Trypanosoma evansi
(protist: flagellate)

Overview

Protists are single-celled organisms with membrane-bound nuclei (eukaryotes). Flagellates are protists that swim using one or more flagella (undulipodia); each arising from a small centriole (basal body, kinetosome) and having a microtubular axoneme core (2+9 configuration). Rather than forming a monophyletic group, flagellates are divided into several disparate groups: metamonads (amitochondriate flagellates), heteroloboseans (amoeboflagellates), euglenozoans (euglenids and kinetoplastids), stramenopiles (heterokonts), alveolates (dinoflagellates) and cercozoans (biflagellates). Most kinetoplastids are parasitic in vertebrate or invertebrate hosts (some in plants) whereas the remainder are free-living aquatic organisms. All species are characterized by the possession of extranuclear DNA in the form of a kinetoplast, a unique structure formed by massed DNA (circles or lattice) within the single large mitochondrion near the flagellar basal body. The flagellates reproduce by longitudinal binary fission and parasitic species may have simple monoxenous (one-host) or more complicated heteroxenous (two-host) life cycles involving different developmental stages. Trypanosomes have a single flagellum and they form four main developmental stages: trypomastigotes (with a posterior kinetoplast and an emergent flagellum forming a long undulating membrane); epimastigotes (with an anterior kinetoplast and an emergent flagellum forming a short undulating membrane); promastigotes (with an anterior kinetoplast and a short emergent flagellum, but no undulating membrane); and amastigotes (with a kinetoplast but no emergent flagellum or undulating membrane). Many trypanosome species are parasitic only in insects whereas others are transmitted by insect vectors to a wide range of vertebrate hosts. Three main groups infect the blood and/or tissues of humans and animals causing severe clinical diseases: including the noncyclic trypanosomes which undergo ‘mechanical’ transmission by biting flies/bats (no development in vector) or by venereal transmission.

Classification:

Domain:	Eukaryota (membrane-bound nucleus)
Supergroup:	Excavata (with conspicuous ventral feeding groove)
Group:	Discoba (diverse group supported robustly by molecular studies)
Phylum:	Euglenozoa (flagella inserted in anterior pocket, some heterotrophs, some autotrophs (with chloroplasts))
Class:	Kinetoplastea (heterotrophs, with extranuclear DNA (= kinetoplast) associated with mitochondrion)
Subclass:	Metakinetoplastina (large polyphyletic group supported by molecular studies)
Order:	Trypanosomatida (parasitic, single anterior flagellum, often forming undulating membrane)
Family:	Trypanosomatidae (monogenetic forms in insects/plants, digenetic forms in vertebrates & arthropods)
Genus:	*Trypanosoma* (vector-borne haemoparasites)
Species:	*T. evansi* (causes surra in animals)

Parasite biodiversity and host range: Protists are unicellular eukaryotes that move using undulipodia (flagella or cilia), pseudopodia (false-feet) or a unique gliding motion. Flagellated species have one or more flagella with an internal microtubular core (in a characteristic 2+9 configuration comprising 2 single central microtubules and 9 peripheral doublets) anchored to a submembranous protein structure (known variously as a centriole, basal body, kinetosome or blepharoplast). Many types of flagellated cells have been described and recent phylogenetic studies have classified them into several disparate groups: including the metamonads (amitochondriate flagellates), heteroloboseans (amoeboflagellates), euglenozoans (euglenids and kinetoplastids), stramenopiles (heterokonts), alveolates (dinoflagellates) and cercozoans (biflagellates). While most flagellated protists are free-living organisms swimming and feeding in aquatic environments, representatives of several groups have developed symbiotic relationships with various hosts; some being endoparasitic in vertebrates (notably anaerobic metamonads in tubular organs, and heterotrophic euglenozoans occurring in blood or tissues), and some being parasitic in invertebrates (alveolates in crustacean tissues) (representatives tabulated below).

