THE EFFECT OF PARASITISM ON NUTRIENT METABOLISM AND PRODUCTIVITY IN SMALL RUMINANTS

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THEOREMS

I

The relationship between digestible organic matter intake (DOMI) and nitrogen retention (NRET) in health and diseases is maintained constant by the subject irrespective of the dry matter intake (DMI).

This thesis

II

Trypanosomiasis does affect the voluntary feed intake of non-lactating trypanotolerant small ruminants.

This thesis

III

From a reproductive point of view, losses attributable to trypanosomiasis in pregnant animals is mediated via inefficient nutrient utilization.

This thesis

IV

Energy equilibrium is attainable over a wide range of dry matter intake in healthy subjects. The definition of maintenance requirements in quantitative terms needs further clarification.

This thesis

V

Increased maintenance requirement is a major route by which infected febrile hosts use nutrients less efficiently during parasitism compared with how starving healthy animals use their resources.

This thesis

VI

In pregnant dams, trypanosomiasis has deleterious effects on performance of offspring while trypanosomiasis in lactating non-dairy dams does not affect offspring performance.

This thesis

VII

For the adequate interpretation of changes in the blood concentration of ß-hydroxy butyrate in relation to the energy balance of high producing dairy cows, knowledge of the ration of cows is most desirable.

Bijlsma, J.G. and Counotte, G.H.M. (1992) Tijdschr. v. Diergeneesk ***117**:627-629*

VIII

Protecting the environment against amonia pollution by feeding high produciong dairy cows less nitrogen relative to energy intake is limited by the restricted efficiency of the nitrogen metabolism in the rumen.

Tamminga, S. (1992) J. Dairy Sci. ***75****:345-357.*

IX

An animal is not solely composed of proteins, fat, ash and water. It is formed by the functional completeness of organs which contain them, plus an abstract component - life.

Kwakkel, R.P. (1994) Multiphasic growth in the layer pullet. Effects of nutrient restriction during rearing. Ph.D. Thesis, Wageningen.

X

A change in the African livestock husbandry system without due regard to the socio-economic component would mean making another Africa. The system of husbandry as well as the diverse breeds bear the African identity.

XI

Animal Agriculture research, from a global perspective is a classical example of the irony of situation. Where it is least relevant is where it is practiced the most.

XII

The escalating global discord is a similitude of the antigenic variation in trypanosomes. It subsides locally only to re-emerge in another place.

XIII

'Not seeing' is believing and not 'seeing' is believing

Stellingen van Yemi Akinbamijo behorende bij het proefschrijft:
'The effect of parasitic diseases on nutrient metabolism and productivity in small rumoinants'.

Wageningen, 27th September 1994.

To Olajumoke, Kasope and Dolapo

PREFACE

Pain past is pleasure; experience comes by it.

C.H. Spurgeon

That this thesis is successfully written and defended is a testimony of God's faithfulness to whom I am eternally grateful. He transformed all the 'perhapses' that so *readily beset a promovendus into 'certainties'*.

It is usual to adopt the path of brevity when it comes to writing an acknowledgement by reason of space, time or both factors in common. This thesis will not be an exception as I find myself contending against both factors in fully expressing the gratitude that has so overwhelmed me to people and organizations that have been of immense benefit to me in the course of preparing this thesis.
I respectfully acknowledge the patience and understanding of my promoter Prof. dr. D. Zwart who initiated me into the world of multi-disciplinary research. He deserves and has my deep gratitude for diligently nursing this work from embryo to full term despite the many threats of miscarriage. In the face of the non-relenting financial constraints world-wide, Professor Zwart saw to the hitch-free execution of my research work in Nigeria, Ethiopia and the Netherlands. His unflinching support saw me through. I would like to convey my gratitude to Dr. Th. Wensing, my co-promoter. In spite of your busy schedule, you made my promotion a priority costing you countless commuting to Wageningen. Thanks for making room for me in my most crucial moments and for painstakingly reading all versions of the manuscript. I thank my promoter and co-promoter who took time to flawlessly translate the summarizing discussion into dutch. I sincerely appreciate all your efforts.

Worthy of distinctive recognition are the enormous contributions of Dr. ir. B.J. Tolkamp *in preparing this thesis. I can feel you on every page of the thesis and* would like to say 'Bert, THANK YOU for being significantly different!'. Your criticisms and advices have greatly improved the quality of the work presented in this thesis. I gratefully acknowledge Professors Allan Lana and Chris Igodan for their constant encouragements and guidance I enjoyed from you both.

The work reported in this thesis was conducted in different experimental stations under different managements. I am most grateful to the Animal Production Systems *section of WAU for providing all the resources required for the experiments* conducted in De Haar and De Ossekampem. I profoundly grateful to all the faculty members who provided me with a friendly and supportive climate in which I conducted my research and writing. I am also indebted to the directorate of Training and Information, the entire staff of the Humid Zone Programme and Animal Health and Reproduction Section of the International Livestock Centre for Africa, Addis Ababa, Ethiopia. I sincerely appreciate the directorate of ILCA for her commitment to the training of African Scientists for Africa. In fostering this noble goal, it is a pleasure to also acknowledge the kindness and understanding of Ms L. Padolina. Letty, your exceptional interest in the success of your trainees is marvelous. The homeliness in your office was always a great succour. The special interests of Dr Len Reynolds (my Team Leader and Supervisor) and Prof. A. Lahlou-Kassi (Head

Animal Science Division) are gratefully acknowledged. I wish to acknowledge the kindness of International Laboratory for Research in Animal Diseases (ILRAD) Nairobi, Kenya a fruitful one.

In the course of my study, I had enjoyed the financial support of the WAU through the West African Dwarf goat Project and in the Sandwich Programme. In particular, I thank the International Education Office (*Bureau Buitenland*-WAU) and the Dean's Office for Foreign Students (DOFFS). I doff my cap indeed. You kept your promises of 'studying without tears but sweat' for which I am most grateful.

While in the Netherlands, a lot of people supplied the midnight oil that I had burnt. They stood by me all the way to the successful completion of this thesis. However, brevity demands that I name just a few of these lovely people without whom it would have been impossible to finish on schedule. The unusual demonstration of love by my brethren Albert-Jan and Wilma Wonnink who denied themselves of appreciable luxury to provide me with a lap-top computer is a confirmation of divine providence. I wouldn't be on schedule without one; Ed and Margreet van Ouwerkerk who gave EVERYTHING so that I can smile with a purpose for life. Now I know the only member of the family who wears a brown (not black!) coat. Wim and Corrie de Brabander are sincerely appreciated for their love and kind gestures. Thanks Trice for giving me Gert Arnold's time at my crucial moments. I thank you all for giving me a home within your home.

The encouragements and support of my pastor Emmanuel Aladiran and brethren Ola Sobamiwa (5 minutes per day is 5 hours in sixty days!) and Kehinde & Funmi Makinde (thanks for 'looking') eased my task. I would not have pulled through but for your unflinching moral, financial and spiritual support.

At the home-front, I thank God for all the members of my immediate family. I thank you Dad for standing behind me until today (even when I remind you that I am also a 'dadje'). I consider myself absolutely lucky to have you as a father. Mum, thank you for your endless prayers. For my brother Olumuyiwa, twin sister Funmi and Idowu my kid sister, I appreciate you all. I felt your hands supporting me as I bored my way through.

It is certain that I never would have ventured this far all alone without the support of my wife Olajumoke. She bore my burden at the heavier end. It has been said that behind every successful man, there is a GOOD woman: if this be true, then I must be successful. You had painfully endured my frequent absence from home. Now I am sure that today, is you heart's desire fulfilled in me. Olajumoke, I thank you for your support.

Finally, I acknowledge with gratitude, the efforts of Mrs Joy Burrough-Boenisch for editorial assistance and Miss Celine van der Hoofd who during the last hours, beautifully but meticulously did the final editings and prepared the text for printing on her own volition.

September 1994, Wageningen. Yemi Akinbamijo

Antithesis

The investigation of the relationship between nutrition and infection is as old as humanity itself. In the quest of finding the interpretation to this age-old phenomenon, the search has been launched in both scientific and mystic dimensions.
In a memoir published in the 16th century, the following lines attest to the existing contentions.

Nature being destrous to preserve life as long as possible
Teaches what rules to follow in times of illness;
For she immediately deprives the sick of their appetite
In order that they may eat but little.
For with little, nature is content.

Luigi Cornaro (1462 - 1566) a venetian who lived healthfully for 104 years. Culled from 'The art of living long'.

The search continues ...

CONTENTS

GENERAL INTRODUCTION

GENERAL INTRODUCTION

PARASITISM AND SMALL RUMINANT PRODUCTION IN AFRICA

Small ruminant husbandry is an essential component of the farming systems in Sub-Saharan Africa (SSA). Further development of the livestock industry in this region, however, has been hampered by technical and socio-economic constraints. Nutrition, with health as a close second, followed by management practices and climate are the major determinants of animal productivity (fig. 1).

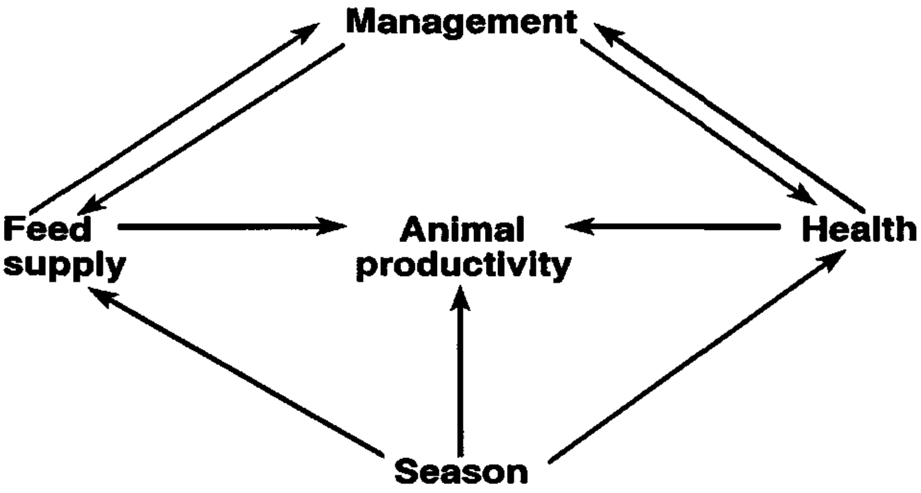

Figure 1 Interactions between factors affecting animal productivity in Sub-Saharan Africa.

SSA (Figure 2) is richly endowed with vast natural resources and livestock population. In relation to land mass, there is one small ruminant to every 10 ha of SSA. However, in terms of output per unit input of land, labour, feed, and capital, there is a great difference between the productivity of small ruminants in Africa and in developed countries. The main cause of this discrepancy is the high incidence of diseases, poor nutrition and the interaction between both factors.

Parasitic and infectious diseases play an important role in the livestock productivity of the Sub-Saharan Africa (Wilson, 1988) and the ecological conditions also support several vectors and parasites of economic significance. According to Ristic and McIntyre (1981), this explains why a considerable proportion of the African livestock persistently subsists under sub-clinical infections.

Figure 2 Sub-Saharan Africa (SSA) Eco-region (*Source: Winrock International, 1992*).

PARASITISM AND THE HOST RESPONSE

Parasitic diseases induce a chain of reactions in the host as soon as the infection is patent. The ensuing metabolic sequelae directly affects the pattern of the host nutrient metabolism pattern. These aspects of feed intake, fever, digestibilty, tissue catabolism and nitrogen retention as they affect animal productivity are reported in this thesis.

During parasitic infections, a sub-optimal performance is often observed in the host. The extent to which performance is depressed depends on the host-immune response. Feed intake influences animal productivity to an important extent and therefore in this thesis, the consequences of non-febrile anorexia in clinically normal subjects are discussed first, followed by an overview of the influence of the infection on host nutrient metabolism. The disturbance of metabolism and the degree of catabolism observed during infection is often proportional to the severity of the disease. To emphasize this relationship, the metabolic patterns during starvation and during the 'infection-starvation' complex are examined and the metabolic abnormalities in each case are related to the changes in the normal physiological pattern.

STARVATION

Energy requirements and the general host nutrient metabolism in health and diseases differ. Fever, for example, accounts for the elevated caloric requirement during infection-induced pyrexia. The pattern of utilization of endogenous energy substrates differs significantly between uncomplicated starvation in healthy and that observed in infected subjects. The most obvious changes are in glycolysis, protein and lipid metabolism (Clowes *et al.* 1976).

To appreciate the significance of the modified energy metabolism during infection, it is essential to briefly highlight the consequences of starvation in the clinically normal, non-febrile fasted state. According to Shetty (1990), during anorexia, several metabolic adaptations occur to reduce energy expenditure, maintain glucose production, stimulate the use of adipose tissue as energy source and minimize the catabolism of endogenous proteins. The major adaptive responses to energy deficit are: an increased metabolic efficiency, decreased metabolically active organ mass and reduced total body mass, ultimately resulting in a reduction in the resting metabolic rate (RMR) (Shetty, 1990; Wolkers *et al.* 1994). The adjustment is achieved by decreasing the energy required for maintenance, lowering the body temperature and reducing the body mass during long-term energy deprivation (Blaxter, 1989; Shetty, 1990; Akinbamijo *et al.* 1992). This conservatory strategy is described as the expected physiological response to caloric insufficiency (Keusch and Farthing, 1986).

In the normal fasted state, the animal switches to the utilization of endogenous reserves to meet body needs. The stored energy available in the body of the normal animal consists of a considerable quantity of fat, protein and a small amount of carbohydrate in the form of glycogen. Being limited, the glycogen reserves are rapidly depleted (in approximately 15-20 hours) and subsequently some protein are mobilized and converted to carbohydrates required by the nervous tissues and blood cells (Linder, 1985).

The adaptation to starvation involves a progressive reduction in amount of protein catabolized for carbohydrate production. The protein reserves are vital for many biological functions and prolongation of life. Therefore, their utilization is minimized by the shifting the energy molecules utilized by different tisssues from glucose to fatty acids and ketone bodies as fuel molecules utilized by different tissues during anorexia in that order. This results in the negative energy and nitrogen balances observed in feed-deprived animals.

The adipose deposits provide the bulk of the caloric requirements. Following the onset of lipolysis, fat is mobilized and utilized as indicated by an increasing availability of free fatty acids (FFA) or non-esterified fatty acids (NEFA) in the peripheral circulation (Linder, 1985). This is usually accompanied by an increase in the utilization of glycerol and ketone bodies (Beta hydroxybutyric acid and acetoactate) by the nervous tissues. The mechanism of this conversion to an economic utilization of fat reserves is dependent on the progressive reduction of insulin in the circulation (Clowes *et al.* 1976).

In effect, the mechanism by which animals overcome shortfalls in exogenous supply of nutrients can be considered as the normal physiological response to caloric insufficiency and the adaptation to fat metabolism. This results in an efficient use of body reserves as a strategy for survival.

Endogenous nutrient mobilization during dietary insufficiency is a complex, hormone-mediated process whose mechanism are not fully understood. Figure 3 gives an overview of the major biochemical events in the liver during endogenous metabolism (Wolkers, 1993). As these biochemical reactions are hormone-mediated, the endocrine system play an important role in the regulation of metabolic process within the body. Insulin, glucagon, glucocorticoid and growth hormones are the most important of these metabolism-related hormones. The levels of blood constituents such as ketone bodies, NEFAs, urea and glucose (least sensitive), reflect the level of starvation. Their concentration in the blood is the result of a finely balanced system of hormonal interactions. The mobilization of body reserves during starvation can therefore also be considered as a reflection of the balance between the anabolic and catabolic hormone-mediated processes within the body.

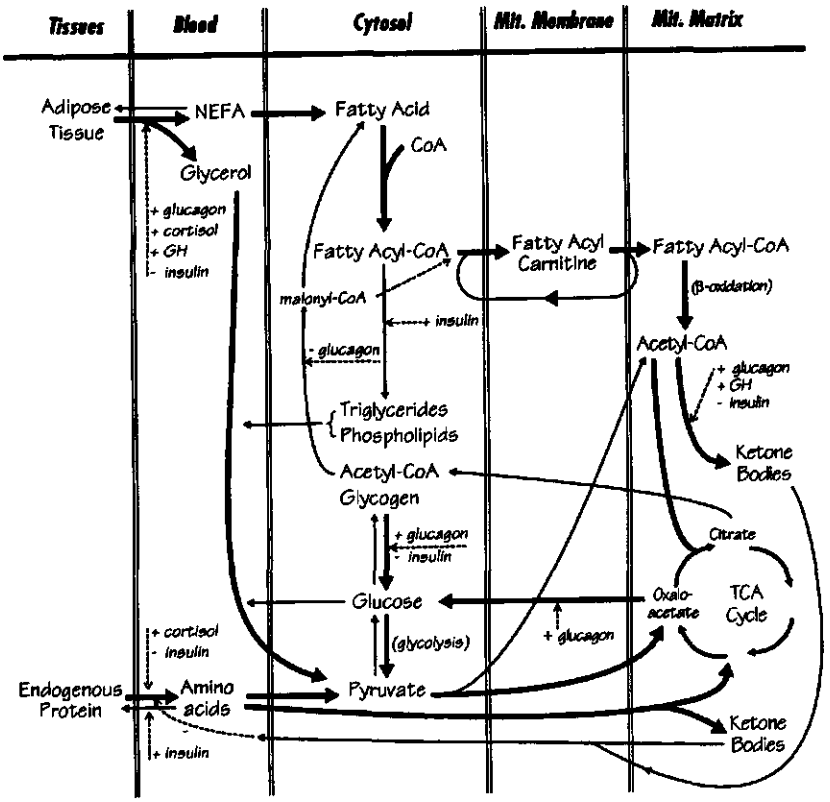

Figure 3 Major hepatic matabolic events taking place during mobilization of body reserves. Heavy arrows indicate dominating reactions. (*Source: Wolkers, 1993*).

NUTRITION AND INFECTION

Most reports in the literature on this subject agree on the fact that infection depresses voluntary feed intake, even though the factor(s) responsible for this decrease remain unclear. Parasitic infections that cause fever, like trypanosomiasis, do not only reduce intake, but also concomitantly increase nutritional requirements by increasing the basal metabolic rate of the host (Keusch and Farthing, 1986).

The mobilization and utilization of endogenous fuel substrates following infection differ from the economic pattern of fat utilization and protein conservation observed in normal fasting and starvation. Metabolic and biochemical responses are generally accelerated during infections and these eventually determine the outcome of each episode of the infection process (Beisel, 1985).

Infections are accompanied by direct or functional losses of the body content of many nutrients. The magnitude of these losses is related to the severity and duration of the infection. When the infection is cured or eliminated by the host's defensive mechanisms (or treatment), lost body nutrients are re-acquired and body reseves are reconstituted to pre-infection levels. However, if the infection continues unabated, becoming sub-clinical, chronic or progressive, the body composition definitely changes and a new relative equilibrium of body nutrient balances emerges (Beisel, 1985; Shetty, 1990; Keusch and Farthing, 1986).

Acute infection causes metabolic rate and oxygen consumption to increase (Beisel, 1985; Keusch and Farthing, 1986; Kaneko, 1989; Shetty, 1990). This initial response leads to a concomitant increase in both anabolic and catabolic processes. Although seemingly incongruous, the catabolic process is more conspicuous. Cells in hepatic and lymphoid tissues rapidly increase the rate at which they synthesize the protein needed for host defence mechanisms (Keusch, 1984). In order to support the anabolic requirements and to provide the energy needed to maintain high metabolic rates in the presence of anorexia, catabolic processes are also accelerated. As demonstrated by Wolkers *et al.* (1994), proteolysis occurs in order to satisfy the body's maintenance requirements for energy, though this does not really start before the terminal stage.

Oxidation of amino acids for energy production has long been recognized as characteristic of infections (Allden, 1970) and a decline in protein synthesis leads to weakness, reduction of immuno-competence and hypoalbuminemia in the host. The state of affairs contrasts with the economic pattern of protein conservation observed following adaptation to non-febrile normal starvation in healthy subjects in which body fat reserves are primarily mobilized to furnish the energy essential for the functioning of the vital organ systems (maintenance). The increased metabolic rate, fever and synthesis of immuno proteins require the supply of metabolic energy and the necessary bio-synthetic precursors. The biochemical adaptations evolved by the body to provide these necessary materials are unique and differ from the physiological responses to fasting in normal animals (Ryan, 1976).

Depending on the pathogenesis of the infection, protein losses through the gastro-intestinal tract may contribute to the poor apparent nitrogen digestion in parasitized animals. This phenomenon is not common in trypanosomiasis, but has been reported in fascioliasis (Dargie, 1980; Dargie *et al.* 1979). Cases of protein loss through the kidneys leading to lower nitrogen retention in infected hosts have also been reported in trypanosomiasis (Ingh *et al.* 1976).

NITROGEN BALANCE

This technique employs a quantitaitive approach to estimate the net nitrogen retained in the body. This is achieved by deducting total nitrogen voided from nitrogen ingested over a specified period of time. The nitrogen balance trial is a classical procedure that has been used to demonstrate in quantitative terms, the indirect evidence for the primary acceleration of muscle protein catabolism post-infection (Dargie *et al.* 1979). Due to the accelerated rate of protein metabolism during the acute phase of an infection, the overall picture is represented by the summation of intake and output of plasma free amino acids (Clowes *et al.* 1976). This technique attempts to estimate the differences between nutrient ingested and total combined losses per unit time. In practice, the retained nitrogen tends to increase during the incubation period of the infecting pathogen, due to the increased acceleration of anabolic processes (Clowes *et al.* 1976). However, shortly after pyrexia starts, coupled with a reduction in voluntary feed intake (VFI) and the increased metabolic rate, the nitrogen balance becomes negative (Beisel, 1985). The nitrogen loss occurs mainly through urine in the form of amino acid nitrogen, creatinine nitrogen, uric acid nitrogen and urea nitrogen which is by far the largest contributor. However, within muscle cells, some branched-chain free amino acids are de-aminated and the carbon skeletons are used as a direct source of cellular energy. When glycogenic amino acids are de-aminated to yield carbon skeletons for glucose production, the nitrogen group enters the urea cycle where it is converted to urea and excreted, mainly in urine. The daily losses of nitrogen in acute infection correlates with the severity and duration of fever (Finco, 1989; Shetty, 1990). In terms of productivity, the net effect of parasitism on nitrogen metabolism would be a reduced performance in terms of nitrogen retention, live weight gain, reproduction potential, milk yield and composition, mediated via lowered VFI. In pregnant animals, foetal growth could be retarded if maternal nutrition is restricted, and this will affect foetal size and birth weight (Gunn, 1983; Ogwu *et al.* 1986; Ikede *et al.* 1988). In the event of severe nutritional stress, abortion will occur (Osuagwuh and Akpokodje, 1986) or death of animal might result.

FEVER AND ENERGY METABOLISM

The accelerated rates of nutrient metabolism during febrile infections are achieved primarily by the oxidation of carbohydrate fuels. This is controlled by a variety of metabolic and endocrine responses that coincide with the initiation of pyrexia. A combination of hormone-mediated biochemical responses at the onset of an acute infection induces pyrexia; in particular the increased glucagon has the net result of accelerating the hepatic production of glucose and the initiation of glycogenolysis (Rayfield *et al.* 1973 cited by Beisel, 1985). The body's ability to generate a febrile response depends on the availability of energy needed to increase cellular metabolism and to slow down the mechanisms needed to reduce heat loss. The degree and duration of fever are two factors responsible for nutrient wasting during the acute infection phase. They are also responsible for the increased respiration rate (oxygen consumption) and hence the increase in metabolic rate by 13 -15% for every degree rise in temperature (Blaxter, 1989; Stephen, 1986) or for causing a 25% rise in maintenance energy requirements (Verstegen *et al.* 1991).

ANIMAL TRYPANOSOMIASIS (Nagana)

Trypanosomiasis has been described as an important factor limiting the productivity of cattle, sheep and goats in SSA (Jahnke, 1982). It is a parasitic disease transmitted by arthropods and largely restricted to areas in which the vector

- tse-tse fly (*Glossina spp.*) can survive. The most pathogenic trypanosomes for ruminants endemic in Africa are Trypanosoma vivax, Trypanosoma congolense and Trypanosoma brucei, either individually or in concert (Murray et al. 1983; Murray & Gray, 1984; Ogwu and Njoku, 1987).

Trypanosomiasis in non-tolerant domestic livestock is generally fatal unless treated. The form of infection may vary from acute with fulminating parasitemia and death within two weeks, to chronic asymptomatic disease which may persist for months or years in large ruminants (Ingh *et al.* 1976; Anosa, 1983; Jordan, 1986). Following infection, the density of parasites in the circulating blood becomes cyclical. It rises to a peak and then declines before increasing again (Kettle, 1990). The infection is often associated with reduced feed intake and sustained pyrexia (Stephen, 1970; Akinbamijo, 1988; Verstegen *et al.* 1991; Zwart *et al.* 1991), loss of protein through the kidneys in acute cases (Ingh *et al.* 1976) and anaemia (Anosa and Isoun, 1974). Loss of protein through the intestines only occurs in rare occasions of a haemorrhagic diathesis (Van Dijk *et al.* 1973; Olubayo and Mugera, 1985). The combination of these processes laeds to loss of condition, high mortality, negative energy and nitrogen balances, unthriftiness, abortion, delivery of weak offspring and reproductive disorders (Ikede and Losos, 1974; Ogwu and Nuru, 1981; Ogwu *et al.* 1986; Ikede *et al.* 1988; Oyejide and Adeyemo, 1989). The affected animals become increasingly emaciated and in the terminal stages may be recumbent or sometimes comatose.

The severity of trypanosome infection is related to the virulence of the parasite stock and relative resistance of the host, mediated through its nutrition level and genotype (Murray *et al.* 1983). Animals on a low plane of nutrition that have to work or trek long distances for grazing or water are more prone to severe infection than animals which are well-fed and relatively sedentary (Stephen, 1986). Stress as a result of poor nutrition, trekking, work (draught), feed shortage or inter-current diseases and pregnancy aggravates the effect of the infection. In cattle, pregnant animals are liable to abortion or to deliver weak offspring (Ogwu *et al.* 1986; Ogwu & Nuru 1981; Stephen, 1986). The situation as regards sheep, goats, pigs, horse, donkeys and camels is probably similar but less well documented in the literature.

