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THEILERIOSIS, GONDERIOSES AND CYTAUXZOONOSES: A REVIEW.*

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INTRODUCTION.

An historical review of the literature dealing with Theilerioses in domestic ruminants was given by du Toit (1931), K. F. Meyer (1931) and Yakimoff (1931) at the Eleventh International Veterinary Congress held in London in 1930. These investigators pointed out that with the gradual accumulation of facts concerning these diseases, it becomes increasingly more difficult to distinguish the species occurring in large and small ruminants. They can hardly be differentiated morphologically, and the available evidence indicates that they represent parasites which merge into one another by gradation in virulence, selection of transmitter, production of symptoms and lesions and development of immunity. Comparative investigations in South and East Africa planned and executed on the same broad basis as those of Sergent and collaborators in North Africa and Dschunkowsky and his colleagues in Southern Russia will undoubtedly clarify the problems at issue. They might prove the identity of *Theileria parva* (Theiler, 1904), with *Theileria dispar* Sergent, Donatien, Parrot, Lestoquard, Plantureux and Rougebief, 1924, *Theileria annulata* (Dschunkowsky and Luhs, 1904) and *Theileria mutans* (Theiler, 1906). Everyone of the diseases produced by these protozoa reveals types of infections which cannot be distinguished readily from that due to *Th. parva,* and it appears imperative that the factors for the decline of virulence be analysed carefully. It was shown by du Toit (1931) that there is a very gradual transition from the most virulent species, *Th. parva* to the avirulent species *Th. mutans.* He expressed his doubts whether the four species already described in cattle are " good " species. He pointed out that the difficulty could be overcome either by regarding all the parasites of cattle as varieties or strains of one species, *Th. parva,* or by creating new species for each one of the unnamed types of *Theileria* described in Morocco (Velu, 1921), Tunis (Brumpt, 1923, 1924), Northern Rhodesia (Turnbull, 1926), India (Cooper, 1926) and Egypt (Doyle, 1924). Neither of these alternatives is recommended by du Toit (*loc. cit.*) and,

* Since going to press it has been established that *Rh. appendiculatus* nymphae that fed on a splenectomized Corridor disease recovered calf (*Bos taurus*) can transmit *Gonderia lawrencei* in the ensuing stage.

In view of this *Gonderia bovis* becomes synonymous for *Gonderia lawrencei.* Corridor disease is the same as Rhodesian malignant bovine gonderiosis.

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therefore, the suggestion is made to follow an intermediate and more conservative course and to accept provisionally the four species named *Th. parva, Th. annulata, Th. dispar* and *Th. mutans.* During recent years Dschunkowsky (1948) and Delpy (1949) have come to the conclusion that *Th. dispar* is synonymous for *Th. annulata.* Neitz described *Theileria lawrencei* as the causal agent responsible for Corridor disease in Southern Africa, and *Gonderia bovis* as the parasite responsible for Rhodesian malignant bovine gonderiosis (*vide infra*).

Neitz and Jansen (1956) considered the views expressed by du Toit (1931), K. F. Meyer (1931) and Yakimoff (1931) as regards the nomenclature of the *Theileria* spp. of cattle, and concluded that the difference between the life-cycle of *Th. parva* and that of the remaining *Theileria* spp., including the two species described in sheep and goats, is a sound reason for revising the classification of the Theilerias. The former parasite multiplies only by schizogony within the lymphocytes, while the remaining *Theileria* spp. described in domestic ruminants reproduce by schizogony within the lymphocytes as well as by binary fission in the erythrocytes. *Th. parva* is retained in the family Theileridae. The remaining *Theileria* spp. are transferred to the redefined and reinstated genus *Gonderia.* This genus together with the genus *Cytauxzoon* which multiplies by schizogony in the histiocytes and by binary fission in the red blood cells are included in the new family Gonderidae. The striking differences between the life-cycles of the family Babesidae and those of the families Theileridae and Gonderidae suggest that the former family should be retained in the sub-order Piroplasmidea Wenyon, 1926, and that the latter two families be included in the sub-order Leucosporidea Neitz and Jansen, 1955. This revision of the classification is based on the fact that members of the Piroplasmidea multiply by binary fission within the erythrocytes, while members of the Leucosporidea reproduce by schizogony in the leucocytes.

The revised classification of the family Theileridae is as follows:—

SUB-ORDER LEUCOSPORIDEA NEITZ AND JANSEN, 1955.

In this sub-order are included certain parasites which inhabit either lymphocytes or histiocytes and erythrocytes, but do not form pigment (haemozoin) characteristic of members of the sub-order Haemosporidiidea Danilewsky. They multiply by schizogony and finally invade the erythrocytes, within which they occur as round ovoid, rod-like or irregular forms. These parasites, as far as is known, are transmitted by ticks of the family Ixodidae Murray, 1877. There are two families in this sub-order, the Theileridae and Gonderidae.

1. *Family:* THEILERIDAE DU TOIT, 1918.—Parasites which multiply by schizogony in the lymphocytes, and finally invade the erythrocytes. The forms in the red blood corpuscles do not reproduce, and are possibly gametocytes or gametes. The family is represented by a single genus, *Theileria.*

Genus:

Theileria Bettencourt, Franca and Borges, 1907.
Theileria parva (Theiler, 1904).

Synonyms:

Piroplasma kochi Stephens and Christophers, 1903;
Piroplasma parvum Theiler, 1904;
Theileria kochi (Stephens and Christophers, 1903).

2. *Family*: GONDERIDAE NEITZ AND JANSEN, 1955.—Parasites which multiply by schizogony in either the lymphocytes (*Gonderia* sp.) or histiocytes (*Cytauxzoon* sp.) and finally invade the red blood corpuscles. The forms in the erythrocytes reproduce by division into two or four daughter individuals, the latter giving rise to the characteristic cross forms. The final stage of the parasites is possibly a gametocyte or a gamete. This family is represented by two genera, *Gonderia* and *Cytauxzoon*.

Genus: *Gonderia* (du Toit, 1918).

Members of this genus multiply by schizogony in the lymphocytes and by fission in the erythrocytes.

Gonderia annulata (Dschunkowsky and Luhs, 1904).

Synonyms:

Piroplasma annulatum Dschunkowsky and Luhs, 1904;
Theileria annulata (Dschunkowsky and Luhs, 1904);
Theileria turkestanica Oboldueff and Galouzo, 1928.

Gonderia mutans (Theiler, 1906).

Synonyms:

Piroplasma mutans Theiler, 1906;
Theileria mutans (Theiler, 1906).

Gonderia lawrencei (Neitz, 1955).

Synonym:

Theileria lawrencei Neitz, 1955.

Gonderia bovis Neitz, 1957.

Gonderia hirci (Dschunkowsky and Urodschevich, 1924).

Synonyms:

Theileria ovis du Toit, 1918;
Theileria hirci Dschunkowsky and Urodschevich, 1924.

Gonderia ovis (Rodhain, 1916).

Synonyms:

Theileria ovis Rodhain, 1916;
Babesia sergenti Wenyon, 1926;
Gonderia ovis Lestoquard, 1924;
Theileria recondita Lestoquard, 1929.

Gonderia tachyglossi (Priestly, 1915).

Synonym:

Theileria tachyglossi Priestly, 1915.

Genus: *Cytauxzoon* Neitz and Thomas, 1948.

Members of this genus multiply by schizogony in cells of the histiocytic series and by fission in the erythrocytes.

Cytauxzoon sylvicaprae Neitz and Thomas, 1948.
Cytauxzoon strepsicerosi Neitz and de Lange, 1956.

A review of the literature shows that much progress has been made in the studies of the biological transmission of the *Theileria* sp. and *Gonderia* spp. These observations have contributed greatly towards the identification of the infectious agents occurring in different countries. The chief differences between the infections occurring in cattle may conveniently be summarized in the appended Table 1.

1. Theileria parva Infection.

Definition.

East Coast fever is a highly fatal tick-borne disease of cattle caused by *Theileria parva* (Theiler, 1904). It is characterised by pyrexia, malaise, anorexia, lachrymation, digestive disturbances, emaciation, dyspnoea, swelling of the superficial and internal lymphatic glands, tumor splenis, tumor hepatis, " lymphomata " in the kidneys, and a multiple, localized, ulcerative abomasitis. Recovered animals develop a solid and a sterile immunity.

Synonyms.

Theileriosis, African Coast fever, Rhodesian tick fever, Rhodesian redwater; Theileriose, Ooskuskoors (Afrikaans); Theileriose van runderen (Netherlands); Theileriose der Rinder, Küstenfieber (German); La fièvre de la côte orientale, Theileriose a *Theileria parva* (French); La febbre della costa (Italian); Amakebe, Matussi, Romatussi, Kivagilira (names given to the disease by the inhabitants of East Africa).

History.

East Coast fever was known to the inhabitants of East Africa long before this territory was occupied by Europeans. Koch (1897), while investigating redwater in cattle in the vicinity of Dar-es-Salaam found the erythrocytes of affected animals parasitized not only by *Babesia bigemina* (Smith and Kilborne, 1893) but also by minute rod-shaped, oval and round organisms. At that time he regarded them to be young forms of *B. bigemina.* The event which first focussed attention on these parasites was the outbreak and subsequent spread of Rhodesian tick fever in Southern Rhodesia and South Africa. The aetiology and transmission of this disease was then studied by Koch, Theiler, Lounsbury, Gray and others. The causal agent was named *Piroplasma kochi* by Stephens and Christophers (1903), and *Piroplasma parvum* by Theiler (1904). The plasma bodies commonly referred to as Koch bodies were described a year later by Koch (1905, 1906). However, it was left to Gonder (1910, 1911) to show conclusively that these bodies actually represented the schizogonous phase of the developmental cycle of *Th. parva.*

Bettencourt, Franca and Borges (1907) compared the life-cycle of *Piroplasma bigeminum* with that of *P. parvum* and *P. annulatum,* and concluded that the presence of schizonts in the life-cycle of the latter two parasites justified their removal from the genus *Piroplasma,* and placed them in a new genus *Theileria.* They were named *Theileria parva* and *Theileria annulata.* Du Toit (1918) placed both species in the family Theileridae. Neitz and Jansen (1956) revised the classification of the Theilerias and in doing so drew attention to the fact that there is a striking difference between the life-cycle of *Th. parva* and that of the other Theileria spp. *Th. parva* only multiplies by schizogony within the lymphocytes, while the remaining *Theileria* spp. multiply by schizogony within the lymphocytes as well as by binary fission in the erythrocytes. This striking difference was used as a basis for the revision of the classification. *Th. parva* was retained in the family Theileridae du Toit, 1918, and the remaining *Theileria* spp. were transferred to the family Gonderidae Neitz and Jansen, 1955, and included in the redefined and reinstated genus *Gonderia.*

TABLE I.

The Characteristic Features of the *Theileria* and *Gonderia* Infections.

Observations.	*Theileria parva.*	*Gonderia annulata.*	*Gonderia mutans.*	*Gonderia lawrencei.*	*Gonderia bovis.*
Pathogenicity as determined by tick transmission	Cattle, water buffalo and African buffalo susceptible. Sheep and goats refractory	Cattle, water buffalo and American bison susceptible. Sheep and goats refractory	Cattle, water buffalo and African buffalo. In sheep and goats only sporozoites and schizonts develop but not the erythrocytic stage	African buffalo. In cattle only sporozoites and schizonts develop but not the erythrocytic stage. Sheep are refractory	Cattle. No information available on the susceptibility of the African buffalo, sheep and goats.
Natural transmission...	Through 7 *Rhipicephalus* spp. and 3 *Hyalomma* spp.	Through 6 *Hyalomma* spp. (Transovarial transmission in *H. savignyi* and *H. excavatum**)	Through 2 *Rhipicephalus* spp...	Through *Rh. appendiculatus*....	Through *Rh. appendiculatus.*
Incubation period following tick transmission	8–25 Days. Average 13 days...	9–25 Days. Average 15 days..	10–20 Days. Average 15 days.	12–20 Days. Average 15 days.	11 Days. Limited information.
Duration of disease....	10–23 Days. Average 15 days.	4–20 Days. Average 10 days..	3–10 Days. Average 5 days...	3–20 Days. Average 10 days..	5–15 Days. Average 10 days.
Erythrocytic stages.....	Over 80 per cent rod-shaped, less than 20 per cent round or oval	About 28–30 per cent rod-shaped, about 70–80 per cent round or oval	About 45 per cent rod-shaped, about 55 per cent round or oval	Pure infection not yet encountered in African buffalo	Pure infection not yet encountered in cattle.
Koch bodies.....	Usually very numerous. Average size 8·0 microns	Usually readily demonstrable. Average size 8·0 microns	Demonstrable in small numbers. Average size 8·0 microns	Demonstrable in small numbers. Average size 5·0 microns	Demonstrable in small numbers. Average size 5·0 microns
Anaemia.....	Oligocythaemia.....	Present.....	Very mild.....	Usually absent, Oligocythaemia may occur	Usually absent.
Icterus.....	May be present.....	Frequently present.....	Sometimes present.....	May be present.....	May be present.
Haematuria.....	Absent.....	Sometimes present.....	Absent.....	Absent.....	Absent.
Mortality.....	90 to 100 per cent. In enzootic regions less	10–80 per cent.....	Less than 1 per cent.....	Approximately 80 per cent.....	Up to 90 per cent.
Lymph glands.....	Always swollen.....	Often swollen.....	Moderately swollen.....	Often swollen.....	Often swollen.
Spleen.....	Often much swollen except in Kenya	Often much swollen.....	Swollen in acute cases.....	Often swollen.....	Often swollen.
Kidneys.....	" Infarcts " usually present (very rare in Kenya)	" Infarcts " often present.....	" Infarcts " seen in some of the acute cases	" Infarcts " seen in some of the cases	Usually absent.
Liver.....	Often swollen.....	Usually swollen.....	Present in acute cases.....	Often swollen.....	Often swollen.
Oedema of lungs.....	Usually present.....	Usually present.....	Present in acute cases.....	Very pronounced.....	Pronounced.
Ulcers in abomasum...	Present.....	Present.....	Present in acute cases.....	Usually present.....	Usually present.
Immunity.....	Solid and sterile.....	State of premunition.....	State of premunition in cattle and buffaloes; needs to be determined in sheep and goats	State of premunition in the African buffalo; needs to be determined in cattle	State of premunition in cattle.
Cross-immunity.....	Absent between *Th. parva* and *G. mutans.* Partial or complete between *Th. parva, G. bovis* and *G. lawrencei*	Absent between *G. annulata* and *G. mutans* and *Th. parva*	Absent between *G. mutans* and *G. annulata, G. lawrencei, G. bovis* and *Th. parva*	Absent between *G. lawrencei* and *G. mutans.* Partial or complete between *G. lawrencei*, and *Th. parva.*	Absent between *G. bovis* and *G. mutans.* Partial or complete between *G. bovis* and *Th. parva*

* The conclusions of Ray *et al.* (1940–41, 1950) and Kornienko and Shmyreva (1944) are not generally accepted.

