CYSTS

One gram of feces is emulsified in 10 cc of iodine solution. The emulsion is transferred with a capillary pipette to a hemocytometer and the cysts counted. At least three or four counts should be made and averaged. The count represents the number of cysts in 0.1 cmm of feces.

## CULTIVATION OF PROTOZOA FROM THE REPRODUCTIVE TRACT

### MORGAN'S MODIFIED SCHNEIDER'S MEDIUM

The medium is recommended for maintaining bacteria-free cultures of Tritrichomonas foetus. It is prepared in two parts. The organisms are incubated at 37° C for 3 to 5 days and then at 30° C indefinitely.

A. Preparation of egg slants.

6 whole eggs
60 cc defibrinated bovine blood (bovine serum may be substituted)
50 cc of "Sodium Citrate Solution" prepared as follows:

| | |
|---|---|
| Sodium Chloride | 5.0 grams |
| Magnesium Sulphate | 0.2 grams |
| Ammonium Acid Phosphate ($NH_4H_2PO_4$) | 1.0 grams |
| Potassium Phosphate Dibasic ($K_2HOP_4$) | 1.0 grams |
| Sodium Citrate | 2.0 grams |
| Glucose | 10.0 grams |
| Distilled water | 1000 cc |

1. Beat up thoroughly with glass beads in a large flask.
2. Filter through cheesecloth or gauze.
3. Distribute in test tubes in 4 to 5 cc quantities.
4. Slant test tubes in previously heated autoclave, turn off completely. Remove when cool.

B. Preparation of liquid portion of the medium for overlaying the egg slants:

1. Sodium Citrate Solution, 1000 cc made from above formula.
2. Bovine serum (sterility not required) 5.0 to 7.5%. The serum is always centrifuged two to three times to remove all cellular material.
3. Adjust pH to 7.2 to 7.4.
4. Add 2 cc of a 1.6% alcoholic solution of bromcresol purple (for observing pH change, indicating flagellate growth in medium).
5. Filter two to three times through double layer filter paper until the solution is clear.
6. Sterilize in autoclave 30 minutes, 15 pounds pressure.

## EXAMINATION OF BLOOD

Venous blood is usually collected for blood studies. The jugular vein of the horse, cow, sheep and goat is used. The radial or saphenous veins may be utilized for removing blood from the dog and cat but the jugular can be used particularly in the dog.

Rabbits, guinea pigs, mink and other small animals may be bled from the heart. Pigs are bled from the anterior vena cava.

Sodium oxalate or sodium citrate can be used as an anticoagulant. From 2 to 4 milligrams per cc of blood is the usual amount. In checking the physical being of an animal suspected of being parasitized a red blood cell count, white blood cell count, hemoglobin, sedimentation rate and differential counts should be run.

In preparation of blood smears an anticoagulant should not be used. A small drop of blood is placed on a grease-free 3 x 1 inch glass slide. Another slide is placed in the center of the drop and is spread thin. A thin uniform smear is best. It is then stained with Wright's or Giemsa's.

There are several important blood diseases of domestic animals in which the veterinary protozoologist should become familiar. Bovine anaplasmosis (Anaplasma marginale), bird malaria, canine piroplasmosis (Babesia canis) and leukocytozoon infections in turkeys are some of the more prevalent forms.

Bone marrow, spleen, liver and lymph nodes may be smeared on slides and stained in a similar manner.

### WRIGHT'S METHOD

1. A blood smear is covered with 10 drops of Wright's stain and allowed to remain for one minute.
2. An equal amount of distilled water is added to the stain and allowed to stand three minutes (the stain should now have a metallic sheen).
3. The sheen is washed off with tap water and the slide allowed to dry.
4. The slide is mounted in balsam, gum damar or clarite if a cover glass is used.

### WRIGHT'S METHOD WITH BUFFER

1. Buffer solution: 6.63 grams of acid potassium phosphate and 3.2 grams of dibasic sodium phosphate are dissolved in 1000 cc of distilled water.
2. Twenty-five to 30 drops of Wright's stain are added to the blood film and allowed to stand one minute. An equal number of drops of buffer solution is then added and allowed to stand four minutes. The slide is washed with distilled water and dried.

### FEEMSTER METHOD

1. Solution No. 1: Enough dry stain is placed in a clean dry bottle to saturate absolute methyl alcohol (not more than 0.3 gram will dissolve in 100 cc). Let stand one or two days with occasional shaking. After filtering add one-fifth of its volume of methyl alcohol.
2. Solution No. 2: In another bottle put 90 cc of 95 per cent ethyl alcohol and 10 cc of distilled water. Add to this 0.3 gram of dry stain. After mixing let stand for two days with occasional shaking. The material should be filtered before using.
3. The blood smear is covered with solution No. 1. The excess is drained off at once and allowed to stand until the slide turns red.
4. Wash blood with distilled water and let stand for one or two minutes.

5. Rinse with solution No. 2 until most of the red precipitate disappears.

6. After washing with distilled water, dry and examine.

### GIEMSA'S METHOD

1. Smears are fixed in methyl alcohol for two to five minutes.

2. The slide is submerged in diluted Giemsa's stain for twenty-five to thirty minutes.

| | |
|---|---|
| Giemsa stain | 1 cc |
| Water (distilled) | 10 cc |

The water should be neutral or very slightly alkaline. It is advisable to test it before use. The stain should be used immediately after diluting.

3. After washing with distilled water the slide is dried thoroughly and examined under the microscope.

### GIEMSA'S STAIN FOR BLOOD PROTOZOA

#### STOCK SOLUTION

| | |
|---|---|
| Powdered Giemsa stain | 1 gram |
| Glycerin (C.P.) | 66 cc |
| Methyl alcohol, absolute, acetone free | 66 cc |

Grind powdered stain and glycerin together. When well mixed, dissolve stain in glycerin in a water bath at 55° to 60° C. When cool add methyl alcohol. Allow to stand for 2 or 3 weeks and filter The stain should be stored in small amber colored bottles.

