Surra

Murrina, Mal de Caderas, Derrengadera, Trypanosomosis, El Debab, El Gafar, Tabourit

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Importance

Surra, caused by *Trypanosoma evansi*, is an important protozoal disease of animals, particularly in tropical and semitropical regions. While clinical cases are most common in equids and camels, many mammals can be infected subclinically, and serious illnesses and deaths have been described, at least occasionally, in a wide variety of species. Surra also results in economic losses from decreased productivity in working animals, reduced weight gain, decreased milk yield, reproductive losses and the cost of treatment. Unlike most other pathogenic trypanosomes, *T. evansi* does not require tsetse flies for its transmission, and it is instead spread mechanically by various biting flies. Together with its extensive host range, this mode of transmission has allowed it to spread into many parts of the world, and to readily become established in new areas after its introduction. Morbidity and mortality can be high during such events, as well as when fully susceptible animals are moved into an endemic region. In the early 1900s, an outbreak in Mauritius killed almost all of the Equidae on the island. More recently, severe outbreaks have been reported in the Philippines, Indonesia and Vietnam.

Etiology

Surra is caused by the protozoal parasite *Trypanosoma evansi* (subgenus *Trypanozoon*, Salivarian section). The two currently known genetic types of this organism are type A, which is recognized by the usual PCR tests designed to detect *T. evansi*, and type B, which is not found by these tests and appears to be much less common. Isolates that could not be recognized as either type A or type B have also been described. Isolates of *T. evansi* can differ in virulence.

T. evansi is very closely related to both *Trypanosoma equiperdum*, which causes dourine, and *T. brucei*, one of the causative agents of African trypanosomiasis, and whether it should be considered a distinct species is controversial. Some genetic analyses suggest that *T. evansi* and *T. equiperdum* evolved independently from *T. brucei* while losing the ability to be transmitted by tsetse flies. Unlike *T. brucei*, these two organisms are usually monomorphic (in most hosts, they appear only as the slender form that replicates in mammals, and do not change to the short, stumpy form that infects tsetse flies); they proliferate only asexually (sexual reproduction occurs in the tsetse fly) and are thus less variable than *T. brucei*; and they have partially or completely lost some of the genetic elements found in *T. brucei*. However, the genetic distance between *T. brucei* and *T. evansi* or *T. equiperdum* is sometimes less than the difference between *T. brucei* isolates from different geographic areas. Various naming systems have been proposed as a result, including some that consolidate all three parasites under a single *Trypanosoma* species, either *T. brucei* or *T. evansi*, though there is, as yet, no consensus.

Species Affected

T. evansi has a wide host range and has been reported to infect all domestic animals as well as many other mammals and marsupials including various large ungulates, suids, felids, canids, primates, lagomorphs, small mammals and bats, either with or without disease. Some birds (e.g., young pigeons, chicks) can be infected experimentally, but their susceptibility in nature is uncertain.

The principal hosts and reservoirs of this organism can differ between regions, but camels, equids, water buffalo and cattle are generally considered to be the major hosts among domestic animals. Equids, Bactrian camels (*Camelus bactrianus*) and dromedaries (*Camelus dromedarius*) are highly susceptible to disease, while infections are usually mild or asymptomatic in cattle, water buffalo and related bovids in Africa and Latin America, though cattle and water buffalo regularly become ill in Asia. Many other mammals and marsupials are also susceptible to varying degrees; clinical cases have been reported in South American camelids, some cervids, sheep, goats, pigs, dogs and other canids, cats and other felids, elephants, Sumatran rhinoceroses (*Dicerorhinus sumatrensis sumatrensis*), Himalayan black bears (*Selenarctos thibetanus*), coati (*Nasua nasua*) and vampire bats (*Desmodus rotundus*), as well as experimentally infected wallabies (e.g., *Macropus agilis* and *Thylogale brunii*) and bandicoot rats (*Bandicota bengalensis*), among others. While understanding of wildlife maintenance

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hosts is incomplete, vampire bats are considered to be reservoirs in South America, and other mammals including peccaries (*Tayassu* spp.) and various small mammals, such as capybara (*Hydrochaeris hydrochaeris*), have been proposed to maintain *T. evansi* in some areas.

