

RESEARCH ARTICLE

History of Trypanosomosis in the One-Humped Camel and Development of its Treatment and Cure, with Special Reference to Sudan

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Abstract

Sudan has one of the largest populations of domestic animals in Africa. One-humped camel (*Camelus dromedarius*) numbers were estimated at 4.5 million in 2009. Once used extensively for military transport they are still used in the transport role by spatially mobile pastoralist households and are a major source of milk and meat for these people. Trypanosomosis, due to *Trypanosoma evansi*, generally known as 'surra' but as 'gufar' in Sudan was first identified in camels in the country in 1902 and is the main cause of disease although *T. vivax* infections have recently been discovered in parts of Sudan. This protozoan disease is the most important health problem in camels, causing high morbidity and huge production losses. The causal organism, unlike most other trypanosomes, is not transmitted cyclically with tsetse (genus *Glossina*) flies as the vector but mechanically by biting flies mainly family Tabanidae but also by others of the Muscidae. Identification of the parasite in camel blood was initially by simple microscopic techniques but biotechnology and molecular methods now enable infection to be diagnosed at an earlier stage and with more accuracy. Prophylactic and curative treatments of trypanosomosis are notoriously complicated and uncertain with the situation in camels being exacerbated because of its peculiar physiology. Many trypanocides have been developed over time but the parasite often develops resistance to these drugs. Some drugs are successful, for some time, as both prophylactics and cures but are often accompanied by undesirable side effects. Other drugs used on conventional domestic stock are ineffective in camels or have lower efficacy. Research on diagnosis and treatment of trypanosomosis is continuing but the disease continues to cause production losses to the detriment of national and household incomes and food security.

Keywords: *Trypanosoma evansi*; *Trypanosoma vivax*; *Camelus dromedarius*; 'surra'; prophylaxis; trypanocides

1. Introduction

A very large and species-diverse array of domestic animals inhabit the Republic of Sudan. According to estimates by the Food and Agriculture Organization (FAO), the Republic of Sudan – since its partition with the Republic of South Sudan in 2011 – has the third largest total livestock population of all African countries. The country ranks second (in this case the one-humped camel *Camelus dromedarius*) in camel numbers and first to third in cattle, sheep, goat and poultry numbers and fifth in donkeys.¹ Official Sudanese data for 2009 for the then northern states of the country indicate national livestock populations of 41.65 million cattle, 51.55 million sheep, 43.27 million goats, 4.52 million camels, 7.51 million donkeys and 784 thousand horses.²

For a considerable time in the late nineteenth and early twentieth centuries the Sudanese veterinary services were mainly staffed by officers of the British Army Royal Army Veterinary Corps.³ This was the case because the country, recently conquered by a joint Egyptian-British expedition, was under military law. Vast numbers of transport and riding animals were required to control the country and to ensure supplies of goods and services. Camels were then extremely important in military operations.⁴ No longer employed by the military, camels continue to have a significant presence in transport operations and in milk and meat production. They contribute to the national economy as earners of foreign exchange when exported live for slaughter to Egypt and Saudi Arabia and as racing animals (Annafi and Bishari breeds) to Saudi Arabia and several Gulf States.^{5,6} They are mainly owned by pastoral nomads in small to very large herds that move seasonally over long distances in search of food and water but fewer animals in smaller units are increasingly being kept by

smallholder mixed farmers and in some suburban and urban areas.

The aim of this paper is to provide details of the occurrence and status of trypanosomosis usually caused in camels by *Trypanosoma evansi* but more recently also by *Trypanosoma vivax* transmitted mechanically by biting flies over the 120-year period from 1902 to 2021. In order to do this a thorough literature view has been undertaken with a focus on the Republic of Sudan but the scope is wider than that as literature on the disease in other countries has also been studied.

