**African animal trypanosomes**

**Life Cycles**

Insects are usually involved in the natural transmission of the African pathogenic trypanosomes with which we are concerned in this field guide. When this is the case, **the life cycle has two phases, one in the insect vector and one in the mammalian host. Transmission by insects may be cyclical by tsetse flies,**[\*](http://www.fao.org/docrep/006/x0413e/X0413E02.htm#ref1.1.1) ***Glossina* species**, or **mechanical by other biting flies** (but apart from transmitting trypanosomes cyclically, tsetse flies can also act as mechanical vectors).

\* We assume that the reader has some knowledge of tsetse flies, as there simply is no scope in this manual for going into details. At present 23 different species and eight subspecies of the genus *Glossina* are recognized, belonging to three groups: *fusca* group or forest group, *palpalis* group or riverine group, and *morsitans* group or savannah group.

**Cyclical transmission**

When a tsetse fly hatches from its pupal case it is free from trypanosomes. Until its first bloodmeal, it is called a **teneral fly**. It acquires a trypanosomal infection when feeding on a parasitaemic (= having parasites in the circulating blood) mammalian host. The trypanosomes undergo a cycle of development and multiplication in the digestive tract of the fly until the **infective metacyclic trypanosomes** **(metatrypanosomes)** are produced. Different trypanosome species develop in different regions of the digestive tract of the fly, and the metatrypanosomes occur either in **the biting mouthparts** or the **salivary glands**. The period from ingesting infected blood to the appearance of these infective forms varies from **one to three weeks**; once infective metatrypanosomes are present the fly remains infective for the remainder of its life. During the act of feeding the fly penetrates the skin with its proboscis. By the rupture of small blood vessels a pool of blood is formed in the tissues and the fly injects saliva to prevent coagulation. Infection of the host takes place at this stage, with infective metacyclic trypanosomes in the saliva.

Although no classical sexual processes in the life cycle of trypanosomes have been described, it has been shown that exchange of genomic material (DNA) between trypanosomes sometimes occurs in the tsetse fly, although it is not clear how significant this is.

1. **Life cycle in the mammalian host**

The infective metatrypanosomes undergo development and multiplication at the site of infection where **a swelling or chancre** may be detected in the skin, and finally the mature blood trypanosomes (or trypomastigotes) are released **via lymph vessels and lymph nodes** into the **blood circulation.**

Reproduction in the mammalian host occurs through a process of **binary division**.

* **Trypanosomes feed by** absorbing nutrients, through their outer membrane, from the body fluids of the host. The proteins, carbohydrates and fats are digested by enzyme systems within their **protoplasm.**
* **Oxygen** dissolved in the tissue fluids or blood plasma of their host is absorbed in a similar manner, to generate the energy necessary for the vital processes.
* **Waste products** are disposed of by a reverse process, through the outer membrane, into the body fluids of the host. They include carbon dioxide formed during respiration, as well as more complex metabolic products.

1. **Life cycle in the tsetse fly**

Blood stream forms (trypomastigotes) ingested by the fly undergo considerable changes, in morphology as well as in their metabolism. They change into long slender forms called **epimastigotes**, which multiply and finally give rise to the infective **metatrypanosomes.**

**Mechanical transmission**

1. **By biting insects**

The process is purely mechanical. A biting insect passes the blood forms from an infected animal to another in the course of interrupted feeding. The time between the two feeds is crucial for effective transmission because the trypanosomes die when the blood dries. The importance of this mode of transmission is variable from place to place, depending on the numbers of hosts and biting insects present, and also on the species of trypanosome. Large biting insects such as tabanids carry more blood and are more likely to act as mechanical vectors than for example mosquitoes. (Tsetse flies themselves can of course also act as mechanical vectors.) This mode of transmission has proved to be sufficiently effective to maintain *Trypanosoma vivax* and *Trypanosoma evansi* in South and Central America, and the latter species in North Africa and Asia as well. No tsetse flies occur outside tropical Africa, apart from small tsetse pockets in the southwest of the Arabian peninsula.