### WRIGHT'S STAIN FOR BLOOD PROTOZOA

#### STOCK SOLUTION

| | |
|---|---|
| Powdered Wright's stain | 0.3 gram |
| Glycerin (C.P.) | 3 cc |
| Methyl alcohol, absolute, acetone free | 97 cc |

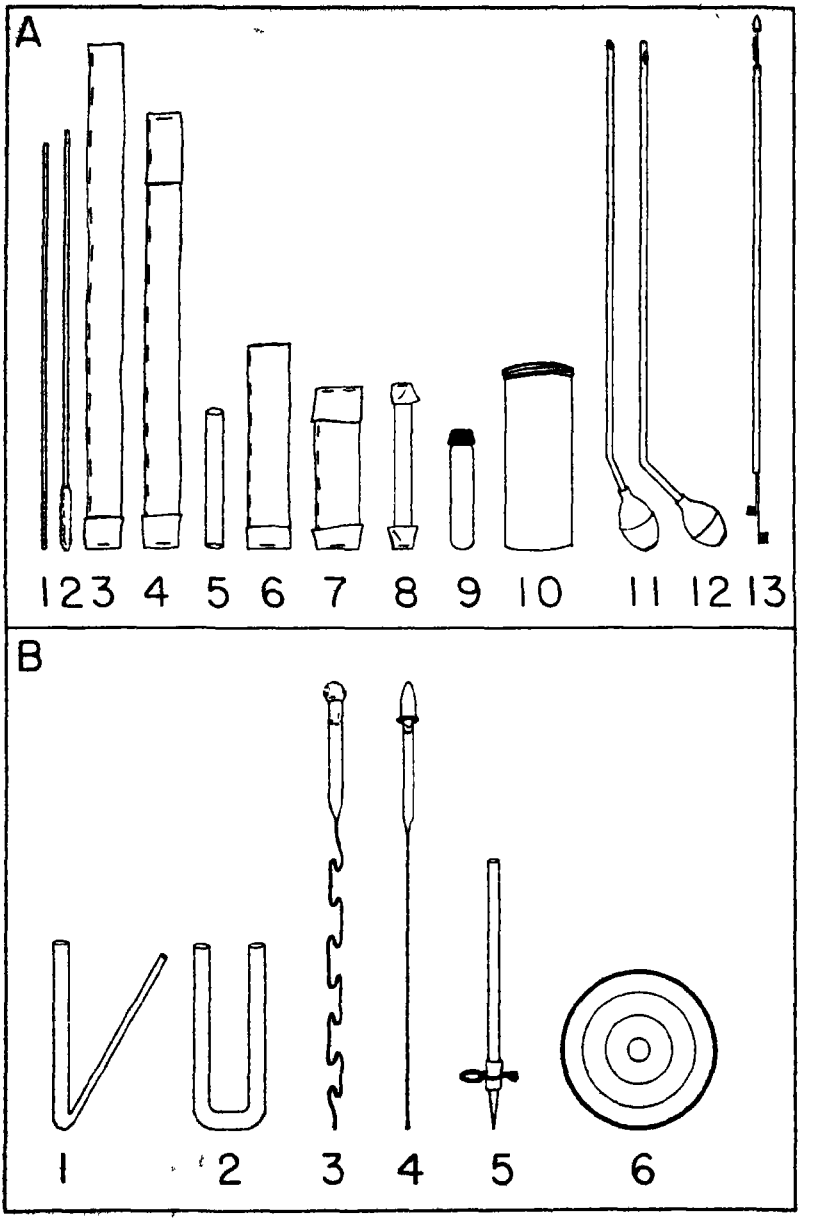
Grind powdered stain and glycerin together. When mixed well add methyl alcohol and stir thoroughly. Place mixture in tightly stoppered amber bottle for 2 or 3 weeks. Filter and stain is ready for use. The stain improves with age.

### COLLECTION OF SAMPLES FOR BOVINE TRICHOMONIASIS

#### COLLECTION OF THE SAMPLE FROM THE FEMALE

Andrews and Miller (1930) devised a pipette for obtaining samples from the vagina. Samples may be collected by cotton swabs moistened with 0.7 per cent saline either on 12 inch applicator sticks or held in a long hemostat. The writer prefers to collect samples with the following apparatus which has been developed at the Wisconsin Experiment Station. A straight steel rod about $\frac{1}{4}$ inch in diameter and 18 inches long is grooved at one end and a cotton pledget attached. The cotton may be held in place either by thread or a rubber band. This swab is wrapped in a heavy paper envelope made for the purpose and autoclaved. The second piece of apparatus is a 6 inch long 3/4 inch diameter pyrex glass tubing, fire polished on both ends. This small glass speculum is also wrapped in a heavy paper envelope and sterilized by autoclaving.

PLATE XXII




A (1-13) Some equipment used for the diagnosis of bovine trichomoniasis.
B (1-6) Some materials used for the isolation of Tritrichomonas foetus free from bacteria.

On use, the glass speculum is placed in the vulva, and the swab moistened in the culture medium, introduced through the speculum up to the cervical region. A circular motion is used to swab the vaginal walls. After withdrawing the swab, it is placed in the culture tube (0.7 per cent saline) and thoroughly shaken. The swab and glass speculum is returned to the paper envelope, taken back to the laboratory, cleaned and prepared for another time. The purpose of the glass speculum is to eliminate any fecal or other contamination that may be present and accidentally be introduced either in the vaginal tract or the culture medium.

Although the infection may be present in the cow, an examination of the vaginal washings, which is the easiest to obtain, may be negative. Exudate obtained directly from the uterus will, in all probability, be more likely to contain trichomonads. In the case of endometritis or pyometra, samples of uterine material may be collected aseptically with a sterile uterine catheter.

A recent report by Bartlett stated the highest percentage of positive samples are taken from the vagina between the 14th to 19th day after the initial exposure.

