Veterinary World, Vol.2(11):435-438 REVIEW

Effect of Trypanosoma spp. on Nutritional status and performance of livestock

A.K. Pathak

Centre of Advanced Studies in Animal Nutrition,
Indian Veterinary Research Institute, Izatnagar-243 122

Abstract

Trypanosomiasis induces loss of body condition in pregnant animals leading to birth of offspring's, with low birth weights foetal and neonatal losses, besides production losses in lactating animals. The consequences of trypanosomiasis are less severe in better-nourished animals but good nutrition does not by itself provide protection. Adequate energy, protein and vitamin nutrition enhances the ability of trypanosome-infected animals to withstand the adverse effects of infection.
Keywords: Nutrition, Livestock, Trypanosoma, Lactation, Fetal.

Introduction

Trypanosoma species occur in vertebrates, principally in their blood and tissue fluids as intercellular. A few may invade the tissue fluids as intracellular parasites like *Trypanosoma cruzi* found within the cells of reticuloendothelial system and in the cardiac muscles. Trypanosomiasis in livestock has a profound influence on productivity (FAO, 2000). Seasonal occurrence of trypanosomiasis, mostly in the dry season (Mochabo *et al*., 2005) and in hot humid climate in tsetse infested areas (Dam *et al*., 1996). The severity of trypanosomiasis depends on the pathogenicity of the *Trypanosoma spp*. and susceptibility and nutritional status of the host. Susceptibility increases to trypanosomiasis depends on malnutrition, overwork, intercurrent infection, pregnancy, parturition lactation, stress and degree of parasitaemia. (katunguka *et. al*., 1995). Species of the genus are the etiological agents of important diseases of animals and man such as Surra, Nagana, Dourine, Mal-decadres in animals and sleeping sickness of man etc. They are transmitted by blood sucking arthropods in which the developmental stages like epimastigote or promastigote occur. A few species are transmitted mechanically without any development in the arthopod vector.

Effect on Feed Intake

Feed intake of *T. vivex* infected sheep was depressed at both plane of nutrition i.e. 12% for males, and 17% and 22% for (pregnant and lactating) females (Reynolds and Ekwuruke, 1988). Dam *et al*.(1997) observed that an average feed intake per unit metabolic body size reduced by 13 to 24% in goat 2 weeks after infection with *T. vivex*. About 20 % reduction in feed intake during febrile trypanosomiasis was recorded in goats (Akinbamijo *et al*., 1992) and in sheep (Akinbamijo *et al*., 1994). Reynolds and Ekwuruke (1988) observed difference in food intakes between *T. vivex* infected and control groups in the last week (LPIP and MPIP) before parturition, with 22% and 19% lower than control groups.

Factors responsible for a reduced feed intake during trypanosomiasis are not known but it may be due to cachectin or TNF, bradykinin, fever and stress produced by the host and toxin released by the *T. brucei*.

Effect on body temperature

Fluctuating body temperature (fever) is a typical symptom of trypanosomiasis, which reflects the response to successive waves of parasitaemia (Stephen, 1986). The body temperature set point in the hypothalamus is then changed under the influence of pyrogenic stimuli released during infection (Baracos *et al*., 1987). Increase body temperature can be increase by increasing heat production without changing heat loss or (by vasoconstriction and high tissue insulation) by reducing heat loss.

Effect on Live weight gain

T. brucei infected boars on the high energy diet had gained a mean of 0.42kg/day or 64.6% of the gains by their control whereas those on low energy diets

gained a mean of 0.09kg/day or 31.0% of the gains by their controls (Otesile *et al*., 1991).

Faye *et al*.(2005) observed that *T. congolense* infected West African dwarf goats on basal diet lost more weight than supplemented ones through out the experiment. *T. congolense* infected sheep (Katunguka *et al*., 1997) and *T. brucei* infected pigs (Fagbemi *et al*., 1990) on low energy intake showed greater retardation of growth than on high energy intake.