Zoonotic potential

Although *T. evansi* is not considered to be a significant zoonosis, rare clinical cases have been reported in humans. It is still uncertain whether all of these cases occurred in people who were unusually susceptible due to various genetic anomalies or other factors, or if the disease is underdiagnosed in people.

Geographic Distribution

Surveillance for *T. evansi* often relies on serology, and its precise distribution can be unclear, especially where its close relatives /*T. equiperdum* and *T. brucei* are also present; however, it is known to occur in parts of Africa, the Middle East, Asia, and Central and South America. *T. evansi* is also established in the Canary Islands off the coast of Africa, a part of Spain, although control programs appear to have greatly reduced its prevalence there in recent years.

The distribution of type A, type B and putative non-A/non-B organisms is incompletely understood; however, type A organisms seem to form the major population in all of the regions where *T. evansi* occurs. Type B organisms were originally described only in parts of Africa, but they have since been found in some Asian countries and might also occur elsewhere.

Transmission

Unlike its close relative *T. brucei*, *T. evansi* does not require a biological vector (tsetse flies) for transmission, and is instead transmitted mechanically by various biting insects. Members of the deerfly and horsefly family, Tabanidae, and flies in the genus *Stomoxys* are thought to be the most important vectors, but transmission by other biting insects (e.g., *Hippobosca* spp., mosquitoes in the family Culicidae, and midges in the family Ceratopogonidae) has been reported experimentally or suspected in the field, and might contribute to local spread. Sucking flies, such as *Musca* spp., might also spread *T. evansi* when they visit contaminated wounds. Other proposed vectors include ticks and leeches, such as buffalo leeches in Asia.

T. evansi can also be spread iatrogenically on contaminated needles or surgical instruments, mechanically via the bites of vampire bats (which can also be infected themselves) and via the ingestion of infected tissues by carnivores. Fresh tissues are most likely to be infectious to the latter hosts, as trypanosomes seem to disappear relatively quickly from the carcass after death. In one study, organisms could be found for up to 13-15 hours in the heart blood of mice, though their viability had decreased to ≤ 5% by this time.

Transplacental transmission has been demonstrated in several species, including ruminants, camels and equids, and the presence of the organism in milk and colostrum was reported in experimentally infected sheep. There is also speculation about the possibility of venereal transmission, given the presence of *T. evansi* in semen and the importance of this route for the closely related organism *T. equiperdum*. However, an attempt to infect ewes via experimentally infected rams, published in 2015, as well as an experiment where infected male rats were co-housed with female rats, found no evidence for sexual transmission of *T. evansi*.

Disinfection

There is limited need for disinfectants, due to the fragility of trypanosomes in the environment, and no studies appear to have specifically examined the disinfectant susceptibility of *T. evansi*. The closely related organism *T. brucei* can be inactivated by various agents including 0.05% sodium hypochlorite, 70% ethanol, 2% TriGene™, 0.1% hand soap, 2% formaldehyde and 0.05% glutaraldehyde. Exposure to 50°C (122°F) is reported to kill 100% of *T. brucei* trypomastigotes.

Incubation Period

The incubation period for surra can be highly variable, as some animals carry the organism subclinically for months or years before becoming symptomatic. Horses have been reported to develop clinical signs approximately one week to 2 months after exposure, with most cases appearing within the first month.

Clinical Signs

Surra can be an acute, subacute or chronic illness. Some animals can die rapidly, especially among highly susceptible species such as horses and camels, while others develop chronic illnesses that may last for months or years. Animals can also carry *T. evansi* subclinically.