Resistant breeds of livestock usually with relatively smaller body frames, include breeds of ruminants in West and Central Africa mainly the West African

Dwarf sheep (Djallonke), goats, N'Dama and the West African Shorthorn (the Muturu) cattle (ILCA, 1979). These animals have a certain degree of innate resistance to trypanosomiasis (ILCA, 1979). This phenomenon of trypanotolerance is the ability of an animal to contain to a great extent the effect of a trypanosomiasis infection and continue growing and reproducing without therapy.

FASCIOLIASIS (Liver fluke disease)

Fascioliasis is the most common form of liver infection, having a cosmopolitan distribution (Boray, 1969) and pathogenic in all classes of livestock (Haroun and Hillyer, 1986). Liver fluke disease is caused by the members of the genera *Fasciola* and *Fascioloides* (Blood and Radostis, 1989). Like trypanosomiasis, the distribution of fascioliasis largely depends on the availability and conditions conducive for the sustenance of the intermediate host of the parasite (Lymnae truncatula).

The host becomes infected by ingesting the infective stages of Fasciola hepatica. The infection load depends on the number of infective metacercariae ingested, species of infecting parasite, age, nutritional and immunological status of the host (Ploeger, 1989). After viable metacercariae have been ingested, the parasites migrate from the gut through the peritoneal cavity and within four to five weeks post-infection reach the liver (El Harith, 1977; Schillhorn van Veen, 1980).

The presence of the juvenile flukes causes hepatic fibrosis. When these flukes enter the bile ducts, calcification and thickening of the bile duct walls commences (Kendall and Parfitt, 1975). During the migratory phase of the parasites, hepatic necrosis and hemorrhage occur, with persistent erosion of the biliary mucosa when the flukes reach the bile ducts. The ensuing hepatic damage can seriously impair host nutrient metabolism (Sykes et al. 1980). In this chronic phase, the flukes become stationed in the bile duct, from where they proceed to shed non-embryonated eggs about 10-12 weeks post-ingestion (Sewell, 1966; Dow *et al.* 1968; Reid *et al.* 1970).

Fascioliasis generally causes a sub-clinical wasting disease typified by anaemia, impaired body condition and hypoalbuminemia (Kaufmann and Pfister, 1990). Other effects attributable to fascioliasis include depressed feed intake, impaired efficiency of utilization of digestible organic matter (Dargie *et al.* 1979, Dargie, 1980, Hawkins and Morris, 1978), destruction of host tissues and elevated enteric loss of plasma protein (Tekelye *et al.* 1992a, b &c).

The derangements in nutrient utilization and the accompanying wasting reportedly affect the production of viable offspring and animal products and may ultimately result in death of the host. (Berry and Dargie, 1976; Hawkins and Morris, 1978; Sykes *et al.* 1980; Dargie, 1980). In the chronic condition, the host may exhibit a slightly impaired body condition resulting in depressed productivity (Holmes, 1986), low milk production (Black and Froyd, 1972; Rond *et al.* 1990), poor growth (Chick *et al.* 1980; Hope Cawdry *et al.* 1977, Abdallah 1989, Parent and Samb, 1984) and reduced fertility (Oakley *et al.* 1979), or may be altogether asymptomatic.

Reports in the literature indicate that in the African highland eco-region, fascioliasis is a major cause of mortality and morbidity in sheep (Tekelye *et al.* 1992a &c). However, the Ethiopian Menz sheep breed has demonstrated high survival rates during fascioliasis where other breeds have succumbed (Njau *et al.* 1989; ILCA, 1992). Although substantial information exists on the life cycle and pathology, not much is known about the interaction of fascioliasis with feed intake and nitrogen retention and productivity of small ruminant animals in SSA.

AIM AND SCOPE OF THE STUDY

This thesis aims at elucidating the role that parasitic diseases play in food intake, nutrient metabolism and productivity in growing, adult, pregnant, and lactating West African Dwarf (WAD) sheep and goats. The effect of trypanosomiasis at different phases of gestation on dam live weight changes, lamb birth weight, lamb performance, milk yield and milk composition following post-partum infection and lamb weaning weight is also investigated. Secondly, the effect of a low to medium level fascioliasis on feed intake and digestibility, nitrogen retention, changes in live weight and packed cell volume (PCV) is studied in open and pregnant Menz ewes. Lamb birth weight in response to artificial challenge by *F. hepatica* was also investigated.

The work reported in this thesis is based on the hypothesis that parasitism would affect the voluntary feed intake and nitrogen balance of the host. Once anorexia is established in the infected animal, fever (in trypanosomiasis), increased host tissue catabolism and high urinary nitrogen losses would set in, typified by increased serum concentration of products of endogenous catabolism. Parasitic infection is potentially capable of inducing a decline in energy and protein balance, leading to the impairment or cessation of productivity. The processes are reported in relation to small ruminant productivity during trypanosomiasis and fascioliasis. The thesis also examines the relationship between VFI and rectal temperature (RT).

GENERAL OUTLINE OF THE STUDY

A preliminary study in which growing goats were used to investigate the consequences of trypanosomiasis on feed intake, digestibility, live weight gain and nitrogen retention is reported (Chapter 1). The confounding effects of infection and feed intake on host metabolism are presented in chapter 2 by simulating the VFI of infected animals in healthy goats. Findings note on the relationship between feed intake and rectal temperature is presented in the third chapter. WAD sheep were used to study the effect of trypanosomiasis on nitrogen retention and productivity in pregnant and lactating animals (Chapters 4 and 5).

Finally, to complement the data collected in the studies on trypanosomiasis, a parallel study was conducted using *F. hepatica* in open and pregnant Menz sheep. This study (Chapter 6) reports the effects of sub-clinical fascioliasis on feed intake, digestibility, nitrogen retention and lamb birth weight.

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Chapter 1

EFFECT OF *TRYPANOSOMA BRUCEI* INFECTION ON LIVE WEIGHT, ORGANIC MATTER INTAKE, DIGESTIBILITY AND N-BALANCE IN WEST AFRICAN DWARF GOATS

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ABSTRACT

In two experiments, half of a group of West African Dwarf (WAD) goats fed pelleted alfalfa *ad libitum* was infected with *Trypanosoma brucei* and live weights were measured for 8 and 11 weeks post infection, respectively. Infected animals lost 30 (s.e. 6) and 18 (s.e. 5) $g.day^{-1}$ while control animals gained 41 (s.e. 3) and 63 (s.e. 2) $g.day^{-1}$, respectively, the differences between groups being significant. In the second experiment, animals were housed in metabolism cages during part of the experimental period and organic matter (OM) intake, N-balance and digestibility of OM and crude protein (CP) were determined in three seven-day periods of one week pre-infection, one and ten weeks post-infection. Rectal temperatures were recorded daily and blood samples for analyses of PCV and buffy coat examination for parasite count were taken twice weekly. All infected animals showed fever and parasitemia and one infected animal died of encephalitis in week 9 post infection. During the first week pre-infection, there were no differences in OM intake, OM digestibility, CP digestibility or N-balance between the control group and the group to be infected. At one week post infection, the infected animals showed significantly lower (-18%) OM intake and lower (-77%) N-balance than the controls but no differences in OM or CP digestibility. Infected animals showed a lower N-balance compared to controls at equal OM intake. During the third balance trial, infected animals showed lower (-63%) OM intake compared to controls and infected animals had a negative but controls a positive N-balance. The effects of infection on OM intake and N-balance were highly variable but OM and CP digestibility were not affected by infection during the third trial. It is concluded that the effect of *T. brucei* infection on live weight in WAD goats is not due to a decreased digestive capacity but mainly the result of a decrease in voluntary feed intake, probably aggravated by an increased N-loss in the urine compared to controls at equal intakes.

Key words: Trypanosomiasis, dwarf goats, feed intake, digestibility, Nitrogen Balance, Trypanosoma brucei.

INTRODUCTION

Trypanosomiasis has a profound influence on livestock productivity of which live weight gain is an important parameter. The underlying patho-physiological mechanisms causing a lowered productivity are, however, poorly understood.

A decrease in organic matter intake (OMI) could be one factor. Changes in organic matter digestibility (OMD) and crude protein digestibility (CPD) or the efficiency of utilization of digestible organic matter (DOM) or digestible crude protein (DCP) for maintenance and gain are other factors which could possibly contribute to the lowered productivity. These changes have been studied during lungworm infections (Verstegen et al., 1990). Ilemobade et al. (1981) studied the effect of trypanosomes in pigs on feed intake and feed conversion efficiency, but very little information regarding these aspects is available on trypanosomal infections in ruminants.

West African Dwarf (WAD) goats are considered to be trypanotolerant (ILCA, 1986), i.e. animals suffer from the infection but morbidity and mortality is reduced. The aim of the present investigation was to study the effect of *T. brucei* infection on live weight development, OMI, OMD, CPD and N-balance in growing castrated WAD goats.

MATERIALS AND METHODS

Experiment 1

Ten castrated WAD goats without previous exposure to trypanosomiasis (initial age between 8 and 9 months) were selected at random from the Wageningen University weaners flock. After pairing the animals on weight basis, the two individuals in each pair were assigned at random to the infection or the control group.

Animals were housed in group pens with straw bedding and received pelleted alfalfa (19.8 % CP), salt lick and fresh water *ad libitum*. A stock of *T. brucei* isolated by the East African Trypanosomiasis Research organization (EATRO) in Uganda and designated as *T. brucei* 1066, was used to prepare stabilates in mice. After an adaptation period of 3 weeks to the feed, goats in the infection group were infected i.v. with approximately 10 million of these parasites per animal.

After recording the live weights weekly for 8 weeks post infection, the infected animals were all treated with 7 mg.kg^{-1} diminazine aceturate (®Berenil).

Experiment 2

Eighteen castrated WAD goats without previous exposure to trypanosomiasis (initial age 8 to 9 months) were selected at random from the Wageningen University weaners flock. After pairing the animals on weight basis, the two individuals in each pair were assigned at random to the infection or the control group.

Animals were housed in group pens with straw bedding, except for two periods of 35 and 17 days, respectively, when three digestibility and nitrogen balance experiments took place and animals were housed in metabolism cages suitable for the separate collection of faeces and urine (see Table I).

Table 1: Experimental time schedule

Experimental day	Activity
-39 to -2	Adaptation to feed in group pens
-24 to -12	Adaptation to cages
-11 to - 1	Balance trial 1
0	Inoculation of infection group
3 to 10	Balance trial 2
11 to 63	Housing in group pens
64 to 73	Adaptation to cages
74 to 80	Balance trial 3

Animals received pelleted alfalfa (19.8 % CP), fresh water and salt lick *ad libitum* during the experimental period. Goats in the infection group were inoculated as described for Exp. 1 on experimental day 0.

Live weights were recorded weekly and rectal temperatures twice weekly from day -11 up to day 80 relative to inoculation.

Blood sampling (5 ml in bottles with 0.048 ml 15 % sol EDTA K3) from the jugular vein of all animals started at experimental day 3 and was continued daily during balance trial 2 and twice weekly (mondays and wednesdays) thereafter up to day 80. During the last 17 days blood was only sampled once a week.

Buffy coat (bc) examinations, to measure parasitemia, and packed cell volume (PCV) were conducted according to Murray *et al.* (1983).

During each 7 day balance trial, the daily amounts of feed offered, feed orts, faeces and urine voided (collected in HCl) were weighed. Representative samples of feed offered, orts and faeces were analyzed for contents of dry matter (DM), ash and N, and samples of urine were analyzed for N-content, all according to A.O.A.C (1975) procedures.

Statistical analyses

Analyses of variance procedures were used to estimate effects of digestible organic matter intake (DOMI) and infection on N-balance. Daily weight gains were estimated from linear regression of live weights on experimental day number. Differences in digestibility and live weight between infected and control groups were analyzed with the Student t-test. All statistical programmes used were available in SAS (1985).

RESULTS

Experiment 1

Group mean live weights with s.e.'s are plotted against time in Figure 1. Average daily gain of control animals from day 0 to day 56 was 41 (s.e. 3) g. Gain of infected animals during the first 4 weeks post-infection(16, s.e. 20, g.day^{-1}) did not differ significantly from 0. Infected animals lost weight fast during the last 4 weeks of this experiment: -91.9 (s.e. 14.8) g.day^{-1}. Due to the difference between groups in initial mean weight and the considerable within group variation, mean live weights differed ($P<0.05$) between treatments during the last 2 weeks only.

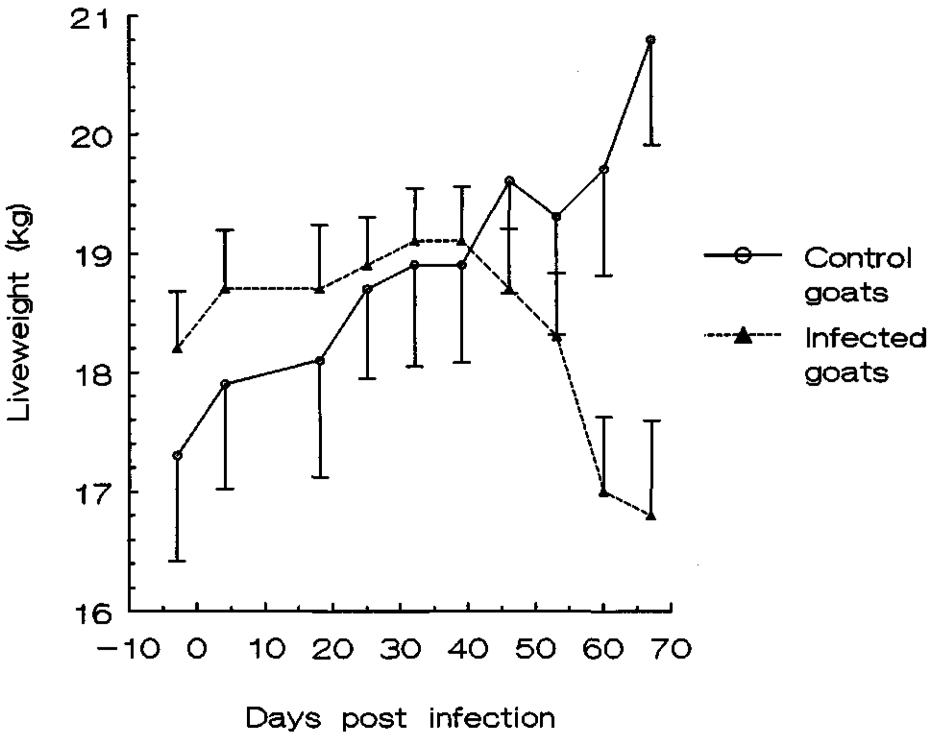

Figure 1 Mean live weight (±s.e.'s) of control and infected group in Exp. 1

Experiment 2

General course of infection

All animals became positive in the bc within one week of infection. This trend was maintained until 23 days post-infection, after which only one or two parasites (and in some cases none) were seen in the bc until the end of the experiment. There were three exceptional cases where the bc was positive even at day 76 post-infection.

PCV dropped from an average of 40 % pre-infection to between 23 and 26 % two weeks after the infection and remained at this level for the rest of the experiment.

All animals developed a meningo-encephalitis, starting approximately 9 weeks after the infection, with mild clinical symptoms in 8 animals. One animal died of a meningo-encephalitis in week 10 post-infection.

Live weights

Group average live weights for infection (n=8, one animal died) and control group (n=9) are plotted against time from experimental day -11 up to day 80 in Figure 2. In the pre-infection period, daily gain averaged 29 and 30 g.d^{-1} for the treatment and the control group, respectively, a non significant difference. Post infection differences in gain were significant between groups ($P<0.05$) after inoculation: 63 (s.e. 2) and -18 (s.e. 5) g.d^{-1} for control and infected group, respectively. During the first 7 weeks p.i., control and infected animals gained 101 (s.e. 5) and 28 (s.e. 6) g.day^{-1} and during the last 4 weeks p.i. control animals gained 39 (s.e. 4) but infected animals lost 59 (s.e. 8) g.day^{-1}, respectively.

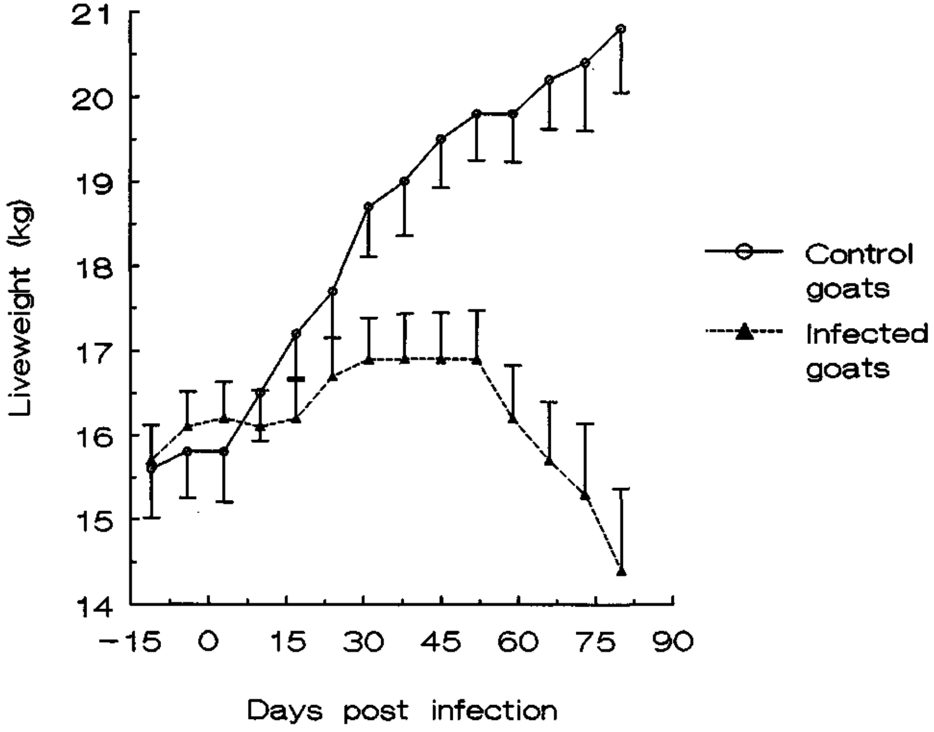

Figure 2 Mean live weight (±s.e.'s) of control and infected group in Experiment 2.

Balance trials

Table 2 shows the results of the three balance trials. During trial 1 (7 days before inoculation), the differences in live weight, OMI, OMD, CPD, digestible OMI (DOMI) and N-balance between infection and control group were not significant. The data in the table show that there was a considerable variation within groups in DOMI as well as N-retention.

Table 2: LW (kg), OMI, DOMI, CPI, DCPI, N-balance (all in g.kg$^{0.75}$.day^{-1}), OMD and CPD (%) for control and infection group during three balance trials. (s.e.'s between brackets).

Trial	1		2		3	
Day	-7		± 7		± 77	
Group	control	infected	control	infected	control	infected
LW	15.7 (±1.6)	15.9 (±1.7)	16.1 (±1.8)	16.1 (±1.7)	20.7^a (±2.3)	14.5^b (±2.4)
OMI	55.3 (±19.5)	59.6 (±21.4)	62.8^a (±10.5)	51.4^b (±9.4)	59.5^a (±5.6)	26.6^b (±23.8)
OMD	54.9 (±4.2)	55.0 (±2.7)	56.8 (±1.7)	56.0 (±2.5)	58.9 (±2.3)	59.8 (±2.4)
DOMI	30.8 (±11.8)	32.7 (±11.7)	35.6^a (±5.4)	28.8^b (±5.3)	35.0^a (±3.2)	14.9 (±15.6)
CPD	63.5 (±4.3)	63.2 (±2.0)	64.5 (±2.0)	65.2 (±2.0)	61.3 (±3.8)	62.4 (±3.1)
N-bal.	0.13 (±0.33)	0.19 (±0.28)	0.31^a (±0.11)	0.07^b (±0.13)	0.16^a (±0.13)	-0.27^b (±0.40)

^{a,b} groups with a different superscript within trials differ significantly ($P<0.05$)

Figure 3 shows the correlation between N-balance and DOMI in trial 1. As expected, the regression of N-balance on DOMI was not affected by group and a single regression line was calculated. The regression equation suggests a zero N-balance for a DOMI of 25.6 g.Kg$^{0.75}$.day^{-1}.

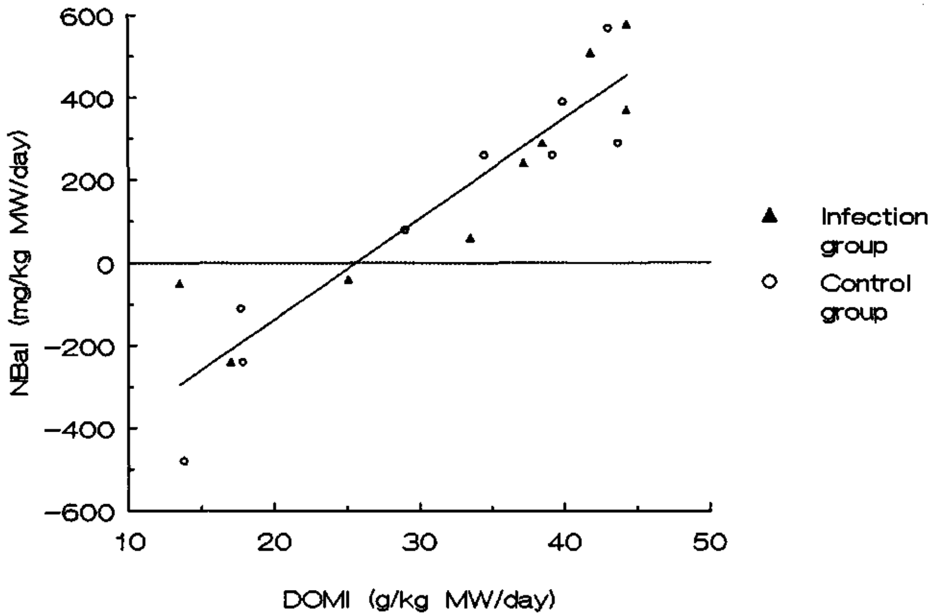

Figure 3 Relation between N-balance and digestible organic matter intake (DOMI) during balance trial 1, N-balance=-624 (s.e 81)+24.3(s.e. 2.5) ∗ DOMI. RSD=116, r^2=0.86.

During balance trial 2 (from 3 to 10 days after inoculation) live weights, CPD and OMD did not differ significantly between control and treatment group. The infected group consumed less OM and DOM ($P<0.05$) and retained less N ($P<0.05$) relative to metabolic size than the control group in this trial. Variation in DOMI and N-balance is again considerable. Group affected the intercept but not the regression-coefficient in the regression of N-balance on DOMI (Figure 4): infected animals showed a significantly ($P<0.05$) lower N-balance at comparable DOMI.

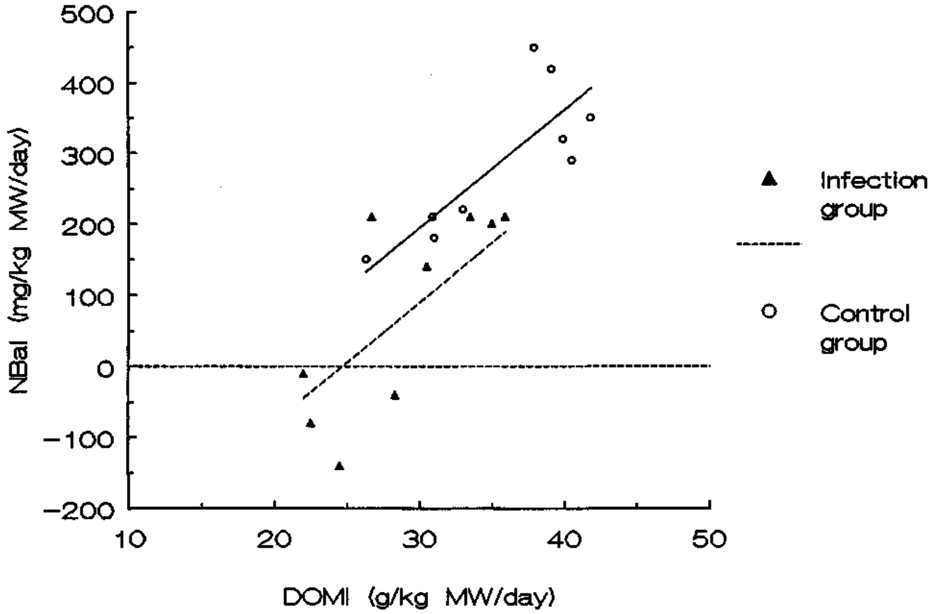

Figure 4 Relation between N-balance and digestble organic matter intake (DOMI) during balance trial 2 i.e. one week post infection.

The intercept differed ($P<0.05$) between control (-308, s.e. 144) and infected (-413, s.e. 118) groups, the regression coefficient (16.7, s.e. 4.0) did not (rsd = 85, $r^2=0.76$).

The differences in OMI, DOMI and N-balance between control and infected group were more pronounced in the third balance trial. Relative to metabolic size, infected animals consumed 63 % less OM than control animals while they showed a negative N-balance instead of a positive one. Again, differences in OMD and CPD were not significant. Due to the high variation in the data, regression analyses of N-balance on DOMI for trial 3 did not result in models suitable for interpretation. In this period, the infection group averages are based on 8 animals only (one animal died).

DISCUSSION

Although WAD goats are classified as trypanotolerant, the experiments show that the effect of inoculation with the strain of *T. brucei* used in this experiment on daily gain is considerable. In both experiments, daily gain decreased to very low levels in infected compared to control animals for the first 4 to 7 weeks after infection while considerable weight loss occurred at later stages after infection.

