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Objekttyp: Article

Zeitschrift: Acta Tropica

Band (Jahr): 45 (1988)

Heft 3

PDF erstellt am: 31.05.2024

Persistenter Link: https://doi.org/10.5169/seals-314080

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# Trypanosome-induced ovarian dysfunction

Evidence of higher residual fertility in trypanotolerant small East African goats

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# Summary

Changes in the length of oestrous cycles, plasma progesterone and oestradiol-17 $\beta$  levels were monitored for 6 months in *Trypanosoma congolense*-infected normocyclic small East African goats obtained from three tsetse-endemic areas and one tsetse-free area of East Africa. Irregular oestrous cycles were observed in all infected goats, before cessation at the second cycle post-infection in the more susceptible and fourth cycle in the more resistant goat groups. A significant decline in the progesterone and oestradiol-17 $\beta$  parameters were observed. The decline in hormonal values was, however, less in the more resistant than in the susceptible goat groups at least in the first 2 months post-infection. Resumption of the ovarian cycle were observed in few resistant goats after 5 months of the infection. It is concluded that clinical tolerance is correlated with residual fertility, i.e., the greater the tolerance the higher the retention of fertility.

**Key words:** ovarian dysfunction; residual fertility; trypanotolerance; *Trypanosoma congolense*.

# Introduction

The use of trypanotolerant livestock is now considered to be an important alternative strategy for increasing animal numbers in tsetse-endemic areas of Africa where other conventional disease control measures have proved uneconomical and/or ineffective. With the increasing reports of the occurrence of severe degenerative lesions of the reproductive organs in trypanosusceptible

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animals (Ikede and Losos, 1972, 1975; Isoun et al., 1975; Ikede, 1979; Anosa and Isoun, 1980; Kaaya and Oduor-Okelo, 1980; Waindi et al., 1986), it has recently become necessary to examine the degree of gonadal dysfunction in trypanotolerant livestock before wide exploitation of these animals is encouraged. To what extent trypanotolerant animals retain their fertility following trypanosome infection is not well known. Since, trypanosome-infected animals develops varying degrees of reproductive impairments, the term "residual fertility" is used in this paper to denote the resulting fertility which potential remain following the infection.

Murray et al. (1981) reported that N'dama cattle can survive and retain a higher degree of residual fertility in tsetse-endemic areas where Zebu perish. Recently, Mutayoba et al. (1987) showed that Small East African breed of goats obtained from Morogoro, a tsetse-endemic area in Tanzania, were more tolerant to experimental chronic *Trypanosoma congolense* infection than goats obtained from Arusha in Tanzania and Lambwe Valley in Kenya (also tsetse-endemic areas). Goats obtained from Imbo, a tsetse-free enclave in Central Nyanza, Kenya, however, were much more susceptible as assessed by parasitaemic and haematic parameters, body weight change and mortality rates. The present paper reports on the changes in reproductive function in the same groups of goats (for which previous data was obtained) following the infection. The relationship between trypanotolerance and higher residual fertility is discussed.

#### **Materials and Methods**

A total of 52 normocyclic Small East African female goats (Mutayoba et al., 1987) aged between 2–3 years were used. Ten female goats obtained from each of the tsetse-endemic areas (Morogoro in Tanzania and Lambwe Valley in Kenya) and tsetse-free (Imbo, Kenya) and 9 goats from Arusha, Tanzania (tsetse-endemic area) were experimentally inoculated via the jugular vein with approximately  $2.5 \times 10^4$  *T. congolense* (EATRO-1753) obtained from Kenya Trypanosomiasis Research Institute (KETRI).

Thirteen uninfected goats from all experimental groups served as controls.

Prior to the infection, all goats were acclimatized for  $1\frac{1}{2}$  months and screened for haemaprotozoa using Giemsa-stained smears and were found negative. At the end of the acclimatization period, male bucks with deviated penises were introduced to monitor overt oestrus. At the same time, all females were bled (5 ml/goat) for two consecutive cycles between 9.00–10.00 a.m. every other day to provide plasma for baseline pre-infection self-control hormonal levels. The bleeding continued throughout the infection period at the same rate except during oestrus when it was done at 6 hourly intervals. The plasma obtained was stores at -20 °C until needed for hormonal analyses. The infection period lasted 6 months.