During abortions and pyometras, trichomonads are at their highest concentration. The various methods as described above may appear relatively simple, but attempts to demonstrate T. foetus in genital exudates are quite frequently unsuccessful. Although trichomonads are more likely to be found from vaginal swabs following a trichomonad abortion, there are cases on record where trichomonads could not be found from 24 to 48 hours after abortion. In some of these cases the fetuses were heavily infected.

## COLLECTION OF THE SAMPLES FROM THE FETUS

If the abortion is complete, that is, with fetal membranes intact, trichomonads may be demonstrated in the fetal fluids. Morgan (1943) obtained pure cultures of T. foetus from aborted fetuses in which the fetal membranes were intact. If the fetal membranes are not available, the swabbing of mucus from around the base of the tongue or the roof of the mouth is recommended. Many investigators have reported the finding of T. foetus in the abomasum of aborted fetuses

## COLLECTION OF THE SAMPLE FROM THE MALE

A pipette or cotton swab is inserted into the prepuce and a sample collected. The bull must be properly restrained. The animal should be secured in a stall and properly restrained by an assistant using a sideline to reduce any exertion to a minimum. The glass speculum is inserted and the swab introduced and moved in a circular motion. The posterior part of the glans penis, and the galea glandis or anterior portion of the penis should be especially swabbed as the two areas are the preferential site of T. foetus in the bull. The writers feel that the sampling should be done without epidural anesthesia.

Mating a suspected bull to several virgin heifers is an aid in making a positive diagnosis. Examinations of vaginal discharges from the mated animals are made between the 14th to 19th day. Negative results do not imply that the bull is free from infection. In many chronically infected bulls attempts to demonstrate trichomonads may result in failure. According to Dikmans (1938), this is probably due to the scarcity of the organisms in the prepuce or that they may have located in some inaccessible portion of the genital tract.

In order to make a complete diagnosis of the bull, two other samples could be taken. With the method described by Andrews and Miller, seminal fluid and semen are collected by rectal massage. The fluids are examined for trichomonads under the microscope. For immediate examination, the mixing of a few drops of 1:500 acetic acid in the semen will stop the motility of the spermatozoa while the trichomonads continue their motility.

### DIAGNOSTIC MEDIA

Many diagnostic media have been evolved for the purpose of maintaining T. foetus in large enough numbers for a positive diagnosis. All of the present methods of diagnosis fall short of this criterion. A 50 to 60 per cent ability to diagnose bovine trichomoniasis with present methods would be a fair estimate. Better methods need to be worked out.

The writers employ an all liquid medium (0.7 per cent saline or Ringer's solution) for diagnostic procedures. The solution is distributed in screw-topped high-pressure inulin tubes and autoclaved at 15 pounds pressure for 30 minutes. Cotton plugged tubes are more apt to lose their contents when on a road trip. This type of tube is also useful for mailing suspected material to a central laboratory for examination. Culture material should be examined as soon as possible, preferably 2 or 3 hours after collection. However, if samples are collected with a reasonable amount of aseptic precaution, the organisms may remain viable for several days.

If trichomonads are present in very large numbers they may be found and identified with ease. On the other hand, if they are present in very small numbers they may be found after a prolonged search or not at all. Occasionally, centrifuging of the sample is done, the supernatant drawn off and the sediment examined. A negative finding does not necessarily mean that an animal is not infected, it means more samples and more microscopical examinations. However, the finding of one living motile trichomonad is all that is required for a positive diagnosis. It is unwise to make a diagnosis on the basis of finding dead trichomonads unless the individual is absolutely certain of his identification.

### SALINE SOLUTION FOR TRICHOMONADS

| | |
|---|---|
| Sodium chloride | 7.0 grams |
| Distilled water | 1000 cc |

### PHYSIOLOGICAL SALINE

| | |
|---|---|
| Sodium chloride | 8.5 grams |
| Distilled water | 1000 cc |

### RINGER'S SOLUTION

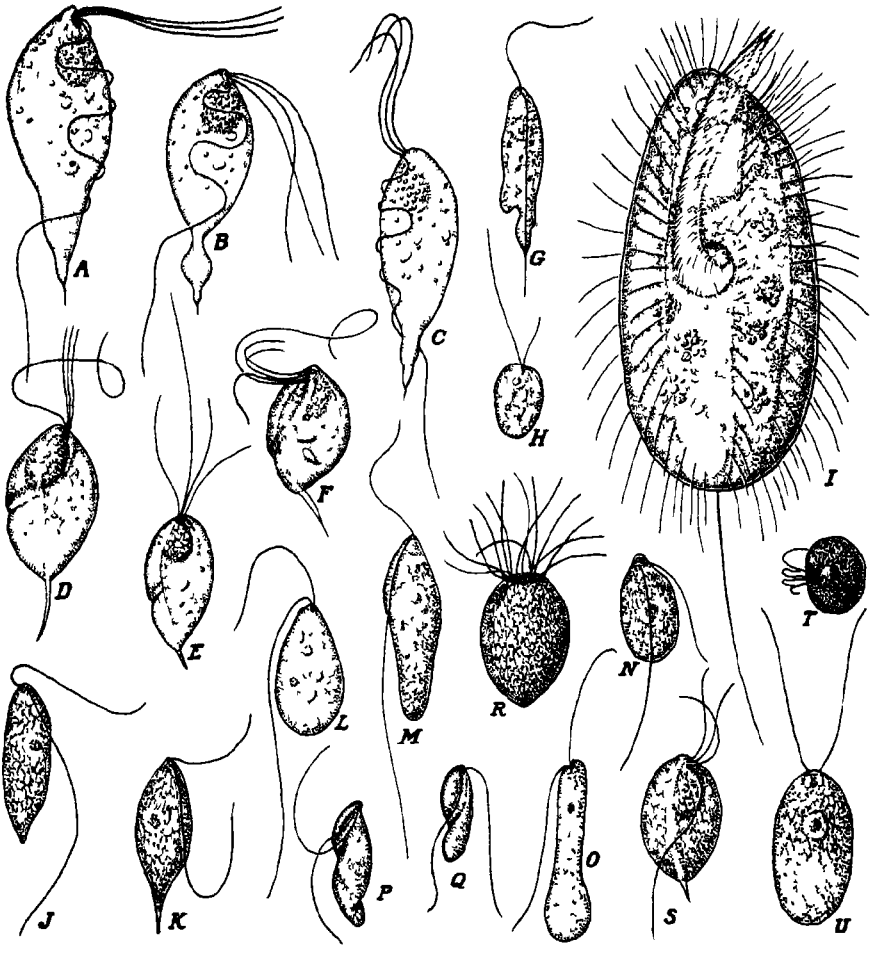
| | |
|---|---|
| Sodium chloride | 4.8 grams |
| Calcium chloride | 0.12 grams |
| Potassium chloride | 0.12 grams |
| Sodium bicarbonate | 0.12 grams |
| Distilled water | 600 cc |