Common clinical signs include fever (which may be intermittent in chronic cases), lymphadenopathy, weight loss or wasting, signs of anemia, lethargy and other nonspecific signs such as decreased milk yield. Dependent edema affecting sites such as the ventral abdomen is common, but some species, such as dogs and elephants, often have edema of the face and/or throat. In rare cases, equids may develop transient edematous patches called “silver dollar plaques” on the skin (usually over the ribs), a lesion that is more typically caused by *T. equiperdum* (dourine).

Some animals may also have ocular signs (e.g., conjunctivitis, keratitis, corneal opacity, anterior uveitis), jaundice and/or signs of a bleeding tendency such as petechial hemorrhages on mucous membranes, nasal or ocular hemorrhages, and bloody urine. There are occasional reports of cases with diarrhea or vomiting, respiratory signs (dyspnea, coughing) or urticaria. Several cats presented with the inability to stand or walk as a prominent sign, in addition to anorexia, anemia and various other signs of surra, but rapidly recovered normal ambulatory function once treatment was started. External or internal abscesses have been documented in some cats, though mainly in experimentally infected animals.

Neurological signs are also possible, particularly in the late stages, and ataxia, with gradually progressive paresis of the hindquarters accompanied by muscle atrophy, is reported to be a common indication of surra among horses in South America. Cardiac abnormalities such as ventricular arrhythmias and dilated cardiomyopathy have been seen in some dogs, and occasionally in other species (e.g., water buffalo), possibly as a complication of anemia, electrolyte imbalance or pericardial effusion. Some animals may also have signs associated with dysfunction of other internal organs, such as hepatitis or nephritis. Pregnant animals sometimes abort or give birth to stillborn or premature, low birthweight offspring, either with or without significant clinical signs in the dam. Reduced fertility from abnormal sperm, as well as rare cases of orchitis, has been reported in some species, including camels and ruminants. In addition, leukopenia caused by *T. evansi* may predispose animals to other conditions or exacerbate concurrent illnesses.

Post Mortem Lesions

The gross lesions tend to be nonspecific and often include wasting or emaciation of the carcass, subcutaneous edema, indications of anemia, enlargement of the spleen, liver and lymph nodes, and petechiae or other hemorrhages on internal organs. Muscle atrophy may be noted, particularly in the hindquarters. Icterus and evidence of hepatitis or nephritis may also be present, and fluid accumulation (e.g., ascites, hydrothorax) is sometimes found in body cavities. Cardiac lesions including hydropericardium, pericarditis and evidence of cardiomyopathy or myocarditis occur in some animals. Respiratory lesions (congestion, consolidation, edema, emphysema, hemorrhages and/or pneumonia) have also been seen occasionally. CNS lesions reported in some horses with neurological signs include subpial hemorrhages, swelling of the cerebral hemispheres with flattened gyri, and severe edema and malacia, with the white matter becoming yellow, gelatinous and friable.

Infected camel fetuses that were either aborted or stillborn had similar lesions that included subcutaneous edema, hemolyzed blood in the body cavities, and moderately to severely congested lungs (bronchopneumonia), liver, spleen, kidneys and/or intestinal mucosa, with pale necrotic foci in the liver and enlarged, soft or pulpy kidneys.

Diagnostic Tests

A presumptive diagnosis can be made if organisms consistent with *T. evansi* are found by direct examination of the blood, lymph nodes or edema fluid, or in various tissues at necropsy, and other trypanosomes do not exist in the area. Blood should be collected from live animals during a febrile period. Parasites may be detected in thick or thin stained blood smears, as well as in wet blood films, where motile organisms can be observed. Thick films have the advantage of being able to detect small numbers of parasites, but the morphology of the parasite is difficult to determine. In thin smears, *T. evansi* normally appears as a monomorphic, slender parasite, a characteristic that helps distinguish it from trypanosomes other that *T. equiperdum*. However, pleomorphic forms of *T. evansi* have been observed in livestock during certain outbreaks (organisms that resembled *T. vivax* during the 2008 surra outbreak in Spain) or in low numbers in some individual animals (e.g., < 0.5% of the parasites in some camels), and seem to be prominent in certain species such as cats and some monkeys.