Higher taxonomy	Class or order	Family	Genera	Hosts (tissues)	Trans-mission*
Supergroup: Excavata (with conspicuous ventral feeding groove)					
Group: Metamonad (amitochondriate flagellates with karyomastigonts)					
Phylum: Fornicata (diplomonads)	Order: Diplomonadida (1-2 karyomastigonts)	Hexamitidae (2 karyomastigonts with binary axial symmetry)	*Giardia*	vertebrates (gut)	direct (f-o)
			Hexamita *Spironucleus*	vertebrates (tissues)	direct (f-o, w)
Phylum: Parabasalia (with parabasal body)	Order: Trichomonadida (3-5 anterior flagella plus recurrent flagellum)	Monocercomonadidae (costa absent, most without undulating membrane)	*Histomonas*	birds (gut, liver)	direct (f-o)
			Dientamoeba	vertebrates (gut)	direct (f-o)
		Trichomonadidae (stout axostyle, costa, undulating membrane)	*Trichomonas*	vertebrates (urogenital tract, gut)	direct (f-o, v)
		Cochlosomatidae (anterior adhesive disc)	*Cochlosoma*	birds (gut)	direct (f-o)
Group: Discoba (diverse group supported robustly by molecular studies)					
Phylum: Euglenozoa (flagella inserted in anterior pocket, heterotrophs, autotrophs)	Class: Kinetoplastea (heterotrophs, with extranuclear DNA (= kinetoplast) associated with mitochondrion)	Ichthyobodonidae (flagellar pocket continues as groove)	*Ichthyobodo* (= *Costia*)	fish (gills, skin)	direct (w)
		Parabodonidae (epizoic or endozoic)	*Cryptobia*	fish (gills, skin)	direct (w)
			Trypanoplasma	fish (blood)	indirect (v-b)
		Trypanosomatidae (monogenetic forms in insects/plants, digenetic forms in vertebrates & arthropods)	*Trypanosoma*	vertebrates (blood, tissues)	indirect (v-b)
			Leishmania	vertebrates (blood, tissues)	indirect (v-b)
Supergroup: SAR (Stramenopiles + Alveolata + Rhizaria) (3 groups unified by molecular studies)					
Group: Alveolata (with cortical alveoli)					
Phylum: Dinoflagellata (with unique mesokaryotic nuclei)	Order: Blastodiniales (uninucleate trophonts with chloroplasts)	Oodiniaceae (trophont with rhizoid-like invasive organelle)	*Amyloodinium* *Crepidoodinium* *Piscinoodinium*	fish (skin)	direct (w)
	Order: Syndiniales (multinucleate plasmodial trophonts)	Syndiniaceae (without chloroplasts)	*Haematodinium* *Ichthyodinium*	crustaceans, fish (tissues)	direct (w)
Phylum: Perkinsozoa (parasitic)	Order: Perkinsorida (released trophonts form biflagellated zoospores)	Perkinsidae (incomplete conoid)	*Perkinsus*	gastropods, bivalves (tissues)	direct (w)

*f-o = faecal-oral transmission; v-b = vector-borne transmission, w = water-borne transmission; v = venereal transmission

Euglenozoans comprise a large group of excavates (with ventral feeding groove), most with 1-2 flagella inserted into an anterior pocket. Many species are free-living aquatic autotrophs possessing chloroplasts while others are free-living or symbiotic heterotrophs feeding on solutes, particles and even other organisms. Kinetoplastids are characterised by the possession of a kinetoplast (containing mitochondrial DNA separate from nuclear DNA), a flagellar pocket, basal bodies with three microtubular roots and paraxonemal (paraxial or paraflagellar) rods, and asexual multiplication by longitudinal binary fission. The unique kinetoplast is formed by massed DNA (circles or lattice) usually closely associated with the flagellar basal body (eukinetoplastic) although some species may be polykinetoplastic (with several kinetoplasts) or pankinetoplastic (irregular kDNA) and some mutants even dyskinetoplastic (without a kinetoplast). Two major kinetoplastid groups are recognized: bodonids with two flagella (most being free-living bactivores in aquatic/terrestrial habitats); and trypanosomes with a single flagellum (most being parasites of animals or plants with monoxenous or dixenous life-cycles). Different kinetoplastid assemblages exhibit increasing morphological/ultrastructural complexity in their cellular organization thought to reflect evolutionary grades or clines. Amastigotes are simple non-flagellated cells, choano-, pro- and opistho-mastigotes are flagellated cells with elongate flagella, while epi- and trypo-mastigotes are flagellated cells with undulating membranes. Most kinetoplastids have amastigote and promastigote developmental stages but monoxenous parasites of insects (e.g. *Crithidia*, *Herpetomonas*) do not have more elaborate forms whereas dixenous parasites of plants or animals with invertebrate vectors (e.g. *Trypanosoma, Leishmania*) do have more morphologically complex forms such as epimastigotes and trypomastigotes.