Development of Anemia

Anemia is recognized as the most important clinical manifestation of animal trypanosomiasis. Richardson and Kendall (1963) observed anemia with reduction in the number of RBC and haemoglobin to the level of 25% of the normal value. Anemia was due to impairment of re-utilization of iron from degraded RBCs as a result of blockage of reticuloendothelial from release. The rate of development of anemia (Little *et al*., 1990) & rate of recovery from the anemia was faster in Ndama cattle supplemented with extra ground nut cake than control. Increase plasma volume (haemodilution), as an important factor in the development of anemia in sheep (LE) infected with *T. congolense* (Katunguka *et al*., 1997). Hypocholesterolaemia in infected animals leads to damage to the integrity of the erythrocyte plasma membrane.

Higher initial cholesterol (LE) tended to have higher parasite numbers and developed more severe anemia (Traore *et al*., 1987). Fagbemi *et al*. (1990) find that adequate dietary energy is a requirement for the reutilization of iron in trypanosomiasis.

Effect on Energy

Heat production of *T. vivex* infected goats was increased by 15.6 kcal /day/ $kg^{0.75}$or about 16% (Zwart *et al*.1991). This increase in heat production (HP) was greater during night (22 kcal/day/ $kg^{0.75}$) than during the day (14 kcal/day/ $kg^{0.75}$). HP increase by (21kj / day/ $kg^{0.75}$) or 10 % per ^{0}C increase in body temperature (Dam *et al*., 1996) in *T. vivex* infected goats. Metabolisable energy for maintenance (MEm) for *T. vivex* infected goats (464kj ME/ $kg^{0.75}$) was 25% greater than for control goats (375kj ME/ $kg^{0.75}$). MEm for *T. vivex* infected goats (464kj ME/ $kg^{0.75}$) was 25% greater than for control goats (375kj ME/ $kg^{0.75}$), (Verstegen *et al*., 1991).

A 15% rise in metabolic rate (Blaxter, 1989) and 25% rise in maintenance requirement (Verstegen *et al*., 1991) for every degree rise in temperature. Fagbemi *et al*. (1990) observed that adequate dietary energy ameliorates the disease produced by *T. brucei* in growing pigs as during 8 week post infection.

Effect on lipids

Lipids constitute 15-20% of trypanpsomal dry weight. They obtain cholesterol from the host by uptake and degradation of low density (Gillet and Owen, 1987; Coppen *et al*., 1987) or high-density lipoproteins (Traore *et al*., 1987). Trypanosome requires cholesterol for growth and multiplication (Black and Vanderweed, 1989) and is the main sterol in trypanosomes (Carrol and McCrorie, 1986). Katunguka *et al*., 1999) observed that sheep infected with *T. congolense* causes hypolipidaemia, hypophospholipidaemia and hypocholesterolaemia in cattle (Traore *et al*., 1987) or *T. b. brucei* (Wellde *et al*., 1974). Belaunzaran *et al*. (2007) identified a Phospholipase- A_1 in *T. cruzi* and they observed that its concentration is high in pathogenic trypomastigote and amastigote trypanosomes. Plase-A_1 could induce cell damage either directly or through free fatty acids (FFA) and lysophosphatidalcholine generated by parasite or host phospholipids.

Effect on protein

Protein also play important role in the course of trypanosomiasis Romney *et al*. (1997).

The infection caused marked reduction of growth in animals fed a low protein ration whereas the infected and the infected and control animals fed a high protein ration grew at similar rates. Hecker *et al*. (1991) observed that sheep supplementation with cotton seed cake and maize bran showed a delayed onset of parasitaemia compared to sheep that were only grazing natural grasslands.

Little, *et al*. (1990) observed that the rate of development of anemia in N'dama cattle inoculated with *T. congolense* and supplemented with extra groundnut cake was slower than in un-supplemented cattle. Fever during infection is associated with increase heat production and increase metabolizable energy for maintenance so that proportion of protein that is used for growth is reduced, as it is metabolized for animals to provide the extra energy required. The increased synthesis of protein occurs at the expense of muscle protein catabolism and loss in body weight (Dargie, 1980).