Plasma concentrations of progesterone and oestradiol- $17\beta$  were determined in 200  $\mu$ l and 500  $\mu$ l plasma aliquots, respectively, by dextran-charcoal radioimmunoassay according to the WHO (1984/85 matched reagent manual) and adopted for use with goat plasma (O'hara et al., 1985; Katongole and Gombe, 1985).

The antisera to progesterone and oestradiol- $17\beta$  were supplied by WHO and had been raised against progesterone 3-(o-carboxymethyl) oximino-BSA and oestradiol-6CMO-BSA, respectively. The <sup>3</sup>H-tracers were obtained from the Radiochemical Centre, Amersham, England.

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Table 1. Estimated parasitaemia scores in the 4 groups of Small East Afri	

	Parasitaemia	scores*						Samples
	0	+	2+	3+	4+	5+	6+	examined
Lambwe Valley	5 (7.2%)	9 (13%)	16 (23.2%)	21 (30.4%)	12 (17.4%)	5 (7.2%)	1 (1.4%)	69
Arusha	12 (16.9%)	16 (22.5%)	13 (18.3%)	18 (25.4%)	9 (12.7%)	3 (4.2%)	0	71
Morogoro	21 (27.3%)	16 (20.7%)	14 (18.2%)	13 (16.9%)	10 (13%)	3 (3.9%)	0	77
Imbo	3 (4.8%)	7 (11.3%)	11 (17.7%)	17 (27.4%)	12 (19.4%)	10 (16.1%)	2 (3.2%)	62
* Parasitaemia scores w	ere obtained us	sing the Darkgro	ound/Phase Cor	ntrast Buffy Me	thod described	by Paris et al.	(1982).	

Three quality control (QC) goat plasma pools containing different concentration of each hormone were included at three places in each assay to monitor the assay drifts and interassay coefficient of variations (CV's). The interassay CV's for progesterone assay were 9.8% (n = 36) at 12.7  $\pm$  0.3 (SEM) nmol/l; 10.4% (n = 12) at 6.4  $\pm$  0.3 nmol/l and 13.3% (n = 21) at 2.7  $\pm$  0.1 nmol/l. The CV's for oestradiol-17 $\beta$  assays were 7.6% (n = 36) at 998.9  $\pm$  17.7 pmol/l; 10.1% (n = 36) at 623.4  $\pm$  10.5 pmol/l and 12.8% (n = 35) at 120.3  $\pm$  3.5 pmol/l. The intraassay CV's were calculated from 25 similar aliquots assayed together for each QC pool and were below 8% for both hormones. The sensitivity of the assays were 0.05 nmol/l (n = 21) and 11.8 pmol/l (n = 29) for progesterone and oestradiol-17 $\beta$  assays, respectively.

The respective hormonal values were averaged on monthly basis for each goat and each group and were analysed by one-way analysis of variance.

The difference between group means were compared by a modified Duncan's New Multiple Range Test (Kramer, 1956) as described by Steel and Torrie (1960).

#### Results

#### Clinical observations

Comparative detailed clinical observations in the same experimental and control groups have been described recently (Mutayoba et al., 1987). Morogoro goats were found to be more tolerant followed by the Arusha, Lambwe Valley and Imbo goats in that order. All infected goats become parasitaemic within 5–8 days and thereafter, the Morogoro goats had intermittent scantly parasitaemias than other groups. Thus, out of 77 samples examined for the presence of trypanosomes during the first two months of the infection in the Morogoro group (Table 1) 21 (27.3%) were negative, whilst, only 5 (7.2%), 12 (16.9%) and 3 (4.8%) of the 69, 71 and 62 samples examined in the Lambwe, Arusha and Imbo goats, respectively, were negative. This was significantly different (p < 0.01). This pattern persisted throughout the course of the infection.

The Morogoro goats had also less severe anaemia (indicated by PCV changes), weight losses and lowest mortality rate compared to the Imbo goats (Table 2). The Lambwe and Arusha goats were intermediate between the resistant (Morogoro) and susceptible (Imbo) goats.

