

## ***Trypanosoma evansi* (surra) in camels: a factor in the weakening of Islam?**

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### **Abstract:**

*Trypanosoma evansi*, the most lethal disease of camels, is caused by blood parasites, which provoke serious and often fatal anaemia. During the last few thousand years, *Trypanosoma brucei brucei*, causing sleeping sickness in tropical Africa, evolved into *T. evansi* in the Sudanic-Sahelian region of Africa, to the north of the tsetse belts. The new disease was transmitted by blood-sucking flies, notably Tabanids. Camels then carried the disease across the Sahara to Morocco around the 7th or 8th century of the Common Era, whence Islamic armies and caravans further dispersed it. Although the details of this process remain to be discovered, the changing size of the parasite yields a very rough chronology. Moreover, the disease remained largely confined to Islamdom in the Old World. As camels and equids were fundamental to Islamic military and economic power, this disease can be seen as having contributed to the weakening of Islamdom in its long contest with Christendom.

### **Introduction**

*Trypanosoma evansi* is the most lethal disease of camels. If untreated, it can result in up to 90% mortality among infected animals, although a partial resistance can build up. (Irwin 2010: 32-3; Röttcher et al. 1987; Leese 1927: 222-4) Also known by its Indian name of *surra*, *Trypanosoma evansi* further kills horses, donkeys and mules across the tropical and sub-tropical world, and can be fatal to dogs and elephants. It weakens cattle and buffaloes, but does not affect humans. A single-cell parasite of the blood, it is almost always transmitted mechanically by blood-sucking flies, notably flies of the *Tabanus* genus. Any kind of direct blood contact can spread the malady, however, and vampire bats are vectors in the Americas. The parasites destroy red blood cells, provoking serious and often fatal anaemia. (Hoare 1972; Luckins 1988)

It is suggested here that the diffusion of *Trypanosoma evansi* was a factor in the decline of the Islamic world, albeit one that has been completely neglected to date. Paradoxically, the great initial military triumphs of the new religion created a perfect environment for the disease to flourish. The intensification of cross-Saharan trade by Muslims probably brought this parasitic disease from Sub-Saharan Africa into the Mediterranean Basin in the first place. Islamic armies and caravans, unifying the desert edges of the Old World as never before, then provided ideal conditions for the further dispersal of *T. evansi*. As the disease only penetrated into the Iberian and Russian fringes of Europe, it contributed to the weakening of Islamdom in its multi-secular contest with Christendom.

### ***Semper ex Africa...: The pre-Islamic origins of Trypanosoma evansi***

*Trypanosoma brucei*, causing *nagana* or sleeping sickness of animals in Africa, at some point evolved into *Trypanosoma evansi*. The two parasites are morphologically virtually indistinguishable, and the evidence points firmly to *T. evansi* emerging from *T. brucei*. Whereas *T. brucei* parasites undergo genetic exchange and development inside tsetse flies, *T. evansi* has lost that capacity, adapting to mechanical transmission by blood contact. *T. evansi* protozoa are thus clonal, reproducing by longitudinal division. (Hoare 1972: 97-8, 582-3, 593; Luckins 1988: 137-8; Gibson 2001: 643-6)

Analysis of the DNA of the parasite's kinetoplast reveals that this was a 'comparatively recent' mutation. (Stevens et al. 1999: 110, 113) More specifically, *T. evansi* parasites 'may have evolved as clonal entities only in the last few thousand years.' (Gibson 2001: 644) The kinetoplast is a tiny organoid, 0.6 microns long. As *T. evansi* came to rely on mechanical transmission, the DNA of the kinetoplast was no longer needed for the protozoa to develop inside tsetse flies, leading to genetically irreversible mutations. (Hoare 1972: 103-6, 568; Gibson 2001)