In the second experiment, the first balance trial (i.e. pre-infection) shows the two groups to be similar with respect to live weight, feed intake, digestive capacity and N-balance, as expected. The digestive capacity of infected compared to control animals was also similar in the last two balance trials, although feed intake was significantly lower in the infected compared to control animals. The absence of an effect of trypanosomes on digestive capacity, despite a decrease in feed intake, has also been recorded by French and Hornby (1934, 1935; cited by Reynolds and Ekwuruke, 1988) in sheep and cattle on roughage diets and infected with *T. congolense* or *T. brucei*. To our knowledge, no other reports with regard to the effect of trypanosomes on digestive capacity are available.

The relation between DOMI and N-balance relative to metabolic size in trial 1 is comparable to earlier observations with West African Dwarf Goats on pelleted feeds (Tolkamp, unpublished results) and to relations found with sheep on roughage diets (e.g. Grenet and Demarquilly, 1977). The line predicts a zero N-balance for a DOMI of 25.6 g.$W^{0.75}$.d-1, which is approximately the estimated maintenance energy requirements for WAD goats (Zemmelink et al., 1985).

From the haematological data and rectal temperatures recorded, it is clear that all infected animals developed parasitemia during the second balance trial. The most prominent effect of infection was on feed intake: infected animals consumed on average 18 % less DOM relative to metabolic size than control animals. Figures 3 and 4 show that also in non-infected animals a lower DOMI is associated with a lower N-balance. The relatively low N-balance of the infected group in trial 2 is therefore probably largely a result of the relatively low DOMI. In this trial, the decrease in DOMI in the infected relative to the control group does not, however, explain all of the decrease in N-balance: treatment significantly affected the intercept but not the regression coefficient of the regression line. This suggests that infection has a negative effect on the utilization of apparently digested protein even if energy (DOM) intake is comparable to control animals. Also it is not unlikely that the energy balance in infected animals will be lower at comparable DOMI, for example because of the energy cost of fever (Baracos et al., 1987), but no data are available in this experiment to verify the expectation. Also, the data should be interpreted with some care. In the first place it should be remembered that all animals were fed *ad libitum* in this experiment. Results might well have been different if also intake in control animals had been limited to levels comparable to the infected group. Another reason for careful interpretation of the data is the fact that N-balance in the control group is unexpectedly high in the second trial compared to earlier experiments and to the first and the third trial in this experiment: e.g. despite similar DOMI, N-balance in the second trial is almost twice as high as in the third trial. The cause of this variation remains unclear, although the difference in daily gain of the control group between the weeks immediately after day 0 and the last 7 weeks of the experiment gives the difference in N-balance some credibility.

The data in this experiment show that one important factor causing the negative effect of infection with trypanosomes on productivity is a decrease in feed intake. The data further suggest that the effect of trypanosomes on productivity may well be extra serious as a result of a stronger decrease in N-balance than could be expected from the decreased feed intake alone. More research is required to elucidate the effect of *Trypanosoma brucei* on the performance of ruminants through the effects on N- and energy-balance.

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Chapter 2

THE EFFECT OF *TRYPANOSOMA VIVAX* INFECTION IN WEST AFRICAN DWARF GOATS ON ENERGY AND NITROGEN METABOLISM

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ABSTRACT

To investigate how *Trypanosoma vivax* affects metabolism in dwarf goats, nine wethers (infection group) given alfalfa pellets *ad libitum* were infected intravenously and food intake was recorded up to 49 days after infection in the infection group and in the control group (n=9). Controls received the same diet, *ad libitum* before infection and in restricted amounts after infection in order to obtain similar intakes in both groups. Digestible organic matter intake (DOMI) and nitrogen balance (NB) were determined during four balance trials. All animals were bled regularly to measure parasitaemia, packed cell volume (PCV) and a number of serum metabolites. All infected animals showed symptoms typical for *Trypanosoma vivax* infection as judged by parasitaemia, PCV and rectal temperature (RT). Infection had a non-uniform negative effect on food intake. Compared with controls at equal DOMI, NB was lower in infected animals, the difference being significant 4 weeks after infection. This was caused by a gradual increase in NB at equal DOMI of the control group. The NB of the *ad libitum* fed infected animals 2 and 4 weeks after infection was comparable to values normally found in healthy *ad libitum* fed dwarf goats with an equal DOMI. NEFA values in serum were significantly elevated after infection. Except for two infected animals with an extremely low food intake towards the end of the experiment, no rise in serum ketone bodies was evident. After infection, serum protein increased, differences with controls being significant 4 and 7 weeks after infection. It is concluded that *T. vivax* infection results in a decrease in energy intake and a decrease in NB up to at least 4 weeks after infection. At equal DOMI, NB of infected animals was not lower than expected for *ad libitum* fed healthy animals but was lower than in healthy controls on a restricted diet, probably as a result of a decrease in maintenance requirements of the latter. The data on NB and serum NEFA concentrations suggest that non-protein energy sources are used to supply the increased energy demand as a result of infection.

INTRODUCTION

It is well documented that trypanosomiasis, mainly an infection with *Trypanosoma vivax*, T. congolense and/or *T. brucei*, affects the productivity of ruminants in the tropics (ILCA, 1986; Jahnke *et al.* 1988; Murray and Dexter 1988).

On the basis of results recently obtained from energy balance studies in dwarf goats with trypanosomiasis, it can be concluded that the reduced productivity at least partly originates from the reduced food intake and the concomittant decrease in nitrogen and energy balance during infection (Akinbamijo, 1988; Verstegen *et al.* 1991; Zwart *et al.* 1991).

In addition, it is well established that there is a great variation in food intake after infection with *Trypanosoma vivax*: some animals may keep their intake at a normal level, while others may decrease their intake considerably (Zwart *et al.* 1991).

The investigations described in this paper aim to elucidate how *Trypanosoma vivax* infection affects nitrogen and energy metabolism and to differentiate between

a) the effects of the reduced food intake after infection and
b) the effects of the infection itself on nitrogen and energy metabolism.

MATERIALS AND METHODS

Animals, feed and housing

Eighteen mature castrated male goats were used from a flock of West African Dwarf Goats with a minimal disease history established about 12 years earlier at the Agricultural University in Wageningen (Montsma, 1986). Animals ranged from 30 to 45 months of age and weighed 20 to 40 kg (28.5±5.0 (sd) kg). Before the experiment, pairs of goats were matched for weight and age. One animal from each of the 9 pairs, was allotted at random to the infection (I) or the control (C) group. Twenty-five days before the day of infection, the animals were placed in individual balance cages in a stable with a light regime set at 12 hours of light (7.00 am - 7.00 pm). Before infection, all animals were given alfalfa pellets (2.9% nitrogen in the dry matter) *ad libitum* (i.e. feed residues were approximately 20% of feed offered) once a day at 0800 h for of 25 days. During the post-infection period (50 days), each animal of the control group was assigned to a separate feeding level, ranging from 40% to 140% of its estimated maintenance requirement for energy (26 g of digestible organic matter per $kg^{0.75}$; Zemmelink *et al.* 1985). Infected animals continued to receive alfalfa pellets, water and salt lick *ad libitum* during the experimental period.

Infection

A stabilate of *T. vivax* Y 486, isolated in Yakawada, Nigeria (Leeflang *et al.* 1976) stored in liquid nitrogen, was used to infect five mice. Fifteen days later, nine goats were infected intravenously with 3 x 10^7 parasites derived from these mice. The number of *T. vivax* in mice and goats were counted as described by *et al.* (1976a). Control animals remained uninfected and none of the animals had been exposed to trypanosomal infections.

Measurements

Animals were weighed once a week and individual food intake (FI) and rectal temperature (RT, measured between 9.00 and 9.30 h) were recorded daily. During the experiment, four balance trials were carried out, each lasting seven days (see Table 1). During each balance trial, a representative sample was taken from the offered food and analysed for dry matter (DM), ash, and nitrogen (N) content. For each animal a composite sample of the bulked weekly orts was taken for every balance trial and analysed for dry matter only. The ash and N content of ort dry matter was assumed to be equal to food dry matter offered. During each balance trial, individual faeces were collected twice daily and the weight recorded. The seven-day collection was bulked at the end of the trial after which a composite sample was taken per animal and analysed for DM, ash and N content. From these data the organic matter intake (OMI), the apparent organic matter digestibility (OMD) and the digestible organic matter intake (DOMI) were calculated. During each balance trial, the urine voided by each animal was collected in a plastic bottle containing about 70 ml hydrochloric acid (HCl 50%) and the weight was recorded daily. Ten per cent (w/w) of a 24-hour urine collection was preserved (daily sample) at 4°C. At the end of each seven day data collection period, all daily samples were bulked per animal and a composite sample (about 80ml) was stored frozen at -20°C and later analysed for N content. The apparent N digestibility (ND) and the N balance (NB) were calculated for each seven day collection period for each animal from N in feed, faeces and urine.

Table 1. Experimental design, indicating the days when food intake (FI), live weight (LW) and rectal temperature (RT) were recorded and showing balance periods and blood and urine sampling days

day	-28	-21	-14	-7	0	7	14	21	28	35	42	49
FI	+++++++	+++++++	+++++++	+++++++	+++++++	+++++++	+++++++	+++++++	+++++++	+++++++	+++++++	+++++++
LW	+	+	+	+	+	+	+	+	+	+	+	+
RT				+++++++	+++++++	+++++++	+++++++	+++++++	+++++++	+++++++	+++++++	+++++++
BP[1]				+++++++		+++++++			+++++++		+++++++	
B&U[2]		+ +	+ +	+ +	+ +	+ +	+ +	+ +	+ +	+ +	+ +	+ +

[1] Balance Periods

[2] Blood and Urine Sampling

Twice weekly, an aliquot (10 ml per animal) of a 24 hour urine sample was analysed for creatinine (Cr), protein (Pr) and urea (Ur). Procedures used for these analyses were as described by Wensing *et al.* (1989). Each animal was bled twice weekly from a jugular vein for blood analyses. Blood (2x5 ml) was collected into tubes containing heparin and into tubes containing heparin and paraoxon as anti-coagulant and anti-lipase factors, respectively. A third tube containing EDTA was filled to estimate the parasitemia (Ingh *et al.* 1976a; Woo, 1970) and to determine the packed cell volume (PCV). The serum was analysed according to Wensing *et al.* (1989) for beta-hydroxy butyric acid (BHBA, mmol/l), non esterified fatty acids (NEFA, mmol/l), total protein (TP, g/l) and creatinine (Cr, mmol/l). After the fourth balance trial, all experimental animals were slaughtered for post mortem examination.

Statistical analyses

The SAS statistical package was used in the regression analysis and general linear models (SAS,. 1985). For differences between treatment groups within periods, the Student's t-test was used for subsequent analysis. Regression analyses (e.g. NB on DOMI) were performed using the following model:

$$Y_{ij} = \mu + a_i + b_i * X_{ij} + e_{ij}$$

(i = group or period; j = animal)

When no significant differences were detected using this model including interaction, the interaction was left out, resulting in the following model:

$$Y_{ij} = \mu + a_i + b * X_{ij} + e_{ij}$$

RESULTS

General course of the infection

Trypanosomes were first detected in the buffy coat of the infected animals on day 6. The number of trypanosomes reached a maximum on day 7 (7.05 ± 0.06 SEM log/ml blood) and showed a steady decline to 5.65 ± 0.26 on day 24. After day 31 the parasites could not be counted, but the buffy coat remained positive during the entire experimental period in all infected goats.

In the pre-infection period, the PCV ranged from 35% to 40%. In the infected group, the PCV dropped after day 6 to stabilise at 21% from day 35 onwards, whereas the PCV in the control animals rose slightly. The rectal temperature remained significantly elevated ($P < 0.05$) in the infected animals during the whole parasitaemic period. The rectal temperatures of the control animals decreased, apart from a peak between day 30 and 35 (Fig. 1). No protein was detected in the urine.

At post-mortem examination, the infected goats showed the typical reaction of chronic trypanosomiasis (i.e. hyperplasia of lymph nodes and spleen and non-specific reactive hepatitis with varying degrees of centrolobular degeneration). The kidneys were normal. No abnormalities were found in the uninfected goats.

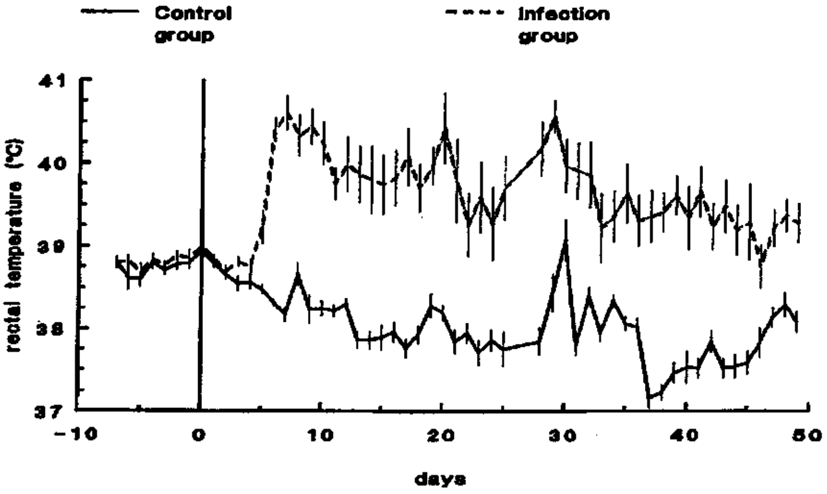

Figure 1 Group mean rectal temperature (^{0}C and s.e.) for control and infection group. Animals were infected on day 0.

Food intake and digestibility

Differences in food intake between groups were not significant before infection. After infection, the mean voluntary intake of the infected animals decreased considerably. The mean intake as well as the range in intake in the food-restricted control group was comparable to that of the infection group, as intended. During the balance periods, OMI did not differ significantly between groups (Table 2). OMD was not significantly affected by infection in any trial (mean value 58.6%, s.e. 0.4). Mean DOMI was lower in the control group in all periods but differences were not significant due to a large intra-group variation (Table 2, see also Fig. 3).

Table 2: Mean live weight (W, kg), organic matter intake (OMI, g/kg$^{0.75}$/d), digestible organic matter intake (DOMI, g/kg$^{0.75}$/d), N-intake (NI, g/kg$^{0.75}$/d), faecal N (FN, g/kg$^{0.75}$/d), urinary N (UN, g/kg$^{0.75}$/d) and N-balance (NB, g/kg$^{0.75}$/d) of dwarf goats in the infection (I, n=9) and control (C, n=9) groups during balance periods 1 (pre-infection) and 2 to 4 (post-infection)

Period	1			2			3			4		
Group	C	I	se	C	I	se	C	I	se	C	I	se
W	28.4	28.8	1.7	27.6	29.2	1.6	27.1	28.1	1.4	27.1	27.5	1.3
OMI	66.1	78.5	4.3	33.9	39.8	4.0	34.1	41.9	4.6	34.1	44.6	6.1
DOMI	38.2	44.8	2.5	20.0	23.3	2.2	19.8	24.0	2.7	20.6	26.6	3.6
OMD	57.9	57.0	0.6	59.3	58.6	0.6	58.1	55.9	1.3	60.6	61.5	1.4
NI	2.28	2.71	0.15	1.17	1.37	0.14	1.17	1.44	0.16	1.17	1.53	0.21
FN	1.07	1.35	0.12	0.43	0.53	0.06	0.43	0.57	0.07	0.41	0.59	0.09
ND	51.7	53.1	1.7	63.3	62.0	1.1	62.5	60.0	2.3	64.9	64.2	1.1
UN	1.01	1.08	0.05	0.74	0.86	0.06	0.70	0.88	0.06	0.67	0.81	0.07
NB	0.20	0.28	0.04	-0.00	-0.02	0.04	0.03	-0.01	0.05	0.09	0.13	0.06

N-balance

The data relevant to N intake (NI), excretion of faecal N (FN) and urinary N (UN) are in Table 2. Apparent N digestibility (ND) did not differ significantly between groups in any balance trial. Also mean N balances (NB) did not differ significantly between groups in any trial due to a large intra-group variation. Intra-group NB was significantly related to DOMI (Figure 2) and group effects were tested in a regression model of NB against DOMI. During the first trial, the group effects on intercept and regression coefficient (b) were not significant ($P>0.7$) and the overall regression equation was $NB = -0.180$ (se 0.116) $+ 0.0101$ (se 0.0029) $*$ DOMI ($r=0.72$, $rsd=0.088$). The regression coefficient was not significantly affected by group in periods 2, 3 and 4 but the intercept was significantly higher in the control group in period 3 (-0.288 versus -0.395 with $b=0.0161$) and tended ($P<0.07$) to be higher in period 2 (-0.289 versus -0.355 with $b=0.0144$). For the fourth period the overall regression equation was $NB = -0.243 + 0.0150 * DOMI$ (no significant differences between groups), showing an increase in intercept for both groups towards the end of the experiment.

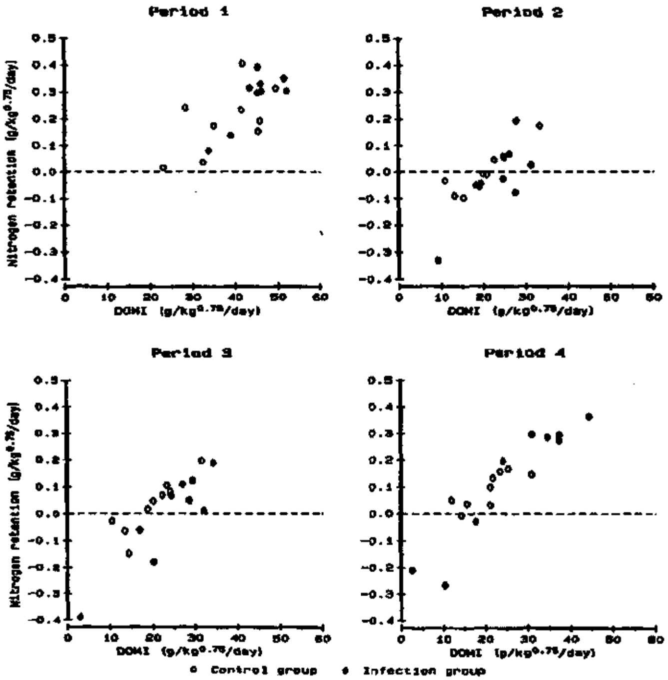

Figure 2 Nitrogen retention plotted against digestible organic matter intake (DOMI) for control and infection group in balance period 1 (one week pre-infection), 2, 3 and 4 (2, 4 and 7 weeks post-infection, respectively).

Energy and protein metabolism

Mean values for blood serum content of NEFA, BHBA, TP, and CR are in Table 3. In the control group, NEFA levels were not affected by period. Infected animals showed significantly ($P<0.05$) higher NEFA levels in the three balance periods after infection than controls (Figure 3). Mean BHBA level was significantly lower in the infection group in period 3 despite the fact that the two infected animals with the lowest voluntary DOMI showed high BHBA values, especially in period 4. Period did not affect TP values in the controls but in the infected animals TP levels were significantly higher than controls in the last two periods. Mean CR levels were increased after the first period in both groups and differences between groups were small and not consistent.

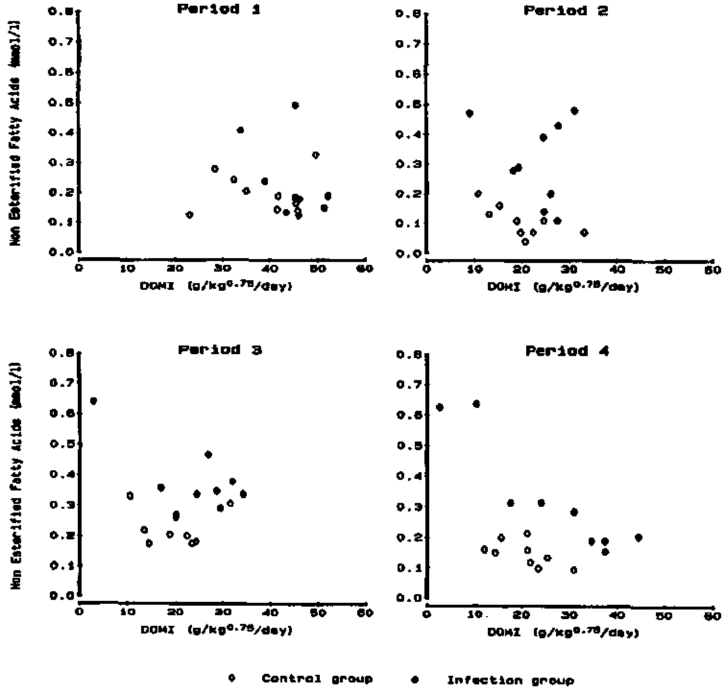

Figure 3 Serum NEFA concentration plotted against digestible organic matter intake (DOMI) for control and infection group in balance period 1 (one week pre-infection), 2, 3 and 4 (2, 4 and 7 weeks post-infection, respectively).

Table 3: Blood serum concentration in dwarf goats of non-esterifeid fatty acids (NEFA, mmol/l), beta-hydroxy butyric acid (BHBA, mmol/l), total protein (TP, g/l), creatinine (CR, micromol/l) and urea (mmol/l) in infection (I, n=9) and control (C, n=9) animals during periods 1 (*pre-infection*) and 2 to 4 (*post-infection*)

Period	1			2			3			4		
Group	C	I	se	C	I	se	C	I	se	C	I	se
NEFA	0.156	0.173	0.024	0.172	0.331	0.025*	0.195	0.359	0.028*	0.137	0.317	0.042*
BHBA	0.165	0.166	0.006	0.159	0.200	0.024	0.174	0.147	0.017	0.176	0.190	0.029
TP	77.5	77.9	1.4	76.4	75.7	1.5	76.6	83.4	1.7*	75.5	95.0	2.92*
CR	77.4	74.9	2.3	95.8	97.1	2.4	99.6	103.1	11.3	106.7	86.1	4.21

*: se indicates a significant ($P<0.05$) differerence between groups within periods

Table 4 shows the amounts of urea and creatinine excreted in the urine. Within periods, differences in urinary urea excretion between groups were not significant at the P<0.05 level. Differences between groups in urinary creatinine excretion were small but significant in periods 2 and 3.

Table 4: Excretion with urine of urea (URUR, g/kg$^{0.75}$/d) and creatinine (URCR, g/kg$^{0.75}$/d) by dwarf goats of the infection (I, n=9) and control (C, n=9) group during balance periods 1 (pre-infection) and 2 to 4 (post-infection)

Period	1			2			3			4		
Group	C	I	se	C	I	se	C	I	se	C	I	se
URUR	1.97	1.84	0.16	1.39	1.51	0.13	1.12	1.56	0.15+	1.54	1.76	0.16
URCR	0.05	0.04	0.004+	0.05	0.06	0.002*	0.04	0.06	0.005*	0.06	0.06	0.004

+: se indicates a tendency ($P<0.10$) for differences between groups within periods

*: se indicates a significant ($P<0.05$) differerence between groups within periods

DISCUSSION

The course of infection with *Trypanosoma vivax* in the goats used in this study, as reflected by an increased rectal temperatures, a high parasitaemia, and a decreasing PCV, did not differ from the pattern described by Ingh *et al.* (1976b) and Vos (1989). The PCV of the infected animals were significantly lower than those of the non-infected animals and did not show any signs of recovery towards the end of the trial. Between animals, the PCV was not significantly affected by DOMI in either group. This is in accordance with the view held by Murray and Dexter (1988), namely that immunological mechanisms and lowered erythropoesis are the most important factors contributing to anaemia in trypanosomiasis. However, this does not exclude the possibility that the level of nutrition plays an important role in the rate of recovery from anaemia caused by trypanosomiasis, as was found by Agyemang *et al.* (1990) in N'dama's under natural tsetse challenge.

From the day of infection, all animals decreased their intake of food but to a variable extent. Some animals continued to show a consumption above the estimated level for maintenance in healthy animals, others decreased their intake to far below this level. The decrease in intake reverted to an increase towards the end of the experiment in some, but not all, animals (see Figure 3), as earlier described (Zwart *et al.* 1991). In order to be able to differentiate between the effects of infection on NB due to anorexia and those due to the infection itself, the control animals were assigned to different feeding levels after infection. Fig. 2 shows that the attempt was quite successful for periods 2 and 3 but that the range in intake was larger in the infected animals in period 4.

Regression analysis of NB on DOMI within periods showed, at equal DOMI, a significantly higher ($P < 0.01$) NB for control animals over infected ones in period 3, a similar tendency in period 2 and no effect in period 4. At first sight, this suggests that infection resulted in a decrease in the ability to retain N for a given energy intake level during the first weeks after infection but that recovery occurred towards the end of the experiment. However, a comparison of NB versus DOMI for the infection group in periods 2 and 3 (i.e. post-infection) with period 1 (pre-infection) suggests no change at all (see Figure 2). This was confirmed by a statistical analyses of group means: for the infection group the overall regression equation in the first 3 periods was $NB = -0.345 + 0.0140 * DOMI$ with no significant period effect. This equation is almost identical to the equation reported by Ketelaars and Tolkamp (1991) for 24 group means of healthy dwarf goats on a variety of diets: $NB = -0.378$ (se 0.045) + 0.0144 (se 0.0014) $*$ DOMI. Also the group mean NB recorded in the first period for the control group (i.e. with food available *ad libitum*) is in accordance with this regression equation. Group mean NB was much higher than expected according to this equation in the control group in periods 2, 3 and 4 (i.e. when food was severely resticted) and in the infection group in period 4. The healthy animals in the experiments reported before (Ketelaars and Tolkamp, 1991) were fed at either an *ad libitum* (18 groups) or a restricted, but not below maintenance (6 groups), level. Apparently, our control animals gradually adapted to the, partly severe, food restriction in the course of the experiment and succeeded in increasing N retention while energy intake remained virtually the same. This could well be the result of a gradual decrease in energy maintenance requirements as the result of food restriction to levels below maintenance, as has been observed before in sheep and cattle (Blaxter, 1989). In healthy *ad libitum* fed dwarf goats, the amount of DOM needed to maintain weight was estimated at 26.0 g/kg$^{0.75}$/d by Zemmelink *et al.* (Zemmelink *et al.* 1985). In the same species, a zero nitrogen retention was found to coincide with a zero energy balance (Raviendran, 1988). From the equation reported by Ketelaars and Tolkamp (1991) it can be calculated that a zero NB is expected at DOMI=26.2 g/kg$^{0.75}$/d, i.e. maintenance requirements, approximately. For the control animals on a restricted diet, zero NB in periods 2, 3 and 4, can be estimated at 20.1, 17.9 and 16.2 g/kg$^{0.75}$/d, respectively. These values are much lower than expected for *ad libitum* fed animals. The idea that maintenance requirements were reduced in the control group on a restricted diet is strengthened by the phenomenon of a decreasing rectal temperature when DOMI decreased as found in this experiment.