1. **By iatrogenic**\***means**

This can occur when using the same needle or surgical instrument on more than one animal, at sufficiently short intervals that the blood on the needle or instrument does not dry. It is not an uncommon occurrence when animals are vaccinated or treated by injection, or when blood is collected from several animals in a row, without changing or disinfecting needles or pins. It may also occur when several animals are subjected at short intervals to a surgical intervention (dehorning, castration, etc.) without properly disinfecting the instruments.

\* Iatrogenic transmission means that it is caused by the (veterinary) operator. Iatrogenic infections are induced (involuntarily) by the operator using unhygienic procedures, such as contaminated instruments.

**3- Transmission by other means**

* It is well known that carnivores may be infected with *T. evansi* and *T. brucei* by ingesting meat or organs from infected animals, as long as these are still sufficiently fresh to contain live trypanosomes. Infection occurs probably through the mucosa of the mouth (in which moreover bone splinters make wounds through which the parasites penetrate even more easily).
* Transmission of *T. evansi* in Latin America by the bites of vampire bats is common. These bats become infected by ingesting blood from infected horses or cattle, the trypanosomes multiply in the bats and these are thereafter able to transmit the disease to healthy animals. The trypanosomes apparently pass readily through the oral mucosa of the bat in both directions.
* All trypanosome species are occasionally transmitted congenitally, from the mother to the offspring, either through the placenta while the foetus is still in the uterus, or when bleeding occurs during birth. Congenital transmission of *T. vivax*, for example, has been observed in Latin America as well as in Africa, but its real importance is not well known.
* Venereal transmission is the normal means by which dourine of equines, caused by *Trypanosoma equiperdum*, is propagated. Because of its presence in the mucous exudate of penis and sheath of the stallion and the vaginal mucus of the mare, *T. equiperdum* is easily transmitted directly during copulation from an infected to a healthy animal and its geographical distribution is not restricted to specific climatic conditions. This species is essentially a tissue parasite and causes at most very low parasitaemias in the circulating blood of equines.

**The pathogenic trypanosomes**

Table 1 indicates the occurrence of the pathogenic African trypanosomes in common domestic animals. Also included are the two non-pathogenic species of *theileri* group mentioned above, which may give rise to confusion; they belong to the subgenus *Megatrypanum* and the section Stercoraria.

Within each species there is a great variety of strains which may be classified in a number of ways. One way of classification is according to the pathogenicity, virulence, or disease-producing potential of the strain, and this can be extremely variable. The course and outcome of trypanosomosis is in addition influenced by a whole range of coexisting factors and influences, which combine and react to exert profound effects. The only indication of pathogenicity in Table 1 is that in the second column the list of livestock species is tentatively ranked in descending order of importance. In the fourth column the susceptibility of the common laboratory animals is indicated, which can be of some importance in certain diagnostic procedures.

**Table 1. The occurrence of African trypanosomes in domestic animals**

|  |  |  |  |
| --- | --- | --- | --- |
| **Trypanosome species** | **Domestic animals affected** | **Reservoir hosts** | **Laboratory animals** |
| *T. congolense* | Cattle, camels[\*](http://www.fao.org/docrep/006/x0413e/X0413E02.htm#note2.1), horses, dogs, sheep, goats, pigs | Several groups of wild mammals | Rats, mice, guinea pigs, rabbits |
| *T. simiae* | Pigs | Wart hog, bush pig | Rabbits, monkeys |
| *T. godfreyi* | Pigs | Wart hog | None susceptible |
| *T. vivax* | Cattle, sheep, goats, domestic buffalo, horses | Several groups of wild mammals | Usually none susceptible |
| *T. uniforme* | Cattle, sheep, goats | Various wild ruminants | None susceptible |
| *T. brucei brucei* | Horses, camels[\*](http://www.fao.org/docrep/006/x0413e/X0413E02.htm#note2.1), dogs, sheep, goats, cattle, pigs | Several groups of wild mammals | Rats, mice, guinea pigs, rabbits |
| *T. brucei gambiense, T. brucei rhodesiense* | Human sleeping sickness; affect domestic animals as *T. brucei brucei*[\*\*](http://www.fao.org/docrep/006/x0413e/X0413E02.htm#note2.2) | Several groups of wild mammals (particularly *T. brucei rhodesiense*) | As for *T. brucei brucei* (after initial adaptation where *T. brucei gambiense* is concerned) |
| *T. evansi* | Camels, horses, dogs, domestic buffalo, cattle | Several wild mammals in Latin America | As for *T. brucei brucei* |
| *T. equiperdum* | Horses, donkeys, mules | None known | As for *T. brucei brucei* (after initial adaptation) |
| *T. theileri* and *T. ingens* (subgenus *Megatrypanum*) | Cattle, domestic buffalo[\*\*\*](http://www.fao.org/docrep/006/x0413e/X0413E02.htm#note2.3) (not pathogenic) | Various wild ruminants | None |