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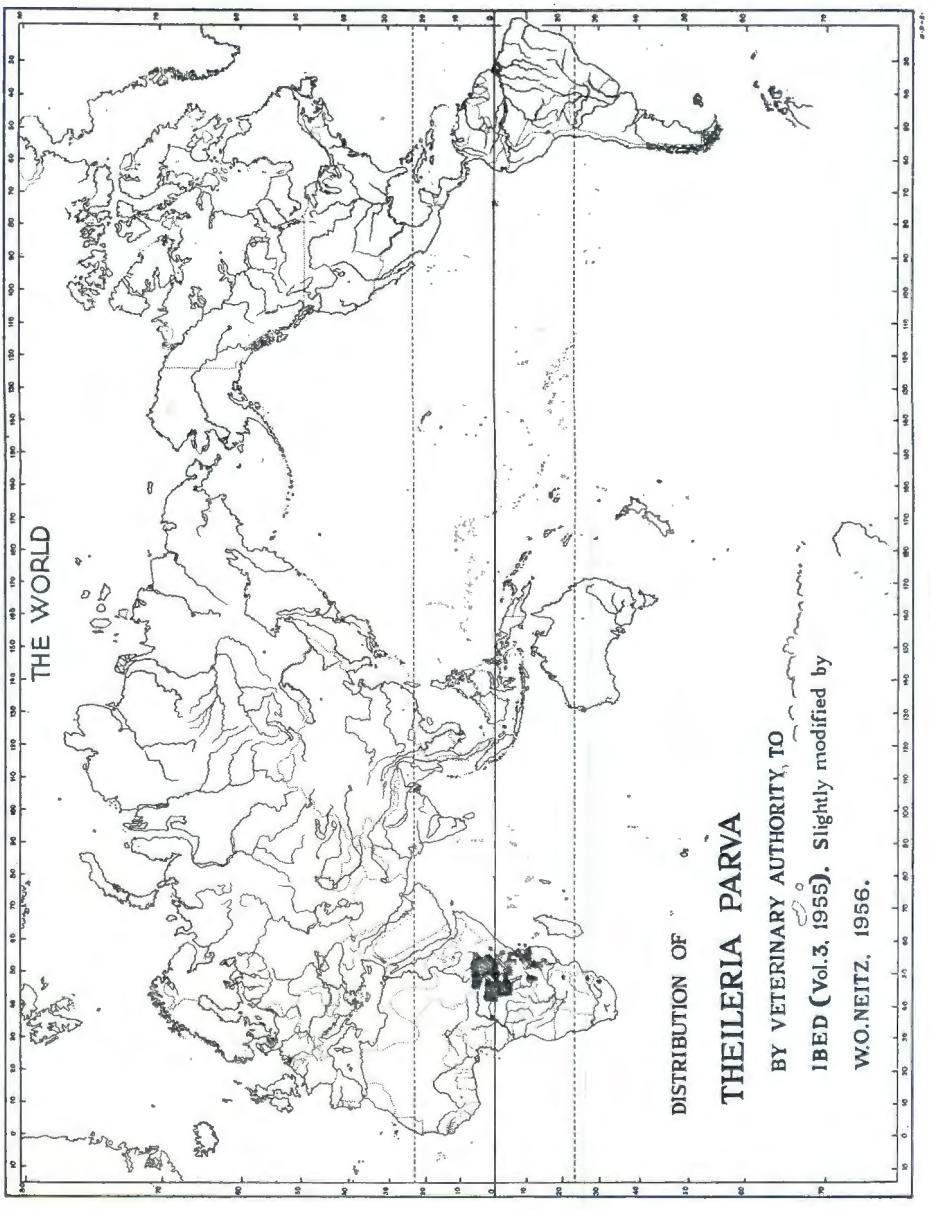
Distribution.

Territories in which *Rhipicephalus appendiculatus* occurs in association with other rhipicephaline vectors (Table III) must be regarded as potential East Coast fever areas. The countries in which this disease has occurred during the last six decades are listed in Table II. This information, however, does not give a true reflection of the present distribution. Prophylactic measures in Southern Africa



MAP No. 1.

have been responsible for the elimination of the infection over large areas. In Portuguese East Africa the disease has been eradicated by slaughter of infected herds in 1917. A recurrence was experienced a few years later but it was also eliminated in the same way (Botelho, 1924; Diesel, 1948). In the Union of South Africa quarantine measures, systematic dipping and slaughter of all cattle connected with isolated outbreaks have resulted in greatly reducing the incidence of the disease (Diesel, 1948). The last outbreaks in Transvaal occurred in 1945 (Diesel, 1948), and in the Transkei in 1954 (Diesel, 1956; Blomefield, 1956). A total of 11 outbreaks was reported from the Vryheid district in Natal during the period 30.5.54 to 28.3.55 (Diesel, 1956; Daly, 1956). In Swaziland van Heerden (1956) diagnosed East Coast fever in the Mbabane district at the beginning of 1954; the last death occurring on 6.2.56. Reports from Southern Rhodesia show that the last outbreak occurred in the Chipinga district on 10.2.54. All cattle on the infected farm were slaughtered, and there has been no recurrence (Adamson, 1954; Lawrence, 1956).

It is interesting to note that Northern Rhodesia has remained free from the disease, unless the tentative diagnosis of East Coast fever made by Hobday (1948, 1950) should be fully confirmed. It appears that this country is entirely free at present as no indication of its occurrence is given on the map showing the geographical distribution of East Coast fever in Africa, South of the Sahara, 1953 (IBED Report, 1955).

TABLE II.

Distribution of East Coast Fever in Africa.

Country.	References.
Belgian Congo.....	Van Saceghem, 1917, 1925, 1931, 1937, 1938; Schwetz, 1932, 1934; Schoenaers, 1951.
Kenya.....	Stordy, 1910, 1915, 1916; Montgomery, 1914–1915, 1917.
Northern Rhodesia.....	Hobday, 1948, 1950.
Nyasaland.....	Stannus, 1910; Griffiths, 1920.
Portuguese East Africa.....	Koch, 1903; Gray, 1908; Botelho, 1924.
Southern Rhodesia.....	Gray, 1902; Robertson, 1903; Koch, 1903; Kleine, 1903; Bevan, 1911; Sinclair, 1912.
Swaziland.....	Elder, 1912.
Tanganyika.....	Koch, 1897; Lichtenheld, 1906, 1907, 1908.
Uganda.....	Bruce, Hamerton, Bateman and Mackie, 1909; Uganda Vet. Rept., 1912; Hutchins, 1913, 1915, 1917.
Union of South Africa—	
Transvaal.....	Irvine-Smith, 1902; Hutcheon, 1903; Theiler, 1903, 1904.
Natal.....	Woollatt, 1906.
Transkei.....	Dixon, 1912; Spreull, 1914.
Eastern Cape Province.....	Dixon, 1912.
Zanzibar.....	Aders, 1916.

According to the annual veterinary reports listed in the literature (*vide infra*), the Belgian Congo, Kenya, Nyasaland, Tanganyika, Uganda and Zanzibar are true enzootic regions. It has not been possible to establish whether or not East Coast fever occurs in Abyssinia and Italian Somaliland (IBED Report, 1955). There is no evidence that East Coast fever occurs in West Africa. (See also Map No. 1.)

Aetiology.

Theileria parva (Theiler, 1904).

Synonyms:

Piroplasma bacilliformis, Koch, 1897.
Piroplasma kochi, Stephens and Christophers, 1903.
Theileria kochi (Stephens and Christophers, 1903).
Piroplasma parvum, Theiler, 1904.
Lymphohaematozoon sp., K. F. Meyer, 1913.

(*a*) *Morphology.*—(i) Erythrocytic parasites.—In blood smears fixed with May-Grünwald and stained with Giemsa *Th. parva* appears in the red blood cells as rod-shaped, round, oval or anaplasma-like organisms. The rod-shaped forms vary from 1·5 to 2·0 microns in length and 0·5 micron in width; oval forms 1·0 to 1·7 microns in length and 0·6 micron in width; round forms 1·0 to 1·2 microns in diameter, and the anaplasma-like forms 0·5 to 0·7 micron in diameter. Approximately 80 per cent are rod-shaped, 12 per cent are round, 6 per cent are oval and 2 per cent have an anaplasma-like appearance.

The cytoplasm stains light blue. The nucleus appears as a deeply stained minute reddish purple granule situated at the wider end of the oval or rod-shaped forms, and on the margin of the round parasites. In the anaplasma-like forms the cytoplasm can hardly be recognized.

(ii) Histiotropic parasites.—In blood and organ smears fixed with May-Grünwald and stained with Giemsa, the schizonts (Koch bodies, corps en grenade, " Plasmakugeln ") appear as masses of blue staining cytoplasm containing one to eighty reddish purple dots. Koch bodies vary in size from 1·0 to 15·0 microns, and in some cases they may be up to 25 microns in diameter. The average size is 8·0 microns. They are seen either free or within the lymphocytes. Neitz (1948) states that they may also appear in cells of the monocytic series. Two types of schizonts commonly referred to as agamonts (macroschizonts) and gamonts (microschizonts) are usually readily demonstrable particularly in the spleen, lymphatic glands, kidney, liver, and lungs (Cowdry and Danks, 1933). The former harbour chromatic granules varying from 0·4 to 2·0 microns (average 1·2 microns) in diameter, while the latter contain granules varying from 0·3 to 0·8 microns (average 0·5 micron). When fully formed the macroschizonts liberate macromerozoites varying from 2·0 to 2·5 microns in diameter. The mature microschizonts liberate micromerozoites 0·7 to 1·0 micron in diameter when round. Some of the forms are ovoid in shape, while others are rod-like, pear-shaped or comma-shaped.

(*b*) *Multiplication.*—*Th. parva* multiplies by schizogony. When schizonts are fully formed they break up into merozoites which either enter lymphocytes to grow and reproduce by schizogony again, or they penetrate the erythrocytes in which they are seen in ordinary blood films. Multiplication does not occur within the erythrocytes (Wenyon, 1926; Reichenow, 1940; Neitz and Jansen, 1956).

(*c*) *Habitat.*—The erythrocytic stages of *Th. parva* can be demonstrated readily in blood smears for periods of up to 10 days after recovery. In typical cases more than 80 per cent of the erythrocytes may harbour parasites. The host cell may contain 1 to 12 organisms. When partially immune cattle are infested with infective ticks up to 30 per cent of the erythrocytes may be parasitized during the reaction period. However, they disappear within a week after recovery. Endoglobular parasites cannot be demonstrated in immune animals. Splenectomy is not followed by a reappearance of parasites in the peripheral circulation as in the case of *Gonderia annulata* and *G. mutans* (du Toit, 1928; Neitz, 1948).

Schizonts can be demonstrated readily in organ smears but only appear in relatively small numbers in the peripheral blood during the course of the reaction. They are found to parasitise lymphocytes but according to Neitz, (1948) they may sometimes also develop in the cells of the monocytic series.

(*d*) *Life-cycle.*—The life-cycle of *Th. parva* in the arthropod vector, *Rh. appendiculatus,* has been studied by Gonder (1910, 1911), Cowdry and Ham (1932) and Reichenow (1937, 1938, 1940). The former three investigators are of opinion that schizogony in the vertebrate host is followed by sporogony in the arthropod. Reichenow (*loc. cit.*), on the other hand, concludes from his observations that asexual and sexual reproduction does not take place. According to him *Th. parva* only multiplies by simple binary fission within the lymphocytes of the mammalian host but not within the erythrocytes. When the endoglobular parasites are ingested by the vector some of them migrate to the salivary glands, and remain dormant within the cells of these organs. Binary fission is resumed by them, when the tick commences to suck blood after attachment on a new host. In other words, Reichenow believes that the life-cycle of *Th. parva* in the invertebrate and vertebrate host is similar to that of *B. canis,* as described by Regendanz and Reichenow (1933) in the tick, *Dermacentor retilculatus* Fabricius.

Cowdry and Ham (*loc. cit.*) have given the following account about the life-cycle of *Th. parva* in the vector:—

> "(1) After the tick takes in blood from a case of East Coast fever the ingested red blood cells can be seen to contain parasites of many shapes and sizes. Some are small and possess a nucleus which stains very deeply and which volumetrically is about as large as the cytoplasm. Others are much larger and contain nuclei which colour less intensely and are embedded in a relatively much greater amount of cytoplasm. Both show indications of division within the corpuscles, but this division is not often repeated. The small ones may be males and the large ones females, but in the absence of evidence no conclusion is warranted.

(2) Within the gut of the tick and after escape from the corpuscles two similar types of parasite can be distinguished, large and small. There is a marked tendency for each to be associated in clumps.

(3) The large and small parasites become applied to the surface of epithelial cells lining the gut and fertilization probably takes place, but we have not discovered convincing evidence of it.

(4) The parasites enter these cells and the small forms rapidly disappear. The large forms grow and give rise to a stage without distinct nuclei and not previously seen, which we refer to as a zygote.

(5) These zygotes increase greatly in size. Nuclei appear within them and some of them exhibit a central concentration of material and an accentuation of their limiting members.

(6) The central concentration becomes more marked and gradually assumes the form of a large elongated nucleated organism which we call an 'ookinete'.

(7) By rupture of the distended limiting membrane of the original zygotes the ookinetes escape into the cytoplasm of the epithelial cells lining the gut. They then make their way into the body cavity, where they may be found in close association with the salivary glands.

(8) The ookinetes enter the cells of the salivary glands where they round up, but may at first be recognized by the characteristic blue staining of their cytoplasm, and their sharply defined nuclei. The cytoplasm of the salivary gland cell in contact with them does not take the stain in the usual way, so that they seem to be surrounded by a kind of halo. The nucleus soon disappears and the parasite increases in size and becomes coloured more intensely but retains its halo.

(9) There is further growth and the cell containing the parasite becomes noticeably distended. The halo persists.

(10) About the periphery of the mass, buds appear: its central part colours less intensely with Giemsa's stain, and the clear outlines of the halo are lost. We designate this stage a 'sporont', and regard the peripheral buds as 'sporoblasts' in process of differentiation.

(11) The sporoblasts become much more distinct and exhibit within their interior irregular masses of chromatin. At the same time the central part of the sporont, which does not contain sporoblasts, loses progressively its affinity for stains.

(12) The sporoblasts develop rapidly. They possess a variable amount of chromatin in their interior. Their peripheral substance gives rise to sporozoites which are oriented rapidly in reference to them. Many of these sporozoites become detached from the parent sporoblasts.

(13) The size of the sporozoites is reduced about one-half. They are very small and consist of a slightly elongated mass of blue-staining cytoplasm, in one extremity of which is situated a deeply red-staining nucleus. A few of the sporoblasts remain. Some of them are still active in the formation of sporozoites, but the majority have lost their function and no longer have sporozoites regularly applied to their surfaces.

(14) The sporozoites, which look very much like the small forms observed both in the red blood cells (1) and free in the gut of the tick (2), are discharged into the lumen of a salivary gland sinus, whence some of them are presumably passed into the animal on which the tick is feeding and to which it is transmitting East Coast fever."