2. Camel trypanosomosis

The most common cause of trypanosomosis in camels is infection by *Trypanosoma evansi* but there is a recent report of infection by *T. vivax* in the east of the country.⁷ In addition to camels this protozoan parasite causes trypanosomosis – which is also widely known as ‘surra’ and more narrowly in Sudan as ‘gufar’ – in equines – known as ‘souma’ in Sudan – and dogs and to a lesser extent cattle, domestic buffalo and elephants. Horses and dogs are severely affected and there is high mortality in these species. Full accounts of the parasite and the disease have been published in other sources.^{8,9}

The parasite was first described from a horse in an obscure report – frequently cited but almost certainly rarely consulted – by an Indian Government pathologist in 1880.¹⁰ The report was later expanded into a more detailed account in a scientific journal.¹¹ The taxonomy of the parasite has always been somewhat complicated and several names were used for the non-tsetse transmitted *Trypanosoma (Trypanozoon) evansi* – it was for long nominally *T. brucei* or *T. soudanense* in Sudan – until it was standardized as *T. evansi* in the 1950s.¹²

In Africa but also elsewhere in the world, trypanosomosis due to *T. evansi* is the most important protozoal disease of camels. The biology of infection by this parasite differs from the trypanosomosis transmitted by blood-sucking tsetse flies of the genus *Glossina*, which is also known as ‘nagana’, as it is transmitted mechanically by biting flies mainly of the families Tabanidae and Muscidae. Tsetse-transmitted trypanosomosis including *T. vivax* and *T. congolense* is cyclical, requiring a period of maturation in the fly before it can again become infective in the animal host whereas in *T. evansi* there is no development stage in the vector and in some cases *T. vivax* can also be transmitted mechanically by tabanid flies.¹³⁻¹⁵

‘Surra’ is seasonal in its occurrence except in riverine or flooded areas, and is associated with an increase in biting fly numbers during rainy periods. The disease affects all ages of the camel but immature, stressed and lactating animals are more susceptible than other categories of stock.^{16,17}

Many factors related to the host and the environment affect the course of the disease. Among these are previous exposure, immunosuppression by other diseases, age, pregnancy, nutritional status and pre-existing (“underlying”) infections. Following infection, the animal typically loses weight, develops a drooping hump, is inflicted with

diarrhoea (Figure 1, Figure 2, Figure 3), walks slowly and only for short distances, develops muscular atrophy especially in the hindquarters, has pale mucous membranes and may have oedema of the feet, brisket, underside and eyelids (Figure 4). A rough coat may be evident. Intermittent fever is accompanied by hyperlacrimation, inappetance, shade-searching behaviour, diarrhoea, late-term abortion (Figure 5) and weak newborn calves. Anaemia is progressive and body temperature fluctuates. Clinical findings include lowered Packed Cell Volume of 18-16 per cent or less and demonstration of the parasite in Giemsa-stained smears from peripheral blood on glass slides. Although this latter is the most certain method of diagnosis of trypanosome infection a large number of infected animals remain undetected. The Woo test was developed later to concentrate the number of trypanosomes present in the blood stream and consists of examining a wet mount of the buffy coat area of a microhaematocrit capillary tube after centrifugation and looking for the actively moving trypanosomes.¹⁸ An alternative inexpensive but still a very effective method of detecting trypanosomosis in animals consists of inoculation intraperitoneally of mice with whole blood of presumed infected camels and subsequent detection of trypanosomes in mice tail blood.¹⁹



Figure 1: Recumbent camel in late stages of infection (Photo: courtesy of Chris Field)



Figure 2: Adult female camel in final stages of chronic trypanosomosis showing extreme emaciation, non-existent hump, severe muscle atrophy and hollow left flank and retracted abdomen due to anorexia (Source: (https://www.google.com/books/edition/Pictorial_Guide_to_Traditional_Managemen/sZAbOhTtBgkC?hl=en&gbpv=0))



Figure 3: Adult female camel in final stages of chronic trypanosomosis displaying muscle atrophy and persistent diarrhoea (Source: as Figure 2)



Figure 4: Male camel 2.5-3.0 years old with abdominal oedema in the form of a ventral plaque just anterior to the prepuce (Source: as Figure 2)



Figure 5: Late term abortion is a very common symptom in females affected by trypanosomosis (Source: as Figure 2)

Camel trypanosomosis due to *T. evansi* (as does *T. vivax* but in cattle) appears on a list of 117 multiple-species animal diseases, infections and infestations that are notifiable to the World Animal Health Organisation (Office International des Epizooties, OIE).²⁰ It is doubtful, however, that most infected countries regularly report its presence to the OIE. This syndrome, as it affects camels, is a chronic wasting disease with high morbidity whose main economic effects are slower growth rates, late-term abortion, reduced milk production and, where relevant, lowered load carrying capacity or tractive effort in agricultural operations. Owners rarely herd their camels in areas infested with tsetse flies but occasionally in severe drought periods when water availability is a limiting factor they may be forced to do so. Camels are then attacked by *Glossina* flies which result in infection by *T. brucei*, *T. vivax* and *T. congolense*. Such infections result in an acute disease reaction and a rapid death.