For the infection group in periods 1, 2 and 3, a zero NB occurred at an energy intake level comparable to the level reported before (Ketelaars and Tolkamp, 1991) for healthy animals (24.9 and 26.0 g/kg$^{0.75}$/d, respectively), even after infection. This suggests that although voluntary intake decreased considerably, infection had no immediate effect on the amount of DOM required for a zero NB although the maintenance energy requirements were higher as a result of fever. The effect of DOMI on NB during the first three balance periods was also comparable to the effect reported before (Ketelaars and Tolkamp, 1991) for healthy animals (increase in NB per g DOMI of 0.0140 and 0.0144 g, respectively). This implies that, apparently, the energy cost of infection was covered by non-protein energy from food or body reserves. Earlier experiments do indicate a preference of dwarf goats under *Trypanosoma vivax* infection to save nitrogen (Verstegen *et al.* 1991). These authors observed fat mobilisation (-94 kJ/kg$^{0.75}$/d) to be of greater importance than protein breakdown (-4 kJ/kg$^{0.75}$/d) to supply energy requirements. The high NEFA values of infected compared with control animals in periods 2, 3 and 4 suggest that lipolysis is indeed a major mechanism to supply the higher energy demands of the fever (Zwart *et al.* 1991), although a disturbance of liver metabolism under the influence of infection may also play a role (Lumsden *et al.* 1972).

High energy demands, coupled with a low DOMI, can result in a rise of ketone bodies (Wensing *et al.* 1989). Increased BHBA levels were only observed in two infected goats with extremely low energy intakes towards the end of the experiment. The energy shortage in the other animals was probably not severe enough to cause a disturbance of normal ketone body metabolism.

The sharp increase in NB of the infected animals toward the end of the experiment (from -0.01 g/kg$^{0.75}$/d in period 3 to +0.13 g/kg$^{0.75}$/d in period 4) is not easily interpreted. From the regression equation for this group during the first three periods, an increase of 0.04 g/kg$^{0.75}$/d could be expected as a result of the increase in DOMI. The remaining increase is associated with a smaller than expected increase in FN (i.e. a higher apparent N digestibility) and especially a decrease in UN (i.e. a more efficient utilisation of apparently digested N). As the effect of DOMI on NB during this period did not differ significantly between groups, this suggests that the infected animals recovered towards the end of the experiment. Indeed, parasitaemia was lower in the fourth compared with the third period. Also the differences between groups in urinary CR excretion, significant in period 2 and 3, ceased to be significant in period 4. Differences in urinary CR excretion between groups and between periods were, however, too small to be indicative of important differences in muscle mass (Kaneko, 1989). A possible recovery was not reflected in changes in PCV between the third and fourth periods in the infected group. Also, rectal temperature remained significantly elevated in the fourth period and NEFA values continued to be higher in infected compared with healthy animals in period 4, which does not indicate recovery. In addition, a relatively high NB not only occurred in infected animals with an increasing energy intake but also in animals consuming considerably less energy than maintenance requires and still decreasing intake. It can be calculated from tables 2 and 4 that the mean urinary urea N excretion as a percentage of total urinary N excretion was 84% (period 1), 83% (period 2) and 78% (period 3) but that this value exceeded 100% for both groups in period 4. This indicates that either the urinary urea N excretion on the two measurement days in period 4 was not representative for the whole week or that total urinary N excretion was underestimated in period 4 in both groups. The increase in total protein in infected animals in period 3 and especially in period 4 is probably the result of a rise in gamma globulin concentration (Anosa and Isoun, 1976).

It is concluded that infection with *Trypanosoma vivax* results in a decrease in food intake and a decrease in NB. As the decrease in NB is not more severe than can be expected from the relation between DOMI and NB recorded with *ad libitum* fed healthy dwarf goats, the energy cost of infection has been covered by non-protein energy sources, as witnessed by the increased serum NEFA levels and the constant level of urinary CR excretion.

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Chapter 3

A NOTE ON THE EFFECT OF LEVEL OF ENERGY INTAKE ON RECTAL TEMPERATURE AND MAINTENANCE REQUIREMENTS OF WEST AFRICAN DWARF GOATS

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A NOTE ON THE EFFECT OF LEVEL OF ENERGY INTAKE ON RECTAL TEMPERATURE AND MAINTENANCE REQUIREMENTS OF WEST AFRICAN DWARF GOATS

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INTRODUCTION

In an experiment reported earlier (Akinbamijo et al., 1992), the effect of *Trypanosoma vivax* infection on energy and nitrogen metabolism in West African Dwarf (WAD) goats was investigated. Rectal temperature (RT) was recorded daily in all animals to monitor the effect of trypanosomiasis in infected animals. It was observed that while the infection lasted, the infected animals depressed their feed intake to a variable extent. In order to obtain a range in energy intake similar to that of infected animals, non-infected control animals were offered a restricted diet that simulated the decreased feed intake of the infected group. The RT of the control animals was considerably lower during the feed-restriction period compared to the preceding ad libitum feeding period (Akinbamijo et al., 1992).

On the basis of the group averages, Akinbamijo et al., (1992) suggested that a reduction in energy intake level resulted in a lower RT. In this note, the analysis of data on individual goats is presented. In addition, estimates of the effect of energy intake level on RT from a subsequent experiment with WAD dwarf goats are reported.

MATERIALS AND METHODS

Experiment 1

This experiment is described in detail by Akinbamijo et al. (1992). Briefly, the relevant details are: 18 adult castrated male WAD goats weighing around 28 kg were divided into two groups of 9 animals. The animals were housed in metabolism cages which allowed faeces and urine to be collected separately. Ambient temperature was maintained at 20 °C. The infection group was infected with *T. vivax*, and digestible organic matter intake (DOMI) and nitrogen balance were measured in four seperate balance trials each lasting zeven days: week 1 pre-infection and at weeks 2, 4 and 7 post-infection. Infected animals received an *ad libitum* diet of pelleted lucerne. During these four periods, DOMI and nitrogen balance were measured in the non-infected control group. Animals in this group received lucerne pellets ad libitum during the first balance trial but restricted lucerne pellets during the last three balance trials. Feed allowances ranged from 0.4 to 1.4 times the maintenance requirements for WAD goats as estimated by Zemmelink et al. (1991). RT was recorded daily at around 0900 h before feeding. The individual means of the seven daily measurements of RT and the mean daily DOMI recorded during each balance trial were used in the present data analysis.

Experiment 2

Four groups of four castrated male West African Dwarf goats weighing around 20 kg were used in the study. The animals were housed in specially designed group pens equipped with four Calan doors (Calan Electronics Ltd, Crossroads, Scotland). Each animal carried a neck transponder which opened only one door to facilitate data collection on individual feed intake within the pens. The minimum in-door temperature was 17 °C. Animals received lucerne pellets ad libitum (group 3), a restricted amount of lucerne pellets, approximately maintenance requirements (group 4), chopped grass hay (group 5) or chopped grass straw (group 6). Individual organic matter intake (OMI) was estimated during one week from the amounts of feed offered and refused and the dry matter and ash contents determined in samples of feed offered and refused. OMI was measured after animals had been on the diet for 90 days. Three additional groups of 4 wethers were kept in metabolism cages to determine the organic matter digestibility (OMD) of the diets during a seven-day collection period following a three-week adaptation period. The DOMI of groups 3, 4, 5 and 6 was calculated from the individual OMI and the mean OMD for each diet. RT was recorded daily before the morning feeding. The individual means of the 7 measurements of RT and the mean daily DOMI estimated during the same week were used for the analyses.

STATISTICAL ANALYSES

The effects of DOMI on RT were estimated using linear regression. The differences between periods, experiments, diet type and method of feeding (ad lib versus restricted) were analysed using the analysis of variance. All means and estimates are presented ± s.e.

RESULTS

The nutritive value of the diets, mean DOMI and mean RT in the two experiments are shown in Table 1. As intake and RT were not affected by balance period during feed restriction in experiment 1, the data presented are for group 1 (recorded in animals fed ad lib during the first balance period) and group 2 (individual means of the three last balance periods when animals were fed restrictedly).

Table 1: Crude protein content (CP, % in DM), organic matter digestibility (OMD, %), mean (±SE) digestible organic matter intake (DOMI, $g.kg^{-3/4}.d^{-1}$) and mean rectal temperature (RT, °C) for the different diets in the two experiments.

Expt. Period	Group	Diet	CP	OMD	DOMI	RT
1	1	Lucerne, ad libitum	18.1	57.5±0.4	42.2±3.1	38.75±0.05
1	2	Lucerne, restricted	18.1	59.3±0.5	21.9±2.3	38.00±0.08
2	3	Lucerne, ad libitum	19.9	54.9±2.7	48.2±2.5	38.68±0.07
2	4	Lucerne, maintenance	19.9	54.9±2.7	24.8±0.5	38.32±0.04
2	5	Grass hay*, ad libitum	12.0	58.7±1.4	34.6±1.9	38.48±0.06
2	6	Grass straw*, ad libitum	5.4	47.1±0.6	25.8±1.5	38.10±0.13

*: mainly *Lolium perenne*

When RT was linearly regressed on DOMI, balance period was found to significantly affect the intercept but not the regression coefficient. At average DOMI, RT was significantly ($P<0.05$) higher in the first period (38.48 °C, ±0.13) compared to the last three periods (mean: 38.08 °C). Within balance periods, RT changed significantly ($P<0.01$) by 0.018 °C (± 0.006) for a change in DOMI of 1 $g.kg^{0.75}.d^{-1}$.

Regression of RT on DOMI in Experiment 2 resulted in the equation:

$$RT=37.79\ (\underline{+}0.17)\ +0.018\ (\underline{+}0.005) * DOMI,\ r.s.d.=0.20,\ R^2=0.49.$$

The results for the two experiments are plotted in Fig. 1. Linear regression, using all the data, resulted in the equation:

$$RT=37.56\ (\underline{+}0.11)\ +0.025\ (\underline{+}0.003) * DOMI,\ r.s.d.=0.21,\ R^2=0.67.$$

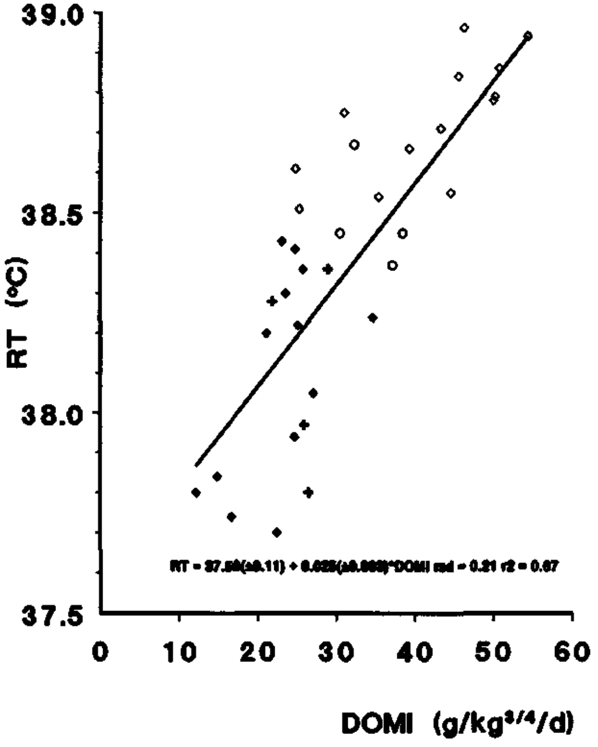

Figure 1 Correlation between digestible organic matter intake (DOMI) and rectal temperature (RT) in West African Dwarf goats.

(◇) = pelleted lucerne ad lib, (◆) = in restricted amounts, (○) = grass hay, (+) = grass straw.

Neither experimental period, nor diet type nor system of feeding affected RT significantly (P>0.2).

DISCUSSION

Figure 1 shows that a considerable range in energy intake levels was obtained in these experiments: from about half up to more than twice the estimated maintenance requirements for WAD goats. This variation in intake was strongly correlated ($P<0.001$; $R^2=0.67$) with variation in RT. As part of the variation in DOMI was experimentally induced, this suggests, at first sight, a causal effect of energy intake level on RT. However, DOMI was measured on a daily basis whereas RT was measured only once per day, i.e. in the morning before feeding. Diurnal variation in RT was, therefore, not taken into account. This may be relevant, as feed consumption itself causes a considerable increase in heat production. The RT in feed-restricted animals was probably measured long after these animals had consumed their last feed. The significant effect of feeding method on the level of RT at the same DOMI in the analyses of the first experiment is consistent with this view.

The effect of DOMI on RT remained significant if only the data of *ad libitum* fed animals were included in the analyses. However, WAD goats fed *ad libitum* usually eat their largest meal after they have received fresh feed in the morning (Mul, 1987). Therefore, in these groups fed *ad libitum*, the early morning RT might not be representative of the mean daily RT. In a recent study, diurnal variation in deep body temperature was measured using an implant which transmitted deep body temperature every seven minutes. A preliminary analysis of these data shows that there is a good correlation between the early morning and average daily deep body temperatures (van Dam, unpublished). The same analyses show, however, that the magnitude of the effect of DOMI on average daily deep body temperature is less than on early morning deep body temperature.

The lower RT (and average deep body temperature) might be related to the decrease in maintenance requirements as suggested by the gradual increase in nitrogen balance at a given level of DOMI, especially in feed-restricted WAD goats (Akinbamijo, *et al.* 1992).

It is therefore concluded that RT recorded in the morning is significantly affected by DOMI in dwarf goats fed *ad libitum* as well as in restrictedly fed animals. This variation in early morning RT or deep body temperature results in variation in average daily body temperature and this may be related to the variation in maintenance requirements.

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Chapter 4

THE EFFECT OF *TRYPANOSOMA VIVAX* INFECTION DURING PREGNANCY ON FEED INTAKE, NITROGEN RETENTION AND LIVEWEIGHT CHANGES IN SHEEP

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THE EFFECT OF *TRYPANOSOMA VIVAX* INFECTION DURING PREGNANCY ON FEED INTAKE, NITROGEN RETENTION AND LIVEWEIGHT CHANGES IN SHEEP

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ABSTRACT

Effects of infection with *Trypanosoma vivax* in mid- or late- pregnancy on food intake and utilization, liveweight changes, abortion rate and lamb growth rate were investigated in West African Dwarf ewes at Ibadan, Nigeria in 1990. Rate of live weight gain by ewes infected during mid-pregnancy (IMHP) was 16 g/day compared with 33 and 37 g/day of the uninfected ewes offered medium (CMP) or high (CHP) plane diet. Although digestibility coefficients were not affected, intake of digestible organic matter was higher in CHP ewes than IMHP and CMP ewes. Nitrogen retention at mid pregnancy on a metabolic size basis was higher in CHP ewes than in CMP and IMHP ewes. Lamb birth weight and survival rate were lower in infected ewes. IMHP and ewes infected at late pregnancy (ILHP) had mean birth weights of 1.4 and 1.0 kg compared with CMP and CHP with mean birth weights of 1.9 and 2.0 kg respectively. Observed survival rates were 63, 15, 75 and 80 % for lambs nursed by IMHP, ILHP, CMP and CHP ewes respectively. During the first 6 weeks after birth, lamb growth rate in all groups did not differ. However, during weeks 7 - 12 post partum, lambs nursed by IMHP ewes had significantly lower growth rates. Weaning weight was also lower in lambs from IMHP (5.0 kg) dams than in lambs from CMP and CHP dams (7.1 kg). Infection during late pregnancy was more severe and all infected ewes lost weight due to reduced feed intake and fever. *Trypanosoma vivax* infection in sheep is responsible for reproductive wastage, abortion, poor lamb growth and ewe mortality.

INTRODUCTION

Although trypanosomiasis research has progressed for several decades, the disease is still one of the major health constraints in the African livestock production system. The presence and significance of trypanosomiasis on the feed intake and utilization in pregnant animals have received minimal attention. Recently, nutrition studies have been conducted in trypanosome infected West African Dwarf (WAD) sheep by Elhassan (1987) and Reynolds & Ekwuruke (1988). These workers reported high reproductive wastage as fundamental hindrance to ruminant livestock productivity (Gunn 1983; Ikede *et al.* 1988). Such reproductive impairment is characterized by embryonic or fetal death, abortion, premature birth, stillbirth, birth of weak offspring and neonatal deaths.

Reduced feed intake and utilization in febrile trypanosomiasis infected small ruminants have been reported in the literature (Akinbamijo *et al.* 1990; Verstegen *et al.* 1991; Zwart *et al.* 1991). This may have profound influence on dam survival, abortion rate, lamb birth and weaning weight (Adu *et al.* 1974). In anticipation of wide variation in feed intake with probable anorexia in *Trypanosoma vivax* infected animals (Akinbamijo *et al.* 1992), a reduced dietary regime was imposed on some healthy animals to simulate feed intake in anorectic infected animals and also to alleviate the likelihood of a confounding feed effect in infected animals.

This study was aimed at investigating the effect of trypanosomiasis on live weight gain pattern, feed intake, nitrogen retention and digestibility of organic matter in pregnant WAD sheep. In addition, abortion rate, ewe mortality, lamb birth and weaning weight as affected by trypanosomiasis during mid and late pregnancy in ewes were investigated.

MATERIALS AND METHODS

Animals

The experiment was conducted at the International Livestock Centre for Africa (ILCA) Humid Zone station Ibadan, SW Nigeria. Thirty-eight West African Dwarf (Djallonke) ewes aged 24 - 36 months were used in the study. All ewes were selected from a closed colony of animals born and raised on station.

The sheep were randomly allotted to infected (n = 20) and uninfected (n = 18) groups. The ewes of the infected group were sub-divided into two groups: IMHP (infected 7th week of gestation, n=10) and ILHP (infected 15th week of gestation, n=10). The uninfected control ewes were divided into two groups: CMP (control offered medium plane diet n= 8) and CHP (control offered high plane diet, n=10).

Oestrus was artificially synchronized in all animals during the late rainy season (August) using dinoprost tromethanine solution - $PGF_2\alpha$ (Upjohn -Tuco

Products, Canada), administered i.m. in two doses of 1 ml given 11 days apart. Rams of known fertility were introduced 24 h after the second prostaglandin administration, and left with the ewes for 2 weeks. A Doppler ultra-sound pregnancy detector (Medata Systems Limited, Pagham West Sussex, UK) was used to monitor pregnancy, fetal resorption and *in-utero* deaths during the gestation period.

Inoculation

All experimental animals were screened for *Trypanosoma vivax* antibodies pre- infection (Katende *et al.* 1987) and for trypanosomes at the start of the experiment and were found to be negative.

Animals in groups IMHP and ILHP were inoculated in the 7th and 15th week post breeding, respectively (see Table 1). The pathogen used was *Trypanosoma vivax* prepared from the Kaban strain isolated from an ox in Kaban village Plateau State of Nigeria. Each inoculated ewe was challenged intravenously using 4×10^6 trypanosomes in heparinized medium. Infection was confirmed by thin/thick blood smears and by the phase contrast buffy coat technique (Murray *et al,* 1983). In order to limit the infection to the physiological phase of interest, all dams were given therapeutic doses of Diminazine aceturate (Berenil, Hoechst, Germany) 3.5 mg/kg body weight shortly after parturition, abortion or withdrawal from the study.

Ewes that aborted or had a Packed cell volume (PCV) of < 12% recorded for two consecutive weeks were removed from the live weight data.

Table 1. Time schedule

Operation	Week of Experiment
Oestrus synchronization	
First dose	-2
Second dose	-1
Breeding	0 - 1
Adaptation to crates	6
Inoculation of Group IMHP	7
Nitrogen retention trial	8
Inoculation of Group ILHP	15
Parturition	20 - 21
Weaning	33

Measurements

Baseline data for analyses were collected for all experimental animals from 5 weeks after breeding. Blood sampling was done twice weekly from the jugular vein into evacuated tubes (Vacutainer, Becton Dickinson). Samples (5 ml) were collected in EDTA tubes to estimate PCV and parasitaemia (Murray *et al.* 1983). Mean values of the twice weekly determination of the PCV was assumed to be the average PCV in the animal for the week of sampling. Rectal temperature (RT) was measured daily for 4 weeks post-infection and weekly thereafter in all animals between 09.00 h and 10.00 h.

All dams were weighed each Monday at 09.00 h before feeding during the entire gestation and lactation period. Liveweight data was analyzed for four periods of early-, mid-, late pregnancy and 10 weeks post-natal. All lambs were weighed within 24 h of birth and liveweight monitored weekly thereafter. Lambs were weaned at 10 weeks after birth. Survival of the lambs was calculated as a ratio of lambs weaned to lambs born alive.

Housing, management and feeding

Experimental ewes were housed in individual pens in two separate, well ventilated, and naturally illuminated housing units. The infected ewes were fly -screened while the uninfected ewes were not screened. During the digestibility trial, animals used for the trial were housed in a double-cabin wooden metabolism crate for 3 weeks from weeks 6 - 8 post breeding. Routine station health management as described by Reynolds & Ekwuruke (1988) was performed.

Every morning, all animals in groups IMHP, ILHP and CHP were offered a basal diet of 4 kg freshly cut *Panicum maximum*. Group CMP received 2 kg *P. maximum* from the 7th week of pregnancy until parturition. The grass was chopped to approximately 2.5 cm length to facilitate feeding. In addition, all animals were supplemented with 550 g/day of *Gliricidia sepium* and *Leucaena leucocephala* as well as 50 g/day dried cassava (*Manihot spp.*) peels throughout the experimental period. Feed refusals were collected and weighed the following morning. After lambing, the dietary allowance of the CMP ewes was upgraded to that offered the IMHP, ILHP and CHP ewes. The composition of feed offered on dry matter basis is presented in Table 2. Experimental animals had free access to drinking water and mineral licks.

Table 2. Composition of feed offered on dry matter basis.

Feed stuff	Dry matter (%)	Nitrogen (%)	Ash (%)
Panicum maximum	17.42	1.70	14.91
Dried Cassava Peels	70.37	0.734	11.59
Gliricidia sepium	31.94	3.69	9.88
Leucaena leucocephala	30.67	3.68	9.21

Digestibility and N balance trial

A seven-day digestibility and N-balance trial was conducted during the 8th week of pregnancy following a 2-week adaptation period. Fifteen ewes (five per group) were randomly selected from groups IMHP, CMP and CHP. Representative samples of feed, refusals and faeces from weekly bulked samples were analyzed for dry matter, ash and nitrogen according to the procedures of the Association of Official Analytical Chemists (1975). Nitrogen retention was calculated from nitrogen in fresh feed, refusals, faeces and urine (Akinbamijo *et al.* 1992).

Statistical analysis

Analysis of variance was used to compare the treatment groups in the metabolism trial. Linear regression models of weight on gestation period and of weight on first 10 weeks post-natal were fitted for each animal. The rate of liveweight change for each ewe were further analyzed using analysis of co-variance for comparing the different treatment groups at pre- and post-natal periods. Corrections were made for the co-variates weight at parturition and for the number of lambs suckled during lactation.

Birth weights of lambs in different groups were compared using analysis of variance. Lamb performance data were analyzed for weeks 0-6 and 7 - 12 after birth. For each lamb, a linear regression model of weight on time was fitted for each period. The regression coefficients were further subjected to analysis of co-variance, using birth weight and gender of lambs as co-variates. Mean values are presented ± standard error (se).

RESULTS

All ewes tested negative for the presence of antibodies against *Trypanosoma vivax* pre-infection and the inoculated ewes remained infected through pregnancy. After infection, packed cell volume (PCV) declined rapidly to 40% of pre-infection values (Fig 1). Two ewes from IMHP and one from ILHP were treated with trypanocide and removed from the experiment due to severe anaemia. In spite of the treatment, at 5 and 4 weeks post infection (p.i.), one of the anaemic animals from IMHP and ILHP ewes respectively, died following 3-4 days of weakness, persistent pyrexia and inanition. Three other ewes (two from group IMHP and one from group ILHP) aborted their pregnancy at 6 and 3 weeks p.i. in IMHP and ILHP respectively after a protracted anaemia (<15%), fever, anorexia and persistent parasitaemia. Abortion rate was 15% in the infected groups and zero in the uninfected animals.

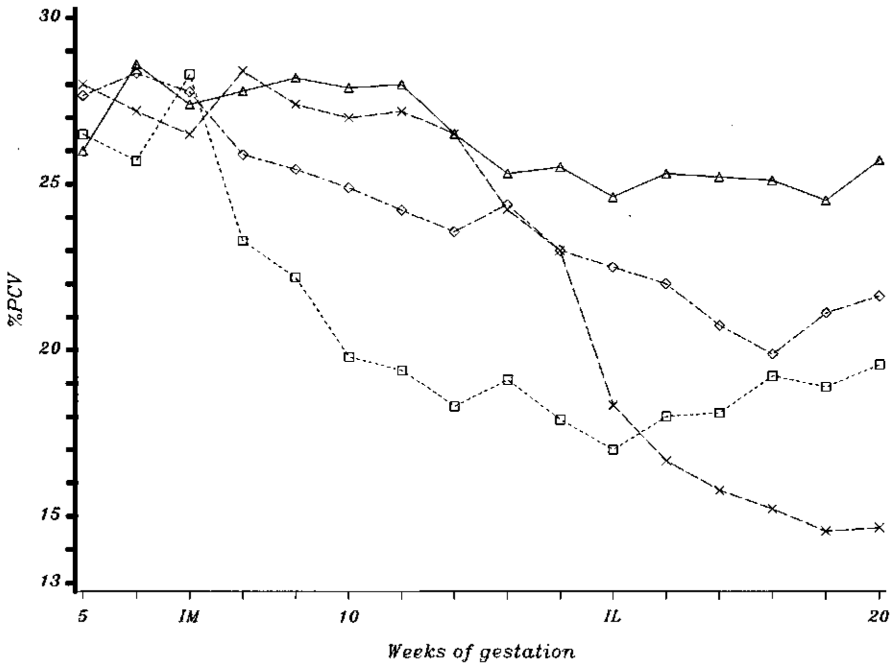

Figure 1 Mean Packed cell Volume of WAD ewes infected with *T. vivax* at mid pregnancy (IMHP □), late pregnancy (ILHP x), uninfected offered medium plane diet (CMP ◇) and high plane diet (CHP ▵).