\*   Camels are highly susceptible to *T. congolense* and to *T. brucei*, but do not usually penetrate into tsetse country.

\*\*   In particular, the behaviour of *T. brucei rhodesiense* in domestic animals is quite similar to that of *T. b. brucei*, whereas *T. brucei gambiense* is on the average more chronic (as it is in humans).

\*\*\* Of the two only *T. theileri* has been reported from domestic buffalo.

**EPIDEMIOLOGY[13](http://www.fao.org/docrep/006/x0413e/X0413E02.htm" \l "ref1.4.1)**

So many factors intervene in the epidemiology of African trypanosomosis that an entire book could be written on the subject. In the context of this guide we cannot possibly discuss all the possible scenarios and have to restrict ourselves to the main factors.

For general principles of epidemiology, we refer to books such as those by Putt *et al.* (1986) and Martin, Meek and Willeburg (1987). It is essential to be familiar with the fundamental terms used in epidemiology, for example, the difference between *prevalence* and *incidence*. It is also essential to distinguish between *disease* as opposed to *infection*; particularly trypanotolerant animals may be infected without having clinical disease, in other words, they may be healthy carriers. *Prevalence* is the frequency of *existing* cases of disease, or of infection, at a certain time. *Incidence* indicates the frequence of *new* cases within a certain period of time. Where it is possible to determine prevalence and incidence by certain tests, it is important to use a correct sample size, and for those with some statistical background considerations on sample size have been included at the end of this manual.

One all-important factor is whether we are dealing with tsetse-transmitted trypanosomosis or not. If so, much depends on the *Glossina* species responsible for transmission. There is a large body of experimental evidence to show that host preferences and vector capacity differ greatly between groups and species of *Glossina*. For example, recent laboratory experiments with teneral tsetse flies in Burkina Faso have shown higher mature infection rates with the savannah type of *T. congolense* in *G. morsitans morsitans*[14](http://www.fao.org/docrep/006/x0413e/X0413E02.htm#ref1.4.2) and *G. morsitans submorsitans* (both belonging to the savannah group of tsetse) than in *G. palpalis gambiensis* and *G. tachinoides* (*palpalis* or riverine group). *G. morsitans submorsitans* was the best vector of both the savannah and the riverine-forest types of *T. congolense*, while *G. m. morsitans* had the lowest vectorial capacity for the riverine-forest type; *G. palpalis gambiensis* was the least effective vector for the savannah type of *T. congolense*.

Savannah species are on the whole better vectors of the pathogenic trypanosomes of livestock. Also, where savannah tsetse (*morsitans* group) are the vectors, the risk of contracting the disease is widespread, although their distribution area in the dry season decreases. When riverine species are the culprits (in many parts of West and Central Africa), transmission occurs particularly along rivers with dense vegetation along the banks (the so-called gallery forests). Some of the forest species (*fusca* group) are confined to dense forest and are therefore not normally in contact with livestock, but some also occur on the forest edge and may locally play a significant role as vectors of AAT.

Populations of savannah species feed mainly on mammalian hosts, particularly bovids (antelopes, buffalo, cattle, sheep, goats) and suids (wart hog and bush pig), while riverine tsetse have a very wide range of preferred hosts, including reptiles and humans. Zebras, certain antelopes and also carnivores have little attraction for tsetse flies. The proportion of a tsetse population found infected with pathogenic trypanosomes therefore depends not only on its vector capability, but also on the hosts on which it mainly feeds. For instance, reptiles do not carry pathogenic trypanosomes,[15](http://www.fao.org/docrep/006/x0413e/X0413E02.htm#ref1.4.3) and there are also major differences between suids and bovids, as the former will infect the flies particularly with *T. simiae* and *T. godfreyi*, while bovids are mainly the source of *T. vivax* and *T. congolense*).[16](http://www.fao.org/docrep/006/x0413e/X0413E02.htm#ref1.4.4)