Effect on Vitamins

Blood stream forms of *T.b.brucei* produce enormous amount of H_2O_2 (Meshnik *et al*., 1977). Increase systemic oxidative stress in *T. gambiense* and *T.b. brucei* infected rat, decrease in tissue ascorbic acid concentration in *T. b. brucie* infected guinea pig and increase susceptibility of RBCs to oxidative haemolysis in *T. b. gambiense* infected rat (Ameh, 1984) and peroxidation in *T. b. brucei* infected mice. *T.b. brucei* infected rabbits were injected (I/P) daily with vitamin C and E @ 100mg/kg bwt and 10mg/kg bwt, on 12 day post infection ameliorate the anemia and reduces oxidative damage (Umar *et al*., 1999). Surra

positive buffaloes had significantly low levels of folic acid and Vit. B_{12} as compared to control (Sangwan *et al*., 1993). Impairment of T and B cells response (Singh *et al*., 1987) aggravates the folic acid deficiency in *T. evansi* infected buffaloes. Vitamin B_{12} is an essential for the normal maturation and development of RBC. Trypanosomiasis causes disturbances in rumen motility, pH and normal micro flora (Kreier, 1977), so micro flora may not be able to synthesize vit. B_{12}. *Trypanosoma equiperdum* and *T. cruzi* infected rats on vit. A deficient diet died sooner than those fed complete diets (Yaeger and Miller, 1963a).

Effect on Minerals

Trypanosoma congolense infection in sheep results in changes in protein metabolism and iron metabolism, and has no, major effects on the concentrations of plasma zinc, copper, calcium, magnesium and inorganic phosphate. Increase serum chloride and calcium concentration in goats infected with *T. vivax* and a gradual fall in the concentration of inorganic phosphate in cattle infected with *T. congolense*. Changes have also been recorded in levels of serum iron, total iron-binding capacity and plasma proteins. Katunguka (1996) study was designed to follow the changes in the plasma concentrations of zinc, copper, calcium, magnesium, inorganic phosphate, serum iron, and total iron-binding capacity during a course of *T. congolense* infection in sheep.

Effect on Animal productivity

Malnutrition increased mortality due to trypanosomiasis (Reynolds and Ekwuruke, 1988). The temperature rise in infected sheep was 1.35^{o}C (Akinbamijo *et al*., 1994) suggesting an increase in metabolic rate and maintenance requirements by 20 and 30%. Increase in maintenance requirements would decreases net energy required for growth, faetal development and milk production. Trypanosomiasis reduced body weight (due to reduced feed intake), milk production and feed conversion efficiency and caused reproductive wastage, abortion, dam mortality and poor weaning weight (Akinbamijo, *et al*., 1990; Verstegen, *et al*., 1991; Zwart, *et al*., 1991). Abortion rate was 15% in the infected ewes with a lamb mortality rate of up to 85%. Productive efficiency of animals modulated by adequate feeding and assists in ameliorating the deleterious effects of trypanosomiasis on production in endemic areas.

Effect on work performance

Infection with *T. evansi* can result in reduced work output of draught animals (Ruckmana, 1979). High mortality in buffaloes infected with *T. evansi* has been associated with periods of intense work (Wells, 1981; Lohr *et al*., 1985). The stress induced by work probably results in increased corticosteroid secretion that could, in turn, aggravate the outcome of infection by depressing the host's immune response (Tizard 1977; Stephens, 1980). *T. evansi* infection had a marked effect on the packed cell volume (PCV) and temperature profiles of the buffaloes (Payne *et al*., 1991). Prolonged fall in PCV in *T. evansi*-infected animals could result in a decline in the oxygen-carrying capacity of the blood. Maintenance of an elevated body temperature in *T. evansi* infected animals results in a reduction in energy available for metabolic activity and work. The estimated energy expenditure ranged from 1.22-1.32 times maintenance energy when the buffaloes traveled a distance of 12-15 km/day at a speed of 1.11-1.39 m/s (Pearson, 1989).

Effect on reproductive performance

In non-pregnant animal, *T. vivax* infection resulted in anoestrus in ewes, varying from 40 to 96 days in duration and characterized by prolonged low levels of progesterone.

T. congolense infection in goats results in anoestrus due to persistant corpora lutea. Osaer *et al*. (1998) suggested that Trypanosome infection can reduce ewe productivity by acting directly on the pregnant uterus and also by disrupting cyclicity and preventing a return to estrus. Both energy-restricted and protein-restricted diets can aggravate the detrimental effects of *T. congolense* infection on growth and productivity in sheep (Katunguka *et al*., 1993, 1995).