IM = Inoculation at mid-pregnancy
IL = Inoculation at late-pregnancy

Mean rectal temperature was 40.04°C (se 0.075, n=20) compared with 38.69°C (se 0.079, n=18) in the controls. Monthly ultrasound pregnancy checks showed that there was no death *in utero* or fetal resorption during gestation.

In the pre-infection period (early pregnancy), all experimental animals were handled as one group as there were no treatment effects imposed during this period. Consequently, liveweight data collected at this period were pooled to obtain a common mean rate of liveweight gain (37.1 g/day). During the mid pregnancy phase, although treatment effect was already introduced, differences in rate of liveweight change were not statistically significant (see Table 3). In the late pregnancy however, the average daily weight gain in IMHP and ILHP ewes declined further while the uninfected ewes (CMP and CHP) gained weight. In the post-partum period, control animals lost more weight than the infected ones (Table 3).

Table 3: Rate of liveweight changes in pregnant West African Dwarf ewes infected with *T. vivax* at mid pregnancy (IMHP), late pregnancy (ILHP), uninfected offered medium plane diet (CMP) and high plane diet (CHP).

	Groups			
Variables	IMHP	ILHP	CMP	CHP
n	6	8	8	10
Mid pregnancy* (g/day)	15.7±8.6	32.9±4.3	37.1±7.1	32.9±4.3
Late pregnancy (g/day)	-27.1±15.7	-21.4±12.9	1.4±12.9	30.0±11.4
10 weeks post-partum (g/day)	-38.6±10	-25.7±8.6	-52.9±8.6	-75.7±7.1

* pooled means for ILHP and CHP ewes (n=18)

Lamb birth weight, growth and survival rates

All lambs were born live in all groups. While differences in lamb birth weight were not influenced by time of infection (i.e between IMHP and ILHP ewes) or feeding level (i.e between CMP and CHP ewes) uninfected dams had heavier ($P<0.05$) lambs compared with their infected counterparts at parturition (Table 4).

Table 4. Birth weight, growth rates and weaning weight of lambs produced by West African Dwarf ewes infected with *Trypanosoma vivax* at mid pregnancy (IMHP), late pregnancy (ILHP), uninfected offered medium plane diet (CMP) and high plane diet (CHP).

	Groups			
Variables	IMHP	ILHP	CMP	CHP
n	8	13	8	12
Birth weight (kg)	1.4±0.17	1.0±0.8	1.9±0.16	2.0±0.13
Early lactation (g/day)	88.0±11.4	ND*	76.0±8.6	77.0±7.1
Late lactation (g/day)	26.0±10.0	ND	39.0±7.1	33.0±7.1
Weaning weight (kg)	5.0	ND	7.1	7.1

* ND = not determined

Lambs from infected ewes were weaker than lambs produced by their control counterparts, judged subjectively by environmental stimuli to sound, light and degree of activity. Consequently, 11 out of 13 lambs in the ILHP group died within seven days post-partum and are therefore not included in the statistical analysis of lamb growth rate.

Liveweight gain of lambs at early and late lactation phases is presented in Table 4. Treatment imposed on the dams during gestation and lamb sex had no effect on lamb growth rate. Lambs from groups CMP and CHP did not differ (P>0.05) in birth weight, growth rate and weaning weight. However, they had significantly higher birth and weaning weight but growth rate was similar in all groups (Table 4). Rate of survival at weaning was 63, 15.4, 75 and 80 % for groups IMHP, ILHP, CMP and CHP respectively.

Digestibility trial (mid pregnancy)

Feed intake and nitrogen balance data is presented in Table 5. Dry matter intake (DMI) and digestible organic matter intake (DOMI) did not differ (P>0.05) between IMHP and CMP ewes. CHP ewes had significantly higher DMI and DOMI than CMP and IMHP ewes. Digestibility coefficients, faecal and urinary nitrogen did not differ (P>0.05) in all the groups.

Infected ewes retained the least nitrogen during mid pregnancy when compared with the control ewes on a metabolic size basis. IMHP and CMP did not differ significantly from each other but both groups are significantly different from CHP.

DISCUSSION

From parasitaemic and haematological assessments p.i., anaemia, fever, and presence of antibodies coupled with anorexia, implicated clinical trypanosomiasis in infected ewes. Control ewes were free of trypanosome infection throughout the study period. In agreement with earlier reports on experimental trypanosomiasis (Anosa & Isoun 1980; Akinbamijo 1988), inoculated animals were anaemic as from week 1 p.i.

The digestibility and nitrogen balance data indicate that the effect of trypanosomiasis on feed intake is significant causing a reduced nitrogen retention and liveweight gain. Although infected ewes were offered a high plane diet, the feed intake was only comparable with that of control ewes offered medium plane diet. This reduction in voluntary feed intake of infected animals is of the same order (20%) as in earlier findings in trypanosome infected goats (Akinbamijo *et al.* 1990; 1992) and sheep (Elhassan 1987).

The DOMI observed in uninfected ewes (CMP, CHP) indicate that intake was high enough to cover the ewes' maintenance requirements for energy and protein and with enough metabolizable energy and protein to gain weight. The DOMI obtained (27.6 $g/kg^{0.75}$) from the CMP ewes also meets maintenance requirements for sheep (ARC 1980). Although digestibility was not affected by infection or feeding level, nitrogen intake and retention was higher ($P<0.05$), in CHP than the CMP or IMHP ewes reflecting the high organic matter intake of the CHP ewes. Data on nitrogen retention indicate that there was no significant difference in urinary and faecal nitrogen voided in all groups. This suggests that the relatively high intake in the CHP group is largely responsible for the difference in nitrogen retention.

Other significant avenues of increased energy expenditure in infected ewes include high basal metabolic rate/ maintenance requirement due to fever. The basal metabolic rate is usually increased in febrile infected animals and hence the need for metabolites is higher than in the control ewes. A 15% rise in metabolic rate (Blaxter 1989) and 25% rise in maintenance requirements (Verstegen *et al.* 1991) for every degree rise in temperature have been reported previously. In the present trial, rectal temperature was 1.35°C higher in infected animals, suggesting that metabolic rate and maintenance requirement would be 20 and 34 % higher than in their control counterparts respectively. In agreement with our hypothesis, it seems that apart from the inappetence in infected animals, trypanosomiasis increases energy expenditure with considerable reduction in the efficiency of utilization of energy for productive purposes.

The most prominent effect of intake and nitrogen retention was seen in the liveweight gain pattern (Table 5). Weight changes observed in uninfected ewes in this study are in accordance with earlier reports in healthy (Orji & Steinbach 1980) and infected pregnant WAD ewes reported by Reynolds & Ekwuruke (1988) under comparable conditions.

Table 5: Nutrient intake, digestibility and nitrogen retention in West African Dwarf ewes infected with *Trypanosoma vivax* at mid pregnancy (IMHP), uninfected offered medium plane diet (CMP) and high plane diet (CHP).

	Group			
Variables	IMHP	CMP	CHP	SE
Intake ($g/W^{0.75}$)				
Dry matter	49.7	50.1	61.2	1.96
Organic matter	44.2	44.5	54.5	1.74
Digestible organic matter	28.5	27.6	35.3	1.35
Crude nitrogen	1.08	1.10	1.28	0.045
Digestibility (%)				
Dry matter	61.7	60.0	62.0	1.21
Organic matter	64.3	62.1	64.7	1.08
Nitrogen	64.2	62.1	64.0	1.30
Faecal nitrogen ($g/W^{0.75}$)	0.38	0.42	0.46	0.020
Urinary nitrogen ($g/W^{0.75}$)	0.61	0.51	0.52	0.053
Nitrogen retention ($g/W^{0.75}$)	0.09	0.17	0.30	0.058

Although during mid pregnancy, the mean rate of liveweight gain did not differ ($P>0.05$) between groups, IMHP ewes tended to have a lower liveweight gain than CHP and CMP ewes. This difference in weight gain was not significant due to large within group variation in IMHP ewes.

During the late pregnancy period, compared with CHP ewes, weight gain was lower ($P<0.05$) in IMHP, ILHP and CMP groups. Although CMP ewes still managed to gain weight, this shows that both IMHP and ILHP ewes lost weight because of the depressed intake and rapid depletion of body reserves to meet the increased requirements for maintenance (Verstegen *et al.* 1991; Zwart *et al.* 1991) and gestation.

The reduction of feed intake, anaemia and the persistent pyrexia facilitated the reproductive wastage of abortion and ewe mortality observed in the study. From the PCV, fever and feed intake records prior to abortion, the three cases of abortion observed were induced by trypanosomiasis. The abortion rate observed in this experiment is considered to be low probably due to the trypanotolerance trait in the breed used. Sanusi (Unpublished) reported 80% abortion in non- trypanotolerant Maradi goats when infected with the same strain of trypanosomes as we have used. Abortions have also been reported in *Trypanosoma vivax* infected WAD ewes (Ikede & Losos 1972; Elhassan 1987; Ikede *et al* 1988), and White Fulani cows (Ogwu *et al.* 1986).

Considering that the lamb birth weight is a reflection of pregnancy nutrition, and that trypanosomiasis imposes a degree of increased energy expenditure in infected hosts, then the infected ewes would have less net energy for production to favour optimal fetal development. Consequently, the smaller ($P<0.05$) lambs from infected ewes were unable to attain birth and weaning weights similar to that of lambs from control dams (Reynolds & Ekwuruke 1988; Elhassan 1987). Although clinical tests were not conducted to confirm transplacental transmission, all lambs from group ILHP ewes were born weak and subsequently 85% died within 7 days after birth. A similar observation was reported during bovine trypanosomiasis when calves born to heifers infected in the third trimester had the lowest birth weights and survival rates (Ogwu *et al.* 1986). Trypanosomiasis induced anorexia during critical fetal development and possible intra-uterine infection of the lambs is suspected to be the main cause of low birth weights (Ikede *et al.* 1988). Although the lamb growth rate was not affected by pre-partum infection in the dams, the weaning weight being a function of birth weight was lower in lambs born by infected dams. In this experiment, milk production was not measured. However, it was obvious that all ewes catabolized body reserves to augment lactation (Adu *et al.* 1974). Another salient factor that contributed to the decline in live weight changes was the low quality dry season *P. maximum* available during lactation.

In conclusion, the evidences suggest that reduced feed intake of *Trypanosoma vivax* infected ewes during pregnancy resulted in lowered dam nitrogen retention, loss of body weight, abortion and low lamb birth weights. It is also believed that the infection was responsible for the ewe mortality and low lamb weaning weight.

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Chapter 5

EFFECT OF POST-PARTUM *TRYPANOSOMA VIVAX* INFECTION ON FEED INTAKE, LIVE WEIGHT CHANGES, MILK YIELD AND COMPOSITION, IN WEST AFRICAN DWARF EWES AND ASSOCIATED LAMB GROWTH RATE

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ABSTRACT

The Effect of trypanosomiasis on digestible organic matter intake, milk yield and composition, dam liveweight changes during lactation and lamb growth rates were investigated at Ibadan, Nigeria in 1992, using 20 West African Dwarf sheep nursing single lambs. Although digestibility coefficients were neither affected by infection nor level of feed intake, organic matter intake during early and late lactation was significantly lower in infected dams. Nitrogen retained in the late lactation was lower in infected animals due to reduced feed intake. Mean daily milk yield of 426, 418 and 399 g/day for groups IHP, CMP and CHP respectively were not affected by the infection during early lactation. However during the second half of lactation, average daily milk yields of 182, 238 and 240 g/day for groups IHP, CMP and CHP respectively was significantly lower in infected animals. Variation in milk component concentrations between groups did not attain statistical significance throughout lactation. While CHP ewes gained 12.1 g/day, IHP and CMP ewes lost 45 and 5.4 g/day respectively during lactation. Live weight gain in the lambs was not affected by infection in the dams. This study demonstrated reduction in feed intake, late lactation milk yield and dam live weight gain with no adverse effect on digestibility coefficients, milk composition, early lactation milk yield and lamb weight gain during *T. vivax* infection of lactating ewes.

INTRODUCTION

The pre-weaning performance of ovine offspring is generally affected by maternal effects such as dam's lactation potential, pre-natal nutrition, health, management, age and body weight of ewes (Robinson *et al.* 1969; Blood & Radostis 1989). The dam's milk production plays a major role in post-natal maternal performance, while failure to lactate usually lead to reduced lamb growth rates and survival (Metz 1990). Although many parasitic infections are known to depress milk yield in dairy cows (Bliss & Todd 1977; Barger & Gibbs 1981), the responses of lactating non-dairy West African Dwarf (WAD) ewes are sparsely reported in the literature (Reynolds & Ekwuruke 1988). Some factors that affect feed intake may influence lactation through depressed appetite in infected animals. Therefore, trypanosomiasis may lower the lactation potential and lamb growth rate in WAD sheep, as it is consistently accompanied by increased maintenance requirement and decreased intake of organic matter (OM) (Akinbamijo *et al.* 1990).

To test this hypothesis in view of the high variation in feed intake in *T. vivax* infected animals (Zwart *et al.* 1991), uninfected animals were assigned to one of two groups that simulate *ad libitum* (high plane) and depressed (medium plane) feed intake in uninfected animals. This study was therefore aimed at investigating the effect of experimental trypanosomiasis on feed intake, milk production and composition of WAD ewes, dam live weight changes and rate of lamb weight gain nursed by infected and healthy dams.

MATERIALS AND METHODS

The investigation was conducted at the International Livestock Centre for Africa (ILCA) humid zone station in SW Nigeria.

Twenty WAD ewes in their second parity (age 24-30 months) born and raised on station were used for the study. Experimental animals were all oestrus synchronized using prostaglandin (PGF_2-α) to facilitate synchronized parturition (Akinbamijo *et al.* 1994). All animals received identical treatment before and during gestation. Animal used in the experiment were screened for *T. vivax* antibodies pre-infection (Katende *et al.* 1987) and for trypanosomes (Murray *et al.* 1983) at the start of the experiment and were confirmed to be without any previous history of trypanosomiasis.

At parturition, animals were randomly allocated into infected group offered high plane (HP) diet (group IHP, n=10) and uninfected control groups fed either medium plane (MP) diet (group CMP, n=5) or fed HP diet (group CHP, n=5). Details of the *T. vivax* strain used have been reported by Akinbamijo *et al.* (1994). In the infected group (IHP), each lactating ewe was challenged intravenously using 4 X 10^6

trypanosomes in heparinized medium 1 week post-partum. Infection was confirmed by thin/thick blood smears and by the phase contrast buffy coat technique described by Murray *et al.* (1983). Infected ewes were treated at the end of the experiment with Diminazine aceturate 3.5 mg/kg body weight (Berenil®, Hoechst, Germany).

To confirm establishment of trypanosomiasis, blood sampling was done twice weekly (Mondays & Thursdays) by jugular venepuncture into evacuated tubes (Vacutainer®, Becton Dickinson). Samples of 5 ml blood were collected in ethylene diamine tetra acetic acid (EDTA) tubes to estimate weekly packed cell volume (PCV) and parasitaemia (Murray *et al.* 1983). Mean values of the twice weekly determination of the PCV was assumed to be the average PCV of the animal for the week of sampling. As a routine practice, rectal temperature (RT) was measured daily in all experimental animals between 08.00 h and 09.00 h before feeding for 4 weeks post-inoculation and weekly thereafter.

All dams were weighed weekly before feeding during the entire lactation period. Lambs were weighed within 12 h of parturition and at weekly intervals afterward.

Housing, feeding and management.

All experimental ewes, managed as a single group before lambing, were housed in individual pens (2 x 1 m) in two separate, well ventilated, and naturally illuminated fly-proof housing units. Animals in groups IHP and CHP were offered freshly cut leaves of *Gliricidia sepium* (3000 g/day) and *Leucaena leucocephala* (1500 g/day). In addition, dried cassava (*Manihot spp.*) peel (50 g/day) was offered as supplement. Animals of the CMP group received 50% of this ration. The chemical composition of the experimental diet is presented in Table 1. Experimental animals had free access to drinking water and mineral licks. Animals were routinely dosed with Oxfendazole (Systamex®; Wellcome Foundation UK Ltd) against internal parasites and with Gamma benzene hexachloride (Gammatox®; Cooper, McDonald and Robertson UK Ltd) against external parasites.

Table 1. Mean composition of feed offered on dry matter (DM) basis.

Feed stuff	Dry matter (%)	Nitrogen (% in DM)	Ash (% in DM)
Dried Cassava Peels	70.37	0.73	11.59
Gliricidia sepium	31.94	3.69	9.88
Leucaena leucocephala	30.67	3.68	9.21

Digestibility trials

Two sets of digestibility trials (early and late lactation) were conducted during the study. During the trials, all animals were kept in twin-cabin wooden metabolism crates. Each trial lasted for 3 weeks, the first 2 weeks being an adaptation period. Data and samples of fresh faeces, feed offered and refused were collected and analyzed as described by Akinbamijo *et al.* (1992) during the 4th and 9th weeks of lactation. In addition, urine samples were collected during the second trial.

Milk sampling

From the 7th day of lactation, weekly milk yields of each ewe were estimated on one sampling day per week for 11 successive weeks. To achieve this, the 24-hour milk production was measured in each ewe as follows: At 18.00 h on the day preceeding the sampling day, udders were evacuated and all lambs were denied access to their dams during the following 24 h. On the sampling day, the milk yield was then estimated in each ewe by adding the weights of milk withdrawn during two sessions (08.00 and 18.00 h) of oxytocin enhanced hand-milking (see Owen 1957; Coombe *et al.* 1960). Each milking session lasted for 15 min or until the udders were empty. Weekly milk yields were then extrapolated from the sampling day's yield multiplied by seven for each ewe. About 80 ml aliquot of the milk withdrawn at each session was preserved with 0.16 g $K_2Cr_2O_7$ in a vial and stored at -20°C until required for chemical analysis. On sampling days, lambs were hand-fed with milk withdrawn from their dams. On other days, the lambs were allowed to suckle at will. Milk yield and composition data reported and discussed in this experiment were based on the following assumptions.

(1) The technique of milk yield estimation employed does not influence the rate of milk production while it is being applied.

(2) The rate of production of milk during the period of observation is the same as for other periods to which the estimate was referred.

(3) The udder is emptied to the same degree at the beginning and end of the sampling period.

Milk samples were analyzed for fat by the Gerber method (BSI 1955). Nitrogen, ash and total milk solids were determined by the methods of the Association of Official Analytical Chemists (1975).

Statistical analysis

Feed intake data was subjected to the analysis of variance. The milk production data was analysed by the analysis of variance in the early (0-6 weeks post-partum), late (7-12 weeks post-partum) and overall (0-12 weeks post-partum) lactation periods. Average daily gain was calculated by linear regression of weight on time for periods 0 - 6, 6 - 12, and 0 - 12 weeks post-partum in each animal. The estimated rates were then subjected to the analysis of variance on group basis.

RESULTS

From day 10 post-infection, parasites were present in the buffy coat until the 5th week of infection after which there were irregular appearances. Inoculated animals with high antibody titres remained infected throughout the trial. PCV declined in infected dams within 3 weeks from 32.5 to 22% and they remained febrile through the study.

Feed intake and digestibility

While intake parameters differed significantly between groups (Table 2), the digestibility coefficients of dry and organic matter did not differ (Table 3). Compared with CHP, the IHP ewes had significantly depressed ($P<0.05$) intakes of dry matter, OM, nitrogen and digestible organic matter (DOM) during early and late lactation. Feed intake of CMP ewes was lower ($P<0.01$) than those of IHP and CHP dams.

Nitrogen balance measured in the late lactation phase was significantly lower ($P<0.01$) in IHP ewes compared with CHP ewes (Table 3).

Table 2. Effect of *T. vivax* infection on dry matter intake (DMI), Organic matter intake (OMI), Digestible organimc matter intake (DOMI) and Nitrogen (N) intake of infected (IHP), control medium plane diet (CMP) and control high plane diet (CHP) WAD sheep during early and late lactation (s.e.'s in brackets).

Group	n	DMI ($g/W^{0.75}$)	OMI ($g/W^{0.75}$)	DOMI ($g/W^{0.75}$)	N ($g/W^{0.75}$)
Early Lactation					
IHP	10	81.6 (3.36)	74.7 (3.06)	47.2 (2.43)	2.53 (0.120)
CMP	5	61.5 (4.76)	55.7 (4.32)	33.9 (3.44)	1.81 (0.169)
CHP	5	96.0 (4.76)	87.9 (4.32)	54.0 (3.44)	3.01 (0.169)
Late Lactation					
IHP	10	76.4 (2.54)	69.1 (2.37)	41.8 (2.01)	2.36 (0.101)
CMP	5	63.8 (3.59)	57.5 (3.36)	35.7 (2.85)	1.91 (0.141)
CHP	5	89.6 (3.59)	81.2 (3.36)	49.5 (2.85)	2.83 (0.141)

Table 3. Mean Dry matter digestibility (DMD), organic matter digestibility (OMD) nitrogen digestibility (ND) and nitrogen balance (NB) of infected (IHP), control medium plane diet (CMP) and control high plane (CHP) in WAD ewes during early and late lactation (s.e.'s in brackets).

Group	n	DMD%	OMD%	ND%	NB$g/W^{0.75}$
Early Lactation					
IHP	10	56.9 (1.35)	63.0 (1.16)	54.5 (2.16)	-
CMP	5	54.1 (1.91)	60.8 (1.64)	54.1 (3.06)	-
CHP	5	54.8 (1.91)	61.3 (1.64)	56.4 (3.06)	-
Late Lactation					
IHP	10	52.6 (1.73)	60.3 (1.29)	42.8 (2.88)	-0.22 (0.069)
CMP	5	54.2 (2.44)	62.0 (1.82)	49.4 (4.07)	-0.06 (0.098)
CHP	5	53.2 (2.44)	60.8 (1.82)	43.8 (4.07)	0.06 (0.098)

Lamb and dam live weight changes

All lambs were born single, alive and healthy. There was no lamb mortality during the experimental period. Birth weight and total body mass accretion gained during early, late and overall lactation phases did not differ between groups (Table 4).

Table 4. Birth weight and Post-natal body mass accretion (kg) of lambs nursed by *T. vivax* infected (IHP) and control WAD ewes offered medium plane diet (CMP) and high plane diet (CHP) (s.e.'s in brackets).

		Period			
Group	n	Birth Weight (kg)	0-6 Weeks (kg)	6-12 Weeks (kg)	0-12 Weeks (kg)
IHP	10	2.13 (0.094)	3.02 (0.209)	2.00 (0.196)	5.02 (0.281)
CMP	5	1.94 (0.134)	3.06 (0.295)	1.90 (0.277)	4.96 (0.398)
CHP	5	1.96 (0.134)	3.34 (0.295)	1.60 (0.277)	4.94 (0.398)

Average daily gains for the dams at early, late and entire lactation period were significantly lower ($P<0.01$) in the IHP group compared with groups CMP and CHP dams (Table 5).

Table 5. Live weight changes of *T. vivax* infected (IHP) and control WAD ewes offered medium plane diet (CMP) and high plane diet (CHP) (s.e.'s in brackets).

		Period		
Group	n	0-6 Weeks (g/day)	6-12 Weeks (g/day)	0-12 Weeks (g/day)
IHP	10	-37.2 (10.82)	-52.0 (6.29)	-45.0 (5.66)
CMP	5	-7.3 (15.30)	6.2 (8.90)	-5.4 (8.00)
CHP	5	3.86 (15.30)	12.3 (8.90)	12.08 (8.00)

Milk Yield and Composition

In general, daily milk production tended to decline after week 3. There was no significant difference in daily milk yield between groups during the first 6 weeks of lactation (Table 6). During late lactation, infected ewes produced less milk (P<0.05) than their uninfected counterparts.

Table 6: Mean daily milk production of *T. vivax* infected (IHP) and control WAD ewes offered medium plane diet (CMP) and high plane diet (CHP) (s.e.'s in brackets).

		Periods		
Group	n	1 - 6 Weeks (g/day)	7-12 Weeks (g/day)	1-12 Weeks (g/day)
IHP	10	426.4 (12.93)	182.0 (9.27)	295.1 (12.48)
CMP	5	418.5 (18.29)	238.0 (13.12)	318.6 (17.65)
CHP	5	399.4 (18.29)	240.1 (13.12)	312.5 (17.65)

Their was no difference in the concentration of milk components between treatment groups and between stages of lactation. The overall (pooled) mean values of milk composition for all groups (g/100 g milk) were 0.94 (se 0.008), 6.39 (se 0.082), 5.56 (se 0.051) and 16.99 (se 0.137) for ash, fat, protein and total solids respectively.

DISCUSSION

The presence of trypanosomes in the infected dams coupled with a decline in PCV and development of fever reported in this study are salient features of trypanosomiasis (Akinbamijo, *et al.* 1990). Two feeding levels (MP and HP) were introduced to simulate the occurrence of large differences in DMI typical of *T. vivax* infected animals (Zwart *et al.* 1991; Akinbamijo *et al.* 1992). This was intended to allow for a fair comparison between infected and control animals without a confounding effect of feed intake. However, because the intake of the CMP ewes was significantly lower than that of the IHP group, comparison of these two groups was no longer justified. Hence, unless otherwise indicated, further reference to the control group will signify the CHP ewes.

In the course of this study, OMI was high even though the infection effect was still reflected in the lower intake observed in infected dams. Lactation is generally associated with an increase in feed intake (Blaxter 1989). It is not unusual therefore, that infected lactating dams consumed more than 1.8 maintenance requirement of DOM per day assuming 26 g DOM/$W^{0.75}$ (ARC 1980). Although the precise factors responsible for trypanosomiasis - induced anorexia remain unclear, the observation that infection depressed intake agrees with our hypothesis.