Traditional classification	Molecular classification	Genera	No. spp.	Vertebrate hosts	Transmission (vectors)
F: Trypanosomatidae	SC: Metakinetoplastina F: Trypanosomatidae	*Trypanosoma*	537	mammals, reptiles, frogs, birds, fish	indirect (arthropods, leeches)
		Leishmania	53	mammals, lizards	indirect (sand flies)
F: Bodonidae	SC: Metakinetoplastina F: Parabodonidae	*Cryptobia, Trypanoplasma*	79	fish	direct or indirect (leeches)
	SC: Prokinetoplastina F: Ichthyobodonidae	*Ichthyobodo* (*Costia*)	5	fish	direct

Conventional taxonomic classification systems divide the kinetoplastids into 2 groups: the free-living bi-flagellated Bodonina; and the parasitic uni-flagellated Trypanosomatina. Over 600 species have been described on the basis of multiple phenotypic characters (host occurrence, geographic distribution, vectors, transmission cycles, morphology, development, pathogenicity, culture requirements, etc.). Modern molecular characterization studies, however, have shown that many traditional groups are polyphyletic and composed of numerous clades. Contemporary phylogenetic classifications recognize 2 main lineages: the Prokinetoplastina represented by 2 diverse genera (*Ichthyobodo* biflagellates ectoparasitic on freshwater and marine fishes, and *Perkinsela* (= *Perkinsiella*) aflagellates endosymbiotic (as parasomes or parasome-like organisms (PLOs)) in amoeba *Paramoeba* and *Neoparamoeba*); and the Metakinetoplastina containing 4 groups, including free-living aquatic eu-bodonids (with one genus *Bodo*), free-living neo-bodonids (with 10 genera, including *Rhynchomonas*), free-living or commensal/parasitic para-bodonids (with 5 genera, including *Cryptobia, Trypanoplasma*), and the parasitic trypanosomatids (containing some 39 genera, including *Trypanosoma* and *Leishmania*).

Trypanosomatids are dixenous (2-host) parasites with indirect transmission cycles between vertebrates and invertebrate vectors. *Trypanosoma* spp. form trypo- and/or a-mastigote stages in the blood/tissues of vertebrate hosts, and epi- or pro-mastigote stages in invertebrate haematophagous vectors. Infections have been found in a range of vertebrate species (mammals, birds, reptiles, amphibians and fish) with many different types of haematophagous invertebrates (leeches, bugs, flies, fleas) implicated as vectors or paratenic hosts. The species found in mammals have been grouped into two Sections (convenient groups without formal taxonomic rank) primarily on the basis of their developmental cycles in their vectors and their modes of transmission. Each Section contains several subgenera, as follows:

Section: Stercoraria (posterior station development in vector, contaminative transmission)

- *T.* (*Megatrypanum*), large trypanosomes with kinetoplast close to nucleus;
- *T.* (*Herpetosoma*), medium trypanosomes with subterminal kinetoplast;
- *T.* (*Schizotrypanum*), small C-shaped trypanosomes with voluminous terminal kinetoplast;

Section: Salivaria (anterior station development in vector, inoculative (some mechanical) transmission);

- *T.* (*Duttonella*), former *vivax*-group, monomorphic forms with large terminal kinetoplast;
- *T.* (*Nannomonas*), former *congolense*-group, small forms with medium marginal kinetoplast;
- *T.* (*Trypanozoon*), former *brucei*-group, pleomorphic forms with small subterminal kinetoplast;
- *T.* (*Pycnomonas*), former *suis*-subgroup, stout monomorphic forms with small subterminal kinetoplast.