#### Oestrous cycles

The lengths of the oestrous cycles between the experimental and control goats prior to the infection period were not markedly different (Table 3) and a length of  $20.1\pm0.2$  days (n = 104) being recorded. The cycle lengths were determined as an interval in days between two behavioural oestrus with the aid of teaser bucks and supplemented by hormonal assays. Following the onset of parasitaemia, irregular and increasingly shorter (p<0.05) overt and silent cycles (detected by hormonal analyses) were noted in all infected groups. In the more susceptible Imbo goats, cyclicity ceased at the second cycle post-infection, whilst in the more resistant Morogoro goats, shorter 3rd and 4th cycles were recorded before cessation. Ovarian cycles ceased at the end of the 3rd cycle in

Table 2. Changes in	n the mean monthly P(	CV% and body weig	hts (BW kg) of the	infected and cont	irol goats		
Duration of infection	Parameters (means ± SEM)	Lambwe Valley	Arusha	Morogoro	Imbo	Control	F ratio
Preinfection means	PCVBW	28.1±0.6 22.8±1.5 (10)	29.2±0.6 19.9±1.0 (9)	$28.9\pm0.5$ 19.3±1.2 (10)	28.8±0.7 21.7±1.2 (10)	28.9±0.7 21.3±0.9 (13)	
June	PCV BW	$-5.8^{a}\pm0.4$ $-1.8^{b}\pm0.3$ (8)	$-4.9^{a}\pm0.5$ $-1.4^{b}\pm0.2$ (9)	$-4.9^{a}\pm0.3$ $-0.6^{a}\pm0.2$ (10)	$-7.7^{b}\pm0.3$ $-0.7^{a}\pm0.3$ (8)	+0.2 <sup>c</sup> ±0.7 +0.1 <sup>c</sup> ±0.1 (13)	10.19*** 3.08**
July	PCV	$\begin{array}{c} -5.8^{a}\pm0.3\\ -3.1^{d}\pm0.5\\ (8)\end{array}$	$-5.7^{a}\pm0.5$ $-2.5^{ad}\pm0.5$ (7)	$-4.8^{a}\pm0.3$ $-0.9^{cb}\pm0.3$ (9)	$\begin{array}{c} -8.5^{b}\pm0.6\\ -1.8^{ab}\pm0.4\\ (6)\end{array}$	+1.5 <sup>c±0.3</sup> +0.1 <sup>c±0.3</sup> (13)	14.81*** 5.41***
August	PCV BW	$-7.0^{bc}\pm0.4$ $-3.0^{a}\pm0.5$ (6)	$-6.3^{ab}\pm0.6$ $-2.3^{a}\pm0.6$ (7)	$-4.8^{a}\pm0.5$ $-1.4^{a}\pm0.4$ (7)	$\begin{array}{c} -9.0^{c}\pm0.7\\ -2.7^{a}\pm0.9\\ (4)\end{array}$	$+1.8^{d}\pm0.4$ $+0.5^{b}\pm0.4$ (13)	9.05*** 1.62 ns
September	PCV	$\begin{array}{c} -6.7^{b}\pm0.5 \\ -4.2^{b}\pm0.5 \\ (6) \end{array}$	-6.8 <sup>b</sup> ±0.6 -3.7 <sup>ab</sup> ±0.7 (6)	$-4.3^{a}\pm0.3$ $-2.3^{a}\pm0.4$ (7)	$-9.1^{c\pm}0.9$ $-4.0^{ab}\pm1.1$ (4)	$+0.3^{d}\pm0.1$ $+0.8^{c}\pm0.5$ (13)	12.29*** 2.10 ns
October	PCV	$\begin{array}{c} -5.8^{b}\pm0.4 \\ -4.5^{b}\pm0.6 \\ (6) \end{array}$	$-6.4^{b}\pm0.5$ $-3.6^{ab}\pm0.8$ (6)	$-4.4^{a}\pm0.3$ $-2.4^{a}\pm0.6$ (7)	$ \begin{array}{c} -8.3^{c\pm 0.3} \\ -3.7^{ba\pm 1.1} \\ (4) \end{array} $	$+0.5^{d}\pm0.2$ +1.3 <sup>c</sup> ±0.4 (13)	10.74*** 1.57 ns
November	PCV	$\begin{array}{c} -5.1^{a}\pm0.3 \\ -3.5^{b}\pm0.5 \\ (6) \end{array}$	−5.0 <sup>a</sup> ±0.6 −2.5 <sup>ba</sup> ±0.9 (5)	$-4.0^{a}\pm0.3$ $-1.5^{a}\pm0.5$ (7)	$\begin{array}{c} -6.3^{b}\pm0.3\\ -1.5^{a}\pm0.9\\ (3)\end{array}$	$+0.6^{\circ}\pm0.1$ +1.4 $^{\circ}\pm0.5$ (13)	4.70*** 2.02 ns
- Values from Tune	e onwards denote loss	(-) or gain (+) from	the nreinfection v	alues			