### SEROLOGICAL METHODS OF DIAGNOSIS

Agglutination, precipitation and complement-fixation tests are rarely used for serological diagnosis of protozoan diseases. The use of these tests are still in the experimental and research stages.

Dourine, caused by a trypanosome, is the only protozoan suitable for the complement-fixation test. A serological test (complement-fixation) may be run as a confirmation check for Endamoeba histolytica.

In Northern Ireland the agglutination and cutaneous tests are utilized for aid in the diagnosis of bovine trichomoniasis. These tests have not been too successful in the United States. A nonspecific precipitin test is sometimes used for aid in the diagnosis of anaplasmosis.




SOME PROTOZOA ENCOUNTERED IN EXAMINATION OF MATERIAL SENT IN FOR DIAGNOSIS OF TRICHOMONIASIS

Fig. A-C. - Tritrichomonas foetus
Fig. D-F. - Monocercomonas sp.
Fig. G. - - Cercomonas crassicaude
Fig. H. - - Monas obliqua
Fig. I. - - Lembus pusillus
Fig. J-O. - Bodo caudatus
Fig. P-Q. - Spiromonas angusta
Fig. R. - - Callimastix frontalis
Fig. S. - - Monocercomonas ruminantium
Fig. T. - - Same, end view
Fig. U. - - Polytoma uvella

## MISCELLANEOUS MATERIAL

### CLEANING SOLUTION FOR GLASSWARE

| | |
|---|---|
| Potassium dichromate | 60 grams |
| Tap water | 300 cc |

The above mixture should be dissolved by heat and then cooled.

Add slowly with constant stirring:

| | |
|---|---|
| Sulphuric acid | 460 cc |

### LOCKE'S SOLUTION

| | |
|---|---|
| Sodium chloride | 9.00 grams |
| Calcium chloride | 0.24 grams |
| Potassium chloride | 0.42 grams |
| Sodium bicarbonate | 0.20 grams |
| Distilled water | 1000 cc |

## APPENDIX

## APPENDIX

### NOMENCLATURE

The scientific names of animals used today, dates back to the time of Linnaeus who first suggested a binomial system for species recognition. The starting point has been set arbitrarily at the year 1758. This was the year Linnaeus' 10th Edition of SYSTEMA NATURAE was published.

The International Rules of Zoological Nomenclature is an attempt by zoologists throughout the world to have a uniform standard in the use of scientific names for animals. These rules are based on the principles of binomial nomenclature of Linnaeus. It involves the concepts of genera and species, how names are originated, methods of determining the priority of names, Code of Ethics and procedures for suspension of the rules in cases when situations may cause confusion rather than clarification.

Each animal has one or more common names and a scientific name. This name is binomial. For example, the protozoan parasite of chickens, Eimeria hagani, belongs to the genus Eimeria and to the species hagani. The name of the person who first named and described the species is placed after the Latinized words representing the genus and species, Eimeria hagani Levine, 1938. The date of the description is usually given.

When a species is placed in a genus which is different from the original description the author's name is enclosed in parentheses. For example, Histomonas meleagridis (Smith, 1895). This parasite was originally named Amoeba meleagridis by Smith, but later Tyzzer, 1920 found the organism to be a flagellate and reclassified it. In the scientific name the species word always beings with a small letter, even if it is derived from a proper name.

The Law of Priority clearly states that the earliest name proposed to a genus or species is the only valid one. Sometimes this rule is suspended in certain cases to prevent confusion. No two different species in the same genus can possess the same scientific name nor can two genera have the same name.

When a new species is described a type specimen is designated and usually sent to a central museum where it is taken care of properly and is available for study by other qualified scientists at any time.

The names for animal groups are as follows phyla, classes, orders, families, genera and species. The terms which designate phyla usually end in -a, classes in -ea, subclasses in -ia, orders in -ida, suborders in -ina, families in -idae and subfamilies in -inae.

The Code of Ethics acts as a personal clearing house concerning scientific names. The important resolution is as follows: "That when it is noticed by any zoologist that the generic or specific name published by any living author as new is in reality a homonym and, therefore, unavailable under Articles 34 and 36 of the Rules of Nomenclature, the proper action from a standpoint of professional etiquette, is for said person to notify said author of the facts of the case and to give said author ample opportunity to propose a substitute name."