3. The disease during the period of the Anglo-Egyptian Condominium, 1898-1955

A formal Veterinary Department was

established in Sudan in 1902. Probably without exception all veterinarians were military personnel of the Army Veterinary Service almost all British (which at that time included Irish) with a sprinkling of Egyptians. Their training and their experience was of a military nature. Some would, indeed, be surgeons in the literal sense as they would have had to put back together animals that had been horribly mutilated in warfare. In Sudan their main concern was to keep the vast numbers of transport animals – oxen, horses, mules, donkeys and camels – fit for work. The diseases they met were of a mechanical nature, mainly saddle and harness sores and poor feet and legs with some relief from these troubles being treatment of colic. None was a scientist or researcher but this was in part overcome by collaboration with the Wellcome Laboratories in Khartoum.²¹ Trypanosomosis was, however, first recorded in 1904 and recognized as a problem only five years after the establishment of the Department in 1907.^{14,22} In 1908 a Dr Wenyon reported deaths from trypanosomosis in a camel caravan in Bahr el Ghazal Province [i.e. outside the normal

range of camels] and again in 1908 the disease had been diagnosed in Kordofan province north of the 12° parallel of latitude, in the whole of the White Nile province and in the area between Suakin and Kassala along the border with Eritrea.¹⁶ In 1914 between 15 per cent and 20 per cent of the camels of the Arab Battalion were infected.¹⁴

By 1915 the Wellcome Tropical Research Laboratories in Khartoum identified the camel parasite as *Trypanosoma soudanense*.²³ Experimental work was being undertaken by Wellcome on the transmission of trypanosomosis and its treatment by arsenic.²³ At this time, however, the arsenical 'atoxyl' (the monosodium salt of para-arsenical acid) was already being recommended for treatment in neighbouring Ethiopia and Eritrea.^{24,25}

In 1913, however, the Sudanese veterinary services had been restructured to form four Sections: Survey, General, Quarantine and Animal Breeding.²⁶ The name of the Survey Section was shortly to be renamed the Research Section but due to reductions in personnel during World War I it confined itself to routine diagnostic work. The war apparently had lesser effect on veterinary work in the neighbouring Italian colony of Eritrea where research had found that the trypanosome of camels there was *T. evansi* and was identical to the parasite found in other parts of North Africa with no appreciable difference in morphology, pathogenicity, transmission by flies other than *Glossina* or clinical symptoms in affected animals.²⁷

It was not until 28 August 1922 that Captain Richard Hall Knowles arrived in Sudan to take up the post of Veterinary Bacteriologist. It was now obvious that if a healthy livestock industry were to be established, adequate research and laboratory

facilities must be provided. With the limited funds at his disposal Knowles began organising a library, replacing equipment and erecting buildings. Rinderpest, Contagious Bovine Pleuro-Pneumonia (CBPP) and camel trypanosomosis were the most urgent problems and, supported by the Acting Director of Veterinary Services, Knowles lost no time in tackling them. He did some "real" research in attempts to produce a vaccine against CBPP. Camels were, however, still a major means of transport and trypanosomosis was their principal health problem. Three of the four papers Knowles produced in the *Journal of Comparative Pathology and Therapeutics* during his tenure until 1927 were on this topic.²⁸⁻³⁰