Reichenow (1940) critically reviews the work of Cowdry and Ham (1932), and points out certain fallacies. In the first place he mentions that within a few days after feeding certain "inclusion" bodies resembling protozoa appear in the epithelial cells of the gut in both clean and infected ticks. These structures were regarded by Cowdry and Ham (*loc. cit.*) to represent zygotes. Reichenow failed to find any structures which could be regarded as being ookinetes. The so-called "sporonts" are according to him nothing else but degenerated tissue cells phagocytosed by the salivary gland cells. The "sporoblasts" in their turn are compact masses of coalesced droplets secreted by the salivary gland cells. He, nevertheless, observed the appearance of sporozoites around the sporoblasts, but states that this developmental stage becomes visible when the secreted mass disappears from the host cell. Reichenow (*loc. cit.*) and Cowdry and Ham (*loc. cit.*), however, agree on one point, namely, that the final stage in the life-cycle of *Th. parva* in the vector is a minute unicellular parasite resembling the forms occurring in the erythrocytes, and that they appear in very large numbers in the salivary gland cells approximately 72 hours after the infective ticks have attached on a new host.

When the ears of susceptible cattle are infested with *Th. parva* infective ticks, schizonts appear in the parotid lymphatic glands either 5 to 13 days (Wagner, 1941), 6 to 15 days (Neitz, 1948), or 8 to 13 days (Reichenow, 1940) later. The Koch bodies are usually demonstrable in these glands before the initial rise in temperature. The fever signifies that an invasion of the blood stream by schizonts has taken place. Koch bodies can then be demonstrated after an interval of 24 hours in the other superficial lymphatic glands. Erythrocytic parasites appear four to five days later and persist in the peripheral blood during the course of the disease, and for periods of up to 10 days after recovery. Thereupon they disappear completely and do not reappear even when such animals are splenectomized. (Du Toit, 1928; Neitz, 1948.)

(*e*) *Cultivation.*—In 1952 Tzur (also known as Tchernomoretz in earlier literature) succeeded in growing Koch bodies of *Th. parva* in lymphocytes according to the method which he evolved for the cultivation of *G. annulata* schizonts in tissue culture.

(*f*) *Action of Physical and Chemical Agents.*—The inability to transmit East Coast fever regularly to susceptible cattle by the injection of infective blood or organ suspensions has greatly hampered determining the keeping qualities of the infectious agent. The immunization process applied on a large scale under field conditions by Theiler (1912) and Spreull (1914) have shown that infective organ emulsions remain potent for at least several hours. Richardson (1930) succeeded in immunizing cattle against East Coast fever by the injection of suspensions prepared from organs collected 24 hours previously from an affected animal.

(*g*) *Biological characteristics.*—Immunologically different strains of *Th. parva* have apparently not yet been encountered in nature. The writer has determined that a complete cross immunity exists between the South African and the Kenya strains.

(*h*) *Maintenance of* Th. parva *Infective Ticks.*—Strains of *Th. parva* are maintained at various laboratories in Africa by alternating the bovine and rhipicephaline passages. This involves infecting an ox with East Coast fever, and infesting its ears with *Rh. appendiculatus* larvae and/or nymphae when the erythrocytic stages of the parasites appear in fairly large numbers in the peripheral blood. The ticks are confined to the ears by means of suitable ear bags, the base of which is firmly secured by means of an adhesive paste around the base of the ears. Engorged ticks detach three to seven days later, and are then stored in suitable containers at a temperature of 26° C. and at a relative humidity of 80 per cent in an acaridarium. In these circumstances the larvae moult 10 days later, and the nymphae after a period varying from three to four weeks. The ensuing nymphae will attach readily on animals after a starvation period of 14 days, while the ensuing adults usually only show an inclination to feed when a period of three months has elapsed.

For many years the practice at Onderstepoort has been to infect fresh batches of *Rh. appendiculatus* larvae at six monthly intervals. This procedure was changed in 1937, after it had been noticed in 1932, that infective ticks may lose the infection six months after moulting. Since then batches of ticks were infected by feeding on reacting animals at four monthly intervals. The strain isolated on the farm Schoonspruit near Waterval Onder in the Eastern Transvaal in 1937, has been maintained in cattle and in ticks for 58 generations, and more than 100,000 larvae have been infected with East Coast fever. No change in the virulence of the parasite has been noticed. The mortality rate in susceptible cattle has been almost 100 per cent.

Transmission.

A. Natural Transmission.

(*a*) *Biological Transmission.*—Ticks responsible for the transmission of *Th. parva* are listed in the appended Table III. Seven *Rhipicephalus* spp. and three *Hyalomma* spp. have been proved experimentally to be transmitters. *Rh. appendiculatus* which is widely distributed in the enzootic regions of Central, East and Southern Africa is undoubtedly the chief vector. Although the remaining *Rhipicephalus* spp. are less important transmitters, they do nevertheless play a significant role in maintaining the infection in nature.

There is no evidence that the *Hyalomma* spp. transmit the disease in nature. Consideration of the biological transmission of East Coast fever and that of tropical gonderiosis (*vide infra*) shows that there is at least one feature common to both diseases, namely that *Hyalomma excavatum* (= *H. anatolicum*) and *H. dromedarii* are both capable of transmitting *Th. parva* and *Gonderia annulata.* Attention is drawn to this fact because in the past (du Toit, 1930) it was assumed that *Th. parva* is only transmitted by certain ticks belonging to the genus *Rhipicephalus,* and that the transmission of *G. annulata* is only effected by some species belonging to the genus *Hyalomma.* This assumption was actually used as one of the criteria for the differentiation between *Th. parva* and *G. annulata* (= *Theileria annulata* = *Th. dispar*). What is even more interesting from an epizootological point of view is that East Coast fever has never spread from East Africa into the adjacent tropical gonderiosis enzootic areas of the Sudan and Egypt even though the proven vectors *H. dromedarii* and *H. excavatum* occur in these countries.

Stage to stage transmission within the same generation has been observed in all the vectors but a transovarial transmission does not take place. It has also been established that infective nymphae lose their infection irrespective of whether they feed on a susceptible, insusceptible or an immune animal.

Theileria parva does not always live as long as the tick (*Rh. appendiculatus*) which it commonly infects. Reichenow (1940) and Wagner (1941) observed that adult ticks were infective for a period of six months after moulting but not after a year. Lewis and Fotheringham (1941) established that when infective ticks were starved for 350 days after moulting, they were no longer capable of transmitting East Coast fever, while ticks from the same infected batches, tested at short periods of starvation, transmitted the disease when fed on susceptible animals. Further investigations by Lewis (1950) showed that not a single batch of ticks, consisting of either nymphae or adults kept unfed for over 350 days after moulting, were capable of transmitting the disease. Ticks starved from 266 to 348 days gave mixed results: Some provoked a fatal attack of East Coast fever; some, a mild form of the disease; and others had clearly lost their infectivity before the expiration of 350 days.

The temperature under which *Rh. appendiculatus* ticks are maintained influences the infectious agent harboured by them. Nuttall and Hindle (1913) showed that when infected *Rh. appendiculatus* ticks were kept at 10°C for 21 days they were incapable of transmitting East Coast fever to a calf. However, when other ticks of the same batch, which had been maintained under the same conditions were exposed for a few days to a temperature of 30°C, they became infective and were capable of provoking a typical East Coast fever reaction in a calf. Fotheringham and Lewis (1937) determined that infected nymphae remained infective after having been subjected to temperatures varying from 4° to 6°C and 8° to 10°C for one, two and three weeks. Lewis (1950) states that exposure of infected nymphae to continuous temperatures of either 31° to 33°C, 34° to 35°C, 35° to 36°C or 37° to 38°C during the moulting period, and to temperatures of 19° to 22°C as soon as moulting was completed, caused the parasite in the tick to either lose its virulence or its viability.

Attempts to transmit *Th. parva* with *Rhipicephalus sanguineus* Laitr., *Rh. pulchellus* Gerstäcker and *Amblyomma variegatum* (Fabricius) have failed (Fotheringham and Lewis, 1937).

(*b*) *Mechanical transmission.*—Blood sucking insects have not been incriminated as vectors.

(*c*) *Intra-uterine transmission.*—This form of transmission has not been recorded.

B. *Artificial Transmission.*

It has been established by K. F. Meyer (1909), Theiler (1912), Wölfel (1912) and Walker (1926), that the intravenous injection of spleen and gland pulp from East Coast fever affected cattle produced the disease in a large number of animals. Sergent, Donatien, Parrot, Lestoquard and Plantureux (1926), and Theiler and du Toit (1929) succeeded in transmitting the disease by the injection of infective blood. The most successful method was the injection of blood both intradermally and subcutaneously. Theiler and du Toit (1928) also showed that transmission was possible, when partially engorged nymphae or adults of *Rh. appendiculatus,* infected with *Th. parva,* were emulsified and injected by the intravenous route into cattle.

Consideration of the results obtained by the above-mentioned investigators shows that there is a marked difference between the naturally acquired and the artificially produced disease. In the former, the course of the disease is fatal in 95 per cent of cases, whereas in the latter more than 50 per cent of animals which react, recover. An analysis of the results obtained by Theiler (1912) was made by Neitz (1946). The different types of reactions in the artificially infected cattle may be briefly summarized as follows:—

1. Typical reactions with a fatal termination were observed in 24·5 per cent of the cattle. (Endoglobular parasites and Koch bodies could be demonstrated.)
2. Typical reactions followed by recovery were noticed in 22·3 per cent of the animals. (Endoglobular parasites and Koch bodies could be demonstrated.)
3. Mild reactions developed in 13 per cent of the cattle which recovered. (Endoglobular parasites and Koch bodies could not be demonstrated.)
4. Irregular reactions from which the animals recovered occurred in 19·2 per cent of cattle. (Endoglobular parasites and Koch bodies could not be demonstrated).
5. In 21 per cent of the cattle no reactions were observed at all.

Animals which survived the artificial infection were all subjected to an immunity test. Generally speaking, it can be stated that the pattern of the different types of reactions in each group, following the immunity tests, had some resemblance to that exhibited by the susceptible cattle after the artificial infection. Deaths occurred in all the groups irrespective of whether typical, atypical or no reactions had been noticed previously. What was even more striking was the fact that as many as 46·7 per cent of the cattle which had not reacted to the artificial infection were found to be either partially or even solidly immune. (See Chart No. 1.)

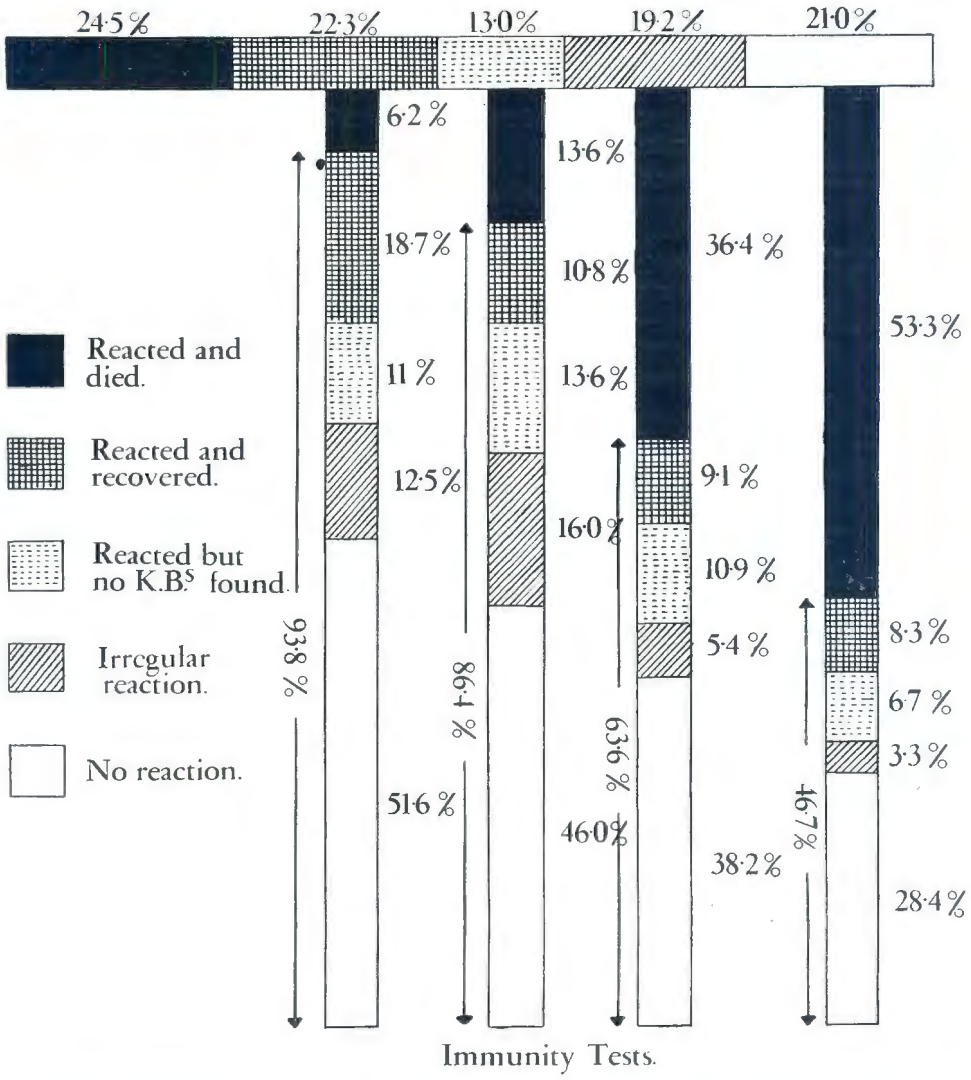
The types of the reactions obtained by Theiler and du Toit (1928, 1929) when either infective blood or emulsified ticks was used as inoculum, and the nature of the immunity as determined by the infestation of the survivors with infective *Rh. appendiculatus* ticks, were similar to those described originally by Theiler (1912). Consideration of these results shows that the variation in the degree of immunity is not in accordance with our present accepted views on protozoal immunity in general. Further studies are, therefore, necessary to determine the reasons for this manifestion. [Richardson (1930) and Neitz (1943, 1946) have suggested the possibility that East Coast fever is caused by the combination of a virus and a protozoon].

Epizootology.

Areas in which the *Rhipicephalus* spp. listed in Table III occur must be regarded as potential East Coast fever areas. Cyclic variations in the seasonal incidence of this disease do occur, but are not so well marked as for instance horsesickness, bluetongue and three-day-sickness. The general experience is that during winter sporadic outbreaks occur, while during other seasons, particularly in summer and autumn, a comparatively larger number of susceptible animals contract East Coast fever. The density of vectors and the presence of susceptible cattle determine the degree of prevalence.

CHART I.

Transmission of East Coast Fever with emulsions prepared from the spleen and gland. Original group artificially injected.



Physiographical factors favourable for the propagation of vectors are of considerable interest from the epizootological standpoint. *Rh. appendiculatus,* the chief vector, requires a warm and relatively humid and bushy grass country for its development, and occurs in areas with an annual rainfall of above 15 inches (Theiler, 1949). Although the other *Rhipicephalus* spp. may also be found under similar conditions, Theiler (1950) determined that *Rh. evertsi* can also exist in regions with a lower rainfall of 10 to 15 inches per annum in South Africa.

