The effect of previous growth retardation on energy and nitrogen metabolism of goats infected with *Trypanosoma vivax*

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The effect of growth retardation, resulting from feed restriction for a prolonged period, on the course of infection with *Trypanosoma vivax* was studied. Twelve male castrated West African Dwarf goats were subjected to a restricted feeding regimen of 55 g pelleted lucerne (*Medicago sativa*)/kg body weight$^{0.75}$ per d for on average 17 weeks. Twelve other animals were fed on pelleted lucerne *ad libitum*, resulting in a normal growth pattern. After this period, all animals were fed on pelleted lucerne *ad libitum*, and six animals of each previous feeding regimen treatment were infected with *Trypanosoma vivax*. The other animals served as controls. In week 2 and 4 post infection (*pi*) energy and N balances were measured. In the week before infection and during infection blood biochemical and clinical variables were measured. At 2 weeks before, and 4 weeks after infection, a liver biopsy was taken for measurement of triacylglycerol. Infection caused intermittent fever and anaemia. The first peak of fever persisted longer in infected animals with normal growth than in infected animals with retarded growth. Gross energy and metabolizable energy intake, and energy retention were reduced in infected animals. Metabolizable energy requirements for maintenance were increased by infection. Plasma non-esterified fatty acids (NEFA) and glucose concentrations were increased in infected animals, whereas serum triiodothyronine and thyroxine concentrations were decreased. Plasma urea concentration and liver triacylglycerol were unaffected. No interaction of growth retardation with infection with respect to blood biochemical variables was found, apart from plasma NEFA in week 2 *pi*. N retention was not significantly affected by treatments. In conclusion, minor indications were found for an interaction between growth retardation, as applied in the present study, and trypanosomiasis infection in West African Dwarf goats with respect to energy and N metabolism.

Trypanosomiasis: Goats: Energy metabolism

Trypanosomiasis, a protozoan disease of (sub-)humid regions of Africa, causes anorexia, anaemia and intermittent fever in domestic animals (Van den Ingh *et al.* 1976; Zwart *et al.* 1991). Energy requirements for maintenance of West African Dwarf goats are increased by approximately 25 % during the acute phase of the infection (Verstegen *et al.* 1991). As a consequence of trypanotolerance, several local breeds of goats, sheep and cattle are able to survive from trypanosome infection (Murray & Morrison, 1981).

Malnutrition often interacts with the severity of disease (Beisel, 1985). Also the degree of trypanotolerance is affected by the nutritional status of the host animal (Murray, 1988).

In tropical countries malnutrition frequently occurs due to shortage of good-quality roughage. In small ruminants malnutrition has been found to be related to increased mortality due to trypanosomiasis (Reynolds & Ekwuruke, 1988). Our group fed fibrous diets with a high or a low nutritional quality for 3 months to trypanotolerant West African Dwarf goats (Van Dam, 1996). In this experiment we observed no interaction between trypanosome infection and fibrous feed quality with respect to N retention, i.e. the negative effect of infection was not greater in animals fed on a poor-quality diet compared with animals fed on a good-quality diet. However, in both feed groups, animals were in good body condition at the start of infection, enabling them to mobilize (part of their) body reserves during infection.

The present trial investigated how dietary limitations (by offering maintenance feed to growing animals) for a prolonged period would affect the course of a subsequent infection with *Trypanosoma vivax* with respect to energy and N metabolism.

MATERIAL AND METHODS

Experimental design

Feed-restriction period. At the start of the feed-restriction period, pairs of goats were selected by matching on a similar body weight, from a group of castrated West African Dwarf goats with a mean age of 4·3 (SD 1·7) months. From each pair of animals, one animal was randomly allocated to restricted feeding (retarded-growth group; R), the other was to receive the ration *ad libitum* (normal-growth group; N). The initial mean body weight of group R was 13·34 (SD 2·49) kg, and of group N 13·24 (SD 1·56) kg. The mean duration of the feed restriction period was 16·5 (SD 3·3) weeks. During this period, the applied feed restriction was 55 g fresh feed/kg metabolic body weight (body weight$^{0.75}$) per d (approximately maintenance level).

Infection period. After the feed-restriction period all animals received pelleted lucerne (*Medicago sativa*) *ad libitum* for a period of 2 weeks, preceding the date of infection. This period of 2 weeks was required for adaptation of the restricted animals to the *ad libitum* feeding regimen which would then be applied to all animals following infection. Half the animals of both feed groups (n 6) were randomly selected to be infected with trypanosomes. Therefore, a 2 × 2 factorial design was used, i.e. infected normal growth (IN; n 6), infected retarded growth (IR, n 6), control normal growth (CN; n 6) and control retarded growth (CR; n 6).

The goats from the IN and IR groups were infected with 1×10^6 *Trypanosoma vivax* (strain Y486, isolated by Leeflang *et al.* 1976). Control animals were sham infected by intravenous injection of 2 ml physiological saline (9 g NaCl/l).

On the day of infection (day 0) the mean body weight of group R goats was 15·60 (SD 2·91) kg, and that of group N goats 21·59 (SD 3·00) kg; mean change in body weight over the feed-restriction period was 17 (SD 7) and 64 (SD 15) g/d for group R and group N animals respectively.

At day 28 post infection (*pi*) all animals were killed by intravenous administration of 5 ml T61 (Hoechst Veterinär GmbH, München, Germany).