Knowles' first paper was about the formol-gel test.²⁸ In this test a drop of serum is added to a drop of formalin which, if it coagulates, confirms the presence of increased proteins in the blood. The findings of this experiment were that the test appeared to be reliable for the diagnosis of *T. soudanense* in camels, that its reliability was not affected by the presence or absence of demonstrable trypanosomes in the blood and that both the tube and slide test could be used and although the former was probably more definite the latter was simpler and quicker and require less apparatus. His second paper examined the possibilities of treating and curing trypanosomosis in infected camels.²⁹ In the mid-1920s a satisfactory treatment had not been found. Some success was claimed in India by using repeated intravenous injections of an aqueous solution of tartar emetic and 'soamin' (an organic arsenical compound) but the treatment needed to be prolonged and the results appeared problematic.³¹ In Sudan this treatment had not proved satisfactory. Apparent cures had been obtained by repeated intravenous administration but in most cases the

trypanosome reappeared in the blood. A new drug, Bayer 205 (also then known as 'naganol' or 'germanin' and now 'suramin') was being marketed as a curative for camel trypanosomosis and was put to the test in Sudan in a 10 per cent aqueous solution administered intravenously. No precautions were taken with regard to the rate of administration and no toxic symptoms were seen. Varying amounts of the drug were later administered with a varying number of doses in order to ascertain the smallest dose and the least number of doses required to effect a cure. It was eventually found that, administered at a rate of 12 mg of active ingredient per kg body weight administered intravenously, it was effective both as prophylactic and curative. The valedictory publication by Knowles in 1927, when he had been promoted Major, was essentially a round up of the work carried out by the Department during his short tenure and some of his thoughts on the future.³⁰

The Research Section was greatly strengthened in 1925 by the arrival of S C J Bennett as Assistant Veterinary Research Officer.³² Bennett was a researcher in the true sense of the word and experimental work became much more thorough. In this endeavour research was also given more prominence by the appointment in December 1924 of Mr William Kennedy as Director of Veterinary Services. Kennedy was the first civilian to be appointed to this position as well as being the first director to have had no previous experience of any kind in Sudan. He had, however, been Chief Veterinary Officer in Kenya where he had been largely responsible for containing camel trypanosomosis through a series of Proclamations and by the imposition of quarantines. In Sudan with its still limited staff numbers and restricted mobility (the more widespread use of motor transport from 1928 improved this situation) it was not

possible to control all the disease problems as many outbreaks were in the remote provinces of Kordofan and Darfur.³³

Sampson Charles Jenkin Bennet was the son of a Veterinary Surgeon. He studied at the Royal Veterinary College (RVC) and was elected Member of the Royal College of Veterinary Surgeons (MRCVS) in July 1916. As a Temporary Lieutenant in the Army Veterinary Corps he served in Mesopotamia before working under Sir John M'Fadyean at the RVC Camden Town in 1920-1922 and then with Sir Arnold Theiler at Ondesterpoort. In 1922 he was as one of JT Edwards' team at the Imperial Institute of Veterinary Research at Mukteswar in India. He resigned from his Indian post early in 1925 to take up an appointment with the Egyptian Army to work as Assistant Research Officer in Sudan where he arrived late in 1925. Bennett threw himself into his work, looking at various diseases in several species of domestic animal. He is credited with making spectacular discoveries and providing first records of many diseases in Sudan. On the departure of Knowles from Sudan in March 1927, Bennett was promoted to Veterinary Research Officer in which capacity he served until the beginning of 1936. He designed many experiments for the most serious diseases such as rinderpest (including preparation of spleen pulp vaccine) and CBPP (for which he developed the broth culture vaccine). It was said of him that in addition to being an eminent bacteriologist and pathologist he was also a virologist and protozoologist of the highest order. Bennett was awarded a D.Sc. from London University in 1933. In 1937 he was promoted to Senior Research Officer and Assistant Director of the Sudan Veterinary Service, posts that he held until his retirement in 1944.³⁴

Bennett published 28 research communications, mostly in scientific journals

and mostly with himself as sole author in the *Veterinary Journal* and the *Journal of Comparative Pathology and Therapeutics* (this in part no doubt reflecting his relationship with Sir John M'Fadyean to which reference has already been made) between 1927 and 1948. Four technical reports and a contribution to a chapter in a book about Sudan agriculture were invaluable for the development of Sudan's livestock sector. His work contributed very considerably to improving the health and welfare of domestic animals and to improved livelihoods for Sudan's people.³⁴