- values from june onwards denote loss (-) of gain (+) from the preintection values.

- Means bearing the same superscripts (a b c d) in the same row do not differ significantly from each other (p < 0.05); control values are significant at

(p <0.01).</li>Numbers in brackets denote surviving goats.

fable 3. Chang	ses in the length (	of oestrous cycles (c	days) of infected ar	id control goats				
Approximate beriod in nonths	Cycles before (–) and after (+) infection	Lambwe Valley	Arusha	Morogoro	Imbo	Control	Level of significance F <sub>1</sub> ratio	Level of significance F <sub>2</sub> ratio
May to	-2	20.0±0.3 (10)	19.2±0.4 (9)	19.5±0.5 (10)	19.1±0.1 (10)	19.8±0.4 (13)	0.85 ns	0.71 ns
Mid-June	-1	$19.8\pm0.4(10)$	19.1±0.8 (9)	19.4±0.3 (10)	19.9±0.7 (9)	20.6±0.4 (13)	0.41 ns	1.32 ns
Mid-June	1	$17.0^{ab}\pm2.3$ (8)	17.2 <sup>ab</sup> ±1.6 (9)	19.6 <sup>bc</sup> ±1.7 (9)	15.5ª±2.0 (8)	20.9°±0.4 (13)	0.91 ns	2.21**
	2	$13.3^{a}\pm1.9$ (8)	15.3a±2.0 (7)	15.9a±2.2 (9)	11.8 <sup>a</sup> ±2.0 (4)	20.7 <sup>b</sup> ±0.9 (12)	0.74 ns	5.56***
	ŝ	9.7 <sup>b</sup> ±1.0 (6)	14.0 <sup>ab</sup> ±2.8 (5)	15.7 <sup>a</sup> ±1.4 (7)	Ĩ	20.6°±0.7 (10)	2.62**	6.21***
	4	1	1	7.5±0.5 (3)	Ĩ	21.3±0.7 (6)		
	5	I	ſ	τ	]	23.0±1.0 (3)		
Mid-October	9	1	1	Ĩ	I	I		
Mid-October	L	Î	Ĺ	19.0±0.5 (2)	20.5±0.5 (2)	21.3±0.5 <sup>n</sup> (9)		
to November	8	1	16.0±0.5° (2)	15(1)	I	19.8±0.5 <sup>n</sup> (8)		
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Values given as means ± SEM

Numbers in brackets denote cycling goats. Goats not included are either acyclic or dead (compare with Table 2).
F<sub>1</sub> and F<sub>2</sub> denote F values between the infected groups, infected and control groups, respectively.
ns, \*\* \*\*\* = nonsignificance, significance at 5% and 1% level of confidence, respectively.

– Means bearing the same superscripts (a b c d) in the same row do not differ significantly (p < 0.05).

- enOne cycling Arusha and 2 control goats, respectively, became pregnant and their values excluded.

Lambwe and Arusha goats. Resumption of the oestrous cycles were recorded in six infected goats following the spell of short rains in mid-October.

Seasonal ovarian activity was observed in the control goats, with highest cyclicity (100%) in May and June and least (0%) in September. Cyclicity was once again high at the start of short rains in late October. Eleven control goats came back on heat within 1½ weeks after the onset of short rains but resumption of cyclicity in the infected goats (two from each of the Arusha, Morogoro and Imbo groups) was delayed to an average of 3 weeks after the onset of rains. However, two of the control and one infected (Arusha) goats became pregnant thereafter.