## PARASITE-HOST LIST

### HORSE

(Equus caballus)

| Name of Parasite | Type of Parasite | Habitat |
|---|---|---|
| Oikomonas equi | Flagellate | Cecum |
| Trypanosoma equiperdum | Flagellate | Blood, reproductive organs |
| Callimastix equi | Flagellate | Colon |
| Tritrichomonas equi | Flagellate | Cecum, colon |
| Endamoeba gedoelsti | Amoeba | Cecum, colon |
| Didesmis ovalis | Ciliate | Cecum, colon |
| D. quadrata | Ciliate | Cecum, colon |
| D. spiralis | Ciliate | Cecum, colon |
| Blepharoprosthium pireum | Ciliate | Cecum, colon |
| Blepharosphaera intestinalis | Ciliate | Cecum, colon |
| B. ellipsoidalis | Ciliate | Cecum, colon |
| Blepharoconus cervicalis | Ciliate | Cecum, colon |
| B. benbrooki | Ciliate | Cecum, colon |
| Bundleia postciliata | Ciliate | Cecum, colon |
| Alloiozona trizona | Ciliate | Cecum, colon |
| Polymorpha ampulla | Ciliate | Cecum, colon |
| Paraisotricha colpoidea | Ciliate | Cecum, colon |
| P. beckeri | Ciliate | Cecum, colon |
| P. minuta | Ciliate | Cecum, colon |
| Blepharocorys uncinata | Ciliate | Cecum, colon |
| B. balbata | Ciliate | Cecum, colon |
| B. jubata | Ciliate | Cecum, colon |
| B. corvigula | Ciliate | Cecum, colon |
| B. angusta | Ciliate | Cecum, colon |
| B. cardio-nucleata | Ciliate | Cecum, colon |
| Charon equi | Ciliate | Cecum, colon |
| Cycloposthium bipalmatum | Ciliate | Cecum, colon |
| C. dentiferum | Ciliate | Cecum, colon |
| C. edentatum | Ciliate | Cecum, colon |
| C. scutigerum | Ciliate | Cecum, colon |
| C. affinal | Ciliate | Cecum, colon |
| C. corrugatum | Ciliate | Cecum, colon |
| Spirodinium equi | Ciliate | Cecum, colon |
| Triadinium caudatum | Ciliate | Cecum, colon |
| T. galea | Ciliate | Cecum, colon |
| T. minimum | Ciliate | Cecum, colon |
| Tetratoxum unifasciculatum | Ciliate | Cecum, colon |
| T. excavatum | Ciliate | Cecum, colon |
| T. parvum | Ciliate | Cecum, colon |
| Tripalmaria dogieli | Ciliate | Cecum, colon |
| Cochliatoxum periachtum | Ciliate | Cecum, colon |
| Ditoxum funinucleum | Ciliate | Cecum, colon |
| Allantosoma intestinalis | Suctoria | Cecum, colon |
| A. dicorniger | Suctoria | Cecum, colon |
| A. brevicorniger | Suctoria | Cecum, colon |
| Sarcocystis bertrami | Fungus? | Striated muscle |

## CATTLE

(*Bos taurus*)

| Name of Parasite | Type of Parasite | Habitat |
|---|---|---|
| *Trypanosoma theileri* | Flagellate | Blood |
| *Monas communis* | Flagellate | Rumen |
| *Callimastix frontalis* | Flagellate | Rumen |
| *Giardia bovis* | Flagellate | Intestine, feces |
| *Monocercomonas ruminantium* | Flagellate | Rumen, prepuce |
| *Tritrichomonas ruminantium* | Flagellate | Alimentary tract |
| *Tritrichomonas foetus* | Flagellate | Reproductive tract |
| *Vahlkampfia lobospinosa* | Amoeba | Rumen |
| *Endamoeba bovis* | Amoeba | Rumen |
| *Eimeria alabamensis* | Coccidia | Intestine |
| *E. auburnensis* | Coccidia | Intestine |
| *E. bovis* | Coccidia | Intestine |
| *E. brasiliensis* | Coccidia | Intestine |
| *E. ildefonsoi* | Coccidia | Intestine |
| *E. bukidnonensis* | Coccidia | Intestine |
| *E. canadensis* | Coccidia | Intestine |
| *E. cylindrica* | Coccidia | Intestine |
| *E. ellipsoidalis* | Coccidia | Intestine |
| *E. subspherica* | Coccidia | Intestine |
| *E. wyomingensis* | Coccidia | Intestine |
| *E. zurnii* | Coccidia | Intestine |
| *Babesia bigemina* | Sporozoa | Blood |
| *Anaplasma marginale* | Uncertain | Blood |
| *Bartonella bovis* | Uncertain | Blood |
| *Eperythrozoon wenyoni* | Uncertain | Blood |
| *Sarcocystis* sp. | Fungus? | Striated muscle |
| *Isotricha prostomata* | Ciliate | Rumen |
| *I. intestinalis* | Ciliate | Rumen |
| *Dasytricha ruminantium* | Ciliate | Rumen |
| *Buteschlia parva* | Ciliate | Rumen |
| *B. neglecta* | Ciliate | Rumen |
| *B. lanceolata* | Ciliate | Rumen |
| *Entodinium bursa* | Ciliate | Rumen |
| *E. minimum* | Ciliate | Rumen |
| *E. caudatum* | Ciliate | Rumen |
| *E. bicarinatum* | Ciliate | Rumen |
| *E. furca* | Ciliate | Rumen |
| *E. rostratum* | Ciliate | Rumen |
| *E. dentatum* | Ciliate | Rumen |
| *Diplodinium magii* | Ciliate | Rumen |
| *D. bursa* | Ciliate | Rumen |
| *D. medium* | Ciliate | Rumen |
| *D. ecaudatum* var. *ecaudatum* | Ciliate | Rumen |
| *D. ecaudatum* var. *bicaudatum* | Ciliate | Rumen |
| *D. ecaudatum* var. *tricaudatum* | Ciliate | Rumen |
| *D. ecaudatum* var. *quadri-caudatum* | Ciliate | Rumen |
| *D. ecaudatum* var. *catanei* | Ciliate | Rumen |
| *D. eberleini* | Ciliate | Rumen |
| *D. dentatum* | Ciliate | Rumen |
| *D. denticulatum* | Ciliate | Rumen |
| *D. minimum* | Ciliate | Rumen |
| *D. clevelandi* | Ciliate | Rumen |
| *D. helseri* | Ciliate | Rumen |
| *D. neglectum* | Ciliate | Rumen |
| *D. multivesiculatum* | Ciliate | Rumen |

| Name of Parasite | Type of Parasite | Habitat |
|---|---|---|
| Ophryoscolex inermis | Ciliate | Rumen |
| O. purkynjei | Ciliate | Rumen |
| O. caudatus | Ciliate | Rumen |
| Elytroplastron hegneri | Ciliate | Rumen |
| Buxtonella sulcata | Ciliate | Rumen |