Repeated sampling may be necessary, as trypanosomes are sometimes difficult to find, especially in mild or subclinical cases, and parasitemia is often intermittent in chronically infected animals. Detection can also be improved with parasite concentration techniques such as mini anion-exchange chromatography, hemolysis methods that use sodium dodecyl sulphate (SDS) to destroy the erythrocytes (wet blood film clarification or hemolysis centrifugation), hematocrit centrifugation (Woo method) or the dark-ground/phase-contrast buffy coat technique (Murray method). Immunostaining can help visualize the organisms in tissues, and has been reported to detect trypanosomes in the brain of some animals with CNS signs even when they were not visible in hematoxylin and eosin stained sections.

Clinical cases can also be diagnosed by PCR, and additional genetic tests such as loop-mediated isothermal amplification (LAMP) methods have been described in the literature. Some PCR tests identify the organism only to the level of the subgenus *Trypanozoon*, while others, such as RoTat1.2 VSG PCR for type A *T. evansi*, or PCRs for type B organisms based on either the JN 2118HU VSG or a mobile genetic element (EVAB), can be suggestive of *T. evansi*. However, most *T. equiperdum* also contain the RoTat1.2 VSG gene (if this organism is considered a separate species), while *T. brucei* is very diverse and incompletely studied, and the possibility that some isolates might react in the type A assay has not been ruled out. A few *T. brucei* isolates are already known to react in type B *T. evansi* tests. Thus, absolute confirmation that an organism is *T. evansi* generally requires a level of genetic analysis not practical for routine diagnosis. Various antigen detection tests have also been published, but are not in wide use and require further evaluation.

Serological tests that can detect antibodies to *T. evansi* include various ELISAs, a card agglutination test (CATT), immunofluorescence assays and the trypanolysis test, which can be used to confirm positive results from other assays. The most commonly used tests are ELISAs and the CATT. The latter test detects IgM and is particularly useful early in the course of the disease. Immunofluorescent assays are generally employed only with small numbers of samples, as they are labor-intensive and require experience to read, while technical aspects and expense limit the use of the trypanolysis test and it is available at very few laboratories. Cross-reactivity, including reactions to nonpathogenic species such as *T. melophagium* in sheep or *T. theileri* in cattle, can be an issue in serological tests. Validated tests may be unavailable for some hosts.

Animal inoculation studies in rats or mice can be used, if necessary, to detect low levels of parasites, such as when importing an animal into a surra-free regions. For animal welfare reasons, they are not generally recommended unless the need is critical.

Treatment

Surra can be treated with antiparasitic (trypanocidal) drugs, though cases with neurological signs can be very difficult to treat due to the poor penetration of most of these drugs into the CNS. Depending on the dose and other factors, treatment may be clinically curative without completely eliminating the parasite, and relapses are possible. Drug resistance is a significant issue in many regions. Treatment protocols for less common hosts should be chosen carefully, as the efficacy and toxicity of a particular drug may differ between species.

Treatment may or may not be permitted in countries where *T. evansi* is not endemic.

Control

Disease reporting

Veterinarians who encounter or suspect a *T. evansi* infection should follow their national and/or local guidelines for disease reporting. In the U.S., state or federal veterinary authorities should be informed immediately.