#### Plasma progesterone levels

No marked regional (group) differences were noted in the monthly and peak luteal progesterone values prior to trypanosome infection (Fig. 1, Table 4). However, a significant difference in the mean monthly and luteal progesterone values were recorded within a month and 2nd cycle postinfection, respectively, between the infected and control goats and between infected groups. Whereas, the control goats maintained high mean progesterone levels  $(10.7 \pm 0.8 \text{ nmol/l})$  in July, one month postinfection (Fig. 1), the mean monthly progesterone values of the infected groups  $(3.0 \pm 0.5 \text{ nmol/l}; 4.7 \pm 0.6 \text{ nmol/l}; 7.1 \pm 0.8 \text{ nmol/l}$  and  $2.2 \pm 0.3 \text{ nmol/l}$  in the Lambwe, Arusha, Morogoro and Imbo groups, respectively) recorded during the same month, were significantly lower (p<0.001).

The decline was, however, least in the Morogoro goats (p < 0.001) when compared to other infected groups. This pattern was maintained up to the end of August when all infected and control goats became acyclic. The mean progesterone levels of the infected groups were still slightly low in September (nonsignificant) and in October (p < 0.01) during the acyclic period. Luteal progesterone values (Table 4) followed the same trend between the infected and control goats from the 2nd cycle postinfection. The differences in hormonal levels among the infected groups were, however, equivocal.

Seasonal ovarian activity in the control goats were marked by a decline in the mean monthly progesterone values in August (p < 0.01) and September (p < 0.001). Following the onset of short rains, the hormonal levels started to rise. However, the mean monthly progesterone values of the pregnant infected Arusha goat was significantly lower (6.9 nmol/l, p < 0.001) than the pregnant control goat values (>20 nmol/l).

# Plasma oestradiol-17β levels

The preinfection monthly oestradiol levels and corresponding preovulatory peaks were not significantly different between the experimental and control groups in May (Fig. 1, Table 5).

A significant difference in the monthly (p<0.01) and peak (p<0.01) values



Fig. 1. Mean monthly progesterone and oestradiol- $17\beta$  in the infected and control goat groups.

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Approximate period in months	Cycles before (–) and after (+) infection	Lambwe Valley	Arusha	Morogoro	Imbo	Control	Level of significance F <sub>1</sub> ratio	Level of significance F <sub>2</sub> ratio
May to	-2	21.3±0.7 (10)	20.2±0.8 (9)	20.8±0.7 (10)	20.4±0.9 (10)	21.8±0.8 (13)	0.46 ns	0.82 ns
Mid-June		21.8 <sup>b</sup> ±0.8 (10)	21.7 <sup>b</sup> ±1.1 (9)	19.0 <sup>a</sup> ±0.7 (10)	19.6 <sup>ab±0.8</sup> (9)	19.3 <sup>a</sup> ±0.6 (13)	2.82**	2.79**
Mid-June	1	$19.6^{a}\pm1.1$ (8)	16.7ª±1.5 (9)	18.3 <sup>a</sup> ±1.2 (9)	16.2 <sup>a</sup> ±2.0 (8)	$19.8^{a}\pm0.8$ (13)	1.08 ns	1.56 ns
, <u></u>	2	$5.4^{a}\pm0.5$ (8)	10.0 <sup>b</sup> ±1.2 (7)	13.8°±1.3 (9)	10.1 <sup>bc</sup> ±1.5 (4)	18.3 <sup>d</sup> ±0.9 (12)	9.89***	22.69***
	S	$4.6^{a}\pm0.6$ (6)	8.7 <sup>b</sup> ±1.5 (5)	10.2 <sup>b</sup> ±0.9 (7)	ľ	18.8°±1.0 (10)	5.93***	27.46***
	4	Ĩ	I	5.6±1.3 (3)	1	19.3±0.9 (6)		
	5	1	1	Ĩ	1	12.5±4.2 (3)		
Mid-October	6	Ĩ	t	ſ	Ę	I		
Mid-October	7	Ĩ	1	7.6±2.7 (2)	8.8±2.1 (2)	16.3±1.2 (9)		
to November	8	I	6.7±2.4 (2)	9.7 (1)	ι	16.9±0.8 (8)		

- Values given as means  $\pm$  SEM. - F<sub>1</sub> and F<sub>2</sub> are F values between infected groups, infected and control groups, respectively.