In infected ewes, there was a considerable nutrient drain due to increased maintenance requirements (Verstegen *et al.* 1991). With the comparable milk production at early lactation in all groups and lower OMI in infected ewes, it is hence logical to expect that body reserves were mobilized to sustain milk production (Gibb & Treacher 1982).

The feed intake, nitrogen balance and liveweight gain data further strengthens the belief that the infected ewes catabolized body reserves to meet increased maintenance requirements of infected animals (Stephen 1989; Verstegen *et al.* 1991; Zwart *et al.* 1991) and lactation. This supports previous views (Kearl 1982) that infection diverts energy that otherwise would serve productive purpose to service the increased maintenance requirements. That the uninfected ewes did not lose weight as much as IHP ewes during lactation could be explained in part by the good body condition of the dam at parturition (Spedding 1962) and good nutrition before and after lambing. In contrast however, despite good nutrition during gestation and lactation, febrile infected ewes still lost weight. This may be an indication of increased maintenance requirement mentioned earlier (Verstegen *et al.* 1991). The changes in live weight pattern support the concept that body reserves were utilized at different rates to sustain lactation. On the average, only the CHP ewes gained weight through the entire lactation reflecting a more efficient utilization of good quality diet.

The absence of an infection effect on milk yield and composition during early lactation implies that neither OMI nor level of infection used in this experiment would affect the lactation potential of WAD sheep. The good pregnancy nutrition and management may have offset infection effects during early lactation agreeing with previous reports by Peart (1967) and Adu (1975). The milk composition of ewes in this experiment was similar to that reported by other workers on non-dairy sheep breeds (Adu *et al.* 1974; Mba *et al.* 1975; Economides 1986) and this supports the findings of Treacher (1970) who demonstrated the importance of late pregnancy nutrition on milk yield and composition.

It has been shown that the factors largely responsible for the growth rate and weaning weight of suckling lambs are lamb birth weight, milk intake and dam's milk production (Robinson *et al.* 1969; Adu 1975; Economides 1986). The birth weights observed in this study are typical of WAD sheep (Adu *et al.* 1974; Orji & Steinbach 1980). Since lambs had similar birth weights and ewes had similar milk production during early lactation, it is therefore expected that liveweight gain by lambs would not be affected by trypanosomiasis in the dams.

Early lactation a period of maximum dependency of the lambs on their dams for nutrients. Since it has been reported that considerable amounts of residual milk are left in dams after suckling (Aboul-Naga *et al.* 1981), it can be deduced that milk yield during the first half of lactation was not limiting to lamb growth.

In conclusion, although trypanosomiasis in lactating dams although depressed feed intake, the milk yield and composition were not affected by the infection. It was also inferred that the infected ewes lost body weight and hence retained less nitrogen during the late lactation to augment cost of infection and milk production.

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Chapter 6

EFFECT OF EXPERIMENTAL FASCIOLIASIS ON FEED INTAKE, NITROGEN RETENTION AND BODY WEIGHT CHANGE IN PREGNANT AND NON-PREGNANT MENZ SHEEP

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ABSTRACT

The effect of a low level fascioliasis infection on feed intake, organic matter digestibility (OMD), nitrogen retention (NR), rate of live weight changes and lamb birth weight was studied in 23 open and 17 pregnant Menz ewes. The infection was imposed by oral dosing with 500 metacercariae of *Fasciola hepatica* in pregnant ewes at six weeks post breeding and in open ewes. All animals were housed in concrete floored pens and assigned to four treatment groups: pregnant infected (PI, n=9), pregnant non-infected (PC, n=8), open infected (OI, n=11) and open non infected (OC, n=12). The infected animals became anaemic from week 8 post infection (p.i.) and *F. hepatica* eggs were first observed in the faeces from the 11th week p.i. During two nitrogen retention trials (at the 7th and 13th weeks p.i.) organic matter intake (OMI) and digestibility coefficients were not affected by fascioliasis. All parasitized ewes voided higher urinary nitrogen resulting in lower nitrogen retention during the acute infection. Infected ewes had lower weight gains than control ewes. Lambs from infected dams weighed less at birth than lambs from control dams ($P<0.05$). It was concluded that under the conditions of the present experiment, although fascioliasis did not affect feed intake or digestibility coefficients, it did reduce the rates of live weight changes. Infected ewes retained less nitrogen, produced lighter lambs, but there was no corresponding reduction in feed intake.

INTRODUCTION

Fascioliasis is a major cause of reduced weight gain, condemnation of organs and carcasses, reduction in animal products and livestock mortality (Roseby, 1970; Dargie, 1980; Hall, 1985). The cause of impaired productivity during ovine fascioliasis is thought to be related to three factors acting singly or in concert. These include the reduced feed intake, poor feed utilization and excessive losses of blood constituents to the gut via the bile (Sykes and Coop, 1976, 1977; Dargie *et al* 1979). The variation in the onset and severity of these disturbances has been attributed to differences in fluke burden, since the number of parasites present is the most important factor (Berry and Dargie, 1976; Hawkins and Morris, 1978).

With the increasing recognition of the role of parasitism on nutrient metabolism, more work is now being focused on the nutritional physiology of the parasitized animal in an attempt to explain the negative effects of parasitism (Berry and Dargie, 1976; Hawkins and Morris, 1978; Dargie *et al* 1979; Symons, 1985).

This experiment investigates the effect of a low to medium (sub-clinical) fascioliasis infection on feed intake, digestive capacity, nitrogen retention and live weight changes in pregnant and open Menz sheep. The foetal survival and lamb birth weight was also studied in infected and control groups.

MATERIALS AND METHODS

The study was conducted at the Debre Birhan Research Station of the International Livestock Centre for Africa, Ethiopia in 1993. The meteorological and geographical characteristics of the experimental site have been described by Njau and Scholtens (1991).

Experimental Design

Animals

Forty adult cycling Menz ewes with a mean live weight of 23.9 kg (range 18.2 - 28.6, SD=2.61) were selected from a flock of on-station reared sheep of the Menz breed. All animals used had been treated with Fascinex during a fascioliasis outbreak 12 months earlier. The animals had been treated quarterly with Fascinex (Triclabendazole, Ciba Animal Health, UK; at the dose of 12mg/kg body weight) during the last twelve months before the experiment.

To provide uniformity in weight and conformation, all the ewes were assigned to 10 blocks of four animals each, on the basis of live weight. In each block, one pair was allotted at random to the 'Infected' group and the other pair to the 'Control' group. Thereafter, these two main groups were randomly sub-divided within blocks into 'Pregnant' and 'Open' ewes. This grouping resulted in pregnant infected (PI), pregnant non-infected (PC), open infected (OI) and open non-infected (OC).

All animals were drenched with fenbendazole against nematodes and triclabendazole against trematodes and vaccinated against pasteurellosis and clostridial infection at the beginning of the study. Animals in groups PI and PC were oestrus synchronized with prostaglandin (Dinoprost thromethanine Lutalyse) and bred instantly after the commencement of oestrus.

Feeding

All ewes were stall-fed and were offered *ad libitum* hay consisting of *Andropogon, Festuca* and *Pennisetum* species (5.4% CP). In addition, each animal received 300 g/d (fresh weight) of supplement consisting of 60% wheat bran + 40% *Guazotica abyssinica* (23.6% CP). Water and salt lick were always available. During the experiment, all animals were fed at 09.00 h daily. The supplementary feed was offered first and basal diet was offered 60 - 90 mins later.

Housing

All animals were housed in pens with concrete floors throughout the experiment, except for two digestibility and nitrogen retention trials, when they were housed in wooden cages. One week before expected lambing date, pregnant ewes were moved to a lambing barn to protect the lambs from inclement weather.

Live body weight and body condition scoring

Live weight changes were assessed by weighing at the beginning of the experiment and at weekly intervals thereafter. Weighing was done after an 18-hour abstinence from food, to avoid variation in gut fill. Body condition scoring, based on Russel (1984) and performed independently by two well trained technicians, was used to assess loss of condition in all animals. Lamb birth weight was measured within 12 hours of birth or as soon as the dam had finished drying the lamb.

Packed cell volume (PCV)

PCV was measured by the capillary micro-haematocrit method, which involved withdrawing blood from the jugular vein into heparinized bottles (Vacutainer, Becton Dickson) at the beginning of the experiment and weekly thereafter.

Parasitological techniques

Laboratory-reared *F. hepatica* metacercariae (Baldwin Aquatics, Calif., USA) were used in the experiment. The metacercariae were kept on moistened filter paper at 4°C before use. At the start of the experiment, animals in groups PI and OI were inoculated orally with 500 metacercariae of *F. hepatica* enclosed in single gelatin capsules.

As from two weeks pre-infection, faecal samples were collected per rectum and analysed for faecal egg count (FEC) every week using a modified McMaster Technique. Zinc sulphate (sp. gravity 1.35) $ZnSO_4$ was used as the floatation fluid. All infected ewes were treated with flukicide after parturition.

Routine on-station herd health and management operations, including weekly clinical checks were conducted.

Digestibility and Nitrogen retention (NRET) procedure

Two digestibility and nitrogen retention studies (Trials 1 and 2) were conducted on all animals during the 7th and 13th weeks post infection respectively. Orts, faeces and urine voided were collected for seven consecutive days after a 14-day adaptation period. Feed refusals were weighed just before fresh feed was offered. Urine was collected in a plastic bowl containing 10 ml of 50% hydrochloric acid.

Urine, feed, orts and faecal samples were collected per period. The samples were frozen (in the form they were collected or offered) until required for analysis. Samples of feed offered and refusals and fresh faeces were analysed for dry matter (DM), ash and nitrogen while urine was analysed for nitrogen only. The procedures of the AOAC (1975) were followed.

Statistical analysis

Comparisons between groups were made for live weight data by fitting linear regression of the form $y = a + bx$ to the sequence of measurements for each animal. Inter-group differences in the gradients were then evaluated using analysis of variance.

The feed intake and NRET data for each group were analysed per period, with infection, pregnancy and interaction as factors in the model:

$$Y_{ijk} = \mu + \alpha_i + \beta_j + (\alpha * \beta)_{ij} + \epsilon_{ijk}$$

where Y_{ijk} = kth measurement on jth physiological status in the ith treatment effect; μ = population mean; α = ith treatment effect where i = infected or non-infected; β = jth physiological status where j = open or pregnant; ϵ = error term associated with Y_{ijk}. Where no significant differences were detected using the model including the interaction term, the model was reapplied without the interaction term.

Student's t-test was used to analyse the effect of pregnancy on faecal egg count and lamb birth weight of dams of infected and control groups. All means and estimates are given ± standard error.

RESULTS

As a result of non-conception, one infected ewe and two uninfected ewes were transferred from groups PI and PC to groups OI and OC, respectively. Pregnancy and infection were confirmed respectively by non-return to oestrus for bred ewes and the presence of eggs in the faeces of infected ewes as from 11th week p.i. The infected ewes did not show overt symptoms of a disease condition and there were no deaths, abortions or still births. No deviation from normal state of health was detected in the control sheep.

The sequential changes in PCV of pregnant and open ewes are presented in figure 1. Parasitized ewes indicated a significantly lower PCV from weeks 8 till 11 p.i. compared with non-parasitized ewes. Towards the end of the experiment, the infected ewes showed an upturn trend in the PCV pattern.

Faecal egg count (FEC)

F. hepatica eggs were not observed in faeces from non-infected ewes during the experiment nor in infected ewes before week 11 p.i. FEC conducted from week 11 through 14 p.i. were higher ($P<0.05$) in the pregnant than in the non-pregnant ewes, and ranged from 60 eggs to 170 eggs per gram. Mean FEC for both groups PI and OI is presented in figure 2.

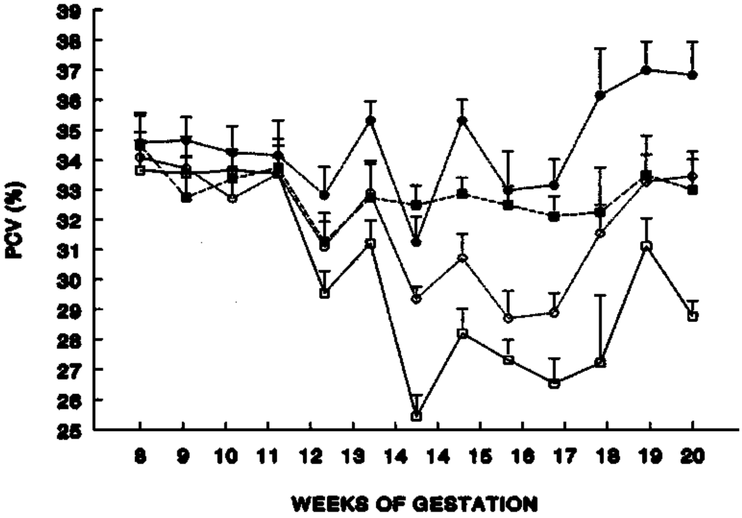

Figure 1 Mean (±SE) sequential changes in packed cell volume of *F. hepatica* pregnant infected (group PI □), pregnant non-infected (group PC ■), open infected (group OI ○) and open uninfected (group OC ●) Menz sheep.

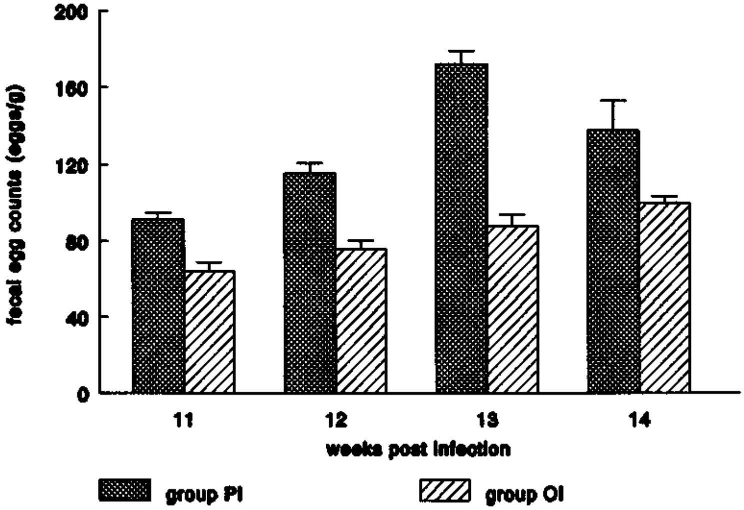

Figure 2 Mean (±SE) faecal egg count for pregnant (group PI) and open (group OI) sheep.

Food intake and NRET

Trial 1 (7 weeks p.i.)

Food intake, digestibility and NRET data at 7 weeks p.i. for all groups are presented in Table 1. Organic matter intake (OMI), Digestible organic matter intake (DOMI) and organic matter digestibility (OMD) did not differ across groups. The proportion of the supplement as percentage of dry matter intake (DMI) consumed did not differ (Table 1). Consequently, it can be inferred that all groups consumed a similar diet.

Table 1: Mean (±SE) estimates of intake, digestibility and Nitrogen retention (NRET) of experimental ewes (Trial 1).

	GROUPS			
VARIABLES	PI	PC	OI	OC
n	9	8	11	12
Metabolic size	11.0 ±0.30	11.3±0.31	10.6±0.27	10.7±0.26
Organic matter intake (g/kg$^{0.75}$)	64.1±1.58	65.7±1.68	64.2±1.43	63.7±1.37
Digestible organic matter intake (g/kg$^{0.75}$)	27.8±1.13	29.5±1.20	30.0±1.02	29.2±0.98
Nitrogen (g/kg$^{0.75}$)	1.02±0.34	1.02±0.37	1.01±0.31	1.01±0.30
Concentrate intake (%DMI)	34.2±1.41	32.2±1.50	35.7±1.28	35.9±1.22
Org. matter digestibility(%)	43.3±1.23	45.0±1.30	46.5±1.11	46.0±1.07
Urinary Nitrogen (g/kg$^{0.75}$)	0.32^{a}±0.27	0.25^{b}±0.29	0.32^{a}±0.24	0.27^{b}±0.23
Faecal Nitrogen (g/kg$^{0.75}$)	0.65±0.22	0.67±0.23	0.69±0.20	0.68±0.19
NRET (g/kg$^{0.75}$)	0.04^{a}±0.013	0.10^{bc}±0.014	0.00^{ab}±0.012	0.06^{a}±0.012

PI = infected pregnant,
PC = pregnant non-infected,
OI = infected open
OC = open uninfected
Means within rows with different superscripts differ ($P<0.05$)

Infected ewes voided more urinary nitrogen than did control ewes. The difference in urinary nitrogen resulted in the lower ($P<0.05$) nitrogen retention in infected ewes. A regression analysis revealed that the relationship between NRET and DOMI showed that there was no significant group effect on either the regression coefficient or the intercept, and therefore the group efect was removed from the model. Further analysis produced a single regression equation describing the relationship between NRET and DOMI using the pooled data from all experimental animals:

$$\mathrm{NRET} = -0.181\ (\pm\ 0.062) + 0.008(\pm\ 0.0021)\mathrm{DOMI}\quad r^2=52\% \qquad (1)$$

Trial 2 (13 weeks p.i.)

Data on feed intake and nitrogen retention at thirteen weeks p.i. are presented in Table 2. Although pregnant ewes consumed slightly more ($P<0.05$) OMI than open ewes there was no statistically significant difference in the feed intake between infection and control ewes. Organic matter digestibility (OMD) and faecal nitrogen did not differ for all groups (Table 2). There was no statistically significant difference in the NRET of infected and non-infected pregnant animals. Open infected ewes, however, retained less ($P<0.001$) nitrogen when compared with pregnant ewes.

The relationship between NRET and DOMI for pooled data from all animals in trial 2 is described by the equation:

$$\mathrm{NRET} = -0.296\ (s\ 0.0554) + 0.012(s\ 0.002)\mathrm{DOMI}\quad r^2=89\% \qquad (2)$$

Table 2: Mean (±SE) estimates of intake, digestibility and Nitrogen retention (NRET) of experimental ewes (Trial 2).

	GROUPS			
VARIABLES	PI	PC	OI	OC
n	9	8	11	12
Metabolic size	11.2±0.37	11.9±0.40	11.2±0.34	10.9±0.32
Organic matter intake (g/kg$^{0.75}$)	$66.8^{a}\pm1.87$	$65.6^{a}\pm1.98$	$60.0^{b}\pm1.69$	$62.8^{b}\pm1.62$
Digestible Organic Matter intake (g/kg$^{0.75}$)	29.1±1.46	29.3±1.56	29.4±1.33	31.1±1.27
Nitrogen intake (g/kg$^{0.75}$)	$1.0^{a}\pm0.33$	$1.0^{a}\pm0.36$	$0.9^{b}\pm0.30$	$0.9^{b}\pm0.30$
Concentrate intake (%DMI)	33.0±1.44	34.2±1.53	35.9±1.31	36.4±1.25
Org. matter digestibility (%)	44.5±1.34	46.6±1.44	48.7±1.20	49.6±1.14
Urinary Nitrogen (g/kg$^{0.75}$)	0.24±0.17	0.23±0.018	0.26±0.015	0.23±0.015
Faecal Nitrogen (g/kg$^{0.75}$)	0.69±0.023	0.68±0.024	0.62±0.021	0.64±0.020
NRET (g/kg$^{0.75}$)	$0.07^{ab}\pm0.021$	$0.10^{a}\pm0.022$	$0.00^{b}\pm0.019$	$0.07^{ab}\pm0.018$

PI = infected pregnant,
PC = pregnant non-infected,
OI = infected open,
OC = open uninfected
Means within rows with different superscripts differ ($P<0.05$)

Live weight changes, body condition and lamb birth weight

The linear regression analysis of rate of live weight change per unit time showed that infection influenced the regression coefficient. Estimated rates of gain were 24.3, 44.3, 14.3 and 17.1 (r^2=92%; RSD = 0.011) g/d for PI, PC, OI and OC groups respectively. The difference in rate of live weight change between open and bred ewes was statistically significant. In general, PC ewes gained more weight than PI ewes but the difference between OI and OC did not attain statistical significance.

At 7 weeks p.i. body condition score in infected animals was lower ($P<0.05$) than that of the control ewes (3.00 versus 3.25). As the pregnancy advanced, the body condition of infected ewes deteriorated. At 13 weeks p.i., the mean body condition score was significantly lower ($P<0.05$) in infected animals than in control animals (2.5 versus 3.25). Infected pregnant ewes also had lower live weights prior to lambing and the birth weights of their lambs were were also affected by fascioliasis.

The birth weights of twins and single lambs observed in this study were compared between groups PI and PC. No significant difference was found between the birth weights of twin lambs because of the small number of animals involved. Mean birth weights differed in the single lambs: 1.63 kg for PI versus 1.91 kg for PC (se = 0.077; n = 7).

DISCUSSION

The number of metacercariae administered in this study was expected to produce a low to medium level infection in the Menz sheep (Hawkins and Morris, 1978). Hence the limited effects of fascioliasis observed in this study were probably due in part to the low dose of single challenge infection. The effect of an undetermined level of pre-infection immunity and the genetic resistance of the Menz sheep cannot be excluded (Boyce *et al.*, 1987; Wiedosari and Copeman, 1990). The delay in appearance of the eggs in the faeces is a function of host susceptibility and intensity of infection (Wiedosari and Copeman 1990). Therefore, the low levels of the FEC and the short excretion time may be due to factors mentioned above. The difference in FEC between groups PI and OI is an indication of the peri-parturient egg rise usually seen during pregnancy (Courtney *et al* 1984).

Generally, helminthic infections tend to reduce the feed intake of the host (Dargie *et al* 1979 and Sykes *et al* 1980). In the present study, voluntary feed intake was not affected by the infection but there was an increase in urinary N losses in trial 1 in the infected group. This resulted in the infected sheep retaining less nitrogen without a concomitant reduction in feed intake and digestibility. Comparing the feed intake data in both trials, it is clear that although a sub-clinical infection developed, its effects on intake, digestive capacity, urinary nitrogen and NRET at the acute and chronic phase (open ewes) and during mid and late pregnancy (pregnant ewes) were limited.

With the low intake of digestible organic matter reported in this study, available DOMI in excess of maintenance requirement was, at best, 15-20%

maintenance requirements, assuming 26 g DOMI/kg$^{0.75}$/d (ARC, 1980) for maintenance.

Previous reports in the literature on digestibility during fascioliasis are conflicting. Hawkins and Morris (1978), Sykes *et al* (1980) and Sinclair (1975) found no effect of *F. hepatica* on the dry matter digestibility (DMD) in sheep carrying up to 400 flukes. In another study with higher level of infestation, significant differences were recorded in the DMD of calves infected with 5000 metacercariae (Canale *et al* 1973, cited by Sykes *et al*, 1980). In this study, no evidence was observed that the digestive capacity was influenced by the low to medium infection. The similarity of faecal nitrogen in the present study strengthens the notion that malabsorption and poor nitrogen digestibility are not involved in the low to medium infection.

The main effect of the infection was the reduction of the efficiency with which apparently digested N was retained. In infected ewes, nitrogen retention was significantly less than in control ewes, due to high excretion of urinary nitrogen (Trial 1). At the 13th week p.i., differences in nitrogen retention between PI and PC ewes were not significant but open infected ewes had lower ($P<0.05$) NRET than open control ewes. Roseby (1970) and Hawkins and Morris (1978) reported that in sheep infected with 500 metacercariae, (an infection dose similar to the one used in this experiment) N utilization was considerably depressed in the acute phase. These researchers found that the rate of live weight gains was also lower, with no corresponding reduction in feed intake, but they reported that loss of condition was less in their controls by visual comparison. This is consistent with our current findings.

There was no effect of infection on slope when NRET was regressed on DOMI ($P>0.05$). The slopes (from trials 1 and 2) are similar to those obtained in healthy and infected West African Dwarf sheep (WADS) (Akinbamijo *et al* 1994) and in goats (Ketelaars and Tolkamp, 1991). This suggests that despite attendant infection, sheep and goats will not easily deviate from the normal relationship between NRET and DOMI.

Considering the level of infection used in this study, the PCV trend is similar to that reported by Ogunrinade and Anosa (1981) in West African Dwarf sheep infected with *F. gigantica*. Reid *et al* (1970) and Sewell (1966), contend that the damage done by the young migrating flukes are healed rapidly and nitrogen is conserved, as indicated by the upturn in PCV pattern.

Although the infected sheep did not lose weight in absolute terms, they failed to match the rate of body weight gain and condition of their control uninfected counterparts. This may be related to the lower efficiency of feed utilization in infected animals.

The mean birth weight of lambs born to infected ewes was 15% lower than that of lambs from uninfected ewes. This difference strengthens the notion that animal productivity in terms of foetal development and birth weight was affected by fascioliasis. In conclusion, feed intake and digestibility coefficients are not affected by a low to medium level of *F.hepatica* infection. However, this subclinical infection still reduces significantly the efficiency of N utilization and productivity, by lowering weight gains, inducing loss of condition and lowering birth weights.

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SUMMARIZING DISCUSSION

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INTRODUCTION

The investigation of voluntary feed intake (VFI) and nitrogen retention (NRET) during parasitic infections in small ruminants is the central theme of this thesis. An attempt was made to examine the effects of trypanosomiasis on feed intake, digestibility, nitrogen retention and animal products. In addition, a similar investigation was conducted during a low to medium level fascioliasis infection in Menz sheep. The relationship between digestible organic matter intake (DOMI), rectal temperature (RT) and maintenance requirements was also studied in healthy goats.

The infected subjects manifested an aggregate of some distinct but not mutually exclusive responses. These include anorexia, fever, increased metabolic rate, lowered nitrogen retention and reduced animal productivity characterized by the host's poor growth rate and weight loss. Although the feed intake appears to be a major factor in the disease process, the observed derangements in productivity and physiological response to parasitic infection could not be fully explained by anorexia. Therefore, it seems that survival and the productive performance of infected animals depended on the adaptive measures they are able to employ to meet nitrogen and energy requirements during the periods of insufficient nutrient supply or inefficient feed utilization.