Feeding and housing

Throughout the experiment, a diet of pelleted lucerne was offered to the animals. The average DM content of the feed was 924 g/kg, with 175 g crude protein (N × 6·25) and 16·2 kJ gross energy (GE)/kg DM. Before infection animals were housed individually in balance cages for at least 3 weeks.

Table 1. *Measurements and time schedule for each pair of West Africa Dwarf goats during the study*

Period after infection (d)	−14	−7	0	7	14	21	28
Housing	Individual pen		| - Dum -	| - RC -	| - Dum -	| - RC -	|
Treatment	B		Inf				Eut;B
Feed intake	+++++++	+++++++	+++++++	+++++++	+++++++	+++++++	+++++++
Rectal temperature			+++++++	+++++++	+++++++	+++++++	+++++++
Body wt	+	+	+	+	+	+	+
Blood samples	+	+	+	+	+	+	+
Energy-balance measurements				|--------|		|--------|	

Dum, dummy chamber; RC, respiration chamber; B, Biopsy; Inf, day of infection; Eut, day of killing; +, measurements made; |---|, period of measurement.

The time schedule of housing and measurements is given in Table 1. During weeks 1 and 3, *pi* animals were housed in a dummy chamber to allow adaptation to housing conditions in the climatic respiration chamber. They were housed in one of two identical climatic respiration chambers (described by Verstegen *et al.* 1987) in week 2 and 4 *pi*. Two goats were housed in each chamber. This was done to prevent stress due to social isolation (Carbonaro *et al.* 1992). The two animals were separated by a wire fence in order to facilitate the individual measurement of feed intake. The space allowance per goat was 1·00 m × 0·40 m × 0·97 m in both the dummy and respiration chambers. The light period was between 07·00 and 19·00 hours. Temperature was maintained at 20°. In the respiration chambers relative humidity was maintained at 65 %. The allocation of animals from the different treatments over the two chambers was balanced. Because only four animals were housed in the respiration chamber at the same time, animals were infected in groups of four, after each other. The sequence of the different treatments was balanced over time, however, to prevent possible bias on the measurements originating from this procedure.

Measurements and calculations

Rectal temperature was measured daily from day 0 onward just before morning feeding, to monitor possible fever during infection. Blood samples were collected on days −7, 0, 7, 14, 21 and 28 in the morning after feeding. Blood was taken from the jugular vein, using Venoject vacuum tubes (Terumo, Leuven, Belgium). Packed cell volume (PCV) was measured in heparinized blood, by means of a microhaematocrit centrifuge. Parasitaemia was measured from assessment of the number of leucocytes per ml heparinized blood using a CoulterCounter, and from the number of parasites relative to leucocytes in a thick smear stained with Giemsa, and was expressed as the number of parasites per ml.

Body weight was measured every week during the restriction period and the infection period. Daily body-weight gain over the infection period (day 0 to day 28) was calculated per animal per d and per kg body weight$^{0.75}$ per d.

From day −14 (i.e. 14 d before infection) onwards, individual daily feed intake was measured by offering lucerne pellets *ad libitum* in the early morning, and subsequent collection of feed residues the next day. DM content of offered feed and feed residues was analysed by drying at 103° for 4 hours using composite samples collected on a per animal per week basis; from this daily DM intake (DMI) per kg body weight$^{0.75}$ was calculated.

A liver biopsy was taken from all animals (method described by Van den Top *et al.* 1995) 14 d before infection. After the death of the animals at 28 days *pi*, another liver sample was taken by incision of the thoracic wall. Liver samples were stored in saline and analysed for triacylglycerol (TAG) with kit no. 405 (Sigma Chemical Co., St Louis, MO, USA) to monitor the effects of treatments on liver fat metabolism.

The following variables were measured in the climatic respiration chambers to calculate the energy and N balance for pairs of goats for a 7 d period. O_2 consumption and CO_2 and CH_4 production were measured during successive intervals of 9 min. For each interval, heat production (HP) was calculated from these gaseous exchanges, using the equation of Brouwer (1965). Faeces and urine, and the water that was used to clean the chamber, were collected and weighed at the end of the balance period and a representative sample was taken and analysed for N by the Kjeldahl method with K_2SO_4 and $CuSO_4$ as catalysts. Gross energy was measured using bomb calorimetry (IKA Analysentechniek GmbH, Heitersheim, Germany). DM of faeces were determined by drying at 103° for 4 h, and ash content by incubation for 2 h at 550°. The weekly amount and composition (DM, ash, N and GE) of offered and refused feed were measured. GE was calculated as the amount of ingested energy in feed. Metabolizable energy (ME) was calculated as GE minus the energy in faeces, urine, expired CH_4 and the energy trapped in cleaning water. Energy retention (ER) was calculated as ME minus HP.

For each observation the ME maintenance requirements (ME_m) were calculated. It was assumed that energy above maintenance had been deposited with a partial efficiency of 0·6, whereas energy mobilization from the tissues could be prevented by offering 1·25 kJ ME/kJ body energy loss (partial efficiency of 0·8; Agricultural Research Council, 1980).