Hoare has often been misunderstood as having written that the mutation giving rise to *T. evansi* took place in North Africa, whereas, in reality, he indicates that the process occurred in the Sahel-Sudanic zone of Africa, to the north of tsetse belts. Anywhere between modern Senegal and Somalia, biting flies could have spread a mutant form of *T. brucei*. Patterns of resistance to the parasite suggest that it was the Horn of Africa, with its notably patchy and discontinuous tsetse belts, that witnessed the birth of *T. evansi*. There exists a degree of resistance to the disease among horses and donkeys in Northern Kenya, Somalia and the Nilotic Sudan, and indigenous breeds of Somali dogs are immune. (Hoare 1972: 557, 579-80, 583-5)

If the disease did emerge in the Horn of Africa, it would easily have spread from east to west along the Sahel-Sudanic zone to West Africa, where it is enzootic and found particularly among camels. (Ford 1971: 92-3, 377) Although little is known about ancient east-west trade routes in the northern half of Africa, camels moved across the southern fringes of the Sahara by the third or fourth century CE. (Bulliet 1975: 132-4) Caravans of donkeys, equally able to transmit the disease, were also widely employed. (Roberts 1987: 14-15, 62-4)

The West African connection is important, because measuring parasites underpins the theory that *T. evansi* first crossed the Sahara along the western routes, arriving in Morocco. The parasite is about 20 microns long at 5 degrees west of Greenwich, corresponding to northern Morocco, and it increases in size eastwards from this point, to reach some 27.5 microns in the Philippines. According to Hoare, this indicates that Morocco was the centre of diffusion north of the Sahara. (Hoare 1972: 568, 579) This evidence on the size of parasites does not support other possible routes, such as the Nile valley, or the short maritime crossing of the Red Sea or the Gulf of Aden.

Moreover, parasitologists believe that *T. evansi* must have been brought to North Africa by camels, because equids die more quickly than camels when they contract the disease, and the Sahara routes are long and slow. (Hoare 1972: 583-6) A strong supporting argument is that equids are not known to have crossed the great desert from south to north after the introduction of camels, although they continued to come from north to south. (Law 1980; Mahadi 1992: 120)

### **The diffusion of *Trypanosoma evansi* across Islamdom**

The most likely time for *T. evansi* to have entered Morocco was when camels began to cross the western swathes of the great desert more regularly and in greater numbers. The establishment of Islamic rule in the Maghrib, from 661 CE, is known to have resulted in a considerable intensification of cross-Saharan traffic. (Bulliet 1975: 138-40) Although it is possible that *T. evansi*'s arrival in the Mediterranean Basin somewhat predated the arrival of Islam, the two processes were probably intimately related. New trading connections provided the necessary density of camels making voyages for the parasite to survive the desert crossing and become established in its new environment. This is in part because the disease is seasonal, often being described as a 'summer' plague. (Irwin 2010: 33) Seasonality is in turn closely related to the appearance of dense swarms of flies, typically during and after heavy rainfall. (Leese 1927: 243) Indeed, the usual Arabic word for *T. evansi* is *dhubâb* (*debab*), which simply means fly. (Leese 1927: 250-1)

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Trading caravans provided ideal conditions for the subsequent dispersal of *T. evansi* out of Morocco, together with equids and camels in Islamic armies, whether as mounts or for logistic purposes. (Cauvet 1926: 28-32; Irwin 2010: 151-3).

Eventually, the disease reached the furthest corners of the Islamic world, and spilled over into the rest of Asia, probably entering the Philippines around 1900 CE.

(Clarence-Smith 2010: 132)

Assuming that the malady arrived in Morocco around 700 CE, and then marched across North Africa and Asia with absolute regularity, it would have advanced about 650 miles, or some 1,000 kilometres, every century. However, it is far more likely that the disease spread quite fast to modern Libya, which may have provided an initial barrier, as Tabanid flies are rare in this arid area. (Leese 1927: 251). Once in Egypt, however, the disease would probably have progressed rapidly to the Indus valley, and then more slowly across South and Southeast Asia.