- Means bearing the same superscripts (a b c d) in the same row do not differ significantly at (p < 0.05); control values in the 2nd and 3rd cycle are significant at (p<0.001).

- ns, \*\* \*\*\* denote nonsignificance, significance at 5% and 1% level of confidence, respectively.

Table 4 Mean luteal plasma properterone levels (nmol/l) of infected and control goals

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Approximate beriod in nonths	Cylces before (–) and after (+) infection	Lambwe Valley	Arusha	Morogoro	Imbo	Control	Level of significance F1 ratio	Level of significance F <sub>2</sub> ratio
May to	-2	896.7±29.8 (10)	899.6±51.0 (9)	808.9±51.2 (10)	785.2±32.4 (10)	847.5±23.5 (13)	2.00 ns	1.76 ns
Mid-June		814.1 <sup>ab±24.3</sup> (10)	741.2 <sup>b</sup> ±32.3 (7)	870.8 <sup>a</sup> ±23.6 (9)	737.3 <sup>b</sup> ±28.4 (9)	786.6 <sup>ab±17.3</sup> (13)	5.59***	4.72***
Mid-June	1	611.3 <sup>a</sup> ±55.1 (8)	582.5 <sup>a</sup> ±62.2 (9)	605.9 <sup>a</sup> ±55.1 (9)	576.1 <sup>a</sup> ±72.5 (8)	792.6 <sup>b</sup> ±22.4 (13)	0.08 ns	3.41**
	2	365.0 <sup>ba</sup> ±52.8 (8)	401.0 <sup>b</sup> ±36.1 (7)	404.9 <sup>b</sup> ±34.9 (9)	262.7 <sup>a</sup> ±18.8 (4)	679.1°±52.5 (12)	2.68**	11.15***
	3	274.4 <sup>a</sup> ±34.3 (5)	$323.6^{a}\pm80.5$ (5)	365.4 <sup>a</sup> ±42.1 (4)	1	588.5 <sup>b</sup> ±29.8 (9)	0.59 ns	11.16***
	4	1	1	297.9 (1)	1	616.8±39.0 (6)		
→	5	Ľ	I	1	1	506.0±93.7 (2)		
Mid-October	6	1	1	Ľ	ľ	I		
Mid-October	7	ť	428.2 (1)	340.9±22.3 (2)	285.9±59.5 (2)	790.3±27.3 (9)		
o November	8	1	1	412.6 (1)	ľ	634.4±30.7 (8)		
- Values given	i as means ± SEN	М.						

Table 5. Mean neak plasma pestradiol-178 levels (mmol/l) of infected and control spats

-  $F_1$  and  $F_2$  denote F values between the infected groups, infected and control groups, respectively. - ns, \*\* \*\*\* denote nonsignificance, significance at 5% and 1% level of confidence, respectively.

- Means bearing the same superscripts (a b c) in the same row do not differ significantly at (p < 0.05). - Numbers in brackets denote cycling goats whose preovulatory oestradiol peaks were determined.

in June were attributed to the missed peak values in the Arusha (two), Imbo (one) and Morogoro (one) goats.

A significant decline in the monthly (p<0.01) and peak values (p<0.05; p<0.01) were recorded in all infected groups within a month, and first and second cycle post-infection, respectively. The monthly levels remained low (p<0.01) during the acyclic period in the infected groups. The Morogoro goats, however, maintained slightly higher monthly values in July and August (p<0.05) than the Lambwe and Imbo goats. Changes in the preovulatory peaks were not significantly different among the infected groups.

Seasonality in the ovarian function in the control goats were marked by a significant decline in both monthly and peak oestradiol levels (p < 0.01) from July to October before increasing in November following resumption of the ovarian cycles. The monthly and peak levels were still significantly lower (p < 0.01) in the few infected goats which came back on heat in November.