N retention (NR) was calculated as the difference between N intake and N losses via faeces and urine, and was expressed in g/kg body weight$^{0.75}$ per d. NR was corrected for N lost from faeces and urine to the air. Protein gain was calculated as the product of retained protein (NR × 6·25) and the energy content of 1 g deposited protein (23·7 kJ). Fat gain was calculated, being the difference between ER and protein gain. NR was expressed in g/kg body weight$^{0.75}$ per d. The ME, HP, ER, ME_m, protein gain and fat gain were expressed in kJ/kg body weight$^{0.75}$ per d.

In the respiration chambers, for pairs of goats, physical activity was measured continuously using Doppler-radar activity meters (Radar MD5; Suther, Vierpool, Amsterdam, The Netherlands). The movements of the animals housed in the respiration chambers were converted into counts per interval of 9 min which corresponded with a HP measurement interval. The relationship between number of activity counts and HP was assessed per goat pair per respiration week by linear regression. These regression estimates were used to calculate activity related HP (HP_{act}). The difference between HP and HP_{act} was HP corrected for activity (HP_{cor}).

The following blood variables associated with energy metabolism were measured. From blood containing lithium heparin and paraoxon, plasma non-esterified fatty acids (NEFA) concentration was analysed enzymically (NEFA C; Instruchemie BV, Hilversum, The Netherlands). From blood containing NaF and potassium oxalate as anticoagulants, plasma glucose concentration was measured enzymically (Boehringer Mannheim GmbH Diagnostica, Mannheim, Germany). Serum triiodothyronine (T3) and thyroxine (T4) were measured using a homologous radioimmunoassay (RIA); serum insulin concentration was measured by means of RIA (Coat-a-Count Insulin, Diagnostic Products Corporation, Los Angeles, USA). Serum urea was measured enzymically (Boehringer Mannheim GmbH Diagnostica, Mannheim, Germany), being an indicator of N metabolism.

After death, gross and microscopic examination was done on all infected animals and four control animals; the methods are described by Van den Ingh *et al.* (1976).

Statistical model

The results were analysed using the general linear models procedure of SAS statistical package (Statistical Analysis Systems, 1990). From preliminary analysis of the results, it was concluded that both respiration chamber, as well as the time sequence in which the animals entered the experiment, did not affect the data. Therefore, these factors were not included in the statistical model.

For energy- and N-balance traits, each pair of goats represented an experimental unit; for all other traits, the experimental unit was the individual animal. Measurements before and after infection were analysed separately.

Treatment effects were tested using two-way ANOVA with repeated measurements; the effect of time after infection was taken up in the model:

$$Y_{ijkl} = \mu + I_i + G_j + (I \times G)_{ij} + e_{1,ijk} + T_l + e_{2,ijkl}, \quad (1)$$

in which Y_{ijkl} is dependent variable; μ is overall mean; I_i is fixed effect of infection ($j = 1, 2$), G_j is fixed effect of growth pattern in pre-infection period ($i = 1, 2$), $(I \times G)_{ij}$ is fixed effect of interaction between infection and growth pattern, $e_{1,ijk}$ is error term 1 which represents the random effect of goat nested within infection × growth pattern treatment ($k = 1, \ldots, 6$), T_l is fixed effect of time after infection ($l = 1, \ldots, 4$ weeks; $l = 1, \ldots, 28$ d), $e_{2,ijkl}$ is error term 2.

The I_i, G_j and $(I \times G)_{ij}$ effects were tested against error term 1; T_l was tested against error term 2. Differences between treatments were significant if $P < 0{\cdot}05$. Differences between treatments tended towards significance if $0{\cdot}5 < P < 0{\cdot}10$.

Preliminary analysis showed that HP, body temperature, DMI, and the blood traits PCV, parasitaemia, NEFA, insulin, T4, T3 were affected by the time after infection. For these traits, the factors $e_{1,ijk}$ and T_l were removed from the model and the results at different times of measurement were analysed separately. This reduced model was also applied for the single measurements: hepatic TAG before and after infection, and body-weight change during the restriction and infection period.

For all energy- and N-balance variables, except HP, no time effect was observed, and model (equation 1) was used with the exclusion of the T_l factor.

To study partial efficiency with which ME is deposited, the relationship between data on ER and ME, pooled per animal, was studied using linear regression. Also the relation between NR and ER was studied, using data pooled per animal.

RESULTS

General course of infection

All infected animals developed intermittent fever about 4 d after inoculation of the *T. vivax* parasites (Fig. 1). The mean rectal temperature from day 4 until day 28 *pi* was 39·89 (SD 0·21) and 38·60 (SD 0·18)° for infected and control goats respectively. The first peak of fever persisted for a longer time in group N animals (with normal growth), compared with group R animals (with retarded growth; $P < 0{\cdot}05$). After this, fever fluctuated independently of effects of growth retardation of animals.