Ignorance about the timing of *T. evansi*'s diffusion across the Islamic world contrasts with the historical record of the introduction and spread of the disease in the Americas. In the sixteenth century, the Spaniards brought it to Mesoamerica, together with equids, but it did not traverse the densely forested Isthmus of Panama. Two further independent importations, into Venezuela and the Brazilian Amazon respectively, can be dated with some certainty to the early decades of the nineteenth century, and the progress of the disease from coastal foci has been charted. (Hoare 1972: 557, 560-1, 569, 587; Canelón and Meléndez 2003)

Even the limits attained by *T. evansi* in Asia remain uncertain, although the prevalence of sufficient concentrations of Tabanid flies was probably the main factor determining the reach of the disease. The furthest north in the world that *T. evansi* has been recorded is around 55 degrees north, on the Volga near the Tatar Islamic centre of Kazan, where it is known as *su-auru*. Hoare declares that the disease did not spread much further east, because only dromedaries suffered from trypanosomes, and not bactrian camels. (Hoare 1972: 559-60, 586) However, other authorities contradict this statement, demonstrating that bactrians are indeed susceptible to *T. evansi*. (Leese 1927: 224; Touratier 1999: 191) Moreover, there would be nothing to prevent horses from carrying the malady further east. Hoare's map further shows southern India and Sri Lanka as unaffected. (Hoare 1972: 559) Although these were not areas suited for camels, equids were present, and were even bred in a few places. (Balfour 1871: *passim*)

Much about *T. evansi* may lie in old Islamic veterinary writings, which appeared from the mid-eighth century CE, in Abbasid times. They were centred on horses and falcons, reflecting the importance of hunting. (Schimmel 2003: 4) Among them was a work by Ahmad b. al-Hasan ibn al-Ahnaf, dating from around 1100 CE, and relating to horses, donkeys and mules. Chapter 27 dealt with the 'emaciation, weakness and anorexia' of equids. (Kamal 1975: 735-6) This could refer to *T. evansi*, although it would be necessary to consult the Arabic text to find out more. Veterinary manuals were particularly common under Mamluk rule in Egypt and Syria, from 1250 to 1517, when cavalry units from the Eurasian steppe came to power. (Kamal 1975: 735; Schimmel 2003: 5) The Ottomans had animal hospitals by the sixteenth century, funded by *awqâf*, charitable trusts, which may have left records. (Foltz 2006: 5)

One detailed examination of alterations in patterns of camel breeding in the lands of Islam concerns the movement of Turkic peoples into northeastern Iran in the eleventh century CE. Richard Bulliet argues that a cooling of the climate in this area was the chief cause of these migrations, especially exceptionally cold winters. (Bulliet 2009: 96-117) However, he does not consider whether a colder climate might have reduced the number of flies spreading *T. evansi*.

In the absence of studies of camel raising, the history of equids in Islamdom may suggest the impact of the disease. Thus, the spread of *T. evansi* may account for a hitherto unexplained crisis in the equine economy of southern Arabia. There is much evidence for horse exports to mediaeval and early modern South Asia from ports in this area, and parts of Yemen enjoyed a great reputation for horse breeding. (Fattah 1997: 160, 235) By the early nineteenth century, this had ceased, and horses in Yemen were rare and of poor quality. (Greely 1975: 22) The 'air' of Yemen was alleged to cause degeneracy in its equids. (Balfour 1871: II, 616) In the 1930s, horses were imported into the north and hardly ever seen in the south. (Coon 1968: 203) However, there are other possible explanations for the crisis, such as surges of African Horse Sickness, a viral ailment, crossing the Red Sea.