## SHEEP AND GOAT

(Ovis aries and Capra hircus)

| | | |
|---|---|---|
| Retortamonas ovis | Flagellate | Feces |
| Eimeria arloingi | Coccidia | Intestine |
| E. ah-sa-ta | Coccidia | Intestine |
| E. crandallis | Coccidia | Intestine |
| E. faurei | Coccidia | Intestine |
| E. granulosa | Coccidia | Intestine |
| E. ninakohl-yakimovi | Coccidia | Intestine |
| E. pallida | Coccidia | Intestine |
| E. parva | Coccidia | Intestine |
| Eimeria intricata | Coccidia | Intestine |
| Balantidium sp. | Ciliate | Intestine |
| Isotricha prostomata | Ciliate | Rumen |
| I. intestinalis | Ciliate | Rumen |
| Dasytricha ruminantium | Ciliate | Rumen |
| Butschlia parva | Ciliate | Rumen |
| B. neglecta | Ciliate | Rumen |
| B. lanceolata | Ciliate | Rumen |
| Entodinium bursa | Ciliate | Rumen |
| E. minimum | Ciliate | Rumen |
| E. caudatum | Ciliate | Rumen |
| E. bicarinatum | Ciliate | Rumen |
| E. furca | Ciliate | Rumen |
| E. rostratum | Ciliate | Rumen |
| E. dentatum | Ciliate | Rumen |
| Diplodinium magii | Ciliate | Rumen |
| D. bursa | Ciliate | Rumen |
| D. medium | Ciliate | Rumen |
| D. ecaudatum var. ecaudatum | Ciliate | Rumen |
| D. ecaudatum var. bicaudatum | Ciliate | Rumen |
| D. ecaudatum var. tricaudatum | Ciliate | Rumen |
| D. ecaudatum var. quadri-caudatum | Ciliate | Rumen |
| D. ecaudatum var. catanei | Ciliate | Rumen |
| D. eberleini | Ciliate | Rumen |
| D. dentatum | Ciliate | Rumen |
| D. denticulatum | Ciliate | Rumen |
| D. minimum | Ciliate | Rumen |
| D. clevelandi | Ciliate | Rumen |
| D. belseri | Ciliate | Rumen |
| D. neglectum | Ciliate | Rumen |
| D. multivesiculatum | Ciliate | Rumen |
| Ophryoscolex inermis | Ciliate | Rumen |
| O. purkynjei | Ciliate | Rumen |
| O. caudatus | Ciliate | Rumen |
| Elytroplastron hegneri | Ciliate | Rumen |
| Globidium gilruthi | Uncertain | Walls of abomasum and intestine |
| Toxoplasma sp. | Uncertain | Throughout the body |
| Eperythrozoon sp. | Uncertain | Blood |
| Sarcocystis tenella | Fungus? | Striated muscle |

## SWINE

(*Sus scrofa*)

| Name of Parasite | Type of Parasite | Habitat |
|---|---|---|
| *Tritrichomonas suis* | Flagellate | Intestine |
| *Trichomonas* sp. | Flagellate | Skin |
| *Chilomastix mesnili* | Flagellate | Intestine |
| *Giardia lamblia* | Flagellate | Intestine |
| *Endamoeba histolytica* | Amoeba | Intestine |
| *Endamoeba coli* | Amoeba | Intestine |
| *Endamoeba polecki* | Amoeba | Intestine |
| *Endolimax nana* | Amoeba | Intestine |
| *Iodamoeba butschlii* | Amoeba | Intestine |
| *Eimeria perminuta* | Coccidia | Intestine |
| *E. spinosa* | Coccidia | Intestine |
| *E. scabra* | Coccidia | Intestine |
| *E. debliecki* | Coccidia | Intestine |
| *E. scrofae* | Coccidia | Intestine |
| *Isospora suis* | Coccidia | Intestine |
| *Sarcocystis miescheriana* | Fungus? | Straited muscle |
| *Balantidium coli* | Ciliate | Intestine |

## DOG AND CAT

(*Canis familiaris* and *Felis domestica*)

| | | |
|---|---|---|
| *Giardia canis* | Flagellate | Intestine |
| *Giardia felis* | Flagellate | Intestine |
| *Pentatrichomonas hominis* | Flagellate | Intestine |
| *Trichomonas canistomae* | Flagellate | Mouth |
| *Trichomonas felistomae* | Flagellate | Mouth |
| *Endamoeba histolytica* | Flagellate | Intestine |
| *Babesia canis* | Sporozoa | Blood |
| *Isospora bigemina* | Coccidia | Intestine |
| *I. rivolta* | Coccidia | Intestine |
| *Isospora felis* | Coccidia | Intestine |
| *Eimeria canis* | Coccidia | Intestine |
| *E. felina* | Coccidia | Intestine |
| *Toxoplasma* sp. | Uncertain | Various parts of body |

## FOX

(*Vulpes vulpes* [Fox])
(*Vulpes velox* [Swift fox])

| | | |
|---|---|---|
| *Isospora canivelocis* | Coccidia | Intestine |
| *Isospora bigemina* | Coccidia | Intestine |
| *I. rivolta* | Coccidia | Intestine |
| *I. felis* | Coccidia | Intestine |

## MINK

(Mustela vison)

| Name of Parasite | Type of Parasite | Habitat |
|---|---|---|
| Eimeria mustelae | Coccidia | Intestine |
| E. vison | Coccidia | Intestine |
| Isospora bigemina | Coccidia | Intestine |
| I. laidlawi | Coccidia | Intestine |