PCV gradually decreased in all infected animals with time after infection ($P < 0{\cdot}001$) to an average 17 % in week 4 *pi* (control animals had a mean PCV of 38 %). Also interaction between infection and growth retardation with respect to PCV was observed in

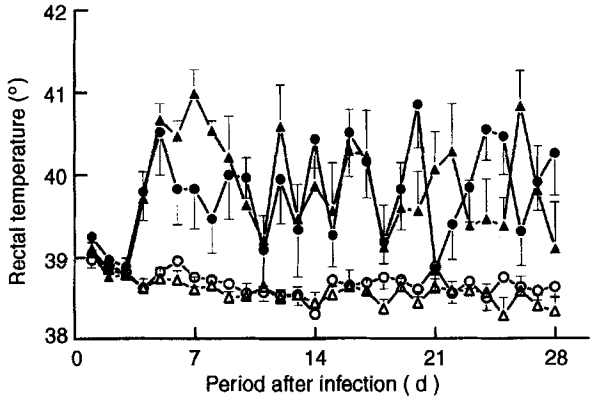

Fig. 1. The effect of *Trypanosoma vivax* infection and different growth patterns of West African Dwarf goats, on rectal temperature. (▲), Infected goats fed on pelleted lucerne (*Medicago sativa*) *ad libitum* (normal growth); (●), infected goats fed on 55 g pelleted lucerne/kg body weight$^{0.75}$ per d (retarded growth); (△), non-infected (control) goats, normal growth; (○), control goats, retarded growth. Points represent mean values with their standard errors represented by vertical bars. For details of treatments and procedures, see pp. 428–429.

week 1 and 2 *pi* ($P < 0.01$), i.e. control animals with retarded growth (group CR) had a PCV that was 5 % points lower than the PCV of control animals with normal growth (group CN), but this difference was not present in infected animals with either treatment.

All infected animals showed parasites in the blood, but parasitaemia followed an irregular course; towards the end of the infection period some animals had undetectable parasite levels. Parasitaemia was not significantly different between group R animals and group N animals ($P > 0.10$).

Dry matter intake and body weight gain

The mean daily DMI of animals in the different treatment groups is presented in Fig. 2; two mean values per week were calculated (3 d and 4 d means). Infection reduced DMI from days 5 to 22 *pi* (at least $P < 0.05$); from day 23 onwards no effects were detected ($P > 0.10$). No interaction between growth retardation and infection was observed ($P > 0.10$).

Body-weight gain during the infection period was affected by growth retardation, i.e. animals from group IR (retarded growth before infection) gained more weight after infection, both per d, and per kg body weight$^{0.75}$ per d, compared with group N animals (normal growth) ($P < 0.05$; Table 2).

Energy and nitrogen balance

Results on energy-balance variables, protein gain, and derived maintenance requirements are presented in Table 3. Infection reduced GE and ME ($P < 0.05$). HP tended to be increased in infected goats ($P < 0.10$). The metabolizability (ME/GE) was not changed by treatments ($P > 0.10$), and averaged 0.56, 0.57, 0.54, and 0.56 for treatments IN, IR, CN and CR respectively. ER was decreased by infection ($P < 0.001$), and ER was lower in group N animals, compared with group R animals ($P < 0.05$). There was no interaction between growth retardation and infection on these variables ($P > 0.10$).

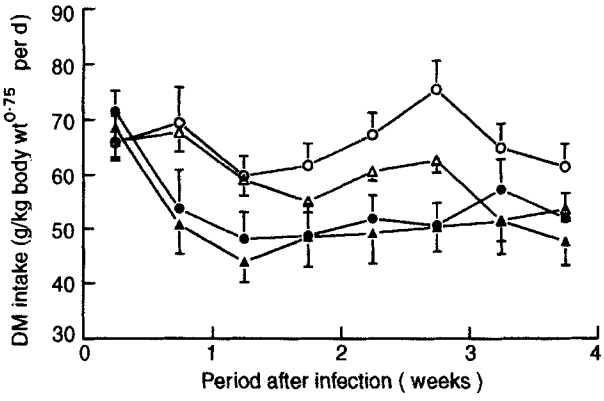

Fig. 2. The effect of *Trypanosoma vivax* infection and different growth patterns of West African Dwarf goats, on DM intake (g/kg body weight$^{0.75}$ per d). (▲), Infected goats fed on pelleted lucerne (*Medicago sativa*) *ad libitum* (normal growth); (●), infected goats fed on 55 g pelleted lucerne/kg body weight$^{0.75}$ per d (retarded growth); (△), non-infected (control) goats, normal growth; (○), control goats, retarded growth. Points represent mean values with their standard errors represented by vertical bars. For details of treatments and procedures, see pp. 428–429.

Table 2. *The effect of* Trypanosoma vivax *infection and different growth patterns of West African Dwarf goats, on body weight gain over the infection period*†

(Mean values and standard deviations for six goats)

Infection status (I)...	Infected				Control				Statistical significance of effect of‡:		
Growth pattern (G)...	Normal		Retarded		Normal		Retarded				
Body-wt gain	Mean	SD	Mean	SD	Mean	SD	Mean	SD	I	G	I × G
g/d	−19·3	41·8	0·8	23·1	−13·4	8·9	14·4	23·9	NS	*	NS
g/kg body wt$^{0.75}$ per d	−1·83	4·40	0·39	2·69	−1·36	0·96	2·05	3·20	NS	*	NS

Normal, pelleted lucerne (*Medicago sativa*) *ad libitum*; Retarded, 55 g pelleted lucerne/kg body wt$^{0.75}$ per d.
* $P < 0.05$.
† For details of treatments and procedures, see pp. 428–429.
‡ NS, $P > 0.10$.

N digestibility was not changed ($P > 0.10$) by treatments and averaged 0·543. The energy deposited in body protein tended to be higher in group N animals ($P < 0.10$). Fat gain was decreased in infected animals ($P < 0.001$) and was increased in group R animals ($P < 0.05$). The calculated ME_m was increased ($P < 0.001$) by 25 % in infected animals. Infected animals either with normal or with retarded growth showed a slightly different ME_m (483 *v.* 452 g/kg body weight$^{0.75}$ per d). This was not significant, however ($P = 0.25$).