Recent molecular phylogenetic studies have validated the separation of mammalian salivarian and stercorarian trypanosomes, but indicated complex placements for trypanosomes from non-mammalian hosts, despite earlier suggestions that they be assigned to separate subgenera (*Trypanomorpha* for those in birds, *Trypanosoma* for those in amphibians, and *Haematomonas* for those in fish). Many clades have been identified, most associated with particular vertebrate or invertebrate hosts, or both, suggesting that 'host-fitting' rather than 'co-speciation' has been the principal mechanism for trypanosome evolution. Comparative studies have found many differences between trypanosomes of aquatic and terrestrial hosts, with several clades found for species in freshwater fishes, marine fishes, amphibians, tortoises and platypuses (all thought to be transmitted by leech vectors). *T. brucei* belongs to the salivarian trypanosomes which exhibit continuous multiplication in the mammalian host and develop in the anterior station in tsetse fly vectors with inoculative transmission (except for *T. evansi* and *T. equiperdum* which are transmitted mechanically by biting flies or by contact). Four salivarian subgenera are recognized: *Duttonella* comprising monomorphic trypanosomes with a large kinetoplast (*T. vivax, T. uniforme*); *Nannomonas* comprising small trypanosomes with a medium-sized kinetoplast (e.g. *T. congolense, T. simiae*); *Pycnomonas* comprising thick monomorphic forms with a small kinetoplast (*T. suis*); and *Trypanozoon* comprising pleomorphic (stumpy to slender) forms with a small kinetoplast (*T. brucei, T. evansi, T. equiperdum*). Species belonging to the former two subgenera develop in the proboscis or midgut of their vectors while those of the latter two subgenera undergo development in the midgut and salivary glands (except for *T. evansi* and *T. equiperdum* which are transmitted mechanically by biting flies or via coitus, respectively).

The species *T. evansi* is thought to be derived from *T. brucei brucei* but is no longer able to undergo development in insect vectors due to the loss of genetic material (maxicircles of kinetoplastic mitochondrial DNA). Instead, T. *evansi* relies on mechanical transmission between hosts via biting flies (passive transfer of blood stages as no development occurs in the flies). Infections exhibit a broad host specificity for vertebrates with severe disease developing in permissive hosts, notably camels, horses and dogs, while other domestic and wild ungulates, carnivores and rodents are more refractory to disease. The disease has many regional names, including surra (meaning 'rotten'), murrina, dehab ('fly'), mbori, guifar, dioufar, menchaca or dubla ('emaciated'), yudleye/yudle ('aimless movement'), dukhan, salaf/salef, tahaga, su-auru, purana ('chronic'), tibarsa ('three-year disease'), makhi ki bimari ('horsefly disease'), peste-boba/derrengadera ('limping'), and mal de caderas ('paralysis'). Infections may be transmitted mechanically by various biting flies, including species in the genera *Tabanus*, *Stomoxys*, *Lyparosia*, *Haematopota,* and *Chrysops*. In the New World, infections may also be transmitted via the bites of vampire bats, which may also act as vertebrate hosts for infections. The species *T. equiperdum* does not utilize invertebrate vectors (not even mechanically), but is transmitted between horses via coitus causing a disease known as dourine.

