

## DOURINE

(Slapsiekte, el Dourin, Mal de Coit, Beschalseuche, Covering Disease)

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### Definition [top](#)

Dourine is a chronic trypanosomal disease of Equidae. The disease is transmitted almost exclusively by coitus and is characterized by edematous lesions of the genitalia, nervous system involvement, and progressive emaciation.

### Etiology [top](#)

Dourine is caused by *Trypanosoma equiperdum* (Fig. 48) (Doflein, 1901), a protozoan parasite related morphologically and serologically to *T. brucei*, *T. rhodesiense*, and *T. gambiense* (of the subgenus *Trypanozoon* of the *Salivarian* section of organisms of the pathogenic genus *Trypanosoma*). Different strains of the parasite vary in pathogenicity (5).

## Host Range [top](#)

Dourine is typically a disease of horses and donkeys. Positive CF tests have been obtained from zebras, although it has not been shown that zebras can be infected with *T. equiperdum* or transmit the disease. The organism has been adapted to a variety of laboratory animals (5,6,9).

Improved breeds of horses seem to be more susceptible to the disease. The disease in these animals often progresses rapidly and involves the nervous system. In contrast, native ponies and donkeys often exhibit only mild signs of the disease. Infected male donkeys, which may be asymptomatic, are particularly dangerous in the epidemiology of the disease, for they may escape detection as carriers.

## Geographic Distribution [top](#)

Once widespread, this disease has been eradicated from many countries. It is currently present in most of Asia, southeastern Europe, South America, and in northern and southern Africa (3)

## Transmission [top](#)

This venereal disease is spread almost exclusively by coitus. Organisms are present in the urethra of infected stallions and in vaginal discharges of infected mares. The organism may pass through intact mucous membranes to infect the new host. Infected animals do not transmit the infection with every sexual encounter, however. As the disease progresses, trypanosomes periodically disappear from the urethra or vagina; during these periods, the animals are noninfective. Noninfective periods may last for weeks or months and are more likely to occur in the later stages of the disease. Thus, transmission is most likely early in the disease process.

It is possible for mares to become infected and pregnant after mating with an infected stallion. Foals born to infected mares may be infected. It is unclear if this occurs in utero or during birth. Because trypanosomes may occur in the milk of infected mares, these foals may be infected per os during birth or by ingestion of infected milk. Foals infected in this way may transmit the disease when mature and develop a lifelong positive CF titer. This method of disease transmission is rare, however. Some foals may acquire passive immunity from colostrum of infected mares without becoming actively infected; in such foals, the CF titer declines, and the animal becomes seronegative by 4 to 7 months of age. Although the possibility of noncoital transmission remains uncertain, it is supported by

sporadic infections in sexually immature equids (1,3,5).

### **Incubation Period** [top](#)

The incubation period is highly variable. Clinical signs usually appear within a few weeks of infection but may not be evident until after several years (1,5,7).

### **Clinical Signs** [top](#)

Clinical signs vary considerably, depending on the virulence of the infecting strain, the nutritional status of the infected animal, and the presence of other stress factors. The strain prevalent in southern Africa (and formerly in the Americas) is apparently less virulent than the European, Asian, or north African strains and produces an insidious, chronic disease. In some animals, clinical signs may not be apparent for up to several years (so-called latent infection). Clinical signs may be precipitated by stress in these animals.

In mares, the first sign of infection is usually a small amount of vaginal discharge, which may remain on the tail and hindquarters. Swelling and edema of the vulva develop later and extend along the perineum to the udder and ventral abdomen. There may be vulvitis and vaginitis with polyuria and other signs of discomfort such as an elevated tail. Abortion is not a feature of infection with mild strains, but significant abortion losses may accompany infection with a more virulent strain.

In stallions, the initial signs are variable edema of the prepuce and glans penis (Fig. 49), spreading to the scrotum and perineum and to the ventral abdomen and thorax. Paraphimosis may be observed. The swelling may resolve and reappear periodically. Vesicles or ulcers on the genitalia may heal and leave permanent white scars (leukodermic patches). Transient cutaneous plaques are a feature of the disease in some locations and strains but not others. When they occur, they are pathognomonic. Conjunctivitis and keratitis are often observed in outbreaks of dourine and may be the first signs noted in some infected herds.