A positive relationship between ER and ME intake was observed (Fig. 3). However, no differences among treatments were detected ($P > 0.10$). A positive relationship was also observed between NR and ER (Fig. 4) without effect of treatments ($P > 0.10$).

HP was affected by time after infection. Therefore results for HP, HP_{cor} and HP_{act} were analysed for week 2 and 4 *pi* separately (Table 4). In week 4 *pi*, but not in week 2 *pi*, HP

Table 3. *The effect of* Trypanosoma vivax *infection and different growth patterns of West African Dwarf goats, on gross energy intake (GE), metabolizable energy intake (ME), heat production (HP), energy retention (ER), protein gain, fat gain and ME for maintenance (ME_m)*†

(Values are means and standard deviations for duplicate measurements for six goats expressed in g/kg body weight$^{0.75}$ per d)

Infection status (I)...	Infected				Control				Statistical significance of effect of‡:		
Growth pattern (G)...	Normal		Retarded		Normal		Retarded		I	G	I × G
	Mean	SD	Mean	SD	Mean	SD	Mean	SD			
GE	885	41	923	161	1017	74	1141	124	*	NS	NS
ME	498	37	530	94	552	43	639	47	*	NS	NS
HP	492	18	480	52	427	22	457	43	t	NS	NS
ER	6[a]	31	50[ab]	43	125[bc]	29	183[c]	24	***	*	NS
Protein gain	13	6	22	13	20	13	33	5	NS	t	NS
Fat gain	−7[a]	28	28[a]	31	105[b]	16	150[b]	22	***	*	NS
ME_m	483[a]	23	452[ab]	32	373[b]	21	376[b]	43	***	NS	NS

[a,b,c] Mean values with unlike superscript letters were significantly different ($P < 0.05$).

Normal, pelleted lucerne (*Medicago sativa*) *ad libitum*; Retarded, 55 g pelleted lucerne/kg body wt$^{0.75}$ per d; t, tendency ($P < 0.10$).

* $P < 0.05$, *** $P < 0.001$.

† For details of treatments and procedures, see pp. 428–430.

‡ NS, $P > 0.10$.

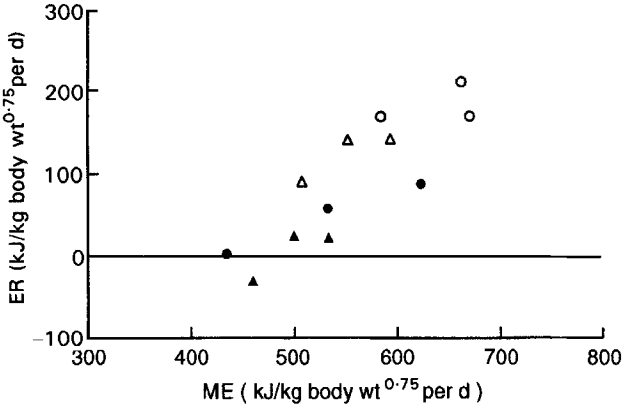

Fig. 3. The effect of *Trypanosoma vivax* infection and different growth patterns of West African Dwarf goats, on the relationship between energy retention (ER) and metabolizable energy intake (ME; both in kJ/body weight$^{0.75}$ per d). (▲), Infected goats fed on pelleted lucerne (*Medicago sativa*) *ad libitum* (normal growth); (●), infected goats fed on 55 g pelleted lucerne/kg body weight$^{0.75}$ per d (retarded growth); (△), non-infected (control) goats, normal growth; (○), control goats, retarded growth.

was higher in infected animals. If HP was corrected for physical activity (HP_{cor}), a stronger significant effect of infection occurred both in week 2 *pi* ($P < 0.05$) and in week 4 *pi* ($P < 0.01$). HP_{act} tended to decrease in infected animals ($P < 0.10$). No effect of growth retardation on the HP traits was found ($P > 0.10$).

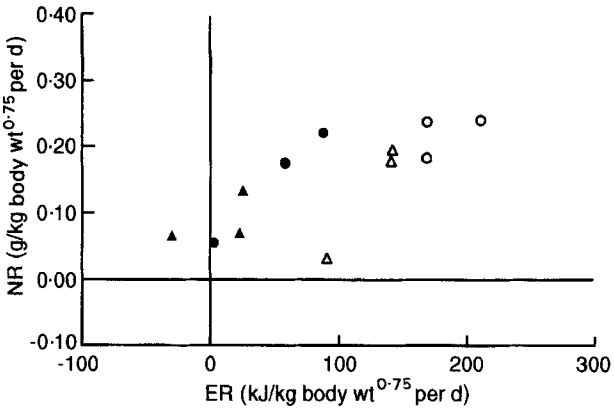

Fig. 4. The effect of *Trypanosoma vivax* infection and different growth patterns of West African Dwarf goats, on the relationship between N retention (NR; g/kg body weight$^{0·75}$ per d) and energy retention (ER; kJ/kg body weight$^{0·75}$ per d). (▲), Infected goats fed on pelleted lucerne (*Medicago sativa*) *ad libitum* (normal growth); (●), infected goats fed on 55 g pelleted lucerne/kg body weight$^{0·75}$ per d (retarded growth); (△), non-infected (control) goats, normal growth; (○), control goats, retarded growth. For details of treatments and procedures, see pp. 428–430.