PARASITIC INFECTION AND FEED UTILIZATION

A depression in feed intake is a common feature in infected subjects, although the mechanisms involved remain unclear. Infection-induced anorexia is a major factor limiting the availability of energy for maintenance and production in parasitized hosts. This phenomenon has been widely reported in both human and animal subjects as one of the earliest signs of a patent infection (Ilemobade and Balogun, 1981; Symons, 1985; Keusch and Farthing, 1986; Holmes, 1987;

Akinbamijo, 1988; Verstegen *et al* 1991; Zwart *et al*, 1991). Anorexia is an important aspect of parasitic infections, but studies using pair-feeding techniques have demonstrated that infected animals also show reduced feed utilization and energy retention relative to their parasite-free counterparts on the same level of feed intake (Sykes and Coop, 1976; Coop *et al*, 1982).

The onset of infection is characterized by the acute phase response during which anorexia, wasting of body tissues and changes in metabolism of carbohydrates, lipids and proteins occur (Baracos *et al*, 1987). Fever, a major factor in energy metabolism, is a conventional occurrence in haemo-protozoan infections such as trypanosomiasis (Holmes, 1987; Akker, 1988; Zwart *et al*, 1991; Verstegen *et al*, 1991) and malaria (Cohen and Lambert, 1982). This rise in body temperature implies an elevation of heat production caused by increased metabolic rates. Lower nitrogen retention has been reported in infected human and animal subjects during most nutrition-infection interactions (Beisel *et al*, 1967; Dargie *et al*, 1979; Morris, 1988). The reduced nitrogen balance observed in parasitized hosts is not only a consequence of the reduced feed intake (Symons, 1985): fever and the related increases in tissue catabolism (Baracos *et al*, 1987), intestinal and renal protein losses (Ingh *et al*, 1976; Holmes, 1987), high urea and total proteins concentrations in the serum (Finco, 1989) and high urinary nitrogen excretion (Roseby, 1977; Akinbamijo, 1988) have all been implicated during parasitism.

The increased catabolism of proteins during the acute infection is reflected in the increased urea production and nitrogen excretion often seen in infected animals (Finco 1989). These wasting events, culminating in lower/negative nitrogen retention reduce the productivity of infected animals.

During parasitic infections in which anorexia and fever occur concomitantly, lipolysis and ketogenesis are common features (Keusch, 1984). After the glycogen reserve has been exhausted, during the first few hours of anorexia, the fat depot is used as an interim measure to meet the body's energy requirements. High concentrations of free fatty acids and ketone bodies in body fluids are an indication of lipolysis and partly oxidation of lipids. They are at a later phase accompanied by protein mobilization, reflected in increased urea in the urine.

The energy deficit during infection boosts the rate of energy needs. Hence, the changes in the composition of the serum often observed during infection-induced anorexia are a reflection of the subnormal caloric intake (Blackburn *et al*, 1991; Keusch, 1984; Bruss, 1989). These physiological changes occurring in concert, with complementary effects on each other, result in a serious negative energy balance, accompanied by a cumulative depletion of body energy stores, precipitating a lowered or negative energy and nitrogen balances (Clowes *et al*, 1976; Beisel, 1985; Keusch and Farthing, 1986; Baracos *et al*, 1987; Blaxter, 1989).

DIGESTIBILITY

In view of their pathogenesis, it is often assumed that endo-parasitism must be associated with changes in the gastro-intestinal function (Holmes, 1987). However, parasitic infections do not normally seem to affect the digestive and/or absorptive capacity of the host, even when infection rates are high (Parkins *et al*, 1973; Roseby, 1977; Reveron and Topps, 1970; Reveron *et al*, 1974). Similar findings have been reported in sheep infected with *Fasciola hepatica* (Hawkins and Morris, 1978) and in calves infected with lungworms (Verstegen *et al*, 1989). Holmes (1987) suggested that the digestion and absorption of amino-acids might decrease as a result of endogenous protein draining into the gut of parasitized animals, but this is not a usual event in helminthiasis. In general, it is not impaired digestion and absorption but rather the increased metabolic demands on the hosts that are the important causes of the low productivity of parasitized hosts (Baracos *et al*, 1987).

INFLUENCE OF PARASITISM ON PRODUCTION

The net effect of infection on productivity is mediated primarily via a reduction in the energy available for productive purposes. Thus, changes in live body weight are a common yardstick for judging the effects of parasitism on animal productivity. It is generally acknowledged that diseased animals lose or gain weight more slowly than their non-infected counterparts. The low weight gains and differences in the productive capacity in bovine (Schillhorn van Veen, 1974; Kroonen *et al*, 1986) and ovine (Roseby, 1970; Hawkins and Morris, 1978; Tekelye *et al*, 1992 a&b) infections are primarily due to reduced voluntary feed intake. In addition to changes in the body weight, alterations in body composition do occur and this can have major implications for productivity. It is well established that under nutrition can cause changes in body composition of healthy red deer (Wolkers, 1993). In sheep, parasitic infection is found to have the same effects (Sykes and Coop, 1976, 1977).

The shortfall in energy required for productivity has a direct quantitative effect on animal products during infection. It has been demonstrated that milk yield and composition are affected during infection with trichostrongylosis (Bliss and Todd, 1976; 1977) and fascioliasis (Randell and Bradley, 1980). Both the quality and the quantity of wool have also been reported to be affected by infection (Bliss and Todd, 1977; Barger and Gibbs, 1981; Leyva *et al*, 1982).

Reproductive disorders in male and female humans and animals caused by *Trypanosoma* infection are widely reported in the literature (Ikede *et al*, 1988; Akpavie *et al*, 1987). The effects of infection have been demonstrated on conception rates and pregnancy in cattle (Ogwu and Nuru, 1981; Ogwu *et al*, 1986; Oakley *et al*, 1979) and sheep (Reynolds and Ekwuruke, 1988) leading to a lower reproductive index[1] (RI) (Gabina, 1988).

Many of the major parasitic diseases such as trypanosomiasis, babesiosis, malaria and fascioliasis are characterized by anaemia. The decline in packed cell volume (PCV) during parasitic infections is often a confirmation that a disease process has set in. The aetiology of parasite-induced anaemia is possibly the oldest and widely recognised clinical sign of a patent parasitic infection (Soulsby, 1976).

OWN INVESTIGATIONS

The derangement in host metabolism is the summation of a multitude of processes which leads to quantitative changes in animal productivity. It consists of the totality of all the modifications that make the difference between health and disease. The effect of a reduction in feed intake in healthy animals is considered first.

At lower levels of food intake, healthy subjects are able to attain energy

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[1] RI = lambs produced per life time • [age in years - 1]$^{-1}$

balance partly by reducing energy expenditure and by mobilizing fat reserves for energy production indicating the use of non-protein energy sources to meet their energy requirements. As demonstrated (see Chapter 2), this was accomplished by the observed rapid fall in maintenance requirements accompanied by lowered body temperature during starvation or feed restriction. The physiological and/or behavioural strategies employed by energy-restricted subjects for survival are principally based on the reduction in energy expenditure. The findings presented in Chapter 3 indicate a positive correlation between body temperature and digestible organic matter intake (DOMI). This conserving process is a regular adaptive measure in healthy subjects during periods of caloric insufficiency. It concurs with the findings of Shetty (1990); Zwart *et al*, (1991) and Ketelaars and Tolkamp (1991). The implication is that feed-restricted animals utilize their nutrient resources more efficiently by reducing body temperature and maintenance requirements to a level proportionate to intake. Several such adaptive physiological responses tend to reduce the metabolic activity of tissues in order to improve the efficient use of resources. The animals are therefore able to maintain nitrogen and energy balance at intake levels reasonably lower than those of *ad libitum* fed animals (Chapter 2).

However, these findings are contrary to what has been reported as occuring during infection-induced fever, where metabolic rate is typically not proportionate to intake (Hayashi *et al*, 1985). Waterlow (1986) showed that the pattern of endogenous fuel substrate utilization differed significantly between healthy feed-restricted subjects and those with a restricted intake caused by an induced infection. Contrary to the depressed values found in *Trypanosoma vivax* infected goats, no deviation from normal levels was observed in the T3 and T4 concentrations in feed-restricted healthy goats (van Dam, Unpublished). This observation further strengthens the hypothesis that thyroid activity declines following infection with trypanosomiasis, suggesting that other mechanisms might be involved in the regulation of energy metabolism during infection.

TRYPANOSOMIASIS

As shown in Chapters 1, 2, 4, and 5 trypanosomiasis studied in different physiological stages of the West African Dwarf (WAD) goats and sheep produces varying responses to feed intake. On average, the infection-mediated anorexia observed in these experiments conforms with earlier findings observed during trypanosomiasis (Ilemobade and Balogun, 1981; Akinbamijo, 1988; Zwart, 1989). The decreased intake is mainly responsible for the negative nitrogen balance and reduced productivity.

No evidence of a haemorrhagic infection was found during trypanosomiasis in the research reported in this thesis. This observation, confirmed by absence of intestinal and kidney lesions at post-mortem examination, makes nitrogenous losses via intestines or kidneys unlikely (Ingh *et al*, 1976). In all the studies conducted, the digestive capacity was not affected by trypanosomiasis. The similar faecal nitrogen output obtained in infected and control animals nullifies the possibility of intestinal nitrogenous losses due to infection. The finding that there was no urinary protein agrees completely with post-mortem observations reported in Chapter 2 and no kidney lesions were found.

In the trypanosomiasis research reported in this thesis, the infection was always accompanied by fever. This is consistent with the fact that protozoal infections are often accompanied by fever which increases the energy expenditure of the infected host. This contention expressed by Baracos *et al*, (1987) and Cohen and Lambert (1982) was also supported by Verstegen *et al*, (1991) who attributed a 25% increase in the maintenance requirement of *Trypanosoma vivax* infected goats to fever. In the present research, all infected animals became febrile from the onset of infection and maintained the pyrexia throughout the infection. Fever, increased basal metabolic rate (BMR) and heat production have been identified as factors contributing to increased energy demands characteristic of infected subjects (Keusch and Farthing, 1986).

This thesis indicates that the metabolic costs accompanying the infection (Baracos *et al*, 1987; Verstegen *et al*, 1991) and anorexia during trypanosomiasis led to increased lipolysis in the host animal. An increased concentration of serum non-esterified fatty acids (NEFA) was observed in infected subjects, confirming that body fat reserves were being used to bridge the energy gap due to anorexia and increased maintenance requirements (Chapter 1). The observed increase in blood urea concentration indicates that a minimal breakdown of body proteins cannot be excluded. This agrees with the findings of Beisel (1985). No evidence was found of increased ketogenesis or excessive breakdown of body proteins, even when feed intake was as low as 40% of maintenance requirements. However, two cases in which increased concentration of serum ketone bodies following extreme anorexia was observed (Chapter 2) deserve mention. A similar metabolic response was described by Symons (1985) and Beisel (1985) during severe starvation.

During the post-infection phase, anaemic and anorexic pregnant ewes tended to reduce rate of weight gain and body condition. These derangements during trypanosomiasis have been attributed to a number of factors: a reduction in dry matter intake (Akinbamijo *et al*, 1990), an increased basal metabolic rate (Zwart *et al*, 1991; Stephen 1986), an increased catabolism of tissue reserves (Akinbamijo *et al*, 1992) a reduced nitrogen and energy balance (Zwart *et al*, 1991; Verstegen *et al*, 1991) and possibly an uptake of host's nutrient by the parasites (Reynolds, Personal Communications). In the acute phase response and the related wasting physiological events that follow, the increased metabolic rate has severe implications for livestock productivity, such as dam mortality, foetal and neo-natal losses. These three factors attained prominence in earlier reviews on the effect of trypanosomiasis on reproduction (Ogwu and Nuru, 1981; Ogwu *et al*, 1986; Ikede *et al*, 1988) and in the findings of Elhassan (1987) and Reynolds and Ekwuruke (1988) in WAD sheep.

In the present research, infected ewes ending pregnancy with low maternal weights and depressed intake, had lambs with lower birth weights that suffered considerable neonatal mortality. Consequently, 85% of lambs from ewes infected at late pregnancy died within seven days after birth. The relation in non-infected ewes, between lamb birth weight and growth rate and maternal effects of pregnancy nutrition and dam weight at parturition are widely reported (Gibb and Treacher, 1980 & 1982, Peart 1967, Treacher 1970, Adu and Olaloku, 1979). In the event of a severe nutritional stress, incidences of sporadic abortion have been reported (Osuagwuh and Akpokodje, 1986; Osuagwuh and Aire, 1990). Hence if dam and foetal nutrition is inadequate, it can induce results such as were obtained in infected ewes (Chapter 4). The maternal effects on the low lamb birth weights were reflected in the low weaning weights of the lambs from infected dams.

In spite of the infection, ewes infected after lambing had sufficient body reserves to meet the daily milk yield observed in all animals during early lactation. Lactation is often associated with an increase in feed intake (Blaxter, 1989), and this may have masked the effect of infection on intake and milk yield during early lactation in the infected dams (Chapter 5). In relation to the live weight pattern, it could be deduced that infected lactating sheep catabolized body reserves to supplement the dietary nutrients required for milk production as established by Gibb and Treacher (1982). Since milk yield did not differ during early lactation, the growth rate did not differ between lamb groups. The difference in milk yield observed during late lactation had no effect on lamb growth rate, as lambs had commenced the weaning process and were supplementing their milk intake with forage.

FASCIOLIASIS

As intended, the clinical observations of the low to medium level infection mimic those of the sub-clinical infection usually experienced by grazing stock. No evidence was found of changes in the feed intake and digestive capacity of the hosts post infection. This concurs with the finding of Hawkins and Morris (1978) obtained using graded levels of infection doses, that there is a negative relationship between fluke burden and digestibility coefficients. Considering the infection dose used in this thesis, the infection level was too low to initiate digestive disturbances. The costs of infection (inefficient feed utilization) were evident. This conforms with the reports of Berry and Dargie (1976) and Dargie *et al*, (1979).

The sub-clinical fascioliasis had no effect on the voluntary feed intake of open and pregnant Menz ewes. However, this finding conflicts with what occurs during parasitism, where anorexia is reported as one of the earliest symptoms (Berry and Dargie, 1976; Murray and Murray, 1979; Symons, 1985, Keusch and Farthing, 1986; Holmes, 1987; Morris 1988). The explanation of this finding can only be a conjecture: in our experiment, it is probably connected with factors such as breed and age of the host, the viability and number of metacercariae administered and the presence of an undetermined level of pre-infection immunity (Sinclair, 1971; Berry and Dargie, 1976; Leyva *et al*, 1982).

The digestive capacity of the ewes was found not to be affected by infection or pregnancy either. This conforms with most findings during parasitism (Dargie *et al*, 1979; Berry and Dargie, 1976) but contrasts with reports by Holmes (1987) who suggested the possibility of impaired digestive and absorptive processes especially due to loss of intestinal proteins into the gastro-intestinal tract during helminth infections. However, as already noted, in the present study, the similarity of the faecal nitrogen in infected and control animals invalidates the chances of such an occurrence.

Despite the low to medium level fascioliasis imposed on them, the infected Menz ewes indicated a decline in PCV about eight weeks post-infection. This is similar to the observations of Sinclair (1971) and Dow *et al* (1968). The decline in PCV post infection substantiated the presence of an infection effect in inoculated ewes.

At comparable levels of voluntary feed intake, infected ewes retained less nitrogen, gained less weight, and produced lighter lambs and had poorer body condition (Chapter 6). This is probably because of differences in the efficiency of utilization or conversion of feed into desired animal products. The difference in growth rate between infected and healthy goats (Chapter 1) illustrates the nutrient drain that accompanies the infection. Inefficient feed utilization and wasting of this type during parasitic infections is also widely reported in the literature quoted above. The nitrogen retention differed remarkably in the early part of the infection and gradually reverted as the infection progressed. An upturn in PCV observed towards the end of the study may also be related to the acquired resistance phenomenon suggested earlier by Sinclair (1971). The reduced nitrogen retention reported by Dargie *et al* (1979) at eight weeks post-infection, was principally due to high urinary nitrogen. In our study, the findings with respect to the time schedule, the lower nitrogen retention and high urinary nitrogen observed in the infected ewes, are similar to those reported by Dargie and his co-workers (1979). The observed similarity in feed intake and digestibility in control and infected ewes indicate that other insidious losses must have had a major effect on the overall nitrogen retention and lower rate of weight gain in infected ewes. This is largely borne out by the nitrogen balance and body weight data that put the infected sheep on the lower limit. Although our study provides no clues about the extra nitrogen excreted by infected ewes, its appearance in the urine rather than in faeces confirms the contention that the host's digestive and absorptive capacity was not impaired during the low to medium fascioliasis.

Live weight gain in the animals was also considerably affected in the infected animals. It has been reported that sheep infected with *Fasciola hepatica* failed to maintain the rate of body weight gain observed in uninfected counterparts (Reid *et al*, 1970; Holmes, 1987; Blackburn, 1991). Lamb birth weight was lower in infected ewes than the control ewes. The low nitrogen retention found in infected pregnant ewes strengthens the contention that there was less nitrogen accretion in the foetus. Such deleterious effects of parasitism on the productive potential and the efficient use of resources have been demonstrated earlier (Ogwu *et al*, 1986;Ikede *et al*, 1988; Reynolds and Ekwuruke, 1988; Akinbamijo *et al*, 1994).

IMPLICATIONS FOR ANIMAL PRODUCTIVITY

In the studies with *Trypanosoma* and *Fasciola spp.* reported in this thesis, absence of digestive disturbances was clear but in neither case did infected animals utilize their food as well as non-parasitized controls. The onset of infection coincided with reduced nitrogen retention resulting from a combined effect of reduced voluntary feed intake and/or increased excretion of urinary nitrogen. As productivity is governed not only by the gross intake but also by the efficiency of conversion into desired products, the evidence obtained in this research suggests that the reduced productivity of parasitized animals is the direct result of infection depressing the utilization of feed intake.

In quantitative terms, less digested nitrogen was retained in the tissues or products of the infected animals. This is typified by the findings reported in the first chapter, where it was shown that trypanosomiasis was responsible for the low growth rate. No carcass analyses were conducted in this research, but other researchers have reported changes in body composition during undernutrition (Wolkers, 1993) and infection (Sykes and Coop, 1976). Both undernutrition and infection were observed in the present research (see Chapters 1 and 2).

Reproductive wastage generally traceable to infections is considered to be substantial during infection (Osuagwuh and Aire, 1990; Osuagwuh and Akpokoje, 1986). It is usually characterized by embryonic or foetal death, abortion, premature birth, still birth, birth of weak offspring and neonatal deaths (see Chapter 4). It seems rational that if the dam is in good body condition pre-partum, then the offspring will have a good chance of surviving. However, this is often not the case during parasitism. Depending on the severity of infection, if the nutritional stress becomes extreme, most or all of the conditions mentioned above will occur, and reproductive wastage will result. The degree of reproductive wastage is therefore related to the level of dam nutrition and parasite load. Considering the findings of Ikede and Losos, (1972) in sheep and Ogwu *et al*, (1986) in cattle, the possibility of intra-uterine infection cannot be ruled out in this study.

At sub-clinical levels, infection resulted in an appreciable degree of poor nutrient utilization that may have serious consequences on productivity (Chapters 4 and 6). Infection of pregnant ewes was characterized by low nitrogen retention, poor weight gains, and poor body condition and foetal development culminating in low lamb birth weight. Even when the dam was treated post-partum, the carry-over effects from gestation were reflected in the lamb performance.

There were no profound effects of infection on lamb growth rates, but infected dams lost more weight at lactation and retained less nitrogen. In the context of livestock health and productivity, parasitism lowers the production potential or offtake of the infected animals. As observed from this thesis, elements of the productivity and reproductive indices mainly growth rate (or weight gain), ewe mortality, abortion rate/foetal loss, birth weight, weaning weight, neonatal loss, nitrogen retention and body condition, and milk yield were appreciably affected by parasitism. Others which should have been included are weight of offspring weaned per dam, the number of lambs produced and number of lambs born per 100 ewes.

Concurrent with findings of Ogwu *et al* (1986) in cattle, Elhassan, (1987) and Reynolds and Ekwuruke (1988) in sheep, abortion occurred in infected ewes during the third trimester. The effect of chronic clinical or sub-clinical trypanosomiasis on reproduction and fertility such as anoestrus, failure to conceive, poor libido have been reported by the authors cited above. These disorders have a direct bearing on the productivity index[2] (PI) described by Bosman *et al* (1988) and the reproductive

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[2] PI = Kg weaned lambs/lambing dams/year

index (RI) described by Burfening *et al* (1993). According to the indices of productivity described by these workers, the incidence of parasitic diseases in small ruminant husbandry has a multiplier effect on productivity as the two indices (PI and RI) are direct determinants of prolificacy[3] (Gabina, 1988). This implies that the factors affected by parasitism are the determinants of productivity and hence are responsible for the reduction in the expression of the genetic potential and the optimum offtake per livestock unit. As shown by this thesis, the consequences of infection do not stop at morbidity alone but also flow through to incidence of mortality (Anene *et al*, 1991). In general, the findings in this thesis corroborate the suggestion of Smith *et al* (1988) that the problem of tropical livestock is clearly not one of inadequate number of livestock units but of low productivity.

The pathogenesis and clinical observations recorded during this thesis are similar to those frequently seen on the field during natural challenges. The sub-clinical infection keeps the host in an apparently healthy state but the overall cost of infection on productivity is substantial. A sub-clinical infection is more important to productivity than clinical infection. The latter, when it occurs, is often easily recognized and treated, or death results. In sub-clinical infections, however, such as are present in some breeds with varying degrees of resistance, the effect on the productivity and economics of production can be very grave.

Based on the findings in this thesis, it can be concluded that feed- restricted healthy animals make physiological adjustments by reducing body temperature and maintenance requirements to compensate for the reduced voluntary feed intake. The body temperature in such subjects is also positively related to the digestible organic matter intake. However, the relationship between DOMI and NRET is not affected by infection or artificial feed restriction.

The VFI was lower in the acute phase of infection in open, pregnant and dry parasitized animals, leading to reduced NRET and productivity. However, this was not the case during a low to medium fascioliasis in adult ewes: in the latter, the digestive capacity was not affected. However, low NRET and productivity were observed in all cases of infection investigated.

The infection of pregnant animals resulted in reproductive wastage and low

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[3] Prolificacy = Number of lambs born per 100 ewes.

productivity. Milk yield and composition of non-dairy sheep during trypanosomiasis did not alter and hence have no effect on the lamb growth rate.

FUTURE PROSPECT

This research has demonstrated the importance of anorexia and the compensatory role of the accompanying lipolysis in parasitized animals. Nutrient wasting is found to be reinforced by the incidence of fever during trypanosomiasis. A prominent feature in trypanosomiasis and fascioliasis is the reduced nitrogen retention caused by anorexia and/or increased losses of urinary nitrogen.

Future work should be directed at unveiling the strategies for feed intake during infection (trypanosomiasis) and the effect of higher levels of infection during fascioliasis. Additional research should be directed towards studies that will identify physiological responses associated with nitrogen metabolism during pathologic conditions.

SAMENVATTENDE DISCUSSIE

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INLEIDING

Het centrale thema van deze dissertatie is het onderzoek naar vrijwillige voeropname (VVO) en stikstofretentie (SRET) gedurende parasitaire infecties van kleine herkauwers. Hierbij is aandacht besteed aan de effecten van trypanosomiasis op voeropname, verteerbaarheid, stikstofretentie en produktie. Tevens is een soortgelijke onderzoek gedaan gedurende een lage tot middelmatige fascioliasis infectie in Menz schapen. Verder is een relatie gevonden tussen verteerbare organische stof (VOS), rectale temperatuur (RT) en onderhoudsbehoefte in gezonde geiten.

De geïnfecteerde dieren manifesteerden een complex van reacties. Deze omvatten anorexia, koorts, verhoogde stofwisseling en verlaagde stikstofretentie. Ook werd vastgesteld dat de produktiviteit afneemt hetgeen blijkt uit een slechte groei of gewichtsafname van de gastheer. Hoewel de voedselopname de voornaamste factor in het ziekteproces lijkt te zijn, kunnen de waargenomen veranderingen in produktiviteit en fysiologie tijdens de parasitaire infectie, niet volledig verklaard worden door anorexia. Er zijn aanwijzingen gevonden die erop wijzen dat overleving en produktie van geïnfecteerde dieren afhankelijk is van aanpassingsmechanismen om de stikstof- en energie behoefte te dekken, gedurende de perioden van onvoldoende voedselvoorziening of bij inefficiënt voergebruik.

PARASITAIRE INFECTIE EN VOEDSELGEBRUIK

Een vermindering in voeropname is een algemeen verschijnsel in geïnfecteerde dieren, hoewel de mechanismen die hiervoor verantwoordelijk zijn, onduidelijk blijven. Een door infectie geïnduceerde anorexia beperkt de energie die beschikbaar is voor onderhoud en produktie. Dit verschijnsel wordt bij mens en dier vermeld als een van de eerste tekenen van een patente infectie (Ilemobade en Balogun, 1981; Symons, 1985; Keusch en Farthing, 1986; Holmes, 1987; Akinbamijo, 1988; Verstegen *et al* 1991; Zwart *et al*, 1991).

Anorexia is een belangrijk aspect van parasitaire infecties, maar bij onderzoekingen waarbij gepaarde voertechnieken zijn gebruikt, is gevonden dat geïnfecteerde dieren vaak een gereduceerde voer- en energiebenutting vertonen, in vergelijking met parasiet-vrije dieren bij dezelfde voeropname (Sykes en Coop, 1976; Coop *et al*, 1982).