TABLE III.

The Biological Transmission of *Theileria parva*.

Vector.	Country.	No. of Hosts.	L.	N.	I.	E.	L.	N.	I.	References.
Rhipicephalus appendiculatus Neum. Syn. (*Rhipicephalus nitens* Neum.)	Central, East and Southern Africa	3	X—	—) X—	—)					Lounsbury (1903); Theiler (1905); Montgomery (1913); Fotheringham and Lewis (1937).
Rhipicephalus ayrei Lewis = ?(*Rhipicephalus compositus* Neum.)	East and Central Africa...	3	X—	—) X—	—)					Wilson (1953).
Rhipicephalus capensis Koch.....	Southern Africa.....	3	X—	—) X—	—)					Lounsbury (1906); Theiler (1907).
Rhipicephalus evertsi Neum.....	Central, East and Southern Africa	2	X—	——	—)					Lounsbury (1906); Theiler (1907); Fotheringham and Lewis (1937).
Rhipicephalus jeanelli Neum. = (*Rhipicephalus kochi* Dönitz)	East and Central Africa...	3	X—	—)						Wilson (1953).
Rhipicephalus neavei Warburton = (*Rhipicephalus neavei punctatus* Warburton = *Rhipicephalus pravus* Dönitz)	East Africa.....	3	X—	—) X—	—)					Lewis, Piercy and Wiley (1946).
Rhipicephalus simus Koch.....	South Africa, East Africa..	3	X—	—) X—	—)					Lounsbury (1906); Theiler (1905); Fotheringham and Lewis (1937); Neitz and Jansen (1956).
Hyalomma anatolicum Koch = (*Hyalomma excavatum* Koch according to Delpy, 1949 and Feldman-Mühsam 1954)	East Africa (Laboratory observations)	3	X—	—) X—	—)					Lewis and Fotheringham (1941).
Hyalomma dromedarii Koch.....	East Africa (Laboratory observations)	3	X—	—) X—	—)					Lewis and Fotheringham (1941).
Hyalomma impressum near *planum* Lewis = (?*Hyalomma transiens* Schulze = *Hyalomma truncatum* Koch)	East Africa (Laboratory observations)	2 or 3	X—	—) X—	—)					Lewis and Fotheringham (1937).

TABLE IV.

Domestic and Wild Members of the Family Bovidae Susceptible to *Theileria parva*.

Host.		Country.	Observations.	References.
Vernacular Name.	Zoological Name.			
Cattle.....	*Bos taurus* Linn.....	East, Central and Southern Africa..	Naturally and artificially infected..	Koch (1897). Theiler (1904).
African Buffalo.....	*Syncerus caffer* Sparrman.....	East Africa.....	Naturally infected.....	Lewis (1943).
Indian Water Buffalo.....	*Bubalus bubalis* Linn.....	East Africa..... South Africa.....	Naturally infected..... Naturally infected.....	Bradshaw (1924). Neitz (1940).

In Kenya the distribution of *Rh. neavei* frequently overlaps with that of *Rh. appendiculatus*, and also extends widely into areas where the latter tick is apparently unable to survive. It thus becomes apparent that *Rh. neavei* can maintain the infection in the absence of other *Rhipicephalus* spp. In outbreaks occuring at very high altitudes varying from 7,000 to 9,000 ft. above sea level, *Rh. capensis* is usually the vector. *Rh. appendiculatus* and *Rh. evertsi* have been recorded at an altitude of 5,000 ft. above sea level in South Africa (Theiler, 1949, 1950), and at 6,000 to 7,000 ft. in Kenya (Lewis, 1950). Schoenaers (1951) states that in the Belgian Congo, East Coast fever does not occur at an altitude of higher than 2,000 metres.

The available information on the source and maintenance of infection in *Rhipicephalus* spp. can be briefly summarized as follows:—

(i) Fully susceptible cattle and buffaloes reacting to East Coast fever:—

In enzootic areas calves from either species of animals sooner or later contract East Coast fever, and thus serve as reservoirs.

(ii) Partially immune cattle (possibly also African buffaloes) that develop a microscopic infection after reinfestation with infected ticks:—

Theiler (1912), Walker (1926), Lewis and Fotheringham, (1941) and the writer have established that endoglobular parasites can appear in the peripheral blood of partially immune animals. It has been found that such animals provide a source of infection to larval and nymphal ticks which can subsequently transmit a virulent disease to susceptible cattle, and that even one infected adult *Rh. appendiculatus* tick can provoke a fatal attack of East Coast fever (Lounsbury, 1904; Lewis, 1950).

(iii) The infective *Rhipicephalus* spp.—Infected nymphae lose their infection irrespective of whether they feed on a susceptible, insusceptible or an immune animal. A transovarial infection does not take place. *Th. parva* remains viable in either *Rh. appendiculatus* nymphae or adults for periods of up to 266 days after moulting. Infected ticks starved for periods varying from 266 to 348 days may or may not be infective, and those kept unfed for 350 days lose their infection completely (Lewis, 1950). After exposing infected *Rh. appendiculatus* nymphae or adults to continuous high temperatures (31° C to 38° C) or the full, but short, period of moulting the parasite loses its virulence or is destroyed (Lewis, 1950).

Factors favourable for the occurrence and the distribution of East Coast fever are—

(*a*) dense cattle population;

(*b*) the partial or complete absence of fences;

(*c*) the uncontrolled movement of stock within an enzootic area or into a potential East Coast fever area. This is often associated with speculators, native customs e.g. "lobola" (marriage custom), funeral ceremonies, etc.;

(*d*) (i) negligence on the part of the owner to report mortality;
(ii) the preparation of faulty smears associated with faulty diagnosis;
(iii) the substitution of smears prepared from healthy slaughtered stock, or from fowls, etc.;

(*e*) high incidence of vectors, no dipping tanks, shortage of acaricides, inadequate supply of water for the dipping tanks, shortage of staff, etc.;

(*f*) uncontrolled movement of hay, skins, etc., from infected farms into potential East Coast fever areas.

East Coast fever often occurs in association with anaplasmosis, babesioses, nagana and heartwater. In these circumstances it is difficult to estimate the direct losses due to East Coast fever unless systematic smear examinations are made from all animals that die within an area. This procedure has been adopted in most of the enzootic or potential enzootic East Coast fever areas of Africa. It entails a great deal of work but stock owners realize the value of this system, and usually collaborate with the veterinary authorities and submit spleen, gland and blood smears from animals that die within the scheduled areas.

The role played by the African buffalo as reservoir of East Coast fever has not been satisfactorily determined. The successful transmission of this disease to the buffalo with infective *Rh. appendiculatus* ticks (Lewis, 1943), suggests that it can be maintained under natural conditions in the complete absence of cattle.

East Coast fever is one of the most important stock diseases of Africa. Its presence has greatly interfered with the establishment of pure-bred stock. The severe losses are well known to farmers. Production of milk and beef could have been very much higher had it not been for this hazard.

Pathogenicity.

Members of the family Bovidae susceptible to *Th. parva* are listed in Table IV. In addition to cattle, the Indian water buffalo and the African buffalo are susceptible to natural East Coast fever infection. The latter species is highly resistant, while the Indian buffalo appears to be as susceptible as cattle.

In Southern Africa the mortality rate in calves and in adult stock is more than 95 per cent. In exceptional cases it may be as low as 78 per cent (Canham, 1951). Theiler, Gray and Power (1914) are of opinion that calves from immune cows are highly resistant. The writer, on the other hand, established, that one to four weeks old calves from either immune or susceptible cows, develop a fatal form of the disease after having been infested with infective ticks.

In the enzootic regions of East Africa there is a marked variation in the mortality both in young and adult stock. It appears that calves and immature cattle under natural conditions possess a greater degree of resistance than mature animals. In calves the mortality varies from 5 to 50 per cent.

According to Lewis (1950) observations in Kenya indicate that while the mortality from East Coast fever among calves varies considerably, the disease in adult stock is usually, but not invariably, fatal. Recoveries among adults are not rare, and instances are known where up to 75 per cent of affected animals recovered. Low mortality among calves and young animals sometimes coincides with a low tick population. It occurs also where tick infestation is heavy or moderately so; and on occasions which appear to be rare a high percentage of recoveries is recorded amongst adult cattle. Lewis (*loc. cit.*) attributes the low mortality to a decrease in the virulence of *Th. parva* in the vector. This reduction in virulence became evident after infective nymphae or adults had been starved for long periods (266 to 348 days), and when engorged larvae and nymphae had been exposed to continuous high temperatures (31°-38° C) for the full, but short,

period of moulting. His field surveys have shown that infective ticks may be exposed to similar conditions in nature, and that an explanation can now be given for the variation in the mortality rate in cattle in the enzootic regions.

Pathogenesis.

The lesions present in the various organs suggest that they are due to a toxin produced by the infectious agent. The endothelial lining of the blood vessels also becomes affected resulting in an oedema of the lungs, subcutaneous and intermuscular tissues. The parasitized erythrocytes liberate coproporphyrin and bilirubin (Roets, 1938, 1943).

Symptoms.

Studies made on naturally and artificially infected cattle, and on naturally infected Indian water buffaloes established that the severity of East Coast fever usually depends upon the intensity, and duration of the parasitic attack. After exposure to tick infestation, the incubation period usually varies from 8 to 25 days with an average of 13 days. After an artificial infection the period varies from 12 to 20 days with an average of 15 days. The administration of either Aureomycin or Terramycin during the incubation period may cause this period to be prolonged by two or three days.

Depending upon the degree of virulence of the parasite, the resistance of the animal, and the mode of transmission, East Coast fever may be classified according to its symptoms into (1) the acute, (2) the subacute, (3) the mild, and (4) the inapparent form.

(1) *The acute form.*—This is the usual type observed. The affected animal exhibits pronounced symptoms usually terminating fatally. There is an elevation of the body temperature varying from 104° to 107° F. The fever may be continuous during the whole course of the disease or alternatively returns to normal or almost to normal after 7 to 11 days, and rises again after one to two days to 106° F. for a period of five to eight days. A rapid drop in temperature takes place before death. Clinical symptoms usually appear a few days after the initial rise in temperature. Koch bodies may be demonstrable in the parotid lymphatic glands as early as four days before the commencement of fever, and the endoglobular form four to five days after the initial rise in temperature. The animal shows inappetence, cessation of rumination, serous nasal discharge, lachrymation, sometimes swelling of the eyelids, ears and jowl region, swelling of the superficial lymphatic glands, frequent pulse, general weakness and a decreased milk production and in some cases icterus. At the beginning of the pyrexial period the faeces are firm but diarrhoea usually sets in six to eight days after the initial rise in temperature. The evacuations are frequently mixed with blood and mucus. The animal becomes markedly emaciated, and assumes a recumbent position and often coughs. The respiration becomes accelerated, and dyspnoea becomes pronounced shortly before death. A variable amount of froth exudes from the nostrils. The course of the disease varies from 8 to 25 days with an average of 15 days.

During the course of East Coast fever, particularly during the period of the second febrile reaction, relapses due to *Babesia bigemina, Babesia bovis* (Babes, 1888), *Anaplasma marginale* Theiler, 1910, and *Gonderia mutans* (Theiler, 1906) may appear. The symptoms of redwater and gall-sickness may obscure the typical clinical manifestations of East Coast fever.

(2) *The subacute form.*—This form is often encountered in calves, and to a lesser extent in adult stock in the enzootic regions of East Africa. It has also been observed in partially immune animals after infestation with infective ticks, and in artificially infected cattle. The symptoms resemble those of the acute form but are not so pronounced. The fever is either continuous or irregularly intermittent, and persists as long as 5 to 10 days. A fairly large number of Koch bodies, and endoglobular parasites can be found in the blood and lymphatic gland smears. Animals usually recover from this form but it may take several weeks before they regain their former condition.

(3) *The mild form.*—In this form the symptoms are a relatively mild fever, which persists for a period of three to seven days, listlessness and swelling of the superficial lymphatic gland. It may be mistaken for benign bovine gonderiosis (*vide infra*). It has been observed in susceptible and in partially immune animals after infestation with infective ticks, and in fully susceptible cattle after an artificial infection. In naturally infected animals lymphatic gland smears reveal a relatively small number of Koch bodies, and in blood smears a few endoglobular parasites may be seen. Their identity, however, can only be determined by making a xenodiagnosis. In artificially infected animals Koch bodies are not demonstrable (Theiler 1912).

(4) *The inapparent form.*—This form has been observed frequently by Theiler (1912), Theiler and du Toit (1928, 1929) and Walker and Whitworth (1929) in cattle which had been infected artificially by the injection of either blood, coarsely ground spleen and lymphatic gland emulsions or suspensions prepared from partially engorged infected *Rh. appendiculatus* ticks. The only way of determining whether an inapparent infection has taken place is to challenge the immunity of such animals with infective ticks.

Pathology.

The lesions of East Coast fever do not vary much and resemble those occurring in either tropical gonderiosis, Corridor disease, *G. bovis* infection or the severe form of benign bovine gonderiosis.

A. *Macroscopical lesions.*—In the acute and subacute forms the carcase is usually emaciated. The skin may show decubital wounds and a variable number of ticks. The perineal region and tail are often soiled with faeces. The visible mucous membranes appear cyanotic. A variable amount of froth may escape from the nostrils. The subcutaneous and intermuscular tissues may be yellow in colour, and are sometimes infiltrated with clear serous fluid, giving them a gelatinous appearance. Degenerative changes and haemorrhages may be seen in the semi-membranosus, semitendinosus and adductor muscles.

The myocardium is flabby and a variable number of petechiae appear on the epicardium and endocardium. Hydrothorax and hydropericardium are not constantly present. The mediastinum may show extensive serous infiltration and petechiae. The visceral and parietal pleura may be spotted with petechiae. The lungs are often congested and oedematous. The mucous membrane of the distal extremity of the trachea and bronchi shows a variable number of petechiae. The liver may be increased in size, friable and brownish yellow to lemon yellow in colour; parenchymatous degeneration is evident. The gall bladder may be markedly distended with dark green viscid bile. The spleen is usually enlarged and the pulpa soft; the Malpighian corpuscles may be prominent. The superficial and internal lymphatic glands are as a rule markedly swollen, and show a variable degree of hyperaemia. The capsula adiposa of the kidneys may contain a large amount of serous fluid. The kidneys are either congested or pale brown in colour, and show a variable number of haemorrhagic " infarcts " or greyish white lymphomata. Petechiae may be present in the cortex of the adrenal glands. The urinary bladder is usually markedly distended with clear yellow urine; haemorrhages may be seen in the mucous membrane. The meninges may be slightly congested but the brain does not show any lesions.

The rumen and reticulum contain a relatively small amount of ingesta, while the contents of the omasum are firm and partially dehydrated. The abomasum shows characteristic ulcers which vary in size from 2·0 to 5·0 mm. Larger sized ulcers may sometimes also be seen. They consist of a central necrotic area, red or brown in colour, surrounded by a haemorrhagic zone. Similar ulcers as well as irregularly disseminated red streaks or patches may be encountered along the entire length of the small and large intestine. Peyer's patches are swollen. The contents of the small and large intestine have a distinct yellow tinge.