Herd management is also important. Daily activity patterns of the tsetse species involved and the grazing patterns of the herds are of great influence. If the herds graze on infested sites at the time of the day that the flies are most active, transmission will occur more frequently. In the Sahel zone, many of the cattle owners (e.g. the Baggara and the Fulani) are transhumant, because in the dry season the pastures and watering places in the Sahel are insufficient to maintain the large livestock populations. The zebu herds, accompanied by small ruminants, are then moved hundreds of kilometres to the south, where they may enter tsetse belts and contract AAT. Although the owners generally know the danger and recognize and associate tsetse flies with the disease, they are not always able to avoid infested areas. Particularly during dry years the southward migration is greater than usual, and the owners may deliberately choose between the risk of starvation of the herd and of tsetse-transmitted trypanosomosis.[17](http://www.fao.org/docrep/006/x0413e/X0413E02.htm#ref1.4.5) At the beginning of the rainy season the transhumants start to move back to the Sahel pastures, in order to arrive when these are sufficiently lush. The animals infected in the tsetse belts are diseased by the time they reach the rainy season pastures, and may even die before, the physical effort of transhumance adversely affecting the outcome. Unless the animals are treated in time, great losses may occur and when there are large numbers of tabanids and other biting flies around during the rains, the infection may be further transmitted mechanically outside the tsetse belts.

Species and breed susceptibility are of course of great importance. Whereas in tsetse areas trypanosomosis is a very obvious problem in susceptible livestock, it may remain practically inapparent where trypanotolerant breeds are concerned (even if these breeds may not be very productive when challenge is high).

The risk to susceptible ruminants living in comparatively free areas surrounded by tsetse-infested regions, or at the edge of tsetse-belts, varies from year to year. Generally, tsetse fly populations during wet years will increase, spread, and persist during the dry season in areas from where they disappear in dry years.

Also, animals used for transporting persons or goods are sometimes particularly at risk. For example, although the classical breeding areas of camels in Africa are north of the tsetse belts, individual camels are used for the transport of merchandise to transhumant animal owners in their dry season grazing grounds in or near tsetse belts, and these camels risk contracting tsetse-transmitted trypanosomosis. The same applies to the riding horses of travellers and of the transhumant cattle owners. Interestingly, in recent years there has been a tendency in Kenya to start keeping camels as far south as the Masai areas, because of the great losses in cattle caused by the severe droughts in the 1980s; this will of course increase contact between camels and tsetse fly and result in more disease.

The epidemiology of non tsetse-transmitted trypanosomosis (*T. evansi*, *T. vivax*[18](http://www.fao.org/docrep/006/x0413e/X0413E02.htm#ref1.4.6)) is also influenced by many factors. There may be seasonal outbreaks, where the populations of biting flies (Tabanids, stable flies, etc.) are influenced by important seasonal climatic differences. The (chronic) disease sometimes becomes more clinically apparent during the dry season, when immunodepressive factors such the poor nutritional state of the animal diminish its defences, even when the initial infection occurred during the rains. The epidemiology is also greatly influenced by host preferences and diurnal (daily) behaviour patterns of the various local species of tabanids and other biting flies (e.g. whether the hours that they are active allow much contact with livestock or not).

The main reservoirs of *T. vivax* infection in Latin America are probably domestic ruminants themselves, but *T. evansi* has found new wild reservoirs such as blood-sucking vampire bats and the capybara, a giant rodent. The peculiar involvement of vampire bats in the transmission of *T. evansi* has been mentioned before in this field guide.

13 The general term *epidemiology* tends to replace *epizootiology*, which was in common use in connection with diseases in animals.

14 *G. m. morsitans* does not occur in Burkina Faso, nor in West Africa as a whole.

15 But tsetse flies do get infected on reptiles with specific reptile trypanosome species, such as *T. grayi* of crocodiles, a species of the subgenus *Megatrypanum* (Stercoraria). Such infections may render the microscopical diagnosis of trypanosome infections in tsetse flies difficult (see Chapter 3 - Diagnosis).