## RABBIT

(Lepus cuniculus)

| | | |
|---|---|---|
| Retortamonas cuniculi | Flagellate | Cecum |
| Giardia duodenalis | Flagellate | Intestine |
| Chilomastix cuniculi | Flagellate | Cecum |
| Monocercomonas cuniculi | Flagellate | Cecum |
| Endamoeba cuniculi | Amoeba | Intestine |
| Eimeria stiedae | Coccidia | Liver |
| E. magna | Coccidia | Intestine |
| E. irresidua | Coccidia | Intestine |
| E. media | Coccidia | Intestine |
| E. perforans | Coccidia | Intestine |
| E. neoleporis | Coccidia | Cecum |
| Toxoplasma sp. | Uncertain | Various parts of body |
| Sarcocystis leporum | Fungus? | Skeletal muscle |

## POULTRY

### CHICKEN

(Gallus gallus)

| | | |
|---|---|---|
| Histomonas meleagridis | Flagellate | Ceca, liver |
| Pleuromonas jaculans | Flagellate | Ceca |
| Pentetrichomonas gallinarum | Flagellate | Ceca, liver |
| Trichomonas gallinae | Flagellate | Mouth, crop, liver |
| Tritrichomonas eberthi | Flagellate | Ceca |
| Monocercomonas gallinarum | Flagellate | Ceca |
| Chilomastix gallinarum | Flagellate | Intestine |
| Endamoeba gallinarum | Amoeba | Ceca |
| Endolimax gregariniformis | Amoeba | Ceca |
| Eimeria tenella | Coccidia | Ceca |
| E. necatrix | Coccidia | Small intestine |
| E. brunetti | Coccidia | Small intestine, ceca |
| E. acervulina | Coccidia | Small intestine |
| E. maxima | Coccidia | Small intestine |
| E. hagani | Coccidia | Small intestine |
| E. mitis | Coccidia | Small intestine |
| E. praecox | Coccidia | Small intestine |
| Cryptosporidium sp. | Coccidia | Intestine |
| Tetrahymena gelei | Ciliate | Intestine, eyelids |
| Toxoplasma sp. | Uncertain | Throughout body |
| Sarcocystis rileyi | Fungus? | Striated muscle |

## TURKEY

(Meleagris gallopavo)

| Name of Parasite | Type of Parasite | Habitat |
|---|---|---|
| Histomonas meleagridis | Flagellate | Ceca, liver |
| Pentatrichomonas gallinarum | Flagellate | Ceca, liver |
| Trichomonas gallinae | Flagellate | Mouth, crop, liver |
| Cochlosoma rostratum | Flagellate | Intestine |
| Hexamita meleagridis | Flagellate | Intestine, ceca |
| Endamoeba gallinarum | Amoeba | Ceca |
| Endolimax gregariniformis | Amoeba | Ceca |
| Eimeria meleagridis | Coccidia | Small intestine |
| E. meleagrimitis | Coccidia | Small intestine |
| Haemoproteus sp. | Sporozoa | Blood |
| Leucocytozoon smithi | Sporozoa | Blood |

## GUINEA FOWL

(Pavo cristatus)

| | | |
|---|---|---|
| Histomonas meleagridis | Flagellate | Ceca, liver |
| Pentatrichomonas gallinarum | Flagellate | Ceca, liver |
| Endolimax numidae | Amoeba | Intestine |

## PIGEON

(Columba livia domestica)

| | | |
|---|---|---|
| Histomonas meleagridis | Flagellate | Ceca, liver |
| Trichomonas gallinae | Flagellate | Mouth, crop, liver |
| Hexamita columbae | Flagellate | Intestine |
| Eimeria labbeana | Coccidia | Intestine |
| Haemoproteus columbae | Sporozoa | Blood |

## QUAIL

(Various Species)

| | | |
|---|---|---|
| Histomonas meleagridis | Flagellate | Ceca, liver |
| Hexamita sp. | Flagellate | Intestine |
| Eimeria dispersa | Coccidia | Small intestine |
| Haemoproteus lophortyx | Sporozoa | Blood |

## GROUSE

(Various Species)

| | | |
|---|---|---|
| Histomonas meleagridis | Flagellate | Ceca, liver |
| Eimeria angusta | Coccidia | Ceca |
| Eimeria bonasae | Coccidia | Small intestine |
| Leucocytozoon bonasae | Sporozoa | Blood |

### PHEASANT

(Various Species)

| Name of Parasite | Type of Parasite | Habitat |
|---|---|---|
| Histomonas meleagridis | Flagellate | Ceca, liver |
| Eimeria phasiani | Coccidia | Small intestine, ceca |

### PARTRIDGE

(Various Species)

| | | |
|---|---|---|
| Histomonas meleagridis | Flagellate | Ceca, liver |
| Hexamita sp. | Flagellate | Small intestine |

### DUCKS

(Various Species)

| | | |
|---|---|---|
| Histomonas meleagridis | Flagellate | Ceca, liver |
| Cochlosoma rostratum | Flagellate | Intestine |
| C. anatis | Flagellate | Intestine |
| Endamoeba anatis | Amoeba | Feces |
| Tyzzeria perniciosa | Coccidia | Small intestine |
| Leucocytozoon simondi | Sporozoa | Blood |
| Toxoplasma sp. | Uncertain | Throughout the body |
| Sarcocystis rileyi | Fungus? | Striated muscle |