B. *Microscopical lesions.*—The histopathological changes of East Coast fever have been described by Steck (1928). His observations can be briefly summarized as follows: The pathological picture is dominated by a proliferation of lymphoid tissue. The kidney shows a variable number of foci in the cortex either around a small artery or in the immediate neighbourhood of a glomerulus. The whole focus consists of a rapidly proliferating lymph node, taking its origin in the adventitia of small arteries. Koch bodies can be demonstrated in many of the lymphocytes. The parenchymatous elements show hardly any change. In a fair number of cases, the veins are compressed when passing through a dense lymphocytic nodule, and distally much widened and filled with blood. In the neighbourhood of distended veins the interstitium often shows extravascular red cells, sometimes in considerable number. However, distended veins and extravasation are also found where no stricture of veins can be detected or even where no lymphocytic proliferation is present.

The most striking changes in the liver are found in the periportal interstitia. There is not only an extensive infiltration of lymphocytes but these cells also show active proliferation. Many of them harbour Koch bodies. The central veins contain an increased number of lymphocytes, many of which show mitosis. The parenchyma cells reveal fairly definite but not extensive degenerative changes. There may be a diffuse fatty infiltration or presence of hyaline droplets. The nuclei may show pyknosis, karyolysis, rarely karyorrhexis.

In the lymph glands the follicles are enlarged, the centre of Fleming indistinct, and a large proportion of the lymphocytes and lymphoblasts is infected with Koch bodies. The capillary vessels of the lung contain a large number of lymphocytes, some infected with Koch bodies, few with pyknotic nuclei. There may also be an accumulation of lymphocytes around the arterioles.

Microscopically the erythrocytes show no degenerative or regenerative changes. An oligocythaemia has been observed by Wagner (1941) and Neitz (1948). The red cell count in affected animals may drop from seven to three million per c.mm. Steck (1928) states that a leucopenia sets in with fever, and that lymphocytes tend to accumulate in the capillary system. Wagner (1941), on the other hand, noticed a decrease in the leucocytic count to commence seven days before death. Roets (1938) isolated coproporphyrin I and coproporphyrin III from the faeces and urine of cattle suffering from East Coast fever. Subsequently he (Roets, 1943) established that bilirubinaemia occurs in affected animals, and that the yellow staining of the fat may be due to the combined effect of bilirubin and carotinoids. The latter pigments are present in green food, and are readily absorbed by cattle (Rimington, 1937).

Diagnosis.

The clinical symptoms presented by cattle suffering from East Coast fever (*Th. parva* infection) are such that they may be confused with those occurring in Corridor disease (*G. lawrencei* infection), Rhodesian malignant bovine gonderiosis (*G. bovis*), benign bovine gonderiosis (*G. mutans* infection), babesioses and anaplasmosis. It is essential that blood and lymphatic gland smears be examined for the presence of Koch bodies and endoglobular parasites. Their presence signifies that the animal is suffering from either theileriosis or gonderiosis. In making a differential diagnosis the epizootology, symptomatology, pathology and the frequency with which Koch bodies and endoglobular parasites appear in blood and organ smears must be taken into account. The mortality rate in East Coast fever is usually more than 95 per cent, in Corridor disease 80 per cent, in *G. bovis* infection 90 per cent and in benign bovine gonderiosis less than one per cent. In the former disease more than 60 per cent of the lymphocytes and more than 50 per cent of the erythrocytes are parasitized, while in the latter three diseases up to 5 per cent of lymphocytes harbour schizonts. The Koch bodies of *Th. parva* and *G. mutans* vary from 1·0 to 15·0 microns with an average diameter of 8·0 microns. The schizonts of *G. lawrencei* and *G. bovis* vary from 1·0 to 10·0 microns with an average diameter of 5·0 microns. Consideration of these criteria makes a differential diagnosis possible.

Difficulty in making a differential diagnosis arises when cattle are suffering from either a mild form of East Coast fever, Corridor disease *G. bovis* infection or a somewhat severe form of benign bovine gonderiosis. In these circumstances cross-immunity tests on recovered animals have to be resorted to. This involves transferring suspected cases to a laboratory, and then infesting them with infective *Th. parva* ticks. Absence of an immunity justifies making a diagnosis of a *G. mutans* infection; presence of an immunity signifies that the animal had reacted previously to either a *Th. parva, G. bovis* or *G. lawrencei* infection. The latter conclusion is based on the fact that a cross-immunity exists between East Coast fever, *G. bovis* infection and Corridor disease (*vide infra*). Consideration of these results together with the history of the farm may allow a final diagnosis to be made. Failing this, the diagnosis is dependent upon the possible appearance of typical cases of the disease at the site of the outbreak.

For the identification of endoglobular parasites present in cattle harbouring Koch bodies at the same time, it may be necessary to apply the xenodiagnosis. This involves feeding clean *Rh. appendiculatus* larvae or nymphae on the affected animals, and the ensuing stages on several fully susceptible calves. In doing so the writer found on one occasion that nymphae, which had fed in their larval stage on an animal harbouring a light infection of *G. mutans* and *Th. parva* endoglobular parasites, transmitted benign bovine gonderiosis to one calf and typical East Coast fever to two animals. When the immunity of the benign bovine gonderiosis recovered calf was challenged subsequently with infective *Th. parva* ticks, it reacted and died from East Coast fever. These observations clearly indicate the importance of using more than one calf for the xenodiagnosis. The fact that sheep and goats are susceptible to the sporozoites and schizonts of *G. mutans* (*vide infra*) makes it apparent that either one or both these species can be included when the xenodiagnosis is applied.

Consideration of these observations suggests that further work be conducted to determine the fate of either *G. mutans* or *Th. parva* within the vector when both parasites are ingested at the same time. It would appear that only one of the two parasites survives, and that this results in a pure infection within the vector.

Lichtenheld (1910, 1911) claims that the complement fixation test can be applied for diagnostic purposes. The antigen is prepared from the lymphatic glands of a beast suffering from East Coast fever. The serum obtained from either sick or recovered animals gave a distinct deviation. The reliability of this test still needs to be determined.

Treatment.

A. *Specific Treatment.*—Drugs administered to cattle during the course of an East Coast fever reaction are listed in the appended Table V. None of them proved to be effective.

During the course of the studies on the chemotherapy of East Coast fever, Neitz (1950) noticed that the intramuscular administration of the 8-aminoquinolin compound, pamaquin (0·5 mg. per Kg. body weight) on alternative days during the incubation period of a sporozoite induced infection, and daily during the reaction period, neither influenced the course of the disease nor the development of the Koch bodies. Careful examination of the blood and the lymphatic gland smears, however, revealed the presence of a comparatively small number of endoglobular parasites, which showed a variable degree of degeneration. They were either contracted or devoid of their cytoplasm. The nuclear rests in the erythrocytes resembled *Anaplasma marginale* in appearance. In order to confirm the histological observations, brown tick larvae (*Rh. appendiculatus*) were allowed to feed on the pamaquin treated animals. Two young oxen were infested subsequently with the ensuing nymphae. They were kept under observation for six weeks but failed to react to East Coast fever. On testing their immunity both animals contracted East Coast fever and died.

TABLE V.

Drugs which Proved to be Ineffective in the Treatment of East Coast fever

Drug.	Effect on the Course of the Disease.	References.
Acaprin	—	Reichenow, 1938.
Acaprin	—	Van Saceghem, 1938.
Ammoniun fluoride	—	Nuttall, 1915.
Arsacetin	—	Nuttall, 1915.
Berenil	—	Bugyaki, 1956.
Calcium chloride	+	Gillain, 1951.
Calcium lactate	—	Nuttall, 1915.
Congo red	—	Nuttall, 1915.
Creosote and oleum	—	Nuttall, 1915.
Copaive	—	
Emetine hydrochloride	—	Nuttall, 1915.
Ethylhydrocupreine	—	Nuttall, 1915.
Gonacrine	—	Els, 1934.
Hoechst 606	—	Nuttall, 1915.
Iboza multiflora	+	Van Saceghem, 1951.
= (*Coleus aromaticus*)	—	Deom, 1952.
Immune serum	+	Koch, 1903.
Immune serum	—	Theiler, 1907.
Mercury salicylate	—	Nuttall, 1915.
Mercury succinimide	—	Nuttall, 1915.
Nivaquine	—	Deom, 1954.
Nuclein	—	Nuttall, 1915.
Phenamidine	—	Piercy, 1951.
Potassium iodide	—	Nuttall, 1915.
Quinacrine	—	Piercy, 1951.
Quinine bihydrochloride	—	Nuttall, 1915.
Quinine hydrochloride	—	Nuttall, 1915.
Soamin	—	Nuttall, 1915.
Sodium salicylate	—	Nuttall, 1915.
Tryposafrol	—	Nuttall, 1915.
Trypan blue	—	Nuttall, 1915.

— = Of no value; + = Beneficial influence.

Further chemotherapeutic studies were conducted on two oxen. One ox received a daily injection of 1·0 mg. pamaquin per Kg. body weight during the periods of incubation and reaction, while the other animal was treated daily with the same dose during the period of reaction. This increased dose also failed to influence the course of the disease. Both oxen died. Smear examination again showed that the development of the Koch bodies was not interfered with, but that the endoglobular parasites revealed a variable degree of degeneration. Similar chemotherapeutic tests were undertaken with the 8-aminoquinolin derivatives, pentaquin and primaquin. It was also noticed that both compounds possessed a similar selective action on the endoglobular parasites of *Th. parva*.

Recognition of the selective action of the 8-aminoquinolin preparations on the erythrocytic parasites of *Th. parva* focussed attention of the writer to the chemotherapeutic studies previously conducted on avian malaria. Kikuth (1931, 1932, 1935) had developed a method by which the schizonticidal action of atebrin or other compounds could be demonstrated by administering plasmoquin as well as the test drug to Java sparrows infected with *Haemoproteus orizivorae*.

The application of Kikuth's conception of selective chemotherapeutic action to East Coast fever prompted the writer to continue the studies on sporozoite-induced *Th. parva* infection. (See Fig. 1.) It was noticed that resochin (4-amino-quinolin preparation) alone or in combination with plasmoquin when injected during the incubation and reaction period, had some action on the schizonts of *Th. parva.* Although two out of five animals recovered it was soon realized that a search for a more specific schizonticidal drug was indicated. The investigation was extended to include Aureomycin. This antibiotic, administered intravenously in repeated large doses during the incubation period of infection, was found to exert a marked effect upon the schizonts of *Th. parva* (Neitz, 1953).

For these tests, nine cattle were used, of which two served as untreated controls. Treatment was commenced 24 hours after tick infestation by the injection of Aureomycin in a dose of 10 mgm. per Kg. body weight. The same dose was repeated at arbitrary irregular intervals as indicated in the appended Table VI until a decrease in the number of schizonts in lymph glands became evident. In each case from this stage the number of schizonts continued to decrease rapidly until they could no longer be found after prolonged search. Endoglobular forms of the parasites persisted from one to five days after disappearance of the schizonts. Apart from an exceedingly mild febrile reaction and swelling of the superficial lymph glands, the animals showed no clinical evidence of the disease. (See Fig. 2.) After complete recovery, it was shown that a solid immunity to re-infection had been induced in all animals tested one to eleven months later.

In the untreated controls the normal fulminating form of the disease was produced, followed by death not later than the twenty-fourth day after attachment of the ticks.

Consideration of these results shows that Aureomycin in a dose of 10 mg. per Kg. body weight administered in repeated doses during the incubation period of *Th. parva* infection is a schizonticide which acts on the early schizonts by suppressing nuclear division. This in turn suppresses the formation of merozoites, which invade either the lymphocytes or erythrocytes. The drug appears to have no effect upon the haemotropic parasites. The result is that completion of the cyclical development of sporozoite-induced infection is inhibited, and the clinical disease fails to develop. Nevertheless, a solid immunity is produced.

Further tests have shown that Terramycin possesses the same chemotherapeutic properties as Aureomycin in the treatment of East Coast fever. The work was extended to determine whether either Aureomycin or Terramycin alone or in combination with pamaquin would have a beneficial influence on the course of the disease if treatment is commenced on the first day of the reaction. It was determined that 10 successive injections of Aureomycin at 24 hourly intervals had no influence on the course of the disease. However, when a total of 10 to 17 injections of either Aureomycin or Terramycin (10 mg. per Kg. body weight) in combination with pamaquin (1·0 mg. per Kg. body weight) was administered intravenously at 24 hourly intervals recovery followed, even though the clinical symptoms were fairly severe.

It is self-evident from these chemotherapeutic tests that this form of treatment cannot be applied in practice. It is nevertheless a useful method for the production of immune animals often required at the laboratory for immunological studies. It is believed that the selective chemotherapy will be of assistance for studying the life-cycle of *Th. parva* in greater detail. The recognition of these specific drugs may also serve as a basis for the synthesis of more potent drugs urgently required in the highly enzootic areas of East Africa.