16 Host preferences of a tsetse population can be determined in specialized laboratories by serologically identifying the species from which the blood in fed flies originates (blood meal analysis).

17 Ironically, the few herds that remain behind in the Sahel during the dry season and live on the few remaining watering places and the pasture available around these, are usually in better shape at the end of the dry season than the herds returning from the coarse vegetation and the unhealthy areas further south.

18 The epidemiology of dourine, as a venereal disease, is of course very different again, and will not be discussed here.

**DISTRIBUTION**

This section is to some extent a continuation of the previous one on Epidemiology, as many of the factors that determine the distribution of a particular trypanosome species are also involved in epidemiology; in fact, distribution dynamics form an integral part of the epidemiology.

As discussed in the previous section, trypanosomes that are normally cyclically transmitted by tsetse flies, can be transmitted mechanically (see Life cycles), and in the presence of large numbers of biting flies trypanosomosis in domestic animals may extend beyond tsetse belts. Horseflies (tabanids) and stable flies (*Stomoxys* species) are particularly important as mechanical vectors. Nevertheless, the distribution of nagana in Africa largely coincides with that of its biological vectors, the tsetse flies, and the disease tends to die out in their absence. The presently accepted approximate distribution of the genus *Glossina* is given in Figure 7; animal trypanosomosis is certainly present in the whole of this area, and in some cases extends to a variable degree beyond it. Within this huge area, the situation is far from uniform. Individual tsetse species (and/or subspecies) are limited to certain regions and have a geographical distribution pattern which is determined by their different climatic and host requirements (just think of the savannah group, the riverine group and the forest group). Trypanosome subspecies, types and even species also have different geographical distribution patterns. For example, *T. godfreyi* is (so far) only known in the Gambia, *T. brucei gambiense* occurs in western and Central Africa, *T. brucei rhodesiense* in eastern and southern Africa, etc.

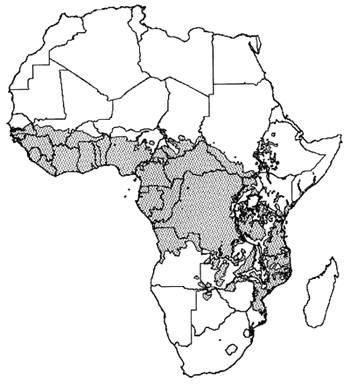
As far as the individual trypanosome species are concerned, seasonal outbreaks of *T. congolense* infection have been reported outside tsetse areas in the southern Sudan, for example, associated with large numbers of tabanids, but normally this trypanosome species is confined to tsetse belts and their near surroundings. It has not managed to escape from its biological vector.

The same holds for *T. simiae*; although it is thought that mechanical transmission by stable flies may be important once the infection has been introduced by tsetse flies into a piggery, the infection is not propagated outside tsetse areas. Knowledge of *T. godfreyi* is still insufficient.

The case of *T. vivax* is different. The infection can be seen in Africa at some distance from the edges of tsetse belts, and the author of this book diagnosed the parasite in the late 1950s in sedentary cattle herds all along the White Nile from Malakal in the southern Sudan up into the semi-desert of Khartoum Province, hundreds of kilometres from any tsetse belt. A similar situation has been reported in Ethiopia, where *T. vivax* is commonly found in highlands too cold for tsetse survival. But the most remarkable fact is that *T. vivax* has been able to establish itself in the western hemisphere, in the absence of tsetse. These American strains of *T. vivax* are thoroughly adapted to mechanical transmission and all attempts to transmit them biologically through tsetse have failed. In the past, *T. vivax* has also been present on the Indian Ocean island of Mauritius, Without tsetse, but has been eradicated there. There are also indications that *T. vivax* may sometimes persist at a low level, because of mechanical transmission, after tsetse flies have been eradicated from an area.