Prevention

Quarantine and testing of imported animals helps exclude surra from uninfected regions. The risk that *T. evansi* will become endemic if infected animals are missed is high, due to the large number of potential hosts and the organism's ability to be transmitted mechanically by many common biting insects. Nevertheless, eradication was successful in a few instances when the outbreak was recognized early. In some cases, these outbreaks were controlled by quarantines, movement controls and the slaughter of infected animals. In 2008, however, an outbreak among camels and equids on an isolated farm in Spain was eradicated by treating the infected camels, with isolation and monitoring in a closed stable for 6 years. Two equids were also treated but had to be euthanized after 4 months due to incomplete responses. An outbreak in France in 2006 was similarly eradicated by isolating and treating camels but euthanizing seropositive sheep.

It is difficult to control the biting flies that transmit *T. evansi* in endemic areas, but some degree of protection might be provided by insecticides/ repellents, traps, insect screens/netting in stables, and other controls. In one outbreak, most cases occurred among horses kept in open paddocks, while horses in nearby fly-proof stables were spared. Flies are most infective soon after feeding on an infected host (e.g., in the first half hour), and the highest probability of transmission is to nearby hosts; tabanids are persistent feeders and do not usually leave one animal to bite another more than 50 meters away. For this reason, it is useful to separate infected from uninfected animals, and to keep highly susceptible species away from herds of cattle, water buffalo or other animals that may be infected subclinically. In South America, animals should also be protected from vampire bats.

Antiparasitic drugs are sometimes used routinely to protect susceptible animals in endemic regions; however, this is likely to contribute to drug resistance. Carnivores and omnivores should not be allowed to eat the carcasses of infected animals.

Morbidity and Mortality

The consequences of infection with *T. evansi* can vary considerably, and include asymptomatic infections, chronic illnesses of varying severity, and acute cases that may be rapidly fatal without treatment. Factors that can affect the severity of the illness include the strain of *T. evansi*, the host species, and various characteristics of the individual, such as previous exposure to trypanosomes, concurrent infections and general health. All ages can be affected when a population is first exposed to this organism; however, clinical cases in endemic areas tend to be more common in young animals. They are particularly frequent in young camels soon after weaning. Stressors (e.g., malnutrition, concurrent illnesses, transport, overwork) may result in clinical signs in subclinically infected animals.

Horses and camels are generally considered to be the most susceptible livestock and sometimes develop life-threatening acute illnesses. The most severe outbreaks, often with morbidity and mortality rates of 50% or more, usually occur when these hosts are exposed to *T. evansi* for the first time. Donkeys and mules seem to be more resistant to clinical signs. Bovids are often asymptomatic carriers; however, cattle and water buffalo in Asia may experience significant outbreaks, especially on first exposure, with reported mortality rates sometimes ranging from 10% to >90%. Why these animals seem to be less affected by surra in Africa and South America is unclear.

Sporadic illnesses and deaths have been reported in other domestic animals and captive wildlife, and probably also affect free-living wildlife occasionally, though many infections seem to be mild or asymptomatic. Cases in some species, such as Asian elephants (*Elephas maximus*), mostly seem to occur after exposure to *T. evansi* carriers such as cattle and buffalo. In Asia, severe outbreaks have been reported in Timor rusa deer (*Rusa timorensis*) and hog deer (*Axis porcinus*).

Information about surra in dogs and cats is still incomplete, though there are a number of published case reports of severe or fatal illnesses in individual dogs, and one report described an outbreak on a South American farm with 38 dogs, where about half of the dogs died before a veterinarian was consulted. Of the remaining 18 animals, 15 responded clinically to treatment, one died, and two had rapid return of parasitemia within a week. One of the latter dogs was eventually treated successfully with a different drug, though the other died of drug toxicity. Other reports describing surveillance of dogs by serology, microscopy or PCR suggest that mild or subclinical infections might not be

uncommon in this species, with reports of seropositivity ranging from < 5% to 25% or more in some regions. There are also some reports of clinical cases in large felids, but very few cases have been published in domestic cats, for reasons that are unclear.