Supplementation has beneficial effect in pregnancy in trypanosome-infected dams including reduced ewe and prenatal lamb mortality (Reynolds and Ekwuruke, 1988). Reduction in both luteal and placental progesterone secretion might include gonadal atrophy due to thrombosis in the gonadal blood vessels, severe anemia and thermal effects of pyrexia (Mutayoba *et al*., 1988). Trypanosomes have been shown to reduce secretion of, and gonadal sensitivity to, gonadotrpphins (Mutayoba *et al*., 1995c, 1996) possibly as a result of hypothalami-pituitary-adrenal activation. Haematic forms of trypanosomes cross the placental barrier possibly leading to disturbance of placental progesterone production and fetal death.

Effect on endocrine system

The thyroid gland is one of the endocrine organs, which affected during trypanosomiasis (Abebe and Eley, 1992). *T. congolense* infection rapidly impaired the function of the thyroid gland in goats as defined by considerable plasma thyroxin (T4) decrease (Mutayoba, *et al*., 1988) and in human sleeping sickness showed decreased in T_3 and T_4 without TSH variation. Plasma concentration of thyroid hormone correlated positively with energy intake (Blum *et al*., 1980; Dauncey *et al*., 1983). Decrease T_4 level in

trypanosome infected animals could have an adverse effect on feed intake and efficiency of feed conversion in spite of apparent good appetite. The effect of trypanosomiasis infection on the endocrine system in general can be direct or indirect. The direct effect might be mediated by biologically active factors of parasite origin such as phospholipase A, protease and peptidase. Proteases of parasitic origin were suggested to cause gonadotrophic dysfunction in rats infected with *T. brucei* (Hublart *et al*., 1990). The indirect effect could be mediated through a highly activated immune response where cells such as macrophages, activated during infection, could release cytokines activating and down regulating the endocrine system. IL-1 and IL-6 are known to be stimulants of the hypothalmic-pituitary axis while TNF (cachectin) is known to inhibit the secretion of pituitary hormones such as growth hormone and TSH.

Future Prospects

The high metabolic cost of fever and reduced feed intake are the major problems. Formulate feeding strategies to assist in minimizing metabolic waste during acute and chronic trypanosomiasis. The control of the parasitemia and anemia that contributes to the survival of animals from trypanosomiasis warrant investigation. Research is needed to study the effect of nutrition (body condition) on the course of the disease.