## Discussion

Chronic trypanosome infection resulted in progressive cessation of oestrous cycles and marked depression of plasma progesterone and oestradiol levels. Changes were initially more marked in the peak hormonal levels, but first decline occurred also in the basal values as the infection period progressed. Trypanosome-induced depression of plasma oestradiol-17 $\beta$  and testosterone have recently been reported in goats (Kivihya et al., 1985; Waindi et al., 1986). Disruption of ovarian activity and prolonged elevation of progesterone levels due to the persistence of corpora lutea have also been reported recently in the British white goats and Boran cows chronically infected with T. congolense (Luckins et al., 1986). A close relationship in the fall of preovulatory oestradiol and luteal progesterone values observed in the infected goats in this study suggested the lack of normal corpora lutea (CL's) formation resulting from inhibited preovulatory oestradiol rise. Histologically, O'hara et al. (1985) and Mutavoba et al. (1988) reported the degeneration of partially lutenized tertiary follicles, absence of CL and lymphocytic infiltration in the degenerating ovarian stroma in the female goats infected with T. congolense. It seems from the above studies that trypanosomiasis causes variable reproductive anomalies in female animals ranging from luteolysis failure to impaired luteogenesis and degeneration of the entire ovarian stroma.

The plasma oestradiol levels obtained in this study were, however, higher than values reported in other breeds of goats but were closely similar to those previously reported in the Small East African goats from Uganda (Katongole and Gombe, 1985). Values ranging between 120–900 pmol/l were reported during normal oestrous cycles of the goats in that study.

The rate and degree of ovarian depression in the infected goats seemed to depend on the susceptibility of individual goats and in resistant goats, they also

depended on duration of the infection. The resistant Morogoro goats were better in maintaining the ovarian cycles and higher hormonal values than other infected groups, at least in the first 2 months of the infection. It was, however, observed from this study, that, chronic parasitaemia even at low levels may exacerbate the underlaying ovarian lesions and reduce the fertility of resistant animals even further as evidenced by the loss of ovarian cycles in the Morogoro goats and levelling of hormonal values between infected groups in subsequent months.

Resumption of ovarian activity was observed in some chronically infected goats during the spell of short rains.

Although, this could have been due to the seasonal effect as also evidenced in the control goats and reported in other studies in the Small East African goats (Katongole and Gombe, 1985), it was an indication that trypanotolerant animals were potentially capable of regaining their ovarian function during the course of the infection. Indeed, one infected goat became pregnant. Quantitatively all goats which survived a six month infection period irrespective of the group were viewed as having been more resistant (trypanotolerant) than those which succumbed. These were 3 (Imbo), 5 (Arusha), 6 (Lambwe) and 7 (Morogoro) goats. The surviving Morogoro goats were in good body condition and had scanty parasitaemias than other infected groups. Their residual fertility by the time the study was terminated was still markedly reduced as indicated by low hormonal values even in the goat which became pregnant. These results, however, help to explain the high level of herd infertility and pregnancy failures often associated with trypanosomiasis in the field (Parkne and Dhake, 1972; Kanyari et al., 1983).

It was also noted in this study that trypanosomiasis delayed the resumption of ovarian cycles in the infected goats following the onset of short rains. In the field conditions, this delay is more likely to be worsened by an increase in the tsetse population and rate of trypanosome infections during the rainy season. To what extent does it affect the onset of goat breeding season in tsetse-endemic areas during rainy seasons is poorly documented and needs to be investigated. It suffices to speculate from the observations obtained from this study that there might be a considerable interference in the breeding season even in resistant infected animals so that young ones are born late when pastures are deteriorating. In this regard, other conventional disease control methods (namely tsetsecontrol and chemotherapy) are quite essential especially during the rains so as to exploit the genetic superiority of resistant livestock in improving their reproductive efficiency.

#### Acknowledgments

We wish to thank Messrs. J. Osaso, D. Ndegwa, L. M'rewa, F. Kariba and H. Odongo for their expert technical assistance and Miss J. Mbena for typing this manuscript. The Research project was partly financed by the German Academic Exchange Service (DAAD/AFAA) in collaboration with Sokoine University of Agriculture, Tanzania.

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