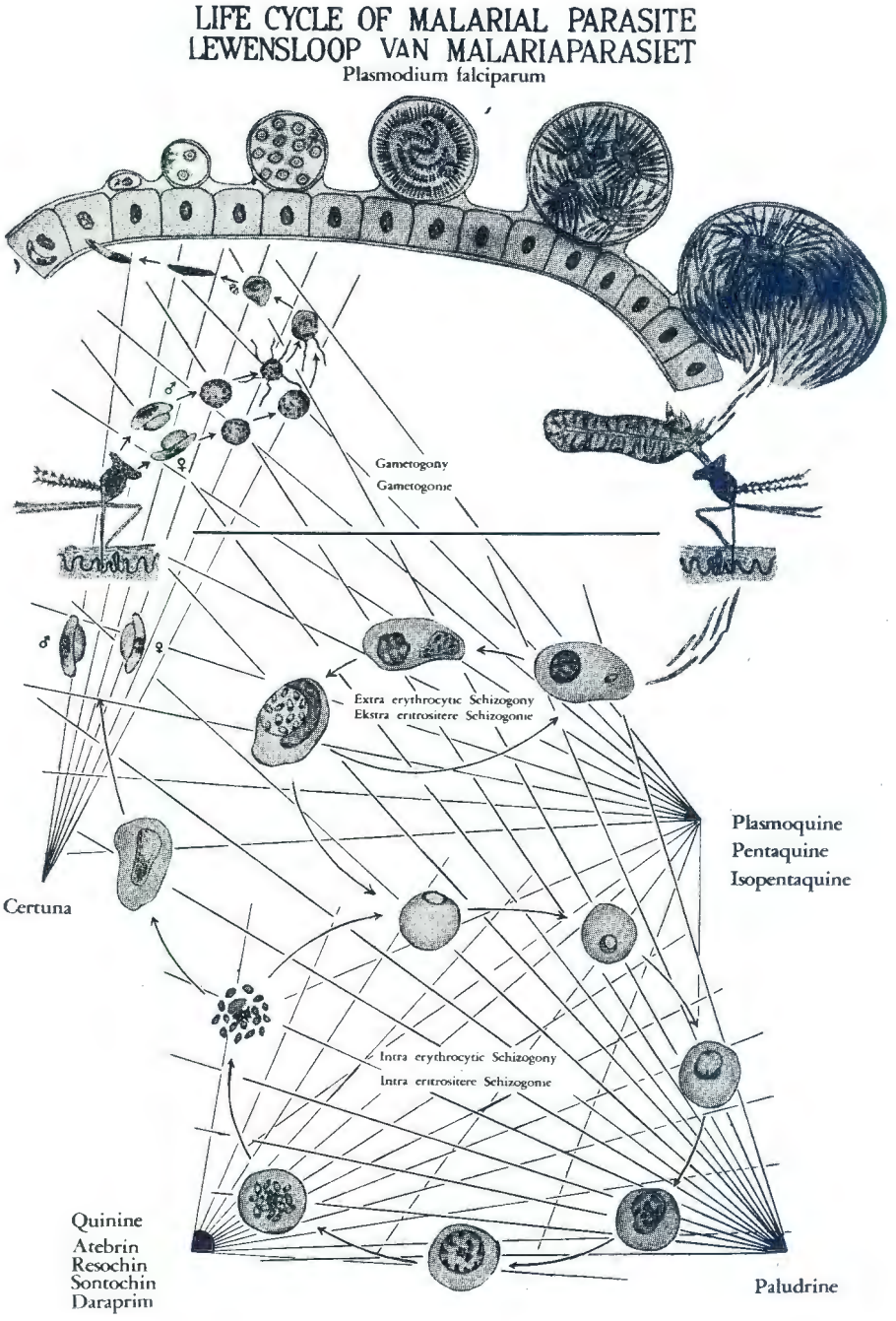

LIFE CYCLE OF MALARIAL PARASITE
LEWENSLOOP VAN MALARIAPARASIET
Plasmodium falciparum
Gametogony
Gametogonie
Extra erythrocytic Schizogony
Ekstra eritrositere Schizogonie
Plasmoquine
Pentaquine
Isopentaquine
Certuna
Intra erythrocytic Schizogony
Intra eritrositere Schizogonie
Quinine
Atebrin
Resochin
Sontochin
Daraprim
Paludrine

FIG. 1.

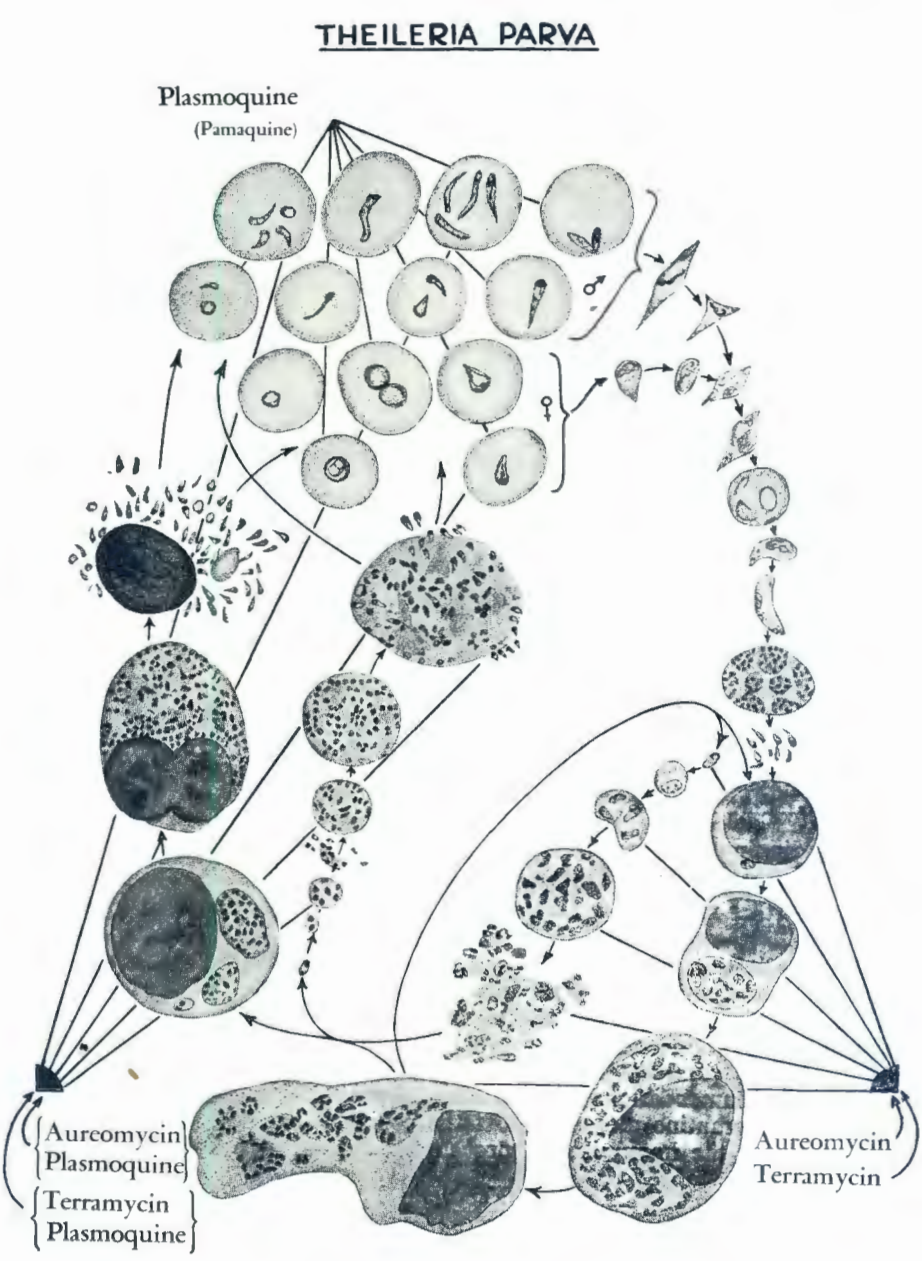

THEILERIA PARVA
Plasmoquine
(Pamaquine)
Aureomycin
Plasmoquine
Terramycin
Plasmoquine
Aureomycin
Terramycin

FIG. 2.

B. *Symptomatic treatment.*—This type of treatment is of no value in the acute form but it should be useful in the subacute form of East Coast fever. Good nursing and the control of intercurrent infections should reduce the mortality rate.

Prognosis.

Prognosis should always be guarded. Not only is the mortality extremely high but the control measures have cost the different African States thousands of pounds. It should, however, be remembered that systematic dipping employed for the eradication of East Coast fever ticks has been of benefit to all countries in that it has also reduced the incidence of other highly fatal tick-borne diseases.

Prophylaxis.

The methods employed for controlling East Coast fever are discussed in the numerous annual reports published by the Veterinary Authorities of Eastern and Southern Africa. A comprehensive survey on the campaign against this disease in South Africa has been given by Diesel (1948). He refers to the various veterinary conferences at which the subject of " East Coast Fever Eradication " was carefully considered, and to the appointment of committees whose function it was to make recommendations for efficient control measures. It is self-evident that prophylactic measures were modified from time to time as the knowledge of the epizootology, biological habits of the infectious agent and the vectors, and the efficacy of acaricides improved. In this connection the conferences resolved that the control of East Coast fever should be arranged in terms of—

(*a*) control over cattle movements;

(*b*) destruction of all cattle connected with isolated outbreaks, with the payment of compensation;

(*c*) fencing and quarantine of infected veld;

(*d*) dipping or cleaning from ticks, of all cattle in the immediate vicinity of infection;

(*e*) further research by all territories.

The application of control measures has been very effective, and in recent years only a few outbreaks (*vide supra* " Distribution ") have been recorded in Natal, Swaziland and Southern Rhodesia. These results show that the efforts of the veterinary staff in collaboration with those of the stockowners have not been in vain. It is beyond the scope of this paper to consider each one of the above mentioned recommendations in detail and hence attention will be paid to the dipping and quarantine measures. At the conclusion of this discussion reference will be made to the immunization process temporarily employed in the Transkei.

(1) *Elimination of Arthropod Vectors.*—The only effective weapon for destruction of ticks is regular systematic dipping or spraying, combined with careful hand-dressing. The dipping of cattle in arsenical dip (0·16 per cent As_2O_3) at five, five and four day intervals has been successfully employed in many of the enzootic East Coast fever regions. Experience has shown that such regions are usually badly infested with various species of ticks, so that it may not be possible to keep animals entirely free from these ectoparasites.

TABLE VI.

Aureomycin Treatment of East Coast Fever during the Incubation Period.

Days.	Treated Animals.							Untreated Animals.	
	5085	5430	5438	5483	5614	5436	5444	5700	5704
1	X	X	X	X	X	X	X	—	—
2	—	—	—	X	X	X	X	—	—
3	X	X	X	X	X	X	X	—	—
4	—	X	X	—	—	—	—	—	—
5	X	—	—	X	X	—	—	—	—
6	—	X	X	—	—	X	X	—	—
7	X	—	—	X	X	—	—	—	—
8	—	X	X	—	—	—	—	—	—
9	—	—	—	X	X S+	X S+	X S+	—	—
10	X	X	X	S+	S+	S+	S+	—	S+
11	—	S+	—	X S+	X S+	X S2+	X S2+	S+	S2+
12	X S+	X S+	X	S+	S3+	S2+	S2+	S+	S3+
13	S+	S+	S+	X S+	X S3+	X S3+	X S3+	S2+	S3+
14	X S2+	X S+	X S+	S2+	S2+	S3+ P+	S4+ P+	S2+	S4+ P+
15	S2+	S+	S2+	S+	S2+	X S3+ P2+	X S4+ P2+	S3+ P+	S4+ P+
16	S2+	X S+	X S2+	S+	S2+	X S4+ P3+	X S4+ P2+	S3+ P+	S4+ P+
17	X S2+	S+	S+	S+ P+	S4+ P3+	S3+ P3+	S3+ P3+	S4+ P2+	S4+ P4+
18	S3+	X	X S+	P+	P+	X S4+ P3+	X S3+ P3+	S4+ P3+	S5+ P4+
19	S2+	P+	—	—	P+	X S4+ P3+	S3+ P4+	S5+ P4+	S5+ P4+
20	S2+ P+	P+	P+	—	P+	X S4+ P4+	S4+ P4+	S5+ P5+	S5+ P5+
21	P+	—	P+	—	P+	S3+ P4+	S+ P4+	S5+ P5+	S5+ P5+
22	P+	—	P+	—	—	S3+ P3+	S+ P3+	S5+ P5+	S5+ P5+
23	P+	—	P+	—	—	S2+ P3+	P3+	S5+ P5+	S5+ P5+
24	—	—	—	—	—	S+ P3+	P2+	S5+ P5+	S5+ P5+
25	—	—	—	—	—	P+	P+	—	—

X, treatment with aureomycin; S2+, schizonts 2+ present in smears; P+, haemotropic parasites present; + very rare; 2+, rare; 3+, fairly frequent; 4+, frequent; 5+, numerous.

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During the last two decades it has been established that in certain enzootic or potential enzootic East Coast fever areas of South Africa (du Toit, Graf and Bekker, 1941) strains of the important vector of anaplasmosis, *Boophilus decoloratus* (Koch) have become arsenic resistant. This observation directed the attention of biologists and chemists to the control of ticks by means of other acaricides. Du Toit *et al.* (1941) successfully controlled the arsenic-resistant *B. decoloratus* strain by the addition of 40 per cent nicotine (as sulphate) in the proportion of 1:1,000 to the arsenical dipping fluid. Bekker (1942) states that nicotine remains remarkably stable in the presence of arsenic but is rapidly destroyed in its absence.

Bekker and Graf (1946) demonstrated that D.D.T. in a concentration of 0·1 to 0·2 per cent para-para D.D.T. plus 0·16 per cent As_2O_3 is a valuable agent for the destruction of the arsenic-resistant *B. decoloratus* strain. D.D.T. alone in dipping tanks is prone to undergo changes which reduces its acaricidal value. Bekker, Graf, Malan and v. d. Merwe (1949) state that at a strength of 0·5 per cent D.D.T. together with 0·04 per cent As_2O_3 (as sodium arsenite) as a preservative for the emulsion, gives complete tick control. The use of D.D.T. at this concentration is, however, uneconomical under South African conditions, and will, therefore, not readily be employed by stockowners.

The gamma isomere of benzene hexachloride (B.H.C.) in a concentration of 0·005 to 0·01 per cent plus seven day arsenical strength (0·16 per cent As_2O_3) is very effective for the control of the arsenic-resistant *B. decoloratus* strain and other ticks. Owing to the relative instability of B.H.C. formulations in dipping tanks, considerably higher concentrations—up to 0·05 per cent—are required if arsenic is absent (Bekker and Graf, 1946). However, in 1948 it was found by Whitnall, Bradford, McHardy, Whitehead and Meerholz (1949) and Bekker *et al.* (1949) that the arsenic resistant strain of *B. decoloratus* had also become B.H.C.-resistant.

In South Africa it was found by Thornburn (1952) that toxaphene is effective against the non-resistant strain of *B. decoloratus* in a concentration of 0·25 per cent, but less effective against the arsenic-resistant strain. Toxaphene used in dipping tanks also has shown indications of losing its biological efficiency as the dipwashes become progressively fouled (Thornburn, 1952). This compound is more toxic to man and animals than is either B.H.C. or D.D.T.

The deterioration of D.D.T., B.H.C. and toxaphene formulations in dipping tanks is a great drawback. For this reason Bekker and Graf (1952) insist that in dipping tanks they should be used only as additions to seven day arsenical strengths. Mönnig (1950) and du Toit (1951) in South Africa recommend that the formulations of the above-mentioned synthetic acaricides be applied in power-operated spray races. They state that the application of freshly prepared formulations by this process is very effective and economical, but this method of application must be regarded as being in the experimental stage (Graf and Bekker, 1952).

In conclusion it should be stated that Alexander (1955) is of opinion that in the control of ticks the benefits that have accrued from the development of the method of using arsenical dip washes in plunge-type dipping tanks can never be estimated but that little has been gained, if anything, from the advent of the new synthetic acaricides, probably because the knowledge of their mode of action is so very incomplete. In actual practice it has been found that the introduction of the new acaricides has coincided with the loss of control of ticks followed naturally by an increased incidence of tick-borne diseases. He (Alexander *loc. cit.*) expresses the hope that further research will rectify the damage that has been done.

Consideration of the above-mentioned facts makes it apparent that Watkins-Pitchford's contribution to the control of ticks has been and still is an effective weapon for the destruction of the transmitters of East Coast fever even though it may not be efficient in controlling the important vector, *B. decoloratus*, of redwater and gallsickness.

(2) *Quarantine measures.*—The danger of introducing East Coast fever into areas free from the disease is fully realized. After an outbreak of East Coast fever the infected and the incontact farms are quarantined. The efficacy of this procedure is dependent on several factors. It is essential that quarantined farms be fenced and that straying or illegal movement of stock be prevented. The source of infection must also be determined. It is self-evident that systematic dipping be persevered with in order to eradicate the infected ticks. Blood, spleen and lymph gland smears, particularly from dead animals, must be submitted to the veterinary authorities so that the cause of death can be determined. Farms are released from quarantine 15 to 18 months after the last death from East Coast fever.