***Trypanosoma* spp.**	**Mastigote length (µm)**	**Vertebrate hosts**	**Disease**	**Transmission**	**Distribution**
NON-CYCLIC TRANSMISSION (no parasite development within vector)					
MECHANICA (mechanical transmission)					
Subgenus: *Trypanozoon* (trypanosomes of former *brucei*-group including *brucei*- and *evansi*-subgroups represented by pleomorphic forms (slender, intermediate, stumpy) with or without free flagellum, small kinetoplast subterminal, development in vector (*Glossina*) in midgut and salivary glands, but other species transmitted mechanically without development in vectors (*T. evansi*) or by contact between hosts (*T. equiperdum*))					
T. (*T.*) *evansi* (syn. *T. aegyptum, annamense, cameli, equinum, elmassiana, hippicum, kirdanii, marocanum, soudanense, suaurii, venezuelense*)	15-33	Artiodactyla: camelid (Bactrian camel, dromedary, llama, alpaca, guanico); bovid (African buffalo, water buffalo, cattle, sheep, mouflon, goat, gazelle, saiga antelope), suid (pig), tayassuid (collared peccary, white-lipped peccary), cervid (sambar deer, rusa deer, hog deer, barking deer, chital/spotted deer, white-tailed deer, brocket deer); Perissodactyla: equid (horse, ass/donkey, mule), tapirid (Asian tapir), rhinocerotid (Sumatran rhinoceros); Proboscidea: elephantid (Indian elephant); Cingulata: dasypodid (armadillo); Lagomorpha: leporid (rabbit, black nap hare), ochotonid (Pallas's pika); Didelphimorphia: didelphid (opossum, short-tailed opossum); Rodentia: murid (rat, tanezumi rat, long-tailed giant rat, chestnut white-bellied rat, red spiny rat, bandicoot rat, New World mouse), cricetid (hamster, Japanese grass vole), caviid (capybara, lesser capybara, guinea pig); Carnivora: canid (dog, wild dog, wolf, red fox, Bengal fox, Azara's fox, golden jackal), hyaenid (hyena), felid (cat, woodcat, leopard, jaguar, tiger, ocelot), herpestid (small Indian mongoose, Javan mongoose), mustelid (ferret-badger), viverrid (civet cat), ursid (Himalayan black bear), procyonid (South American coati); Diprotodontia: macropodid (agile wallaby, dusky pademelon); Chiroptera: phyllostomid (Latin America vampire bat, broad-nosed bat, short-tailed bat), vespertilionid (mouse-	surra, murrina	Diptera: tabanid (*Tabanus*, *Stomoxys*, *Lyperosia*, *Haematopota, Chrysops*) plus Chiroptera: phyllostomid vampire bats in New World (*Desmodus rotundus*)	Asia, India, North Africa, Americas

		eared bat); Primates: atelid (Colombian red howler monkey, ursine howler), cercopithecid (rhesus macaque), hominid (orangutan); Galliformes: phasianid (chicken)			
T. (*T.*) *equiperdum* (syn. *T. equi, rougeti*)	15-36	Perissodactyla: equid (horse, donkey, mule); plus experimental infections in Lagomorpha: leporid (rabbit), Carnivora: canid (dog); Rodentia: murid (rat, mouse)	dourine	coitus	North Africa, Eurasia, Americas

Parasite morphology: *T. evansi* and *T. equiperdum* belong to a small group of trypanosomes apparently related to *T. brucei* but considered to be 'petite mutants' due to the loss of genetic material (kinetoplast DNA maxicircles) required for cyclic development in tsetse flies (unable to transform into procyclic stages). Due to this loss, they only form blood stages (trypomastigotes) in vertebrates. Trypomastigotes are elongate slender stages ranging in length from 15-36 μm. They have a central ovoid basophilic nucleus as well as another smaller basophilic organelle (containing mitochondrial DNA) known as the kinetoplast which is located subterminally posterior to the nucleus. Flagellar basal bodies (microtubular complexes) are located adjacent to the kinetoplast and give rise to a long flagellum which emerges and runs the length of the body forming a conspicuous undulating membrane before extending freely beyond the cell margin. Observations of live motile parasites show they move in the direction of the free flagellum so that cellular aspect is designated anterior (the flagellum is not recurrent or trailing like that of the bodonids *Cryptobia* and *Trypanoplasma*). During their development, some trypomastigotes form slightly smaller intermediate forms with the kinetoplast located almost terminally and the free flagellum being shorter (but not as small as the stumpy metacyclic forms observed for other *Trypanosoma* spp.). Transmission and culture experiments have sometimes induced profound pleomorphic changes in trypomastigotes involving size disparities, abnormal growth patterns and the development of dyskinetoplasty. A few experimental studies (notably on carnivores and primates) have also described the occasional appearance of sphaeromastigote-like (rounded with short emergent flagellum) and amastigote-like (rounded with non-emergent flagellum) stages (usually in organ smears), but it is not known whether they represented true developmental stages or were degenerate forms.