Public Health

Humans possess innate resistance to many species of trypanosomes, including *T. evansi*, due to the trypanolytic activity of the human serum protein apolipoprotein L-I. Nevertheless, there have been a few reports of clinical cases caused by this organism. Whether such illnesses are underestimated due to lack of diagnostic testing or occur only under unusual circumstances (e.g., rare genetic anomalies) is currently unclear. Some *in vitro* research also suggests that *T. evansi* isolates may differ in their innate resistance to human serum and that some isolates might become more resistant with prolonged exposure to human serum. Most of the patients described to date, including one with an apolipoprotein deficiency, were treated successfully with suramin, though deaths were also seen.

One historical, laboratory-acquired infection was reported by several sources as having occurred in 1977, but appears to be much older, and whether the organism was actually *T. evansi* is unclear. This person, who was apparently exposed while mouth pipetting contaminated blood, was reported to have developed insomnia, memory loss, tachycardia and an enlarged liver, spleen and lymph nodes, which resolved after treatment with an antitrypanosomal drug.. A definitive diagnosis was established in a few subsequent cases by PCR and/or sequencing, in addition to the observation of trypanosomes in the blood. One of these cases occurred in a 45-year-old Indian farmer who was later found to have a genetic defect in apolipoprotein L1. His symptoms included intermittent fever, chills, sweats and neurological signs. Another case, with few reported details, was reported from Sri Lanka in a patient with a headache and intermittent fever. More recently, *T. evansi* was found in an apparently healthy Vietnamese woman with normal apolipoproteins who developed a fever, severe headache, arthralgia, hepatomegaly and pancytopenia about 3 months after giving birth.

A few additional cases were also suspected to be caused by *T. evansi*, but were diagnosed only by the observation of trypanosomes in the blood. They occurred in countries such as Egypt and India, where *T. evansi* is thought to be the only pathogenic trypanosome. The patients exhibited various symptoms, including neurological signs in one person and recurrent episodes of fever in another. While there are few details about most of these cases, one published report described an HIV-infected pregnant woman who also had disseminated tuberculosis and an upper respiratory tract infection, and presented with severe anemia, hepatosplenomegaly, pedal edema and a history of abdominal pain and persistent low grade fever. The trypanosomes detected in her blood included both slender forms and short, stumpy forms, which are not usually characteristic of *T. evansi* and might indicate another organism; however, this finding does not necessarily rule it out either, as morphological anomalies have been seen occasionally in *T. evansi*-infected animals.

A few surveys examined people in endemic regions for evidence of infection with *T. evansi*. In Egypt, PCR and microscopic examination of blood smears revealed no evidence for this organism in camel owners, though 10-46% of their camels were infected. Another study, from the village in India where the farmer with the apolipoprotein L1 defect lived, also did not find any individuals with trypanosomes in the blood; however, some people were seropositive in the livestock CATT. Because this test has not been validated for humans, and cross-reactivity could also be an issue, the significance of this finding is still unclear. A later study from India reported that some people in another rural area were also seropositive with this test. In addition, these researchers tested blood samples with 3 PCR assays for different trypanosome proteins, all of which should have detected *T. evansi*. They reported that 3% of their subjects were positive in one test, but negative with the other two tests. Whether this discrepancy was due to differing test sensitivities, as suggested by the researchers, or for another reason (including false positives), appears uncertain. There was no mention of clinical signs in any of the individuals with positive PCR tests, and no further investigations were apparently done.

Internet Resources

Center for International Collaboration on Agricultural Research for Development (CIRAD), France. Atypical-Human Trypanosomoses.

Food and Agriculture Organization of the United Nations (FAO). Uilenberg G. A field guide for the diagnosis, treatment and prevention of African animal trypanosomosis (older reference, but contains some useful information including life cycles and trypanosome morphology)

The Merck Veterinary Manual

World Organization for Animal Health (WOAH)

WOAH Manual of Diagnostic Tests and Vaccines for Terrestrial Animals

WOAH Terrestrial Animal Health Code

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