Although these procedures have been applied successfully in most instances, Sinclair (1914) and Diesel and van Drimmelen (1948) state that a recrudescence of East Coast fever in old centres of infection has occurred after a lapse of periods varying from 2 to 13 years. In these cases accidental introduction of East Coast fever could be excluded with certainty.

The reason for the sudden reappearance of the disease is still obscure and several theories have been advanced. It has been suggested that recovered animals may serve as reservoirs for the infection of ticks. Theiler (1921) refuted this assumption after having considered the epizootology very carefully. Du Toit (1928), Walker and Whitworth (1929), and Neitz (1948) concluded from their splenectomy experiments on recovered East Coast fever cattle that a sterile immunity exists, and hence such animals cannot serve as reservoirs for the infection of ticks. These observations fully support Theiler's views.

Another theory about the maintenance of the infection in nature is that partially immune cattle on reinfestation with infective ticks develop a mild form of the disease accompanied by a microscopic infection of *Th. parva* endoglobular parasites, and thus serve as temporary reservoirs. Such a possibility needs to be considered carefully as Lewis (1950) has shown in his experiments that *Th. parva* may become attenuated within the vector when infected ticks are exposed to high temperatures during the full, but short, period of moulting or alternatively when such ticks are starved for long periods after moulting (*vide supra*). Should conditions in nature be favourable for the attenuation of *Th. parva*, a high percentage of recoveries from East Coast fever can then be expected. If the cyclical development of *Th. parva* in the partially immune vertebrate, and in the invertebrate host is accepted as a reason for the maintenance of the infection in nature, it stands to reason that it must also be accepted that infected ticks feed selectively on either immune or partially immune animals over a period of several years. It is difficult to conceive that infected ticks are endowed with such an instinct. The inference is drawn that even though partially immune animals on reinfection may serve as the source of infection, they cannot be accused of exclusively maintaining the infectious agent for periods of up to 13 years before a fresh outbreak occurs. The source of infection still needs to be determined.

Infected farms are also quarantined when the "slaughter-out" policy is applied. In these circumstances cattle are removed from infected into clean paddocks through a series of camps in which they are retained for 21 days. During this period the temperatures of the animals are recorded at certain times and those showing a thermal reaction are destroyed, and the remainder dipped before removal to an abattoir. The farm may then be restocked when an interval of 15 to 18 months has elapsed. A recrudescence of the disease on such farms has not yet been recorded (Diesel, 1956; Daly, 1956).

(3) *Immunization.*—This prophylactic measure was adopted on a large scale, but for a short period, in the Transkei. (Theiler, 1912; Wölfel, 1912; Spreull, 1914). According to Spreull (*loc. cit.*) a total of 280,000 head of cattle was subjected to the immunization process which consisted of artificially infecting animals by the intravenous administration of suspensions prepared from spleen and lymphatic glands of affected East Coast fever animals. The results were not satisfactory in that approximately 25 per cent of treated animals died from the disease, and of the survivors about 70 per cent proved to be completely or partially immune. This prophylactic measure was discontinued when it became evident that systematic dipping was more effective, and if persevered with would eventually eradicate all infected ticks. The immunization process was not only a costly method but it was also responsible for creating a large number of reservoirs on which ticks could infect themselves.

Immunity.

Naturally recovered animals develop a durable and sterile immunity (du Toit, 1928, 1931; Neitz, 1948). Its duration, however, varies a great deal.

According to du Toit (1931) naturally recovered animals in the absence of infected ticks, *never* show a breakdown in immunity with reappearance of the parasite. Du Toit (1928) tried in many ways to break down the immunity but never succeeded. Attempts to determine whether the infection could ever be carried from immune to susceptible cattle under natural conditions i.e. in the presence of numerous ticks, never resulted in a transmission. The recovered animals can thus not serve as a reservoir.

At one time it was believed that the immunity in East Coast fever is solid and permanent (Richardson, 1930). Du Toit (1931) also stated that the immunity is "solid and sterile" but that in only very few cases do recovered animals show a second infection when exposed to tick infestation. Piercy (1947), in his review on the immunity to East Coast fever, refers to several observations made in South and East Africa. Theiler (1917) noticed that "salted" oxen on re-exposure to infected ticks may show a breakdown in immunity. Purvis (1937) believes that immunity to East Coast fever depends on constant re-exposure to infected ticks, and that its duration in young animals is a matter of a few weeks only in the absence of reinfestation. Walker (1925, 1926), on the other hand, describes an experiment wherein ten adult cattle, which had recovered from natural infection, were subsequently re-exposed after living under tick-free conditions for considerable periods. Only one showed a reaction and it recovered. Daubney (1932) carried out a similar trial with eight adult animals, and found that none died on re-exposure, although three showed mild reactions, Koch bodies being absent in smears. Piercy (1947) records an instance where a naturally recovered Ayrshire bull calf died approximately two years later from East Coast fever. Smear examination revealed a large number of Koch bodies and erythrocytic parasites.

The writer has challenged the immunity of ten recovered cattle by infesting their ears with large numbers (150-250) of infective *Rh. appendiculatus* nymphae. The animals had been maintained under conditions free from infective ticks for periods varying from one to five years. With one exception all animals developed a swelling of the parotid lymphatic glands, sometimes accompanied by a swelling of the remaining superficial glands. A relatively mild fever (104°-105°F) which persisted for four to seven days was observed in three of these animals. Examination of the swollen parotid lymphatic glands invariably revealed a relatively small number of Koch bodies but schizonts were not always found in the remaining superficial glands. The febrile reaction in the three animals was accompanied by the presence of erythrocytic parasites in the peripheral blood, thus showing that the degree of immunity had dropped to such a level that the Koch bodies were capable of completing their life-cycle in the vertebrate host. These three animals had recovered either one, two-and-a-half or four years previously from a natural sporozoite induced infection. The latter two animals were capable of infecting *Rh. appendiculatus* larvae which in the ensuing stage transmitted typical East Coast fever reactions to susceptible calves.

Consideration of the above-mentioned observations permits the conclusion that a good immunity develops in East Coast fever recovered animals which may persist for periods of up to five years—limit of the observation. It may commence to wane as early as one year after recovery but the residual immunity is sufficient to protect against a fatal infection under laboratory and field conditions. However, in some cases the immunity may disappear completely rendering the animal fully susceptible as early as two years after recovery from a natural infection.

The immunity following an artificial infection may be complete or partial (Theiler, 1912; Walker, 1926; Neitz, 1943, 1946).

Animals which have recovered from a natural infection are fully susceptible to benign bovine gonderiosis (Neitz, 1956), tropical gonderiosis (Sergent, Donatien, Parrot and Lestoquard, 1945; Neitz and Jansen, 1956). However, a complete or partial cross-immunity exists between East Coast fever and Corridor disease (Neitz, 1956), and *G. bovis* infection (Lawrence, 1939).

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2. GONDERIA ANNULATA INFECTION.

Definition.

Tropical gonderiosis is a peracute, acute, subacute or chronic disease of cattle caused by *Gonderia annulata* (Dschunkowsky and Luhs, 1904). It is characterised by pyrexia, malaise, anorexia, anaemia, icterus, haemoglobinuria in some cases, digestive disturbances, dyspnoea, swelling of the superficial and internal lymphatic glands, tumor splenis, tumor hepatis, " lymphomata " in the kidneys, and a multiple, localized ulcerative abomasitis. Recovered animals develop a durable premunity.

Synonyms.

Tropical piroplasmosis, Tropical theileriosis, Egyptian fever, Mediterranean Coast fever; Tropiese piroplasmose, Tropiese theileriose, Tropiese gonderiose, Kwaadaardige gonderiose (Afrikaans); Tropiesche piroplasmose, Tropiesche theileriose, Tropiesche gonderiose (Netherlands); Tropische Piroplasmose, Tropische Theileriose, Tropische Gonderiose (German); Theiléríose a *Theileria dispar*, Theiléríose a *Theileria annulata*, Gonderiose a *Gonderia annulata* (French); Febbre della costa mediterranea (Italian).

History.

Dschunkowsky and Luhs (1903) encountered a fatal disease in Transcaucasian cattle which they named " Tropical Piroplasmosis ". Examination of blood smears revealed endoglobular parasites resembling but not identical with those seen by Koch (1897) in East Coast fever. A striking morphological difference was observed. The erythrocytic stages of the East Coast fever parasite are mainly rod-shaped, while those of tropical piroplasmosis are mostly round or oval.

Dschunkowsky and Luhs (1904) named the parasite of the latter disease *Piroplasma annulatum*. Further investigations also revealed the presence of schizonts in smears prepared from various organs (Dschunkowsky and Luhs, 1909). The first attempts of transmitting the disease artificially failed. This mode of transmission, however, was successful when Dschunkowsky and Luhs used cattle derived from non-enzootic areas (Tartatowsky, 1905).

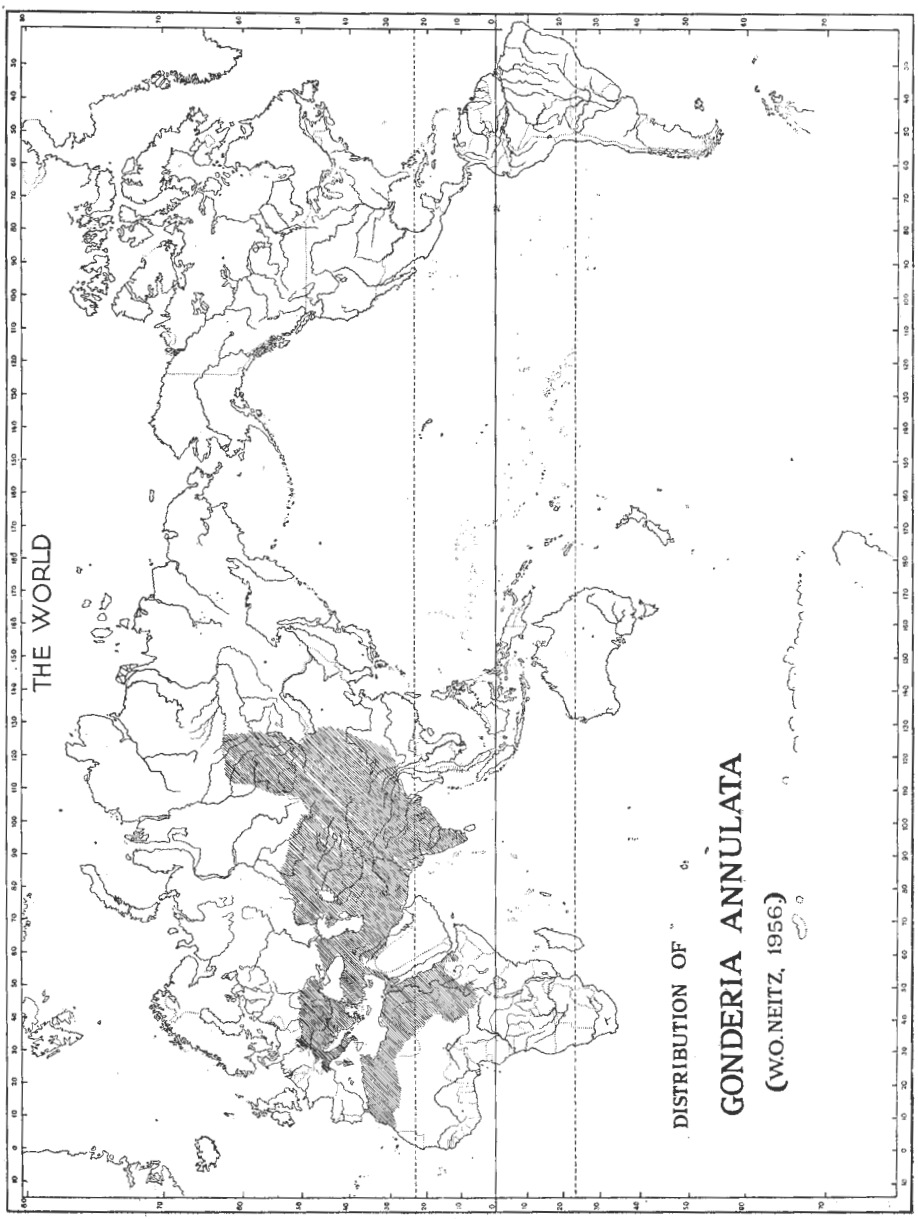
Bettencourt, Franca and Borges (1907) compared the life-cycle and morphology of *Piroplasma bigeminum* Smith and Kilborne, 1893, with that of *P. annulatum*, and concluded that the presence of schizonts in the developmental cycle of the latter protozoon justified its removal from the genus *Piroplasma* and placed it in the genus *Theileria*. It was thereupon named *Theileria annulata*.

Investigations in North Africa (Bitter, 1905; Ducloux, 1905; Balfour, 1908; Littlewood, 1915, 1916; Pricolo, 1914, 1915; Carpano, 1912, 1915; Velu and Eyraud, 1915; Brumpt, 1923, 1924); Asia (Baldrey, 1910; Yakimoff, Schokhor, Koselkine and Paroisky, 1917; Stefko, (1917) and Europe (Cardamatis, 1912; Carpano, 1915; Behn, 1919) showed that an unidentified type of theileriosis was widely distributed on these three continents. As nothing was known about its biological transmission and its immunogenic relationship to the diseases caused by either *Th. annulata* or *Th. parva*, a great deal of speculation arose as regards the identity of the infectious agent. Some investigators considered the causal agent to be *Th. annulata*, while others believed that it was *Th. parva*. However, it soon became apparent that the latter parasite was not concerned in the aetiology. Sergent, Donatien, Parrot, Lestoquard, Plantureux and Rougebief (1924) concluded from their studies that the Algerian *Theileria* sp. is a distinct parasite, and named it *Theileria dispar*. For many years *Th. annulata* and *Th. dispar* were regarded as distinct species (du Toit, 1930; Sergent, Donatien, Parrot and Lestoquard, 1937). Oboldoueff and Galouzo (1928) considered the protozoon responsible for theileriosis in Turkestan to be a distinct species, and named it *Theileria turkestanica*. Yakimoff and Dekhtereff (1930) described *Theileria sergenti* as a new species responsible for theileriosis in East Asia.

A great deal of controversy arose as regards the validity of *Theileria* spp. described from Transcaucasia, Algeria, Turkestan and East Asia. It became apparent from the cross-immunity tests conducted by Sergent, Donatien, Parrot and Lestoquard (1937), Sergent, Parrot, Lestoquard and Delpy (1939), Adler and Ellenbogen (1935, 1936) and Yakimoff, Gusev, Pelevin and Monechikova (1940) that an immunogenic relationship exists between *Th. dispar* and the *Th. annulata* strains occurring in Russia and Asia Minor. Furthermore it was also determined that the biological transmission of these protozoa is effected by various *Hyalomma* spp. Consideration of these facts together with the epizootology caused Dschunkowsky (1948), Delpy (1949) and Richardson (1939) to conclude that there was no justification for retaining all these species as distinct entities. By following the nomenclatorial rules they decided that *Th. annulata* is the correct name and that *Th. dispar, Th. turkestanica* and *Th. sergenti* are synonyms. Sergent, Donatien, Parrot and Lestoquard (1945) admit their identity, but claim that the name *Th. dispar* be adopted, as the description of *Th. annulata* by Dschunkowsky and Luhs (1904) was in fact a description of a mixed infection of a *Babesia* sp. and a *Theileria* sp.