A similar straw in the wind is that Ottoman statesmen became increasingly worried that they disposed of insufficient horses for military purposes, as Austrian and Russian armies pressed ever further into the empire in the eighteenth century. These fears resulted in sporadic prohibitions on horse exports from Iraq to India, even though the Porte was also greatly concerned about the 'drain' of bullion to South Asia. The Crimean War of 1854-56 caused a renewed spurt of Ottoman anxiety. (Fattah 1997: 74-5, 105-6, 111, 160-1, 173-6, 183; Ammon 1983: 278-82; Barendse 2001: 30) Breeding of horses in the Iraqi-Syrians borderlands prospered, however, possibly because flies were scarce in this dry zone. Lady Blunt reported that horses were exceptionally healthy in this region in the 1870s. (Blunt 1968: II, 263) In contrast, camel trypanosomiasis was prevalent in the areas flooded by the Tigris and Euphrates rivers. (Leese 1927: 224)

Conditions were less favourable in Persia, where a disease of livestock was reported in 1876, which was later demonstrated to have been *T. evansi*. (Luckins 1988: 139) It was probably no coincidence that, from around this time, prices of equids soared in Persia, exports dwindled, and some tribes faced acute shortages of horses. (*Encyclopaedia Britannica 1911*: XXI, 196) That said, drought may also have played its part.

Moving to South Asia, strong resistance to the parasite demonstrated by bovids prompts a suggestion that the disease had been present for a long time. (Leese 1927: 223) However, this is insufficient to justify Hoare's claim that *T. evansi* dated back to 'the remote past' or 'time immemorial,' given that he notes that a qualified bovid immunity to *T. evansi* could be acquired in as little as twenty years. (Hoare 1972: 555, 581).

The numerous names for the malady in the nineteenth century again suggest that the disease had been present for many generations. Griffith Evans, the scientist who discovered the parasite in 1880, noted that the common Hindustani term was *surra*, meaning 'rotten' or 'withered.' (Evans 1881: 1-2, 84) Although it was this term that entered the literature, a plethora of other names were in use. Some twenty were recorded in the northwest of British India alone, where both camels and equids were present in significant numbers. (Leese 1927: 224)

It may be that *T. evansi* spread during the Delhi Sultanate of the thirteenth century CE, when a Mamluk cavalry regime greatly strengthened links with the Islamic world. A Middle Eastern or Inner Asian origin for the disease is certainly propounded, inasmuch as the word *surra* allegedly derives from the Arabic word for epilepsy, *sara*. (Phillott 1911: 39) This derivation may be spurious, however, as there is no indication that speakers of Arabic ever used this word to designate *T. evansi*. (Adel Aulaqi, personal communication) Nevertheless, the fact that an Arabic etymology was suggested probably reflected the belief that the disease reached South Asia from the Middle East, either traveling with camel and equid caravans, or brought by horses coming by sea.

### Colonial triumphalism: quarantine, entomology and drugs

As Western colonialists triumphed over Islam from the 1750s, the new masters of the world developed techniques with which to fight *T. evansi*. Although the disease was by no means eradicated, control improved the West's military and economic position in the lands of Islam. In particular, from the beginning of the British conquest of India in the mid-eighteenth century, horses and camels played a significant part in Western military campaigns over a vast area. (Cauvet 1926: 42-52; Bulliet 1975: 237-58) At the same time, inroads into the malady formed part of a broader scientific justification for formal and informal imperialism.

Simply knowing more about the disease was an important step forward. European scientists first observed trypanosomes in the blood of trout and frogs in 1843, leading up to Griffith Evans' great discovery in the Punjab in 1880. India's Imperial Bacteriological Laboratory then investigated *T. evansi* extensively in its early years. (Ware 1961: 25, 32) This helped to avoid incorrect diagnosis, for example by confusing *T. evansi* in horses with equine piroplasmiasis [horse tick fever; Texas fever], caused by two other protozoan blood parasites, *Babesia equi* and *Babesia caballi*, transmitted by blood-sucking ticks. (Lloyd 1987: 433-5)

Early twentieth-century successes in preventing the entry of *T. evansi* into areas of European settlement were mainly due to rapid diagnosis, followed by the slaughter of infected animals, and the imposition of strict quarantine. Imported camels were responsible for outbreaks in Namibia [German South West Africa], the United States, and Western Australia in the decade of the 1900s. In none of these three cases was *T. evansi* allowed to gain a permanent foothold. (Hoare 1972: 560; Leese 1927: 223; Reid 2002: 223; Röttcher et al. 1987: 464)