The distribution of *T. brucei* seems to be closely associated with that of its *Glossina* vectors (Figure 7), but it should be remembered that *T. evansi*, and probably also *T. equiperdum*, appear to have been derived from *T. brucei* and have adapted to mechanical and venereal transmission, respectively. *T. evansi* has been spread widely by biting insects outside tsetse-infested regions in Africa, and also outside Africa; it is present in tropical and subtropical areas of Africa north of the equator, in Asia, and in South and Central America from Panama to Argentina. *T. equiperdum* infection, as a venereal disease, is even less restricted by climate and in the past has spread as far as Canada and Russia in the northern hemisphere, and as far to the south as Chile and South Africa. Its present distribution is not very well known; *T. equiperdum* is sometimes difficult to distinguish from *T. evansi*. It has been eradicated from North America and most of Europe. It is certainly present in northern and southern Africa, and in tropical Africa at least in Ethiopia and probably the Sudan. It has made a comeback (or perhaps has been rediscovered) in Europe (Italy, Russia, possibly other countries), and is still present in parts of Asia, including Ouzbekistan and China. It is also believed to be still present in parts of South America, but there is little reliable information.

**Figure 7  
Tsetse distribution in Africa**



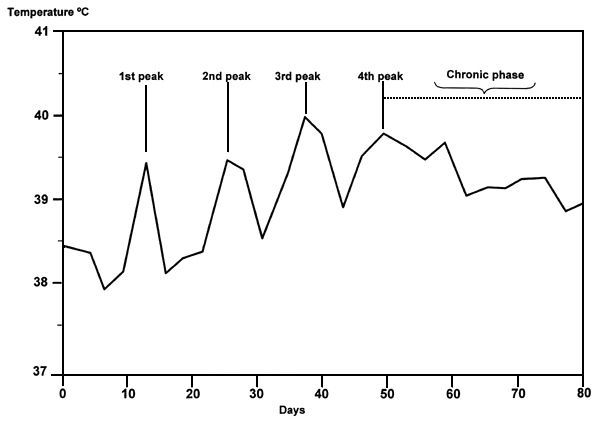
## PATHOGENESIS

When the tsetse fly injects infective metacyclic trypanosomes into the skin of the host, there is a phase of local inflammation and a swelling, a so-called chancre, develops. The metatrypanosomes divide and multiply in the chancre and give rise to the typical blood forms which invade the lymphatics and lymph nodes, and then the blood stream.

Trypanosomosis, like other infectious diseases, starts with an increase of the body temperature, a hyperthermia. This is the result of the contact between the trypanosomes multiplying in the host and the defence system of the host. The surface proteins of the trypanosomes provoke the host in making specific antibodies against these proteins, and after a few days almost all of the trypanosomes in the blood are destroyed by these antibodies and the body temperature drops. However, a few parasites survive as they have been able to replace their surface proteins by different ones, against which the antibodies cannot act. These surviving trypanosomes are able to multiply, and cause a new peak of parasitaemia and hyperthermia, until the organism of the host makes specific antibodies against the new surface proteins. This seesawing process continues for a long time, as the trypanosome is able to make an almost unlimited number of antigenic variants, and the host responds to each of them, until either the antigenic repertoire of the trypanosome is finally exhausted, in which case self-cure of the host follows, or the ability of the host to react to all of the antigenic variants is overwhelmed, and the host dies.

A typical aspect of trypanosomosis therefore is the temperature curve: there are peaks every few days, particularly in the beginning. Antigenic variation becomes slower as the disease progresses and the intervals between temperature peaks become longer and the peaks are less high. Figure 8 illustrates this.

**Figure 8  
Temperature curve in a bovine suffering from *T. congolense* infection**



One of the main symptoms of the disease is *anaemia* (decrease of haemoglobin in the blood). There are various theories on the pathogenesis of anaemia. In the early stages of the disease, it is believed to be caused in part by phagocytosis of red cells (their removal by a certain type of the host's white cells, the phagocytes). The red cells apparently become coated with material from lysed trypanosomes which tricks the phagocytes into mistaking them for foreign invaders and remove them. (This phenomenon is also called auto-immunity = immunity directed against cells of the host itself.) It is possible that the anaemia caused by phagocytosis is increased by toxic substances emanating from the trypanosomes which destroy red cells directly by lysis (haemolysis). The haemopoietic system (the system which produces red cells, mainly in the red bone marrow), tries to compensate for the loss of erythrocytes by increasing its activity but, later, in the chronic stages of trypanosomosis, othertoxins from the parasites have a depressing effect on the haemopoietic system, and the host is unable to produce as many red cells as are removed (even normally these cells have only a limited life span, and now they are removed even faster because of auto-immunity and haemolysis).