Very shortly after his arrival in Sudan, Bennett started to publish a spate of papers mostly on equine diseases and afflictions resulting almost certainly from a quest to discover and describe new diseases. He was also involved in vaccine development and effectiveness against rinderpest in cattle. His first paper on camel trypanosomosis, with a laboratory technician as second author, questioned the efficacy of the formol-gel test as he considered (from some evidence) that some infected animals never showed a positive reaction to the test and proposed the use of the mercuric chloride test for diagnosis of *T. evansi*.³⁵ Although this test was more accurate with positive reactions being referable to relative and absolute increases in the euglobulin of the serum it was found to be a rather delicate test to administer.³⁶ A third paper written together with a bacteriologist from the Wellcome Research Laboratories serve to confirm earlier findings.³⁷ Camel trypanosomosis – its most widespread local name there being 'dukkan' – was first confirmed in British Somaliland in 1926 in both native animals and in those of the Camel Corps. A British veterinarian there was aware of the articles of Knowles and Bennett (and, indeed, visited Bennett in Sudan) and had some success in identifying the disease with

the Mercuric Chloride Test and curing it by the use of 'naganol' administered intravenously initially at 10 g but later at doses lower than this.³⁸ There were further ramifications of the Mercuric Chloride test when it was used to study diagnosis and infection of *Trypanosoma* species in Guinea pigs.³⁹

In the meantime, Bennett had attended a conference in South Africa where he reported that "trypanosomiasis" of camels in the Anglo-Egyptian Sudan was mainly caused by *T. soudanense* but outbreaks due to *T. brucei* and *T. congolense* occasionally occurred and that camels had been infected experimentally with *T. vivax* and with a human strain of *T. rhodesiense* from southern Sudan.⁴⁰ Two papers were published by Bennett in 1933. The first, as recorded by Bennett, was a "record of work carried out in the Sudan since early 1927, the object of which [...was...] to develop a routine system of camel trypanosomiasis control which [...could...] be generally applied in the field."⁴¹ The second paper extols the virtues of the Mercuric Chloride Test and the curative effect of 'naganol' which does not confer permanent immunity but may protect for up to two month with a dose of as little as 4 g.⁴² This theme is again given prominence in the annual report of the Veterinary Department for 1935 in which it is recommended, in order to provide a longer period of immunity, the routine dose in Sudan had been raised to 5 g.⁴³ In what appear to be terminal papers by Bennett, written in collaboration with a specialist in trypanosome studies details are given of the lack of a kinetoplast in *T. evansi*.^{44,45}

Bennett's success was greatly facilitated by the empathy of the Directors of the Veterinary Department during his period of tenure, especially Harold Baskerville

Williams (Director 1934-1940) and Claude Percy Fisher (Director 1940-1944) Prior to and during their tenure the Department had mutated from an essentially supporting service for transport animals to a fully fledged preventative and curative agency not only for transport stock but for the country's ruminant animals which not only provided food for internal consumption but also as an important mediator in exporting animals to generate revenue. Bennett departed Sudan during 1944 at about the same time as Director Fisher who was replaced by Waldo Hearne Glanville (Director, 1944-1952) who had been in the country since 1928. Joshua Timothy Richard Evans was the last of the expatriate Directors, serving in that capacity from 1952 until 1 December 1954 on the eve of Sudan's independence: he had, however been in the country since 1930. Evans was promoted to Senior Research Officer in 1945 on the departure of Bennett and then Assistant Director (Research) of Veterinary Services in 1947 when he also became Commissioner, Animal Trypanosomiasis Control. Having previously been mainly interested in rinderpest, during World War II, Evans became aware of the importance of trypanosomosis although he was mainly concerned with tsetse-born trypanosomes in cattle. He nonetheless continued to support and implement the prophylactic and curative treatment of camels.⁴⁶

Somewhat surprisingly in view of its importance there is very little information on actual losses of camels due to trypanosomosis in official documentation with losses in Rizaygat herds Darfur in 1946-1947 "possibly due to the disease" being a cause for concern.⁴⁷