Preventing transmission by insects was a second measure, although it did not take Western scientific advances for local peoples to make the connection between biting flies and infection. In what is today northern Nigeria, smoke and herbal remedies served to discourage flies from biting animals in the 1820s. (Law 1980: 81-2) Evans noted that Punjabis blamed biting flies for spreading *T. evansi*, although he himself equivocated, opining that infected water was the ultimate source. (Evans 1881: 8-9) Research on tsetse-borne trypanosomes helped to focus scientific attention on flies, and the role of Tabanids in passing on *T. evansi* was first scientifically described in 1901. (Hoare 1972: 571)

Veterinarians then concentrated on insect control. Australia's Northern Territory, home to significant numbers of camels and horses, was seen as vulnerable to spread from Indonesia's ponies. This led to experiments with 'large fly-proof stables,' and washing animals with an emulsion of castor oil and soda. (*Bulletin of the Imperial Institute*, 12, 1914: 116) In northwestern British India, 'mosquito curtains' were found to be effective experimentally, but not practically. Other strategies were avoiding areas where flies were known to abound, burning greenwood fires to windward of stock, covering vulnerable areas on beasts with sacking, and 'clumping' animals to improve their own herd defences. Biological control was tried, using hornets and dragon-flies against flies, and chalcids against eggs. (Leese 1927: 242, 247-9)

As the sheer abundance and variety of biting flies made insect control difficult, chemical treatments moved to the fore. Alfred Lingard first demonstrated the specificity of arsenic in treating trypanosomiasis in India. (Ware 1961: 37) Combinations of arsenic and antimony soon proved effective, especially if the disease was quickly diagnosed. (Leese 1927: 218, 241-2, 247, 254) The main drug to emerge was Suramin, also called Naganol or Bayer 205. India's Imperial Bacteriological Laboratory reported in 1927 that it had achieved a 94% success rate, when administering Naganol 'by the intra-theal route.' (Ware 1961: 39) Injected

intravenously as a prophylactic, Naganol protected the equids of Burma's military police in the 1930s. (Rippon 1961: 70)

Chemical treatments are no magic bullet, however, and the tendency has been for *T. evansi* to remain enzootic in its domain. Resistance has built up in camels to Suramin, and to the more recently introduced Quinapyramine family of drugs. (Röttcher et al. 1987: 466-7; Wernery and Kaaden 2002: 280-2) After initial successes, there followed decades of stagnation in the production of new trypanocides, and research and experimentation have remained inadequate. (Touratier 1999)

## Conclusion

For the emergence of *surra* from the mists of the deep past, historians will have to remain reliant on other disciplines. Better techniques of analyzing the DNA of the parasite's kinetoplast may enable more precise dates to be attributed to when this scourge arose in Africa, as well as more exact locations of origin. (Canelón and Meléndez 2003) Taking more measurements of the size of the parasites might also suggest an improved chronology for its dispersion. Whether archaeologists can throw light on this conundrum remains to be seen.

For documented history, and especially for the period of the growth and decline of Islamdom, the focus for new research should be on veterinary materials, particularly those in Arabic, Persian, Turkish, Armenian, and other regional languages. This requires considerable linguistic skill, and depends on painstakingly gathering fragmentary scraps of surviving evidence. Moreover, there is a need to carefully interpret the formulations of Islamicate veterinarians, who worked in conditions preceding the scientific revolution.

Concerning the central suggestion of this paper, namely that *T. evansi* undermined the economic and military efficacy of Islamic states in their contest with the West, this paper has not been able to do more than formulate a working hypothesis. There is a need to scour documents that reveal official or mercantile preoccupations about the state of camels and equids. The most promising sources are probably military in nature, with an emphasis on horses, but the proverbial Arab love of camels may also yield useful results.

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