References

1. Abebe G. and Eley R.M. (1992): *Br. Vet. J.* 148: 63-69.
2. Akinbamijo O.O., et.al.(1990): *Trop. Vet.* 8: 140-148.
3. Akinbamijo O.O., et.al. (1992): *Vet. Quart.* 14: 95-100.
4. Akinbamijo O.O., Reynolds L., Sherington J. and Nsahlai I.V. (1994): *J. Agric. Sci.* 123: 387-392.
5. Ameh D.A. (1984): *IRCS Med. Sci.* 12: 130.
6. Baracos V.E., Whitmore W.T. and Gale R. (1987): *Can. J. Physiol. Pharmacol.* 65: 1248-1254.
7. Belaunzaran M.L., Wainszelbaum M.J., Lammel E.M., Gimenez G., Aloise M.M., Florin-Christensen J. and Isola E.L.D. (2007): *Parasitol.* 134: 491-502.
8. Black S. and Vanderweed V. (1989): *Mol. Biochem. Parasitol.* 37:65-72.
9. Blaxter K.L. (1989): Energy Metabolism in animals and man. Cambridge: Cambridge University Press.
10. Blum J.M., Gingins M., Vitins P. and Bickel H. (1980): *Acta endocr.* 93: 440-447.
11. Carrol M. and McCrorie P. (1986): *Comp. Biochem. Physiol.* 83B: 647-651.
12. Christensen K. (1983): Dynamic Biochemistry of Animal Production World Animal Science, A3 (edn. P.M. Riis). Elsevier Science Publisher BV, Amsterdam, The Neetherlands. pp. 215-279.
13. Coppens I., et.al.(1987): *J. Parasitol.* 34: 465-473.
14. Dam van J.T.P., et.al. (1996): *Vet Quart.* 18: 55-59.
15. Dam van J.T.P., et.al. (1997): *Br. J. Nutr.* 77: 427-441.
16. Dargie J.D. (1980): Immunoglobulin metabolism in trypanosomiasis. Proceedings of the 5th International Symposium on Ruminant Physiology, MTP Press, Lancaster, pp.349-371.
17. Dauncey M.J., et.al. (1983): *Metab. Res.* 15: 499-502.
18. Fagbemi B.O., et.al.(1990): *Vet. Parasitol.* 35: 29-42.
19. FAO 2000. A field Guide for the diagnosis. Treatment and Prevention of African Animal Trypanosomiasis. 2nd edn. (FAO, Rome).
20. Faye D., et.al. (2005): *Acta Tropica*. 93: 247-257.
21. Gillet M.P.T. and Owen J.S. (1987): *Biochem. Soc. Trans.* 15: 258-259.
22. Hecker P.A.,et.al. (1991): Proceedings of the 21st Meeting of the International Scientific Council for Trypanosomiasis research and control, 21-25 Oct., Nairobi. 23. Hublart M., et.al. (1990): *Acta Trop*. 47: 177-184.
25. Katunguka-Rwakishaya E., et.al. (1997): *Res. Vet. Sci.* 63: 273-277.
26. Katunguka-Rwakishaya E., Murray M. and Holmes P.H. (1999): *Vet. Parasitol.* 84: 1-11.
27. Kreier J.P. (1977): Parasitic Protozoa. Vol. I Academic Press, New York, San Francisco, London. pp 277- 288.
29. Little D.A., et.al. (1990): Proc. Nutr. Soc. University of Strathclyde, March 29-30. pp 2.
30. Lohr K.F., et.al. (1985): *Trop. Anim. Health Prod.* 17: 121-25.
31. Meshnick S.R., Chance K.P. and Cerami A. (1977): *Biochem. Pharmacol.* 26: 1923.
32. Mochabo M.O.K., et.al.(2005): *Trop. Anim. Health Prod.* 37: 187-204.
33. Osaer S., et.al. (1998): *Anim. Reproduc. Sci.* 51: 97-109.
34. Otesile E.B., Fagbemi B.O. and Adeyemo O. (1991): *Vet. Parasitol.* 40: 207-216.
35. Payne R.C., Djauhari D., Partoutomo S., Jones T.W. and Pearson R.A. (1991): *Vet. Parasitol.* 40: 197-206.
36. Pearson R.A. (1989): *Anim. Prod.* 49: 355-363.
37. Reynolds L. and Ekwuruke J.O. (1988): *Small Rum. Res.* 1: 175-188.
38. Romney D.L.N., et.al.(1997): *J. Agric. Sci.* 129:83.
39. Ruckmana D.W. (1979): Ph.D. Thesis, University of Padjadjaran, Bandung, Indonesia.
40. Sangwan N., et.al.(1993): *Trop. Anim. Health Prod*. 25: 79-84.
41. Singh V. and Raisinghani P.M. (1987): *Indian J. Parasitol.* 11:131-135.
42. Singh V., Sharma K.N. and Raisinghani P.M. (1987): *Indian J. Comp. Microbiol. Immunol. Infect. Disease.* 8:55-59.
43. Stephen L.E. (1986): Trypanosomiasis- A Veterinary Perspective. 1st edn. Pergamon Press, New York.
44. Stephens D.B. (1980): *Adv. Vet. Sci. Comp. Med.* 24: 179-210.
45. Tizard I.R. (1977): An introduction to Veterinary Immunology. Saunders. Philadelphia, pp. 551.
46. Traore-Leroux T., Fumoux F. and Pinder M. (1987): *Acta Trop.* 44:315-323.
47. Umar I.A., et.al. (1999): *Vet. Parasitol.* 85:43-47.
48. Verstegen M.W.A., et.al. (1991): *J.Anim. Sci.* 69:1667-1677.
49. Wellde B., et.al.(1974): *Exp. Parasitol.* 36: 6-19.
50. Wells E.A. (1981): Report on the consultancy on the control of Trypanosomiasis in domestic buffalo in North Vietnam. FAO, Rome, pp. 18.
51. Yaeger R.G. and Miller O.N. (1963a): *Fed. Proc.* 22: 435.
52. Zwart D., et.al. (1991): *J. Anim. Sci.* 69: 3780-3788.