4. The post independence period, 1956-2021

A new Ministry of Livestock

Resources was created on 9 January 1954 with a Sudanese national as Minister. The veterinary services became the Department of Animal Production in this Ministry with Evans continuing as Director. A Veterinary Council – the first Sudanese council created specifically to organize and regulate the practice of a profession – came into being in 1954. Once again Evans was a prime mover in this development which laid down a new policy for disease control and animal production and underlined Evans' great abilities and sound judgement of concepts. When he eventually retired at the end of 1955 he bequeathed a strong and flourishing Department to his Sudanese successor.²⁶

The new Director was content to let the new Department get on with its work. And its work was very much as before. The standard treatment for camel trypanosomosis continued to be 5 g of suramin' (now marketed as 'antrypol' by Imperial Chemical (Pharmaceuticals, Ltd)) dissolved in 50 ml of sterile water to produce a 10 per cent solution and injected intravenously but repeated injections could lead to scarring and abscesses (Figure 6). Higher concentrations such as 20 per cent may be used but are irritant and phlebitis may occur (Figure 7). An incorrect method of administration, such as intramuscular or paravenous also causes problems (Figure 8). In areas where relapses were common retreatment with 2 g of Antrycide Methylsulphate dissolved in 20 ml of sterile water injected subcutaneously was carried out. Camel owners were quick to recognize the efficacy of this latter drug and demanded its universal administration. As 'antrypol' was still effective in most cases, however, this was the drug of choice by the veterinarians and in order to curb the enthusiasm of the nomads the price of this was maintained at one third that of the Methylsulphate.⁴⁸



Figure 6: Phlebitis of the right jugular vein caused by injection of a too highly concentrated solution of isometamidium chloride ('samorin') (Source: Schwartz, HJ and Dioli, M. *The one-humped camel in Eastern Africa. A pictorial guide to diseases, health care and management.* Margraf Scientific Book, Berlin. 1992)



Figure 7: Scars and abscesses on the right jugular vein caused by repeated intravenous injections of a too highly concentrated solution of isometamidium chloride (Source: as Figure 6)



Figure 8: Adult male camel with abscesses caused by incorrect routes of administration of isometamidium chloride: intramuscular (right hind leg) and paravenous (jugular area) (Source: as Figure 6)

A trypanosomosis outbreak among working camels belonging to the Public Health Department occurred in the autumn of 1956. The opportunity was then taken, when lapses occurred after ‘antrypol’ treatment, to compare the two standard drugs with ‘berenil’ (diminazene aceturate), which had recently been introduced as a trypanocidal.⁴⁸ It was later established that 10 mg/kg of ‘berenil’ was highly toxic to camels, the main signs of poisoning being hyperaesthesia, salivation, intermittent convulsions, frequent urination and defecation, itching and sweating with damage to the liver and kidneys.⁴⁹

A case of *T. congolense* was recorded in a camel at 12° N in 1960 – when the Mercuric Chloride Test was still the main means of diagnosis -- this being thought due to this animal (and others) being used for transport in the tsetse area as the owners were now less cautious of tsetse-borne infection due to the use of effective curative drugs.¹⁴

Camel trypanosomosis was then – and still is – considered to be most prevalent in a belt between 15° and 18° North latitude, north of which there were very few biting flies and south of which camels were not herded because of the presence of tsetse flies.⁵⁰

Veterinary education had begun in Sudan in 1938 with the establishment of the Khartoum Veterinary School. The school was administered by the Department of Veterinary Services until 1945 when it united with four other higher educational establishments under the umbrella of Gordon Memorial College. Successful students were awarded a Diploma after two years of study. The College was upgraded to a university college in 1947 and affiliated with the University of London. In July 1956, after the independence, the Gordon Memorial College was transformed to the University of Khartoum, ending the "special relationship" with the University of London. The first BVSc degrees were awarded to three

graduates in 1959. In 2021 the original three Departments had been expanded to ten. Faculty staff after independence were mainly Sudanese although there has always been a minority of expatriate lecturers.. The effectiveness of the Faculty was enhanced by collaboration with external veterinary colleges and most notably with the Royal “Dick” Veterinary School at the University of Edinburgh.⁵¹⁻⁵⁶ The main thrusts of this collaboration were detection of infection by *T. evansi* and development of resistance and of cross-resistance to various drugs.