Serum and hepatic metabolic variables

Plasma glucose concentration was increased by infection ($P < 0·001$) and a tendency towards higher values in group N goats was observed ($P < 0·10$). No effect of the time after infection was detected ($P > 0·10$). Mean glucose levels were 3·796 (SD 0·173), 3·683 (SD 0·132), 3·492 (SD 0·176), and 3·358 (SD 0·200) mmol/l for treatments IN, IR, CN, and CR respectively.

Compared with controls, serum insulin concentrations (Fig. 5) were lower in infected animals in week 1 *pi* ($P < 0·05$) and tended to be lower in week 2 *pi* ($P < 0·10$). In week 2 *pi* growth retardation tended to affect serum insulin ($P < 0·10$). Also a tendency towards an interaction between growth retardation and infection was observed ($P < 0·10$). After week 2 *pi* no effect of treatments was detected ($P > 0·10$).

Plasma concentrations of NEFA (Fig. 6) were increased in infected animals ($P < 0·001$) and NEFA concentration tended to be higher in IN animals than in IR animals ($P < 0·10$). Plasma NEFA concentration was positively correlated with plasma glucose (Pearson's correlation r 0·44; $P < 0·01$).

The T4 concentration was lower in infected animals. Mean values for infected and control goats were 81 (SD 16) and 149 (SD 19) mmol/l respectively ($P < 0·001$). Also T3 concentration in infected animals was lower with mean values of 1·31 (SD 0·33) and 2·19 (SD 0·48) mmol/l for infected and control goats respectively ($P < 0·001$). No effect of growth retardation was detected ($P > 0·10$).

Serum urea concentration was not affected by the week *pi*, infection or growth retardation ($P > 0·10$). The average concentration was 7·6 (SD 0·8) mmol/l.

Hepatic TAG concentration (Table 5) was higher in animals with a normal growth pattern at day 14 before infection ($P < 0·05$). At day 28 *pi*, only a tendency toward a higher TAG concentration in group N goats was observed ($P < 0·10$). Overall TAG concentration at day 28 *pi* was higher than at day −14 ($P < 0·001$). Liver TAG was positively correlated with plasma NEFA concentration (Pearson's correlation r 0·53; $P < 0·001$).

Table 4. *The effect of* Trypanosoma vivax *infection and different growth patterns of West African Dwarf goats on heat production (HP), heat production corrected for physical activity (HP_{cor}), and heat production attributed to physical activity (HP_{act}) in weeks 2 and 4 after infection*†

(Values are means and standard deviations for three observations expressed in g/kg body weight$^{0.75}$ per d)

Infection status (I)...	Infected				Control				Statistical significance of effect of‡:		
Growth pattern (G)...	Normal		Retarded		Normal		Retarded		I	G	I × G
	Mean	SD	Mean	SD	Mean	SD	Mean	SD			
Week 2											
HP	489	19	461	47	433	20	449	52	NS	NS	NS
HP_{cor}	437	10	405	49	361	16	379	53	*	NS	NS
HP_{act}	52	9	56	14	71	17	70	24	NS	NS	NS
Week 4											
HP	495	18	500	57	421	24	464	34	*	NS	NS
HP_{cor}	448	3	442	59	354	12	389	41	**	NS	NS
HP_{act}	47	17	57	3	67	15	75	23	t	NS	NS

Normal, pelleted lucerne (*Medicago sativa*) *ad libitum*; Retarded, 55 g pelleted lucerne/kg body wt$^{0.75}$ per d; t, tendency ($P < 0.10$).

* $P < 0.05$, ** $P < 0.01$.

† For details of treatments and procedures, see pp. 428–430.

‡ NS, $P > 0.10$.

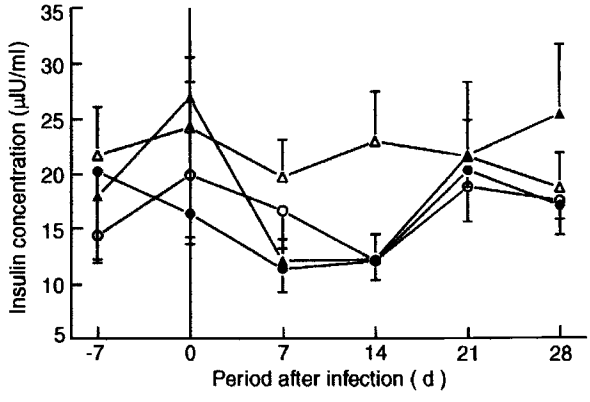

Fig. 5. The effect of *Trypanosoma vivax* infection and different growth patterns of West African Dwarf goats, on serum insulin concentration (μIU/ml). (▲), Infected goats fed on pelleted lucerne (*Medicago sativa*) *ad libitum* (normal growth); (●), infected goats fed on 55 g pelleted lucerne/kg body weight$^{0.75}$ per d (retarded growth); (△), control goats, normal growth; (○), control goats, retarded growth. Points represent mean values with their standard errors represented by vertical bars. For details of treatments and procedures, see pp. 428–430.