Nervous disorders may be seen soon after the genital edema or may follow by weeks or months. Initially these signs consist of restlessness and the tendency to shift weight from one leg to another followed by progressive weakness and incoordination and ultimately by paralysis and recumbency. Anemia and emaciation sometimes accompany development of clinical signs even though the appetite remains unaffected.

Dourine is characterized by stages of exacerbation, tolerance, or relapse that may vary in duration and occur several times before death or recovery. The course of

the disease may last several years after infection with a mild strain. Experimentally, horses have survived for up to 10 years after infection. The course is apparently more acute in the European and Asian forms of the disease in which the mortality rate is higher (1,5).

### **Gross Lesions** [top](#)

Anemia and cachexia are consistent findings in animals that have succumbed to dourine. Edema of the genitalia and ventral abdomen become indurated later in the course of the disease. Chronic lymphadenitis of most lymph nodes may be evident. Perineural connective tissue becomes infiltrated with edematous fluid in animals with nervous signs, and a serous infiltrate may surround the spinal cord, especially in the lumbar or sacral regions (1,5,7).

### **Mortality** [top](#)

Although the course of the disease may be long, it is usually fatal. Uncomplicated dourine does not appear to be fatal unless the nervous system is involved. The progressive debilitation associated with the neurological manifestation of the disease predisposes infected animals to a variety of other conditions. Because of the long survival time in some experimental cases, reports of recovery from dourine should be regarded with skepticism.

### **Diagnosis** [top](#)

#### **Field Diagnosis** [top](#)

Diagnosis on physical signs is unreliable because many animals develop no sign. When signs are present, however, they are suggestive of a diagnosis of dourine. If "silver dollar plaques" occur, they are pathognomonic for dourine.

#### **Specimens for the Laboratory** [top](#)

Detection of trypanosomes is highly variable and is not a reliable means for diagnosis of dourine. The following specimens should be submitted: serum, whole blood in EDTA, and blood smears.

#### **Laboratory Diagnosis** [top](#)

A reliable complement-fixation test (CFT) has been the basis for the successful eradication of dourine from many parts of the world. The antigen used in the CFT

is group-specific, leading to cross-reactions with sera of horses infected with *T. brucei*, *T. rhodesiense*, or *T. gambiense*. The test is therefore most useful in areas where these parasites do not occur. Indirect fluorescent antibody, card agglutination, and enzyme-linked immunosorbent assay test (ELISA) have also been developed for dourine but have not replaced the CFT (1,3,4,5,6,10,11).

### Differential Diagnosis [top](#)

The perineal and ventral abdominal edema characteristic of dourine may also be seen in horses with anthrax. These signs may also resemble infection with equine infectious anemia or equine viral arteritis. Coital exanthema and purulent endometritis (as occurs in contagious equine metritis) should also be considered.

### Treatment [top](#)

Although there are reports of successful treatment with trypanocidal drugs (e.g., suramin at 10 mg/kg IV, quinapyramine dimethylsulfate at 3-5 mg/kg SC), treatment is more successful when the disease is caused by the more virulent (European) strains of the parasite. In general, treatment is not recommended for fear of continued dissemination of the disease by treated animals (1,6). Treatment may result in inapparent disease carriers and is not recommended in a dourine-free territory.

### Vaccination [top](#)

Immunity to trypanosomiasis is complicated. *T. equiperdum* has the ability periodically to replace major surface glycoprotein antigens, which is a strategy supporting chronic infections (2). No method of immunization against dourine exists at present.

### Control and Eradication [top](#)

The most successful prevention and eradication programs have focused on serologic identification of infected animals. Infected animals should be humanely destroyed or castrated to prevent further transmission of the disease. Some geldings may still show service behavior and constitute a risk. All equids in an area where dourine is found should be quarantined and breeding should be stopped for 1 to 2 months while testing continues.

Sanitation and disinfection are ineffective means of controlling the spread of dourine because the disease is normally spread by coitus.

**Public Health** [top](#)

Humans are not susceptible to infection with *T. equiperdum*.

**GUIDE TO THE LITERATURE** [top](#)

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