The anaemia means a reduction in haemoglobin and therefore in the oxygen-carrying capacity of the blood. Insufficient oxygen is available to the cells for their efficient functioning and the efficiency of their normal activities is reduced. A slow process of deterioration of health and condition sets in.

Trypanosomosis is also associated with *immunodepression*,[19](http://www.fao.org/docrep/006/x0413e/X0413E03.htm#ref2.3.1) i.e. the host's immune system becomes less efficient to deal with infections. Although this can perhaps be explained in part by the depression of the haemopoietic system, which not only provides red cells but also white ones involved in the immune response, immunodepression occurs also in the acute stage; the ability of the immune system to react to invaders is already diminished before the haemopoietic system is depressed. Animals affected by trypanosomosis often develop a lower antibody titre after vaccination against other diseases, and secondary infections which the host would normally control may also crop up during the disease. For example it is common to find considerable numbers of *Babesia*, *Theileria* and/or *Anaplasma* in bloodsmears of animals suffering from AAT, in situations where normal animals are healthy carriers of these tickborne infections. Trypanosome infections disrupt the balance. Such concurrent diseases may also affect necropsy findings.

Various organs are affected by AAT, to some extent depending on the species involved. While (more or less hypothetical) toxins may be involved, as well as the anaemia (see above), the trypanosomes may also be more directly responsible. *T. congolense* is mainly confined to the blood, while *T. vivax* and *T. brucei* also invade the tissues. *T. vivax* is found in the lymph and even in the chamber of the eye and *T. brucei* is well known to invade the central nervous sytem in human sleeping sickness (*T. brucei gambiense* and *T. brucei rhodesiense*), but also in animals such as horses, goats and dogs. The nervous system is also affected in the later stages of dourine (*T. equiperduni*).

The *heart* is often affected by a myocarditis (inflammation of the heart muscle), and heart failure is often the direct cause of death. However, this depends to a large degree on the effort the heart muscle has to provide. Extensive myocarditis with the presence of trypanosomes in the heart muscle has been found in highly susceptible European cattle infected experimentally in Europe with pathogenic West African *T. vivax*, kept at rest in a stable and on a good level of nutrition, and which had shown no obvious signs of distress before they were slaughtered at the end of the experiment. Such infections could easily have caused fatal heart failure in African zebu cattle subjected to the stress of poor nutrition and walking long distances to watering points or during transhumance.

*Oedemas* (subcutaneous swellings caused by accumulation of tissue fluid) are often present in trypanosomosis, particularly in horses and dogs. There is evidence of increased permeability of blood capillaries, and therefore leakage of blood plasma leading to the swellings.

In chronic trypanosomosis the animal loses condition, there is *wasting*. During the acute stage, the appetite is variable, being decreased during the fever peaks. But in the chronic stage, when the fever reactions are less pronounced, the appetite is usually normal, almost until death, even when extreme weakness prevents the animal from rising. The pronounced wasting is therefore not caused by starvation. There is consumption of the fat reserves during the recurrent bouts of fever, but there are also severe degenerative changes of the muscle cells and other tissue cells, and there is an increased breakdown of protein in muscles and elsewhere, leading to atrophic degeneration (the cells are reduced in size and efficiency). The decreased supply of oxygen because of the anaemia is also an important factor (see above).

19 Also called immunosuppression, but as there is never a total suppression of the immune system, the word immunodepression may be more appropriate.

**ECONOMIC ASPECTS**

**Tsetse-transmitted trypanosomosis**

In high challenge areas, and in the absence of trypanotolerant breeds, tsetse flies and AAT prevent the keeping of livestock, at least of those species that are affected by AAT. In that case, the economic impact on livestock production is most pronounced. In several African countries livestock (draught oxen) and their products (manure) play an important role in crop production, and the integration of livestock in crop agriculture is therefore severely affected.

Keeping non-susceptible livestock, in particular poultry, may be the best answer to solving the problem of providing sufficient animal proteins in the human diet. Or game farming may be a feasible option. Sometimes the area is more suitable for the production of valuable agricultural crops which can be sold and the income used to buy animal products from elsewhere.