Site of infection: Trypomastigotes are extracellular parasites usually found intravascularly in the circulatory system of vertebrates, but they may sometimes invade host tissues and organs where they are found in extravascular fluids (including lymph and spinal fluid). The species *T. evansi* has a broad host specificity, with infections found in 78 mammalian species, including ungulates (perissodactyls, artiodactyls), elephants, carnivores, marsupials, opossums, cingulates, lagomorphs, rodents, bats, and primates. Trypomastigotes may also be found contaminating the mouthparts of various secondary hosts which act as mechanical paratenic vectors, mostly tabanid flies (*Tabanus*, *Stomoxys*, *Lyperosia*, *Haematopota, Chrysops*) but occasionally phyllostomid vampire bats (*Desmodus rotundus*). Infections by the species *T. equiperdum* are found primarily in the tissues of equine hosts, rarely in the blood, and trypomastigotes are present in genital secretions of both infected males and females (no vectors are involved).

Pathogenesis: Infections by *T. evansi* vary markedly in their severity depending on host susceptibility (due to species, breed, age, nutritional and immune status) and parasite pathogenicity (strain variation, intensity of infection). Infections cause severe disease in permissive hosts, especially camels, horses and dogs, while other domestic and wild ungulates, carnivores and rodents are more resistant to disease manifestations. Trypomastigote stages occur in the blood where they grow and divide by binary fission, sometimes causing high parasitaemias. However, these stages differ from other African trypanosomes in that they are also able to invade host tissues, including the nervous system. Infections may therefore occur in intra- and extra-vascular fluids and elicit a range of clinical signs which can result in high mortality in some hosts, but more chronic production-limiting diseases in others. Infections of the nervous system may mimic those occurring in sleeping sickness (caused by *T. brucei*). There are many regional names for the disease(s) caused by *T. evansi* (and its many synonyms) reflecting clinical observations, including surra (meaning 'rotten'), murrina, dehab ('fly'), mbori, guifar, dioufar, menchaca or dubla ('emaciated'), yudleye/yudle ('aimless movement'), dukhan, salaf/salef, tahaga, su-auru, purana ('chronic'), tibarsa ('three-year disease'), makhi ki bimari ('horsefly disease'), peste-boba or derrengadera ('limping'), and mal de caderas ('paralysis'). When disease is apparent, it may manifest as acute, subacute or chronic disease. Infections in camels, horses and dogs may have a short incubation period of 1-4 weeks producing acute disease and even death over 2-8 weeks (esp. dogs and horses) or becoming chronic and persisting for 2-3 years (esp. camels). Clinical signs include fever, anaemia, jaundice, pale mucous membranes with petechial or ecchymotic haemorrhages (incl. eyelids of horses) and anterior chamber of eye (horses and dogs) producing ocular signs with conjunctivitis, lachrymation, keratitis and corneal opacities (trypanosomes can be found in gelatinous material from inner canthus). Animals develop ventral oedema involving the abdomen, brisket, legs scrotum and udders (also head and larynx in dogs), highly coloured urine (odiferous in camels) and sometimes transient local or general cutaneous eruption (in horses). They exhibit progressive loss of condition with weakness, lethargy, staring hair, weight loss with cachexia and emaciation (so-called 'living skeletons') despite variable loss of appetite, and marked loss of productivity (meat, milk, power and even abortion in pregnant animals). Infections may lead to neurological signs with alterations in locomotion (forelimb incoordination and hindlimb paresis, known as mal de caderas in horses) and sometimes periodic convulsions prior to death. Animals may die unexpectedly or develop delirium and struggle for hours. All age groups may be affected, but most

