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# Reproductive disorders in African trypanosomiasis: a review

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## Summary

Reproductive disorders are frequently seen in human beings and in animals infected with tsetse-transmitted (African) trypanosomiasis. The disorders include irregular menstrual (or oestrus) cycle, infertility, abortion and impotence. Intrauterine infections occasionally occur, resulting in still birth or neonatal mortality. The changes are essentially reversable after treatment, although recovery may take several months.

**Key words:** trypanosomiasis; reproductive disorders; pathophysiology of reproduction; infertility; abortion.

## Introduction

Reproductive disorders have been recognised in human and animal African trypanosomiasis since the early part of this century. However, the pathogenesis of these disorders is not well understood. We believe that this aspect deserves closer study especially in livestock where reproductive performance is the cornerstone of productivity. Both males and females are affected by the disease.

Pituitary hormones (gonadotropins) play a major role in the reproductive process. In particular, follicle stimulating hormone (FSH), interstitial cell stimulating hormone (ICSH) and growth hormone (GH) play an important part in the spermatogenic cycle in males and oestrus cycle in females. Consequently, absence of pituitary gonadotropins (e.g. following hypophysectomy) leads to a block in sperm maturation in rats (Clermont and Morgentaler, 1955) or to marked testicular degeneration and abortion in man and animals (Steinberger and Duckett, 1967; Apted, 1970; Ikede and Losos, 1975).

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*Pituitary damage*

Human patients with sleeping sickness due to *Trypanosoma gambiense* and *T. rhodesiense* may exhibit impotence, gynaecomastia, feminine distribution of fat and infertility or sterility (Apted, 1970). Although some of these symptoms have been attributed to hypopituitarism probably related to pituitary fibrosis described in two patients by Hawking and Greenfield (1941), no definitive tests have been carried out to confirm the role of the pituitary or to determine the exact mechanism in man.

Studies in infected domestic and laboratory animals have provided more evidence of specific damage to the pituitary gland. Extensive mononuclear inflammation of the gland has been described in cattle, sheep, goats, donkeys and dogs experimentally infected with *T. brucei* (Losos and Ikede, 1970, 1972; Ikede et al., 1977; Moulton and Sollod, 1976; Morrison et al., 1981) and in a naturally infected horse (Ikede et al., 1973). In addition, 10 out of 19 sheep experimentally infected with *T. brucei* showed extensive coagulative necrosis and fibrosis of the adenohypophysis (Ikede and Losos, 1975). These lesions were in most cases associated with extravascular localization of trypanosomes in the gland. However, pituitary function is yet to be correlated with these lesions through sequential hormonal assays.

*Reproductive disorders in males*

Clinical orchitis has been described in human sleeping sickness (Apted, 1970). In male animals infected with the brucei group, the lesions are a combination of scrotal dermatitis, orchitis and periorchitis. Soon after infection the parasites localize in the scrotal skin and hydrocoel fluid and also invade the tunica vaginalis, testis, epididymis and spermatic cord, provoking a nonpurulent granulomatous inflammation in monkeys (Peruzzi, 1928), sheep (Ikede, 1979), rabbits (Van den Ingh and Van Dijk, 1975; Ikede and Akpavie, 1982) and mice (Anosa and Kaneko, 1984). Thrombosis of the vessels of the pampiniform plexus has also been reported in *T. brucei* infected male dogs (Morrison et al., 1981). These lesions lead to degeneration of the seminiferous tubules, aspermatogenesis and aspermia in severe cases. Other factors such as fever may also be responsible for the testicular degeneration.

*T. vivax* and *T. congolense* are also associated with infertility in animals but the mechanisms appear different. Inflammatory changes in the genital organs are usually mild or absent but there is progressive and marked testicular degeneration that can lead to atrophy and aspermia (Isoun and Anosa, 1974; Isoun et al., 1975; Anosa and Isoun, 1980; Kaaya and Oduor-Okelo, 1980; Masake, 1980; Anosa, 1983). The cause of the testicular lesion is believed to be due to the effect of prolonged fever, thrombosis of spermatic blood vessels, and the general wasting of body organs (Anosa and Isoun, 1980; Anosa, 1983).