Pathology

Gross and microscopic examination after death revealed hyperplasia and a plasmacellular reaction of lymph nodes and hyperplasia of the spleen in all twelve infected animals. Mononuclear infiltration of kidneys (ten of twelve), brain (six of twelve) and heart (six of twelve; moreover three of four control animals) was observed. The thyroid showed active

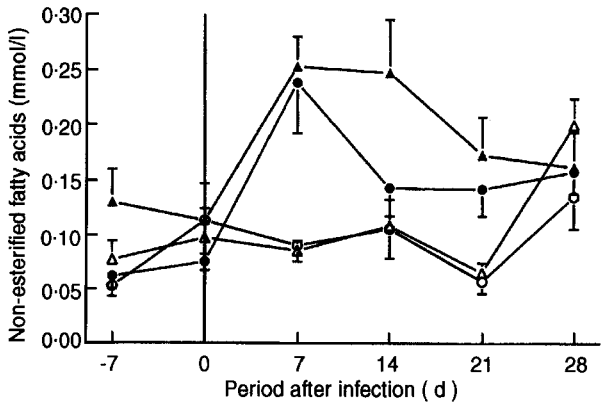

Fig. 6. The effect of *Trypanosoma vivax* infection and different growth patterns of West African Dwarf goats, on plasma non-esterified fatty acids concentration (mmol/l). (▲), Infected goats fed on pelleted lucerne (*Medicago sativa*) *ad libitum* (normal growth); (●), infected goats fed on 55 g pelleted lucerne/kg body weight$^{0.75}$ per d (retarded growth); (△), control goats, normal growth; (○), control goats, retarded growth. Points represent mean values with their standard errors represented by vertical bars. For details of treatments and procedures, see pp. 428–430.

Table 5. *The effect of* Trypanosoma vivax *infection and different growth patterns of West African Dwarf goats, on liver triacylglycerol (TAG) concentration (mg/g) before and 28 d after infection*†

Infection status (I)...	Infected				Control				Statistical significance of effect of‡:		
Growth pattern (G)...	Normal		Retarded		Normal		Retarded		I	G	I × G
	Mean	SD	Mean	SD	Mean	SD	Mean	SD			
day −14	15·9	3·1	14·0	2·3	15·0	1·5	12·6	1·1	NS	*	NS
day 28	22·6	5·8	19·3	3·2	23·3	3·7	20·2	3·6	NS	t	NS

Normal, pelleted lucerne (*Medicago sativa*) *ad libitum*; Retarded, 55 g pelleted lucerne/kg body wt$^{0.75}$ per d; t, tendency ($P < 0.10$).
* $P < 0.05$.
† For details of treatments and procedures, see pp. 428–430.
‡ NS $P > 0.10$.

epithelial cells with many follicles present, in one infected animal cuboidal cells, and in one control animal inactive epithelial cells. The liver of many infected animals showed some fat accumulation (ten of twelve). An ischaemic hepatitis was observed in one infected animal. Another infected animal showed polymicrocavitation of the cerebrum.

DISCUSSION

Effect of T. vivax *infection*

In the present study, infection affected most variables with respect to energy and N metabolism, and pathology of disease. Intake of GE and N was reduced by infection, and this led to changes in retention and in the blood metabolic profile, which are typical for

sub-optimal nutrition; however, most infected animals still showed a positive energy balance.

All infected animals developed anaemia to the same extent, irrespective of growth retardation. The PCV level after 4 weeks of infection was very low, compared with the results of Verstegen *et al.* (1991) and Akinbamijo *et al.* (1992) on West African Dwarf goats, and observations of Paling *et al.* (1991) on infected N'Dama cattle. Parasitaemia fluctuated with time, which is a normal phenomenon in trypanosome infections (Stephen, 1986). Also other pathological findings at autopsy revealed the typical picture associated with *T. vivax* infection (Van den Ingh *et al.* 1976).

Feed intake was reduced; DMI was about 16 % lower in infected animals; this is a smaller decrease than 35 % reduction in intake that we found in a previous experiment (Van Dam, 1996). Metabolizability of GE, and digestibility of N were not affected. This result corresponds with previous studies (Verstegen *et al.* 1991; Akinbamijo *et al.* 1992; Van Dam *et al.* 1996*b*) and suggests that kidneys and intestines were intact, a finding confirmed at autopsy. Moreover, no indications for a decrease of NR at a given ER level (Fig. 4) were found.

The observed reduction in intake affected the decrease in insulin. The increased plasma NEFA concentration demonstrates that this decrease in insulin has induced lipolysis (Payne, 1989). Wassink *et al.* (1993) reported a negative correlation coefficient (r −0·76) between NEFA and DMI in *T. vivax*-infected West African Dwarf goats. This is in agreement with the present study. The observation that liver TAG at day 28 *pi* was not increased by infection may imply that the lipolysis, as induced by the reduction in ER, was only mild to moderate. Post-mortem analysis, however, indicated some zonal fat accumulation in livers of infected goats. Van den Top *et al.* (1995) found increased liver TAG in peri-partum goats, probably due to negative energy balance and substantially increased NEFA supply from the blood to the liver. Feingold *et al.* (1990) described increased hepatic TAG under the influence of immunological products such as tumour necrosis factor during infection.

The increase in plasma glucose of infected animals was unexpected, given the results from previous studies (Akinbamijo *et al.* 1992; Van Dam *et al.* 1996*b*). In animals with a negative energy balance the glucose level is often decreased (Payne, 1989). However, in our study most animals showed a positive energy balance.