All kinds of situations exist between this extreme case and the absence of trypanosomosis; the economic impact varies accordingly. Many factors are involved when economic aspects are considered, such as:

* Species, type, productivity, value and susceptibility of livestock. For instance: exotic dairy cattle with a high production are both very valuable and very susceptible; exotic breeds in general are of high monetary value and more susceptible than indigenous ones having been exposed to the disease since many generations. Nevertheless, indigenous draught oxen are valuable and, when worked hard, have an increased susceptibility to the effects of the infection. Horses are particularly susceptible to trypanosomes of the subgenus *Trypanozoon*, cattle much less so. Trypanotolerant breeds can survive and even produce where other breeds can only be maintained under intensive and expensive chemoprophylactic or chemotherapeutic regimens.
* Challenge, which depends on species of fly present (vectorial ability, host preferences), density of the fly, their daily activity patterns and the grazing patterns of the livestock, and wild reservoir hosts).
* Presence of drug resistance. (As we shall see further on, very few drugs against trypanosomosis remain commercially available.)
* Type of production (commercial, subsistence, transhumant).
* Economic situation and management of the country (part of national budget and foreign exchange allotted to livestock resources).
* Commercial factors, such as “dumping” of surplus meat by the European Union (which decreases the price of locally produced animals), or an unrealistic exchange rate, may bias cost-benefit aspects of trypanosomosis control.

The economic impact is made up of direct losses (consisting of loss of production, mortality, abortion), as well as the cost of control (which includes the cost of drugs, their transport to the field site, the salaries of the operators, etc.).

The loss of *potential* production (i.e. the production that could be achieved if trypanosomosis did not occur) are indirect losses. At present unused grazing areas in many of the tsetse-infested areas of Africa could support a large ruminant livestock population. However, the control of the fly should only be envisaged when really needed, and then only when proper and sustainable land-use plans have been elaborated, and when the political will and legislative means to carry such plans through are assured. If not, overgrazing is bound to occur, followed by erosion and, depending on the climatic zone, by desertification, leading to permanent loss of the land. In the meantime, the presence of tsetse fly preserves these areas.

Because livestock keepers avoid certain tsetse-infested areas, cattle distribution is often imbalanced or even distorted. From the continental cattle distribution we know that this indirect effect of AAT is very important; only 10 million out of 165 million head of cattle in the tsetse-infested countries of sub-Saharan Africa are distributed within the limits of the continental fly belt while most of the remainder is distributed at the perimeter of the fly distribution. At the local level, it is extremely difficult to clarify this point because nobody is sure about the magnitude of these indirect losses. Still, the collective, indirect AAT losses are estimated by FAO to be in excess of the 0.6 to 1.2 hundred thousand US dollar direct losses incurred by trypanosomosis-affected cattle.

Increasingly, tsetse and trypanosomosis control schemes become concentrated in selected areas of high priority. These are areas where control is technically feasible, where the economic returns are considerable and where the transformation of the landscape, from bush to farmland, already occurs because of demographic pressure. It is in such dynamic environments, which become progressively less suited for tsetse survival, that it is economically attractive to intervene.

It is important to monitor the changes during such interventions. Data have to be collected to check on what happens to tsetse-transmitted trypanosomosis, how farming practices and the landscape change. Close monitoring makes it possible to adjust control programmes for technical reasons, or make the programme more efficient in agricultural (economical) terms, or adapt to environmental degradation risks.

Simulation models are useful to examine the economic impact and to decide upon suitable control strategies to achieve a positive cost-benefit result, taking into account the range of various parameters in any particular situation (see, for example, Brandl, 1988). Because of the land-use aspects, there is a growing tendency towards the collection of georeferenced data, which may be plotted on maps; computerized versions may be examined in the so-called geographical information systems (GIS).

**Mechanically transmitted trypanosomosis**

Studies on *T. vivax* and *T. evansi* in Latin America show that their economic impact can be quite severe. Trypanosomosis of domestic animals has been ranked as third in importance in Colombia, after ticks/tickborne diseases and liver fluke. Even the inapparent losses of subclinical infections by *T. vivax* may be considerable and the same certainly applies to mechanically transmitted trypanosomosis in Africa; futher economic studies are necessary in order to obtain reliable figures.