The severity of testicular and epididymal lesions is reflected in poor quality of semen and the high percentage of abnormal spermatozoa present in the

ejaculate of bulls (Isoun et al., 1975; Grundler, 1985) and rams (Isoun and Anosa, 1974; Anosa and Isoun, 1980; Akpavie et al., 1987) experimentally infected with *T. vivax, T. congolense* or *T. brucei.* It has also been shown in rabbits and sheep that successful treatment with trypanocides will lead gradually to normal spermatogenesis over a period of several months if the original lesions have not been complicated by secondary bacterial infections (Ikede and Akpavie, 1982; Akpavie et al., 1987).

*Reproductive disorders in females*

Females infected with trypanosomes show irregular oestrous cycle and may be infertile or sterile. Infection during pregnancy may lead to foetal death, abortion, still birth and neonatal death. Macfie (1913) was among the earliest workers to report amenorrhoea in women suffering from Gambian sleeping sickness, while Hornby (1921) first described abortions in pregnant cattle infected with African trypanosomes.

Abortions, still birth and neonatal deaths occur sporadically in infected women. Manson-Bahr (1966) reviewed the few reports of human congenital African trypanosomiasis in the former French Congo, Cameroun and Western Germany. Later, Olowe (1975) described a case of congenital *T. gambiense* infection in a 17-day-old girl who died a few weeks later of meningoencephalitis. The organism was detected in the cerebrospinal fluid but not blood. Recently, Emeh and Nduka (1983) showed that serum gonadotropins (FSH and luteinizing hormone) were significantly depressed in a study of 11 cases of advanced Gambian sleeping sickness. A year after antitrypanosomal treatment the values returned to normal. Single, rather than sequential samples were apparently taken before and after treatment. Further studies are required for a clearer picture of the role of gonadotropins.

Although abortions are often reported in clinical cases of African animal trypanosomiasis (Leeflang, 1975), organisms are rarely detected in the foetus or newborn (Woo and Limebeer, 1971). Some of the reports of intrauterine infections in animals include those on *T. vivax* in the blood of a newborn lamb (Ikede and Losos, 1972) and a calf (Ogwu et al., 1985), and *T. congolense* in mice (Griffin, 1983).

Vohradsky (1971) reported the presence of cystic ovaries and endometritis in cattle infected with *T. vivax.* Isoun and Anosa (1974) also observed numerous ovarian cysts containing trypanosomes in two sheep experimentally infected with *T. vivax.* One of the sheep also had trypanosomes in the amniotic fluid. Apparently neither the cysts nor the parasites in the amniotic fluid adversely affected pregnancy in the ewes.

Ige and Amodu (1975) reported reproductive disorders in N'dama cattle experimentally infected with *T. vivax, T. congolense* or *T. brucei* or a mixture of all three. They observed that irregular oestrus and sterility persisted for 3–16 months after self-cure or Berenil treatment. Ogwu et al. (1984) found that

infection of zebu heifers with *T. vivax* before first mating resulted in infertility. When pregnant heifers were infected, the calves born had lower birth weights than controls (Ogwu et al., 1985). They also observed that infection of pregnant heifers in the first trimester resulted in one abortion and three normal deliveries, whereas, heifers infected in the second trimester all had normal births. On the other hand, of the 4 heifers infected in the third trimester, three calved prematurely while the fourth died shortly after a full-term calf had been pulled out. Control heifers had normal gestation and parturition.

In general, the foetus or newborn from an infected dam may be anaemic, but no significant placental lesions have been described. However, many aborted foetuses do not always show evidence of intrauterine infection. Such cases may be related to maternal hormonal imbalance from pituitary damage, to the stress of infection and to the effect of hyperthermia on the conceptus.

## Conclusion

During the course of infection, trypanosomes cause specific and non-specific damage to some of the organs involved in the reproductive process as well as the foetus. The organs include the pituitary gland, testis, epididymis, ovary and uterus. Lesions in the gonads lead to infertility while those in the foetus lead to foetal death, and/or neonatal death. Superimposed on these changes is damage to the pituitary gland. The sequential pathophysiology of endocrine imbalance in trypanosomiasis is yet to be adequately studied.


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