Serum T3 and T4 were decreased in the infected animals; this corresponds with earlier findings (Abebe & Eley, 1992; Van Dam *et al.* 1996*b*). No effect of growth retardation was detected.

HP in infected animals was increased by about 10 %. This is lower than values reported by Verstegen *et al.* (1991), who demonstrated a 16 % increase in HP in West African Dwarf goats due to *T. vivax* infection. HP due to physical activity, measured with Doppler-radar activity meters, tended to be reduced in week 4 *pi* in infected animals. Van Dam *et al.* (1996*a*) reported a reduction in standing time of West African Dwarf goats, due to *T. vivax* infection. Lying down costs less energy due to a lower muscle tone and an increased thermal insulation (Hart, 1985). We did not monitor postural behaviour in the present study.

Interaction between nutritional history and infection

The ME_m was increased by 25 % in the infected group. This can be referred to as the metabolic costs of infection. Both the absolute level of ME_m and the increase due to infection agrees with the findings of Verstegen *et al.* (1991). Baracos *et al.* (1987)

attributed the increment of ME_m to an increase in BMR due to fever, and to other metabolic costs, e.g. mounting of the immune response and increased protein turnover. However, it appeared that infection caused a smaller increase of ME_m in group R goats than in group N goats (i.e. 20 % increase in group R animals compared with 29 % in group N animals) but this was not significant.

Figure 3 shows the relationship between ER and ME. Regression analysis of this relationship can also provide an estimate for ME_m, i.e. the point of intersection with the x-axis; however, no statistical effects of treatments were observed, which is at least partly explained by the low number of observations. The same problem prevented statistical analysis of the relationship between NR and ER, although it can be derived from Fig. 4, that NR was at least not decreased at a given ER in infected animals. This implies that maintenance requirements for N probably were not increased by infection, as was also reported by Verstegen *et al.* (1991).

The ER appeared somewhat higher in IR animals than in the IN animals. This was also reflected in the higher NEFA concentration at day 14 *pi* of the IN group, compared with the IR group, indicating a higher lipolysis in IN animals (Payne, 1989).

Apart from the tendencies found for ME_m and ER, only minor (if any) carry-over effects of previous growth check on the course of infection were observed. Katunguka-Rwakishaya *et al.* (1995) reported a more severe anaemia and greater growth retardation after *T. congolense* infection in sheep on a low energy intake compared with those on a high energy intake. Reynolds & Ekwuruke (1988) observed an increased mortality of *T. vivax*-infected West African Dwarf sheep if fed at a low plane of nutrition. This corresponds with the view of Murray (1988) that shortage of nutrients negatively affects the immune response during infection.

An experiment with chickens, however, provided no evidence that nutritional stress in early life has an effect on subsequent disease resistance (Zulkifi *et al.* 1994). Kim & Lovell (1995), reported reduced resistance of 1-year-old catfish (*Ictarulus punctatus*) to infection with *Edwardsiella ictaluri*, if they had experienced a period of starvation. However, they observed the opposite for 2-year-old catfish, which showed reduced mortality if subjected to starvation before infection. The latter result is in line with observations by Murray & Murray (1979) who reported a sharp increase in mortality of mice after infection with *Listeria monocytogenes* when they were force-fed, compared with their anorectic congeners. These studies emphasize a negative relationship between intake level and disease resistance during early infection, possibly by reducing the available nutrients for the invading micro-organism, and/or by production of specific substances that slow down development of the infectious agent. This was also concluded by Isoun (1972), who found evidence that malnutrition leads to lower parasitaemias and increased survival times in rats, infected with *T. brucei*, and fed on a diet deficient in protein, thiamine or vitamin A, by comparison with controls adequately fed. In the present experiment, no such effect of growth retardation treatment on parasite counts of infected goats was observed. The shorter duration of the first peak of fever in animals with growth retardation, however, may indicate a quicker clearance from the blood of the first parasite peak (Stephen, 1986).

Effect of growth retardation on intake and energy balance

The differences in feeding regimen in the restriction period led to large differences in body weight at the time of infection, but apparently the applied feed restriction did not induce substantial lipolysis, because 2 weeks before infection liver TAG was not increased in group R animals compared with group N animals (Table 5).

Although DMI during the infection period was not significantly affected by growth retardation, body-weight gain over the infection period was higher in these group R animals than in group N animals (Table 2). According to Hogg (1992) this may be caused by compensatory mechanisms taking place in animals, just after a period of feed restriction, i.e. a higher feed intake, an increase of gut fill, together with initially lower maintenance requirements. In infected animals only a slightly higher DMI was observed in animals with retarded growth. Minor indications for a reduced ME_m due to previous growth retardation were found in infected animals (not significant) but not in control animals. It is also possible that gut fill gradually increased in group R animals in the course of the experiment, due to adaptation from restricted to *ad libitum* feed intake.

Although indications were found for some compensatory intake and body-weight gain in group R animals, these may have been only minor effects compared with the effect of trypanosome infection.

Conclusions

The course of a *T. vivax* infection in West African Dwarf goats with a retarded growth pattern generally was not different from that in West African Dwarf goats with a normal growth pattern. Feed intake and ER was reduced. Blood biochemical variables of infected goats were consistent with the reduced energy balance; blood glucose, however, was increased in infected animals. HP and ME requirements for maintenance were increased.

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