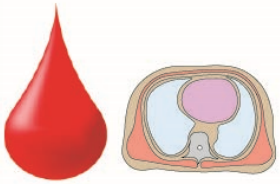
clinical infections start around weaning. Parasites often exhibit antigenic variation of their surface glycoproteins to evade host immune responses, which contributes to frequent relapses of parasitaemia and remittent clinical signs. Infections have also been associated with immunosuppressive effects that may influence intercurrent diseases or interfere with vaccination campaigns. Nonetheless, animals in endemic regions may develop some resistance to disease but continue to act as asymptomatic or subclinical carriers. Resistance appears to involve both innate (complement, macrophages) and adaptive (humoral and cell-mediated) responses leading to premunitive immunity. Infections in other herbivores are less severe, often persisting as latent-chronic infections making the hosts carriers or reservoirs of infection. Bovids are usually refractory to disease, but some strains in cattle and buffalo in India appear to be more virulent sometimes causing clinical signs including hyperthermia, anaemia, oedema (legs), weight loss, production losses (meat, milk, draught power), neurological signs (usually meningoencephalitis), dyspnoea, recumbency, abortion and sometimes death. Cervids exhibit variable signs, ranging from subacute to acute and fatal, and pigs are highly susceptible to infection but not disease. Infections in Asian elephants have been associated with severe symptoms, fever, anorexia, oedema of face, trunk, neck, brisket, lower abdomen and limbs, weakness and restlessness. Experimental transmission studies have also shown that macropod marsupials are very susceptible to infection and disease, with several kangaroo and wallaby species developing acute life-threatening diseases. Infections are occasionally detected in humans but they rarely cause disease as most possess natural trypanolytic proteins (apolipoprotein L1). Infections by *T. equiperdum* cause a disease in equids known as dourine characterized by fever, local oedema of the genitalia and mammary glands, oedematous cutaneous plaques, anaemia, ocular lesions, facial and lip paralysis, incoordination, paraplegia and emaciation, with periodic exacerbation and relapse. Infections in horses are often fatal, but spontaneous recoveries do occur although animals generally remain as latent carriers. Donkeys and mules are more resistant to infections and disease than horses but may also act as asymptomatic carriers.

Developmental cycle and mode of transmission: Trypomastigote stages feed on nutrients in intravascular and extravascular fluids by absorption across their surface membranes. They divide asexually by transverse binary fission involving the replication of organelles followed by karyokinesis and then cytokinesis. Infections by *T. evansi* are usually transmitted mechanically via contamination of the mouthparts of biting tabanid flies, and sometimes via contaminated mouthparts of vampire bats in the New World. The parasites do not undergo further development in these vectors so they are not considered to be true intermediate hosts but rather paratenic transport hosts. Other minor transmission routes have occasionally been reported, including oral transmission to carnivores consuming infected prey, vertical transmission in some hosts (transplacental or transmammary), and even iatrogenic transmission (when butchering animals). In contrast, the species *T. equiperdum* does not utilize invertebrate vectors (not even mechanically), but is transmitted exclusively between horses via coitus (not during every copulation). The incubation periods and duration of the diseases vary considerably between host and parasite species.

Differential diagnosis: Infections may be indicated in some animals by the occurrence of various clinical signs, such as *T. evansi* causing emaciation (surra) or neurological conditions (mal de caderas), or *T. equiperdum* causing oedematous cutaneous plaques (dourine) in horses, but other conditions cannot be discounted. Diagnosis is therefore made by the direct microscopic demonstration of parasites in host fluids (blood, lymph, cerebrospinal fluid) or centrifugal concentrates. Wet mounts may be examined by high-contrast microscopy for live motile trypanosomes, or fixed smears examined after staining with Giemsa or Leishman's stains. Unfortunately, test sensitivities are generally low as parasites may be sequestered extravascularly in host tissues rather than being abundant in the circulation. Parasites may be cultivated *in vitro* using various axenic media (e.g. Hirumi's modified Iscove's medium-9) although *T. equiperdum* appears to prefer soft agarose underlays. Parasites can also be propagated *in vivo* in laboratory mice, rats or rabbits, although some isolates do not readily adapt. A range of serological tests (complement fixation, immunofluorescence, agglutination, enzyme immunoassays) have been developed to detect specific host antibodies against crude and refined parasite antigens, but it is often difficult to distinguish between recent and chronic infections. Molecular biological techniques have been used to detect and characterize parasite isolates following the polymerase chain reaction (PCR) amplification of nuclear gene sequences (small subunit (18S) ribosomal DNA, internal transcribed spacer regions, and some maxicircle DNA regions).