Various methods of detection of *T. evansi* infection have been developed. In Sudan, these include, in addition to the basic microscopic techniques, the Card Agglutination Test for Trypanosomosis (CATT), MicroHaematocrit Centrifugation Technique (MHCT), Enzyme-Linked Immunosorbent Assay (ELISA), DNA hybridisation and the Polymerase Chain Reaction (PCR).^{7,57-60} The last of these is highly sensitive and detects trypanosomes in the blood three days before they can be detected by microscopy. PCR has been used in eastern Sudan to detect *T. evansi* infection and which trypanocides are effective in curing it. Several strains of the parasite were resistant to quinapyramine sulphate, the reasons being the use of expired drugs, sub-curative doses and improper administration.⁶¹ This confirmed the much earlier reports of resistance to quinapyramine sulphate in Sudanese camels.^{51,52} Developed in the 1980s to overcome resistance to quinapyramine sulphate and ‘suramin’ it was found in eastern Sudan 30 years later that ‘cymelarsan’ (melarsomine dihydrochloride) was still largely effective.⁶²

Serological tests have proved effective in detecting infection elsewhere in Africa (and beyond). In Mauritania, for example, the prevalence of infection

identified in blood smears was 1.3 per cent, by CATT was 16.2 per cent and by the Immuno Fluorescence Antibody Test (IFAT) was 25.2 per cent.⁶³ In Chad a Buffy-Coat Technique (BCT) parasite test gave an indicated infection rate of 5.3 per cent in 2831 camels from 136 herds whereas using the antibody CATT test the apparent rate of infection was 30.5 per cent.⁶⁴ In Mali, CATT revealed a global serological prevalence of 30.6 per cent in 1903 sera samples.⁶⁵ The CATT test used in Somaliland on 2575 camels indicated an overall animal-level prevalence of 26.4 per cent.⁶⁶

During the 1980s the Faculty of Veterinary Science of the University of Khartoum was becoming increasingly independent and influential. The Departments of Medicine, Pharmacology and Toxicology and of Preventive Medicine were particularly active in research on camel trypanosomosis. They contended that specific recommendations for the camel were rare and where present tended to be extrapolated from those for other species. Most reported research was on the chemotherapeutic efficacy of a few drugs and that the pharmacology and toxicity of drugs likely to be used in the camel needed further study to ensure their efficacy and safety. Areas most deficient were pharmacodynamics, pharmacokinetics and drug metabolism and the anatomical, physiological and biochemical peculiarities of the camel warranted more pharmacological and toxicological studies. They contended that camels were more susceptible than other domestic stock to the toxic action of some trypanocidal drugs and in some cases these drugs might be metabolized differently. ‘Samorin’, for example, is curative against *T. evansi* in acute cases but not effective in chronic cases as it does not cross blood-brain

barrier. Drugs such as ‘berenil’ and furazolidone had been proved to be effective against *T. evansi* in mice but ineffective or toxic in camels.⁶⁷⁻⁷³

A camel research unit was established in the University of Khartoum in 1982 with assistance from the University of Hanover. In 1989 a camel research station was established at El Shuwak in Gadarif State to support the activities of the camel research unit and there is a dedicated laboratory at the University. The unit was promoted to a Camel Research Centre (CRC) in 1995. The CRC supports researchers from various universities and technical units in ministries and encourages collaboration with foreign scientists. Since its inception in addition to research on systems and production the Centre has continued research on diseases. Further (very belated) support for camels is evident in the setting up of a Camel Research and Development Committee, The National Council for Camel Research and Development, The Butana Camel Research Project in El Shuwak and Camel Research Centres in Tambul and El Rahad.⁷⁴ In recent years much of the research has been on aspects of drug resistance, on biotechnology and on molecular aspects of disease identification, prevention and cure.⁷⁵⁻⁸² It is to be expected that this type of research will continue into the future.

5. Discussion

It is commonplace to assert that trypanosomosis is the most important single cause of morbidity and mortality in camels. The organism invoked is generally *T. evansi* although other trypanosomes, especially *T. vivax*, can also be implicated. Control of the disease and its protozoan cause have attracted a great deal of attention not only in Sudan but also wherever camels are a constituent of the

array of domestic animals kept in the service of humanity.