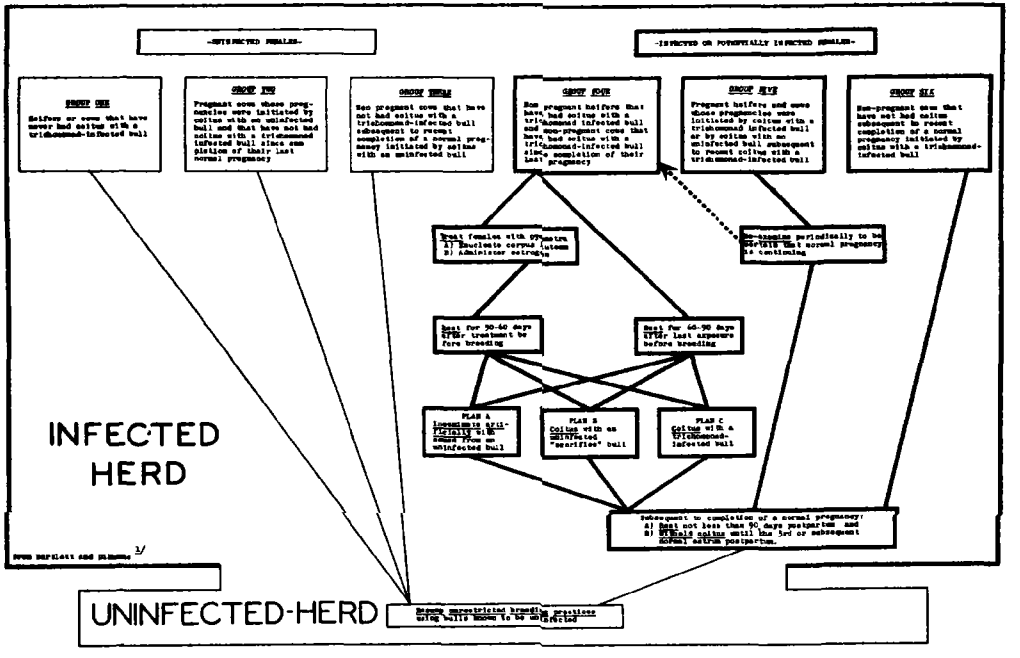
## TREATMENT SCHEDULES

| Name of Parasite of Disease | Treatment |
|---|---|
| 1. Bovine trichomoniasis *Tritrichomonas foetus* | 5 doses intravenously at intervals every other day; each dose 5 grams of sodium iodide per 100 pounds of body weight, dissolved in 500 cc of sterile distilled water. (For the bull.) |
| 2. Bovine coccidiosis *Eimeria* spp. | Sulfaguanidine; 0.1 gram per kilogram of body weight, daily for 3 weeks or 5 gram doses for 8 days. |
| 3. Bovine anaplasmosis *Anaplasma marginale* | Intravenous injection of 1000 cc of 5 per cent dextrose in distilled water to which has been added enough sodium cacodylate to make the dosage of 30 grains per 100 pounds of body weight. |
| | Transfusions of 2000 to 4000 cc of citrated blood. |
| 4. Ovine coccidiosis *Eimeria* spp. | Prevention. 2 per cent sulfur in grain mixture. Treatment. sulfaguanidine, 2 grams daily for one month. |
| 5. Porcine coccidiosis *Eimeria* spp. | Sulfaguanidine, 1 gram per 10 pounds of body weight for 3 days after symptoms. |
| 6. Canine babesiasis *Babesia bigemina* | Trypan blue, 1 to 2 per cent solution, 5 to 6 cc, 1 or 2 treatments. Intravenous. |
| | Trypaflavine, 0.3 cc of a 2 per cent solution per pound of body weight. Intravenous. |
| 7. Histomoniasis, blackhead *Histomonas meleagridis* | Phenothiazine, $\frac{1}{2}$ to 1 gram per bird to remove cecal worms, *Heterakis gallinae*. This will aid as a preventative measure. There is no treatment for the disease. |
| 8. Avian trichomoniasis of the lower digestive tract *Pentatrichomonas gallinarum* | Fever therapy, 3 to 6 treatments in an incubator at 106.5° for 1 to 2 hours. |
| | Enteric coated tablets of gentian violet, 1/16 grain to 1 pound poult, 1/8 for 3 pound, $\frac{1}{4}$ for 5 pound, $\frac{1}{2}$ for 8 pound, 1 for 10 pound and $1\frac{1}{4}$ for 12 pound poult. Give every other day for 3 days. |
| | Or, solution of gentian violet, 1 gram of the drug with 1000 cc of water and given every other day as follows: 5 cc to 1 pound poult, 10 cc for 3 pound, 15 cc for 5 pound, 20 cc for 8 pound and 25 for 10 to 12 pound poults. |
| 9. Avian trichomoniasis of the upper digestive tract *Trichomonas gallinae* | Copper sulfate in drinking water (1.4 to 4.2 grams per gallon of water). |

| Name of Parasite of Disease | Treatment |
|---|---|
| 10. Avian cecal coccidiosis<br>Eimeria tenella<br>Chronic intestinal coccidiosis<br>Eimeria necatrix | Preventative: flowers of sulfur, 5 per cent of the ration for 3 days, then 2 per cent until hot dry weather. |
| | Sulfaguanidine, 1 per cent in ration or ½ per cent intermittently at 3 to 4 day intervals. |
| | Treatment: Sulfamethazine ½ per cent in mash. |
| | Sulfamerazine ½ per cent in mash or 0.2 per cent in drinking water. |
| | Sulfaquinoxaline .0125 to .033 per cent in mash. |
| | Sulfadiazine, saturated solution in drinking water. |
| | Borax, 2 per cent in mash or 0.3 per cent in drinking water. |
| 11. Liver coccidiosis of rabbits<br>Eimeria stiedae | Preventative. Sulfasuxidine, 0.625 grams in feed for 13 days then 0.75 grams daily. |
| 12. Intestinal coccidia of rabbits<br>Eimeria magna | Phthalysulfathiazole 0.2 to 0.25 grains per pound of body weight per day. |

It must be remembered that the treatments listed above are mostly in the experimental and research stages. More improvements are desired as better treatments are in demand. After this book is printed new treatments will appear in the various journals. Progress can only be made by the endless and never tiring efforts of the researcher.

PLATE XXIV




Diagrammatic representation of a hygienic breeding program for elimination of bovine venereal trichomoniasis from an infected herd.

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