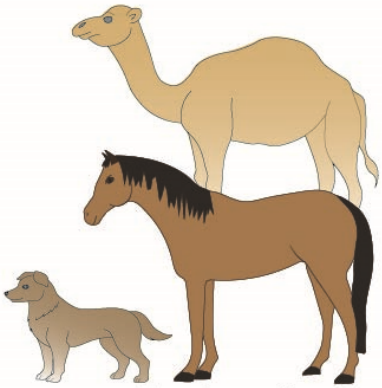
Treatment and control: A range of drugs have been used to treat clinical infections in animals, including trypanocides (quinapyramine, isometamidium chloride, suramin, cymelarsan), diamidines (diminazene aceturate) and even arsenic-based anthelmintics (melarsomine). Treatments have exhibited variable efficacies in different hosts (e.g. diminazene aceturate often ineffective in cattle, horses, pigs and elephants), and the incidence of drug-resistant strains of parasites appears to be increasing. Infections are deemed notifiable diseases in many countries with legislation governing the importation of livestock, diagnosis, quarantine, chemotherapy and chemoprophylaxis. There are no vaccines available as immunological responses are confounded by rapid changes in parasite surface coat antigens (variable surface glycoproteins). Preventive control measures adopted for *T. evansi* are based around controlling vector populations (using physical barriers, chemical insecticides or biological agents), while those adopted for *T. equiperdum* involve selecting uninfected horses for mating or promoting good hygiene during assisted matings. In many situations, the only effective control is achieved by culling infected animals.

Trypanosoma
(mammalian 'mechanica' species)
(e.g. *T. evansi, T. equiperdum*)

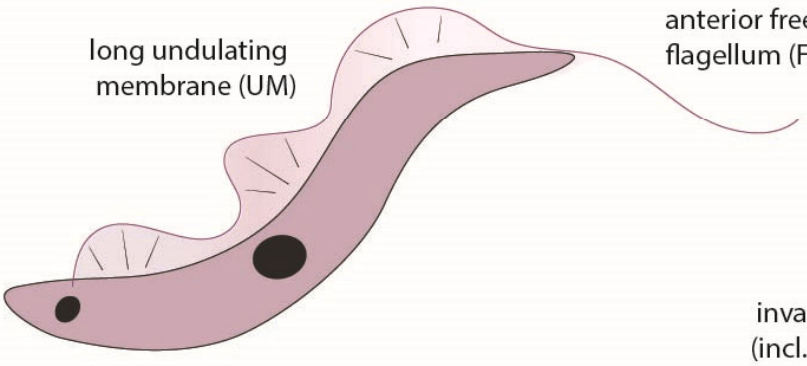


blood, tissues
(fever, anaemia,
jaundice, oedema
(surra))

division by
longitudinal
binary fission



Vertebrate Hosts
(mammals)



long undulating
membrane (UM)

anterior free
flagellum (FF)

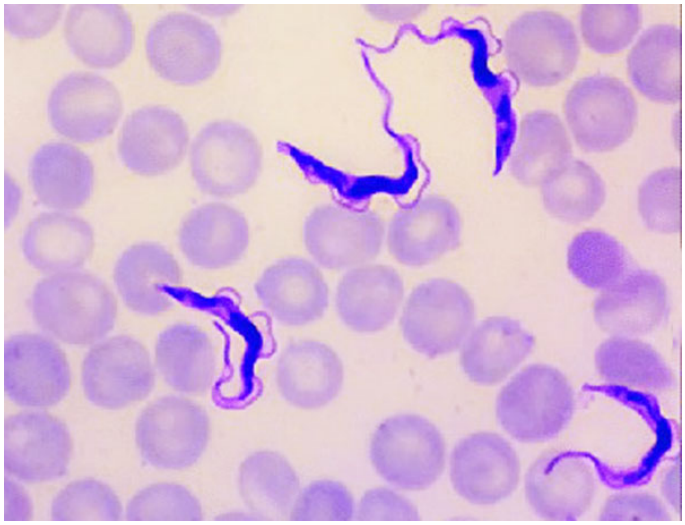
posterior
kinetoplast (K)

invade tissues
(incl. central
nervous system)

trypomastigote (15-36 µm)

T. evansi
mechanical transmission between hosts when
trypomastigotes contaminate mouthparts
of tabanid flies (sometimes vampire bats)

T. equiperdum
venereal transmission between equine
hosts via coitus



Trypanosoma evansi trypomastigotes