Anaemia is a major factor in the pathology of disease. Its development and persistence induce anoxic conditions that are manifested in the dysfunction in various organs as a result of changes in tissue and vascular damage. Large quantities of cytoplasmic and mitochondrial enzymes are released into the serum, causing further cellular and tissue damage. An important effect of these changes is immunosuppression which predisposes the animals to the other infections that are associated with morbidity and death and, in practical terms, reduce the value of the animal in reduced output of the edible and other economic products of the camel that support human livelihoods.

For more than a century emphasis has been placed on diagnosis of the disease in the animal with a view to controlling or eliminating it with trypanocides for prophylaxis and cure. Sudan has played a large part in all of these facets from demonstrating the parasite in the blood, initially though basic microscopic techniques on blood smears to sophisticated and cutting edge biotechnology and molecular techniques. Not the least of Sudan’s contribution has been collaboration with the international research and development communities in the identification and development of trypanocidal drugs (Table 1). Many of these have proved effective prophylactics or curatives but *T. evansi* has proved evasive in efforts to overcome it to which may be added the peculiar physiology of the camel which means that drugs used effectively in other domestic species may be less useful and even harmful in the camel. The struggle to overcome camel trypanosomosis will continue.

Table 1: Characteristics of trypanocides used as prophylactics and curatives for *Trypanosoma evansi* infections in the one-humped camel

Chemical compound (commercial drug)	Dose rate and method of application	Effects and notes
(suramin, Bayer 205, naganol, antrypol, germanin)	10-12 mg/kg live weight; intravenous injection as 10% solution (may be increased to 20%)	in common use, resistance common, strictly intravenous application; locally irritant; may cause phlebitis at higher concentrations; some prophylactic effects for 2-3 months; curative against <i>T. evansi</i>
4 melaminophenylarsine (cymelarsan)	0.25-0.5 mg/kg; intramuscular injection	resistance common; higher doses may cause systemic side effects (muscle tremors, increased defecation and urination, salivation); curative against <i>T. evansi</i> , less effective against <i>T. vivax</i>
Quinapyramine sulphate (antrycide, trypacide, noroquin, quintrycide, triquin)	3-5 mg/kg; subcutaneous injection	resistance rapidly acquired; may cause abscess at injection side; higher doses may cause systemic side effects (muscle tremors, salivation); curative against <i>T. evansi</i> and <i>T. congolense</i> , moderate efficacy against <i>T. vivax</i>
Quinapyramine sulphate + Quinapyramine chloride (trypacide prosalt, antrycide prosalt)	5-8.3 mg/kg; subcutaneous injection SC	resistance rapidly acquired; may cause abscess at injection side; higher doses may cause systemic side effects (muscle tremors, salivation); prophylactic effects for 3-6 months; curative against <i>T. evansi</i> and <i>T. congolense</i> , moderate efficacy against <i>T. vivax</i>
Isometamidium chloride (trypamidium, samorin, veridium, securidium)	0.5-1 mg/kg intravenous injection as 2% solution	strictly intravenous; locally irritant: may cause phlebitis; higher doses may cause systemic toxicity (salivation, tachycardia, profuse diarrhoea, hindleg weakness); curative against <i>T. evansi</i> , <i>T. congolense</i> and <i>T. vivax</i> ; not effective in chronic cases since does not cross blood-brain barrier
Homidium bromide (ethidium) Homidium chloride (novidium)	1mg/kg intramuscular injection	not curative in camels
Duminazene aceturate (berenil, veriben, trypan, ganaseg, azidin)	4-8 mg/kg intramuscular injection	not recommended for camels; high doses may cause death due to low therapeutic index

Source: Constructed by the authors from citations in the text and from additional references⁸³⁻⁸⁸

6. Conclusions

In the 120 years since ‘surra’ was first identified as a major disease of camels, thousands upon thousands of hours have been spent on laboratory research and field work, hundreds of reports and scientific papers have been written and countless millions of pounds, dollars, francs, riyals and other

currency have been disbursed in attempts to overcome the disease. There have been some successes, mostly ephemeral, rarely lasting and unable to be sustained. If a solution seems to have been found the parasites institutes counter measures. *T. evansi* has refused to accept defeat, the fight goes on and the war is yet to be won.

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