Recently Neitz and Jansen (1956) revised the classification of the Theilerias. In order to avoid unnecessary confusion in the nomenclature, they redefined and reinstated the generic name *Gonderia* to include parasites which multiply by

schizogony in the lymphocytes, and by binary fission in the erythrocytes. This genus was placed in the family Gonderidae Neitz and Jansen, 1955. Since the life-cycle of *Th. annulata* is typically that of a *Gonderia* sp., they renamed the protozoon *Gonderia annulata* (Dschunkowsky and Luhs, 1904). In the family Theileridae du Toit, 1918, they retained a single genus and a single species *Theileria parva,* which only multiplies by schizogony in the lymphocytes and not by fission in the erythrocytes. (See Map No. 2).



MAP No. 2.

Distribution.

The distribution of *G. annulata* is given in Table VII.

TABLE VII.

Continent.	Country.	References.
Africa.....	Algiers.....	Donatien, Plantureux, Rossi and Esperandieu, 1923; Sergent. Donatien, Parrot, Lestoquard, Plantureux and Rougebief, 1924.
	Egypt.....	Piot-Bey, 1903; Bitter, 1905; Dreyer, 1910; Littlewood, 1915, 1916; Mason, 1922; Daubney and Sami Said, 1951.
	Eritrea.....	Carpano, 1912.
	Lybia.....	Carpano, 1915.
	Morocco.....	Velu and Eyraud, 1915; Velu, 1921, 1923; Grimpet, 1937, 1938.
	Sudan.....	Balfour, 1908; Littlewood, 1915, 1916; Mason, 1922; Bennet, 1930, 1931.
	Tripolis.....	Pricolo, 1914, 1915, 1923.
	Tunis.....	Ducloux, 1905; Pricolo, 1914, 1915, 1923; Brumpt, 1923, 1924.
Asia.....	Central Asia.	Oboldoueff and Galouzo, 1928.
	East Siberia..	Yakimoff and Dekhtereff, 1930.
	East Asia....	Springholz-Schmidt, 1938.
	India.....	Baldrey, 1910; Edwards, 1925; Cooper, 1926; Ajwani and Subbarayudu, 1934; Sen and Srinivasan, 1937; Datta, 1940; Raghavachari, 1944.
	Iran.....	Delpy, 1946, 1949.
	Iraq.....	MacHattie, 1935.
	Israel.....	Adler and Ellenbogen, 1934, 1935, 1936.
	Kazakstan...	Tselishcheva, 1940.
	Palestine.....	Doyle, 1924; Gilbert, 1927; Ashbel, 1935; Sturman, 1935.
	Turkestan....	Yakimoff, Schokhor, Koselkine and Paroisky, 1917.
	Turkey.....	Stefko, 1917; Schern, Mavrides and Major, 1920; Tüdzil, 1946.
	Uzbekistan...	Lavrent 'ev, 1938.
Europe.....	Bulgaria.....	Angeloff, 1921; Tomoff, 1938; Pavlov, 1942, 1948/49.
	Cyprus.....	Doyle, 1929.
	Greece.....	Cardamatis, 1912; Cardassis, 1956.
	Italy.....	Carpano, 1915; Dini, 1934; Mannuci, 1932.
	Macedonia...	Behn, 1919; Mlinac, Petrovic and Babouder, 1934; Yaneff, 1942.
	Roumania...	Iriminoiu, 1948; Metianu, 1950.
	Sardinia.....	Cerruti, 1934.
	Transcaucasia	Dschunkowsky and Luhs, 1903, 1904; Tartatowsky, 1905.
	Yugoslavia...	Mlinac and Romic, 1948.

Aetiology.

Gonderia annulata (Dschunkowsky and Luhs, 1904).

Synonyms:—*Piroplasma annulatum* Dschunkowsky and Luhs, 1904.
Theileria annulata (Dschunkowsky and Luhs, 1904).
Theileria dispar, Sergent, Donatien, Parrot, Lestoquard, Plantureux and Rougebief, 1924.
Theileria sergenti Yakimoff and Dektereff, 1930.
Theileria turkestanica Oboldoueff and Galouzo, 1928.

(*a*) *Morphology.*—(i) Erythrocytic parasites:—In blood smears fixed with May-Grünwald and stained with Giemsa, *G. annulata* appears in the red blood cells as round, oval, comma-shaped or anaplasma-like organisms. The round forms have a diameter varying from 0·5 to 1·5 microns; oval forms are 0·6 micron in width and 2·0 microns in length; comma-shaped forms 0·5 micron in width and 1·6 microns in length and anaplasma-like forms 0·5 micron in diameter.

Approximately 50 per cent of the parasites are round, 0·5 per cent have an anaplasma-like appearance, while the remainder are either oval or comma-shaped.

The cytoplasm stains light blue. The nucleus appears as a deeply stained minute reddish purple granule situated at the wider end of the oval or comma-shaped forms, and on the margin of the round parasites. When division takes place two, three or four chromatin granules are observed. In the anaplasma-like forms the cytoplasm can hardly be recognized.

(ii) Histiotropic parasites:—In liver, spleen and lymphatic gland smears fixed with May-Grünwald and stained with Giemsa, the schizonts (Koch bodies, corps en grenade) appear as masses of blue staining cytoplasm containing one to eighty reddish purple dots. Koch bodies vary in size from 1·0 to 15·0 microns, and in some cases they may be up to 27·0 microns in diameter. The average size is 8·0 microns. They are seen either free or within the lymphocytes. Sergent, Donatien, Parrot and Lestoquard (1945) state that they may also parasitize monocytes. Two types of schizonts commonly referred to as agamonts (macroschizonts) and gamonts (microschizonts) are usually readily demonstrable. The former harbour chromatin granules varying from 0·4 to 1·9 microns (average 1·2 microns), while the latter contain granules varying from 0·3 to 0·8 microns (average 0·5 microns). When fully formed the macroschizonts liberate macromerozoites varying from 2·0 to 2·5 microns in diameter. The mature microschizonts liberate micromerozoites 0·7 to 1·0 micron in diameter when round. Some of the forms are ovoid in shape, while others are rod-like, pear-shaped or comma-shaped.

(*b*) *Multiplication.—G. annulata* multiplies by schizogony. When schizonts are fully formed they break up into merozoites which either enter lymphocytes to grow and reproduce by schizogony again, and they penetrate the erythrocytes in which they are seen in ordinary blood films. Multiplication also occurs within the erythrocytes. Division into two, giving rise to two daughter cells (du Toit, 1918), or alternatively into four takes place (Sergent, Donatien, Parrot and Lestoquard, 1945) resulting in cross forms, in which four minute pear-shaped individuals radiate from a central point.

(*c*) *Habitat.*—The erythrocytic stages of *G. annulata* can be demonstrated readily in blood smears for periods of up to eight weeks after recovery. In acute cases of the disease up to 95 per cent of the erythrocytes may harbour parasites. The host cell may contain 1 to 10 organisms. In the chronic form 10 to 40 per cent of the red blood cells may be parasitized. In premune animals it is usually difficult to demonstrate the infectious agent. Splenectomy of such animals is followed by a relapse within three weeks after the operation. Smear examination reveals that more than 25 per cent of red blood cells become parasitized, and a microscopic infection persists for very long periods.

Schizonts can be demonstrated readily in the liver, spleen and lymphatic gland smears and may also appear in fairly large numbers in the peripheral blood during the course of the reaction. They are found mostly in the lymphocytes but according to Sergent, Donatien, Parrot and Lestoquard (1945) Koch bodies also develop in the cells of the reticuloendothelial system.

(*d*) *Life-cycle.*—The life-cycle of *G. annulata* (= *Theileria dispar*) has been studied by Sergent, Donatien, Parrot and Lestoquard (1936) in the two host tick, *Hyalomma mauretanicum* (= *Hyalomma detritum*). They are of opinion that schizogony in the vertebrate host is followed by sporogony in the invertebrate host. The infection is acquired by the immature stages, and transmitted by the ensuing adults.

When the endoglobular parasites are ingested by the larvae they escape from the erythrocytes, and congregate in masses in the intestine. It is believed that a process of syngamy takes place within the intestine 13 hours after tick attachment. Zygotes appear in the epithelial cells of the gut seven hours later. They become encysted during the period of the larval moult. After moulting the zygotes emerge from the cysts, and develop into ookinetes which enter the body cavity. From there they migrate to the salivary glands and invade the cells of these organs. After penetration the next stage is referred to as a beginning " sporont ". Before and during the nymphal moulting period, the sporonts develop into multinucleate masses, the sporoblasts. When the adult tick attaches itself to a new host and commences to suck blood, the sporoblasts break up into numerous uninucleate sporozoites which enter the salivary ducts, and are injected into the new host. This process of sporozoite production from the sporoblasts takes 72 to 96 hours, so that transmission is not effected for three of four days after the attachment of the vector. In a later report Sergent, Donatien, Parrot and Lestoquard (1936) state that the infection may be given off 60 hours after tick attachment. Sporozoites entering the lymph stream migrate to the regional lymphatic gland, and invade lymphocytes in which reproduction takes place.

When the ears of susceptible cattle are infested with *G. annulata* infective ticks, schizonts appear in the parotid lymphatic glands 7 to 18 days later. The thermal reaction which commences after two or three days signifies that an invasion of the blood by schizonts has taken place. Koch bodies can then also be demonstrated 24 hours later in the other superficial lymphatic glands. The endoglobular parasites appear three to four days after the initial rise in temperature. In recovered animals the erythrocytic parasites persist throughout life. Sergent *et al.* (1945) have established that the immature stages of *H. mauretanicum* were capable of infecting themselves when they were allowed to feed on a premune animal which had recovered from tropical gonderiosis eleven years previously.

(*e*) *Cultivation.*—Tchernomoretz (1945) succeeded in growing Koch bodies of *G. annulata* in lymphocytes cultivated in tissue culture.

(*f*) *Action of physical and chemical agents.*—Adler and Ellenbogen (1935) found that defribinated blood kept at 12° C proved to be infective for at least 19 days. Sen and Srinivasan (1937) established that when infective defibrinated blood was stored at room temperature (21°—22° C) it remained viable for four days and when kept in a refrigerator it lost its infectivity within six days. Sergent *et al.* (1945) found that when citrated or defibrinated blood is stored at temperatures varying from 0° to 25° C, *G. annulata* remained viable for periods of up to nine days. They recommend that infective citrated blood for the immunization of cattle be administered within three days after issue. The infectious agent remains viable for this period even when it is exposed to ambient temperatures in North Africa.

(*g*) *Biological characteristics.*—Experimental and field observations have shown that there is a wide variation in the virulence between the various *G. annulata* strains. Not only does this difference exist between strains of different countries but it has also been observed between strains isolated from the same enzootic region. Sergent *et al.* (1945) and Rampon (1948) established that the " Jacquot ", " Brunette " and " Kouba " strains isolated in Algeria produced a mortality rate of 50, 13 and 3 per cent respectively. Yakimoff, Gusev, Pelevin and Monechikova (1940) state that the Algerian strains are less virulent than the Russian strains. The average mortality in the North African cattle is 30 per cent,

TABLE VIII.

The Biological Transmission of *Gonderia annulata*.

Vector.	Country.	No. of Hosts.	L.	N.	I.	E.	L.	N.	I.	References.
Hyalomma detritum Schulze Syn. (*Hyalomma mauretanicum* Senevet)	North Africa.....	2	X—	—X—	—)					Sergent, Donatien, Parrot and Lestoquard (1928).
	Russia.....		X—	—X—	—)					Galuzo (1935).
	Kazakstan.....		X—	—X—	—)					Galuzo and Bespalov (1935). Tselishcheva (1940).
Hyalomma dromedarii Koch Syn: (*Hyalomma dromedarii asiaticum* Schulze and Schlottke, according to Delpy, 1949. = *Hyalomma dromedarii* Koch, according to Feldman-Mühsam 1954).	Central Asia.....	2 or 3	X—	—X—	—)					Galuzo (1934).
			X—	—X—	—)					Tselishcheva (1940).
Hyalomma excavatum Koch Syn. (*Hyalomma anatolicum* Koch, *Hyalomma turkmeniense* Olenev, according to Delpy, 1949 and Feldman-Mühsam, 1954)	Asia Minor.....	3	X—	—)						Delpy (1949).
				X—	—)					
					X—	——	——	——	—)	Kornienko and Shmyreva (1944).
Hyalomma impressum near *planum* Lewis (= ?*Hyalomma transiens* Schulze according to G. Theiler = *Hyalomma truncatum* Koch according to Feldman-Mühsam, 1954)	East Africa (Laboratory observations)	2 or 3		X—	—)					Fotheringham and Lewis (1936).
Hyalomma rufipes glabrum Delpy. (= *Hyalomma turanicum* Pomerantzer)	Asia Minor.....	2	X—	—X—	—)					Delpy (1949).
Hyalomma savignyi (Gervais)..... (= *Hyalomma marginatum* Koch according to Feldman-Mühsam, 1954)	Asia Minor.....	2 or 3		X—	—)					Delpy (1949)
	Kazakstan.....			X—	—)					Tselishcheva (1940).
Hyalomma savignyi (Gervais)—Referred to originally as *Hyalomma aegyptium* Neum. but according to Ray (1950) identified by Adler and Feldman-Mühsam as *Hyalomma savignyi* (Gervais)	India (Hereditary transmission through four generations)	2 or 3			X—	——	——	——	—)	Ray (1940–41). Raghavachari, Shah and Ray (1944).
					X—	——	——	——	—)—	
			——	——	——	——	——	——	—)—	Ray (1950).
			——	——	—)					

TABLE IX.

Domestic and Wild Members of the Family Bovidae Susceptible to *Gonderia annulata*.

Host.		Country.	Observations.	References.
Vernacular Name.	Zoological Name.			
Cattle (European, Asiatic and African breeds including the Zebu cattle	*Bos* spp.....	North Africa, Southern Europe and Asia	Naturally and artificially infected...	Dschunkowsky and Luhs (1904, 1909); Knuth and du Toit (1921); Sergent *et al.* (1945); Cordier, Ménager and Delorme (1936); Yakimoff, Gouseff and Nezwetaieff (1932).
Water Buffalo.....	*Bubalus bubalis* Linn.....	Algeria, Egypt.....	Naturally and artificially infected..	Sergent *et al.* (1945); Mason (1922).
American Bison.....	*Bison bison* Linn.....	Egypt (Zoological Gardens, Cairo).	Naturally infected and died.....	Carpano (1937).