Het begin van een infectie wordt gekenmerkt door de acute fase reactie. Hierbij treden anorexia, verval van lichaamsweefsels en veranderingen in het metabolisme van koolhydraten, lipiden en eiwitten (Baracos *et al*, 1987). Koorts, een belangrijke factor in het energie metabolisme, is een algemeen verschijnsel in haemo-protozoaire infecties zoals trypanosomiasis (Holmes, 1987; Akker, 1988; Zwart *et al*, 1991; Verstegen *et al*, 1991) en malaria (Cohen en Lambert, 1982). Deze verhoging van lichaamstemperatuur, impliceert een verhoging van warmteproduktie via een intensivering van het metabolisme. In geïnfecteerde mensen en dieren is een verlaagde stikstofretentie gevonden (Beisel *et al*, 1967; Dargie *et al*, 1979; Morris, 1988). De verminderde stikstofbalans, zoals is waargenomen gedurende parasitaire infecties, is echter niet alleen een gevolg van de verminderde voeropname (Symons, 1985): Ook koorts, en de daarmee samenhangende verhogingen in de weefsel stofwisseling, (Baracos *et al*, 1987), eiwitverlies via nieren en ingewanden (Ingh *et al*, 1976; Holmes, 1987), hoge ureum- en eiwitconcentraties in het serum (Finco, 1989) alsmede de hoge stikstofuitscheiding in de urine (Roseby, 1977; Akinbamijo, 1988) kunnen betrokken zijn bij een parasitaire infectie.

Dat het catabolisme van eiwitten gedurende de acute infectie verhoogd is blijkt uit een verlaagde produktie en verhoogde stikstofexcretie (Finco, 1989). Deze effecten, culminerend in een verlaagde c.q. negatieve stikstofretentie, verminderen de produktiviteit van de geïnfecteerde dieren.

Gedurende parasitaire infecties, waarbij anorexia en koorts gelijktijdig voorkomen, zijn lipolyse en ketogenese een algemeen verschijnsel (Keusch, 1984). Nadat de glycogeen reserve is uitgeput, dit is al het geval na enkele uren van anorexia, worden vetzuren gemobiliseerd om aan in de energiebehoefte te voorzien. Hoge concentraties van vrije vetzuren en keton lichamen in de lichaamsvloeistoffen zijn een aanwijzing voor lipolyse en een gedeeltelijke oxidatie van lipiden. In een latere fase kan ook eiwitmobilisatie optreden. Dit blijkt uit een verhoging van het ureum in de urine.

De grotere energiebehoefte tijdens een infectie verhoogd de snelheid van de energieproduktie. De veranderingen in de samenstelling van het serum die vaak waargenomen worden gedurende een door de infectie geïnduceerde anorexia zijn vergelijkbaar met die van een sub-normale calorische opname (Blackburn *et al*, 1991; Keusch, 1984; Bruss, 1989). Deze veranderingen die tegelijkertijd plaats vinden en elkander complementeren, resulteren in een energie opname die aanzienlijk lager is dan het energie gebruik. Dit leidt dan tot een verdere toename van de mobilisatie van uit depot vet en eiwit resulterende in verlaagde of negatieve energie en stikstofbalansen (Clowes *et al*, 1976; Beisel, 1985; Keusch en Farthing, 1986; Baracos *et al*, 1987; Blaxter, 1989).

VERTEERBAARHEID

Op grond van de pathogenese wordt vaak aangenomen dat endoparasitisme vaak gepaard gaat met veranderingen in de gastro-intestinale functies (Holmes, 1987). Parkins *et al*; 1973; Roseby, 1977; Reveron en Topps, 1970; Reveron *et al*, 1974 vermelden echter dat parasitaire infecties normaliter de digestie en absorptie van de gastheer niet beïnvloeden, zelfs niet wanneer de besmetting hoog is. Soortgelijke bevindingen worden ook vermeld in schapen geïnfecteerd met *Fasciola hepatica* (Hawkins en Morris, 1978) en in kalveren met longwormen (Verstegen *et al*, 1989). Holmes (1987) suggereerde dat de opname van aminozuren kan verminderen door endogeen eiwit verlies in de darm van met parasieten geïnfecteerde dieren. Bij helminthiasis is dit echter geen regel. In het algemeen kan gesteld worden dat niet de verminderde digestie en absorptie maar de verhoogde metabole eisen van de gastheer de lage produktiviteit van geparasiteerde gastheren veroorzaken (Baracos *et al*, 1987).

INVLOED VAN PARASITAIRE OP PRODUKTIVITEIT

Het netto effect van een infectie op de produktiviteit loopt primair via een reductie in de energie die beschikbaar is voor produktieve doeleinden.

Veranderingen in lichaamsgewicht zijn in dit verband een algemene maat om de effecten van parasitisme op de dierlijke produktiviteit te meten. Het is algemeen bekend dat zieke dieren gewicht verliezen, of minder snel in gewicht toenemen, dan niet geïnfecteerde dieren. De geringe gewichtstoename en de verschillen in produktiecapaciteit van geïnfecteerde runderen (Schillhorn van Veen, 1974; Kroonen *et al*, 1986) en schapen (Roseby, 1970; Hawkins en Morris, 1978; Tekelye *et al*, 1992a&b) worden in de eerste plaats veroorzaakt door een vermindering in vrijwillige voeropname. Naast veranderingen in het lichaamsgewicht, ontstaan er ook veranderingen in de lichaamssamenstellingen. Dit kan belangrijke gevolgen hebben voor de produktiviteit. In gezonde herten leidt ondervoeding tot duidelijke veranderingen in lichaamssamenstelling (Wolkers, 1993). In schapen lijdende aan een parasitaire infectie is hetzelfde gevonden (Sylkes en Coop, 1976, 1977).

Een energietekort heeft direct gevolgen voor de produktiviteit. Zo is duidelijk aangetoond dat de melkopbrengst en -samenstelling negatief beïnvloed worden gedurende een infectie met trichostrongylosis (Bliss en Todd, 1977) en fascioliasis (Randell en Bradley, 1980). Bliss en Todd (1977), Barger en Gibbs (1981) en Leyva *et al* (1982), rapporteren dat zowel kwaliteit als kwantiteit van wol beïnvloed worden door infectie.

In de literatuur worden herhaaldelijk reproduktie stoornissen bij mens en dier van beide geslachten vermeld die veroorzaakt zijn door trypanosomiasis (Ikede *et al*, 1988; Akpavie *et al*, 1987). Effecten van de infectie zijn aangetoond op conceptie en drachtigheid van runderen (Ogwu en Nuru, 1981; Ogwu *et al*, 1986; Oakley *et al*, 1979) en schapen (Reynolds en Ekwuruke, 1988) resulterend in een lagere reproduktie index[4] (RI) (Gabina, 1988).

Veel van de belangrijkste parasitaire ziekten, zoals trypanosomiasis, babesiosis, malaria en fascioliasis worden gekenmerkt door anaemie. De daling van de hematocrit waarde (PCV) gedurende parasitaire infecties is vaak een bevestiging dat een ziekteproces is begonnen. De aetiologie van een parasitair-geïnduceerde anaemie is waarschijnlijk het oudste en meest herkenbare klinisch teken van een patente parasitaire infectie (Soulsby, 1976).

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[4] RI = lammeren geboren gedurende het leven* (leeftijd in jaren-1)$^{-1}$

EIGEN ONDERZOEKINGEN

De verstoringen die tijdens een infectie optreden in het metabolisme van de gastheer zijn het resultaat van een aantal van processen, die leiden tot kwantitatieve veranderingen in de produktiviteit. Dit scala van processen bepalen het verschil tussen gezondheid en ziekte. In eerste instantie zullen de effecten van voeropname in gezonde dieren besproken worden.

Bij een verlaagd niveau van voedselopname zijn gezonde individuen in staat zich te handhaven, door het energie verbruik te verminderen en door het mobiliseren van vetreserves. Zoals beschreven in hoofdstuk 2 gebeurde dit gedurende vasten of bij verminderde voeropname door een snelle daling van de onderhoudsbehoeften en door een verlaging van de lichaamstemperatuur. De fysiologische en/of gedrag strategieën die voor overleving gebruikt worden door individuen die in hun energie-opname beperkt worden zijn gebaseerd op vermindering van het energiegebruik. De bevindingen zoals beschreven in hoofdstuk 3 geven een positieve correlatie aan tussen lichaamstemperatuur en verteerbaar organische stofopname. Dit energiebesparende proces is een regulier aanpassingsmechanisme in gezonde individuen en is ook gevonden door Shetty (1990); Zwart *et al*, (1991) and Ketelaars en Tolkamp (1991). Het betekent dat voer beperkte dieren hun reserves efficiënter gebruiken door hun lichaamstemperatuur en onderhoudsbehoeften op een niveau te brengen dat in overeenstemming is met hun opname. Dergelijk adaptieve processen zorgen ervoor dat de metabole activiteit van de weefsels verminderd wordt, zodat het gebruik van reserves efficiënter wordt. De dieren zijn daardoor in staat hun stikstof en energiebalans te handhaven bij opname niveaus die lager liggen dan *ad lib* gevoerde dieren (hoofdstuk 2).

Deze bevindingen zijn echter tegengesteld aan die, welke gevonden zijn bij dieren met koorts. Bij deze dieren past het metabolisme zich niet aan de voeropname (Hayashi *et al*, 1985). Waterlow (1986) toonde aan dat het patroon van endogeen energie substraat gebruik significant verschilt tussen gezonde dieren met een beperkte voeropname en dieren waarbij de voeropname was verminderd door een infectie. In tegenstelling tot verminderde T3 en T4 waarden in met *T. vivax* geïnfecteerde geiten, werden door Van Dam (ongepubliceerd) geen veranderingen gevonden in voerbeperkte gezonde geiten. Deze observatie ondersteunt de hypothese dat de schildklieractiviteit vermindert gedurende trypanosomiasis, maar suggesteert tevens dat andere mechanismen betrokken zijn bij de regulering van het energie metabolisme gedurende infectie.

TRYPANOSOMIASIS

Zoals beschreven wordt in de hoofdstukken 1, 2, 4 en 5, veroorzaakt trypanosomiasis verschillende reacties in West Afrikaanse dwerggeiten en schapen. In zijn algemeenheid bevestigt de door de infectie veroorzaakt anorexia in deze experimenten, de vroegere bevindingen van Ilemobade en Balogun, 1981; Akinbamijo, 1988; Zwart, 1989. De verminderde voeropname is voornamelijk verantwoordelijk voor de negatieve stikstofbalans en de verminderde produktiviteit.

Dus onderzoek leverde geen aanwijzingen op voor het bestaan van een haemorragische infectie gedurende trypanosomiasis. Deze waarneming, gecombineerd met de afwezigheid van nier- en darm afwijkingen tijdens de sectie, maakt stikstofverlies via de darmen of nieren onwaarschijnlijk (Ingh *et al*, 1976). Geen van onze experimenten leverde aanwijzingen op voor de veronderstelling dat de verteringscapaciteit veranderd was gedurende trypanosomiasis. Dat de stikstofuitscheiding door geïnfecteerde dieren niet verschilde van die van de controle dieren bevestigt dat er geen stikstofverlies heeft plaatsgevonden gedurende de infectie. Het feit dat er geen verhoging van eiwit in de urine werd gevonden, bevestigt de sectie resultaten van hoofdstuk 2 en ondersteunt de stelling dat er geen sprake is geweest van een nierdysfunctie.

De trypanosomiasis in onze onderzoekingen ging altijd gepaard met koorts. Dit is in overeenstemming met het feit dat protozoaire infecties vaak vergezeld gaan met koorts, waardoor de energie behoefte van de geïnfecteerde gastheer zal toenemen. Dit verband, dat eerder vermeld is door Baracos *et al*, (1987) en Cohen en Lambert (1982) wordt ook ondersteund door Verstegen *et al* (1991), die 25% van de toename aan onderhoudsbehoefte van met *Trypasosoma vivax* geïnfecteerde geiten toeschrijven aan koorts. In het huidige onderzoek, hadden alle dieren koorts en hielden dat gedurende de hele infectie. Koorts, een verhoging van het basaal metabolisme en een hogere warmteproduktie dragen allen bij tot een verhoging van de energiebehoefte van geïnfecteerde individuen (Keusch en Farthing, 1986).

Deze dissertatie geeft aan dat de metabole kosten die gepaard gaan met een infectie (Baracos *et al* 1987; Verstegen *et al*, 1991) en anorexia, leiden tot een verhoging van de lipolyse in de gastheer. Een verhoging in het serum van geïnfecteerde dieren van vrije vetzuren bevestigen dat lichaamsvetreserves werden gebruikt om de gevolgen van de negatieve energiebalans, die ontstaan is door de anorexia en de verhoogde onderhoudsbehoefte te bestrijden (hoofdstuk 1). Omdat de ureumconcentratie in het bloed wat verhoogd is moet rekening worden gehouden met de mogelijkheid dat ook lichaamsweiwitten worden gecataboliseerd. Dit komt overeen met de bevindingen van Beisel (1985). Er zijn geen aanwijzingen gevonden voor een duidelijke verhoogde ketogenesis of een overmatige afbraak van lichaamseiwitten, zelfs niet bij een voeropname die gelijk was aan 40% van de onderhoudsbehoefte. Slechts bij twee dieren werd een verhoogde concentratie aan betahydroxybutyraat gevonden (hoofdstuk 2). Een dergelijke metabole reactie werd door Symons (1985) beschreven gedurende ernstige uithongering.

Bij de anaemische en anorexische drachtige ooien was er gedurende de post-infectie fase een tendens om de gewichtstoename en lichaamsconditie te verminderen. Deze storingen gedurende trypanosomiasis zijn door andere onderzoekers toegeschreven aan een reeks van oorzaken, zoals: een vermindering in droge stof opname (Akinbamijo *et al*, 1990), een verhoging van het basaal metabolisme (Zwart *et al*, 1991; Stephen 1986) een verhoogd catabolisme van weefsel reserves (Akinbamijo *et al*, 1992), een verminderde stikstof en energie balans (Zwart *et al*, 1991; Verstegen *et al*, 1991) en een mogelijke opname van gastheer nutriënten door de parasieten (Reynolds, persoonlijke mededeling). In de acute fase en de daarbij horen de fysiologische slijtage processen, heeft het verhoogde metabolisme ernstige gevolgen voor de dierlijke produktie, zoals sterfte van de moeder, en foetale en neo-natale verliezen. Deze drie factoren zijn prominent aanwezig in vroegere overzichten van het effect van trypanosomiasis op de reproduktie (Ogwu en Nuru, 1981; Ogwu *et al*, 1986; Ikede *et al*, 1988) en in de bevindingen van Elhassan (1987) en Reynolds and Ekwuruke (1988) bij West Afrikaanse dwergschapen.

De geïnfecteerde ooien in ons onderzoek hadden aan het eind van de dracht lagere lichaamsgewichten en een verminderde voeropname. De lammeren hadden verlaagde geboortegewichten en er was een aanzienlijke neo-natale sterfte. Als gevolg hiervan stierf 85% van de lammeren geboren uit ooien die laat in de dracht waren geïnfecteerd, binnen 7 dagen na de geboorte. In het algemeen is de relatie tussen geboortegewicht en groei van lammeren enerzijds en het effect van maternale voeding tijdens de dracht en het gewicht bij de partus anderzijds in verschillende publikaties vermeld (Gibb en Treacher, 1980 & 1982, Peart 1967, Treacher 1970, Adu en Olaloku, 1979). In het geval van ernstige voedingsstress zijn gevallen van sporadische abortus gerapporteerd (Osuagwuh en Akpokodje, 1986; Osuagwuh en Aire, 1990). Als de voeding van moeder en foetus inadequaat is, kan dit leiden tot resultaten die verkregen zijn in dit onderzoek bij geïnfecteerde ooien (hoofdstuk 4). De maternale effecten op de lage geboorte gewichten weerspiegelden zich in de lage speengewichten van lammeren van geïnfecteerde moeders.

Ondanks het feit dat de ooien geïnfecteerd waren na het lammeren, hadden ze voldoende lichaamsreserves om te voldoen aan de dagelijks behoefte aan melk gedurende het begin van de lactatie. Lactatie is vaak geassocieerd met een toename van de voeropname (Blaxter, 1989) en dit kan het effect van infectie op voeropname en melk opbrengst gemarkeerd hebben gedurende de vroege lactatie (hoofdstuk 5). Gerelateerd met de ontwikkeling van het gewicht, kan afgeleid worden dat geïnfecteerde lacterende schapen hun lichaamsreserves aanspreken om de voedingsnutriënten te supplementeren die nodig zijn voor de melkproduktie zoals ook vastgesteld door Gibb en Treacher (1982). Omdat de melkopbrengst geen verschillen vertoonde tijdens de vroege lactatie, verschilde de groei toename ook niet tussen de groepen lammeren, daar deze reeds begonnen waren met afspenen en hun melkopname supplementeerden met voeder.

FASCIOLIASIS

De klinische observaties van de laag tot middelmatige met fascioliasis geïnfecteerde dieren, kwamen overeen met de doelstelling om een sub-klinische infectie na te bootsen die gewoonlijk voorkomt bij grazende dieren. Er zijn geen aanwijzingen gevonden voor veranderingen in voeropname en digestie capaciteit van de gastheren na infectie. Dit stemt overeen met de bevindingen van Hawkins en Morris (1978) die verkregen werden bij dieren waarbij infecties met verschillende gradatie waren gedaan. Zij vonden dat er een negatieve relatie is tussen de hoeveelheid leverbotten die voor het induceren van de infecties werden toegediend en de verteringscoëfficienten. De infectie dosis van ons onderzoek in beschouwing nemend, mag men aannemen dat het infectie niveau te laag is geweest om verteringsstoornissen te initiëren. De kosten van de infectie in de norm van inefficiënt voer gebruik waren wel overtuigend. Dit stemt overeen met de rapporten van Berry en Dargie (1976) en Dargie *et al* (1979).

De sub-klinische fascioliasis had geen effect op de vrijwillige voeropname van drachtige en niet-drachtige Menz schapen. Dit is in tegenstelling met wat er gebeurt tijdens een parasitaire infectie, waarbij anorexia als een van de eerste symptomen wordt vermeld (Berry en Dargie, 1976; Murray en Murray, 1979; Symons, 1985, Keusch en Farthing, 1986; Holmes, 1987; Morris 1988). Over een verklaring van deze bevinding in ons experiment kan allen maar gespeculeerd worden. Het kan verband houden met factoren zoals ras en leeftijd van de gastheer, de vitaliteit en het aantal toegediende metacercariae en aan het bestaan van een onbekend niveau van immuniteit na de eerder doorstane infectie (Sinclair, 1971; Berry en Dragie, 1976; Leyva *et al*, 1982).

De verteringscapaciteit van de ooien was niet door de infectie en/of drachtigheid beïnvloed. Dit bevestigt de meeste bevindingen gedurende parasitisme (Dargie *et al*, 1979; Berry en Dargie, 1976) maar is in tegenstelling met die van Holmes (1987). Deze suggereerde de mogelijkheid van gestoorde verterings- en opnameprocessen, vooral ten gevolge van verlies van darmeiwitten in de gastro-intestinale tractus tijdens worminfecties. Zoals echter al werd vermeld, de gelijke hoeveelheid faecale stikstof in alle experimentele groepen ontkracht het bestaan van een dergelijke proces.

Ondanks het lage tot middelmatige niveau van fascioliasis infectie waaraan de Menz schapen in dit experiment werden blootgesteld, trad er acht weken na infectie een daling op van de PCV. Dit komt overeen met de waarnemingen van Sinclair (1971) en Dow *et al* (1968). De daling in PCV post-infectie toont aan dat de geïnoculeerde ooien inderdaad geïnfecteerd waren.

Op vergelijkbare niveaus van vrijwillige voeropname, hielden de geïnfecteerde dieren minder stikstof vast, verminderde hun gewichtstoename, produceerden ze lichtere lammeren en hadden ze een slechtere lichaamsconditie (hoofdstuk 6). Dit wordt waarschijnlijk veroorzaakt door verschillen in efficiënt gebruik of omzetting van voer in de verlangde dierlijke produkten. Het verschil in gewichtstoename tussen geïnfecteerde en gezonde geiten (hoofdstuk 1) is een illustratie van de nutriënten onttrekking die een infectie begeleidt. Inefficiënt voer gebruik en gewichtsverlies gedurende parasitaire infecties wordt ook in de bovenstaande literatuur vermeld. De stikstofretentie verschilde aanzienlijk in het eerste deel van de infectie en keerde langzamerhand terug naarmate de infectie voortschreed. Een stijgend PCV die waargenomen werd tegen het eind van de proef, kan ook verband houden met de verkregen resistentie zoals eerder vermeld door Sinclaer (1971). De verminderde stikstofretentie op acht weken na infectie zoals vermeld door Dargie *et al* (1979), werd voornamelijk veroorzaakt door een verhoogd stikstof gehalte in de urine. In onze studie, het tijdschema in beschouwing nemende, stemmen de lagere stikstofretentie en hoog urine stikstof in de geïnfecteerde ooien, overeen met die van Dargie *et al* (1979). Dat de voedselopname en de vertering in de controle dieren gelijk was aan die in de geïnfecteerde ooien, suggereert dat er andere, onbekende, verliezen een rol spelen bij de verlaagde stikstofretentie en de verlaagde gewichtstoename in geïnfecteerde ooien. Dit wordt grotendeels ondersteund door de data van de stikstofbalans en het lichaamsgewicht, die de geïnfecteerde dieren in de laagste categorie plaatsen. Hoewel ons experiment geen aanwijzingen verschaft over de oorzaken van de extra stikstof die uitgescheiden wordt door de geïnfecteerde ooien, bevestigt de aanwezigheid van stikstof in de urine en niet in de faeces, de veronderstelling dat de verteringsopname capaciteit van de gastheer niet was aangetast gedurende de lage tot middelmatige fascioliasis infectie.

De toename in lichaamsgewicht was ook aanzienlijk aangetast in de geïnfecteerde dieren. Reid *et al*, 1970; Holmes, 1987; Blackburn, 1991 rapporteren dat schapen geïnfecteerd met *Fasciola hepatica* niet in staat waren de gewichtstoename te handhaven in vergelijking met controle dieren.

Het geboortegewicht van de lammeren van de geïnfecteerde ooien was lager dan die van de controle ooien. De lage stikstofretentie, gevonden in de geïnfecteerde drachtige schapen, sluit aan bij de veronderstelling dat stikstof afzetting in de foetus verminderd zou zijn. Zulke schadelijke effecten van parasitisme op het produktie potentieel en het efficiënte gebruik van reserves werden eerder gedemonstreerd (Ogwu *et al*, 1986; Ikede *et al*, 1988; Reynolds en Ekwuruke, 1988; Akinbamijo *et al*, 1994).

IMPLICATIES VOOR DIERLIJKE PRODUKTIVITEIT

Bij de proeven die in het kader van deze dissertatie zijn uitgevoerd met *Trypanosoma* en *Fasciola spp.* hebben zich géén verteringsstoornissen voorgedaan. In vergelijking met de controle dieren was de voerbenutting bij de geïnfecteerde dieren lager. Bij de geïnfecteerde dieren viel een vermindering van de stikstofretentie, die het resultaat was van een gereduceerde vrijwillige voeropname en/of een verhoogde excretie van stikstof in de urine altijd samen met het begin van de infectie. Omdat de produktie niet alleen afhankelijk is van de opname maar ook van de doelmatigheid waarmee de gewenste produkten worden gemaakt kan uit de resultaten het onderzoek afgeleid worden dat de verminderde produktiviteit in geparasiteerde dieren het directe resultaat is van door de infectie veroorzaakt vermindering van de voerbenutting.

In kwantitatieve termen wordt daardoor minder stikstof achtergehouden in het weefsel of in de produkten van de dieren. Dit wordt bevestigd door de bevindingen zoals beschreven zijn in hoofdstuk 1, waar wordt aangetoond dat trypanosomiasis verantwoordelijk is voor de lage groeisnelheid. In het kader van dit onderzoek is er geen karkas analyse gedaan, maar andere onderzoekers hebben veranderingen gerapporteerd in lichaamssamenstelling gedurende ondervoeding (Wolkers, 1993) en infectie (Sykes en Coop, 1976). In ons onderzoek was sprake van zowel ondervoeding als infectie (zie hoofdstuk 1 en 2).

Er bestaat een duidelijk verband tussen een verminderde reproduktie en infecties. Deze vermindering wordt als aanzienlijk beschouwd (Osuagwuh en Aire, 1990; Osuagwuh en Akpokoje, 1986). Het produktieverlies wordt in het algemeen gekenmerkt door embryonale of foetale sterfte (zie hoofdstuk 4). In het algemeen geldt dat indien de moeder in goede lichaamsconditie is voor de partus, de nakomelingen een goede overlevingskans hebben. Als moederdieren lijdende zijn aan parasitaire infecties is er bijna altijd sprake van een verminderde conditie. Als de infectie ernstiger is en wanneer de voedingsstress extreem, ontstaan de bovengenoemde condities en zal verandering van de reproduktie het resultaat zijn. De mate waar dit zo is gerelateerd aan het niveau van de voeding van de moeder en aan de parasitaire infectiedruk. Lettend op de bevindingen van Ikede en Losos, (1972) in schapen en van Ogwu *et al*, (1986) in rundvee mag aangenomen worden dat in onze studie ook een intra-uteriene infectie niet kan worden uitgesloten.

Vastgesteld is dat een infectie op sub-klinisch niveau de nutriënten benutting in sterk negatieve zin beïnvloedt. Dit kan ernstige consequenties hebben voor de produktiviteit (hoofdstuk 4 en 6). De geïnfecteerde drachtige ooien hadden een lage stikstofretentie, vertoonden een matige gewichtstoename en hadden een slechte lichaamsconditie en foetale ontwikkeling. Dit culmineerde in een laag geboortegewicht van de lammeren. Zelfs indien de moeder na de partus behandeld werd, ijlden de effecten tijdens de drachtigheid nog na en werd dit weerspiegeld in de ontwikkeling van de lammeren.

Hoewel er geen ernstige effecten waren van een infectie post-partum op de groeicurves van de lammeren, verloren de geïnfecteerde ooien meer gewicht tijdens de lactatie en hielden minder stikstof vast. In de context van vee gezondheid en produktiviteit verlaagt parasitisme het produktie potentieel en de opbrengst van geïnfecteerde dieren. Zoals in deze dissertatie werd waargenomen, worden door parasitisme een aantal parameters aanzienlijk beïnvloed. Het gaat hierbij om groeisnelheid (of gewichtstoename), abortus, geboortegewicht, speengewicht, neonatale verliezen, stikstofretentie, lichaamsconditie en melkopbrengst. Andere variabelen die in dit verband meegenomen zouden kunnen worden zij het gewicht van de nakomelingen die per moederdier gespeend worden, het aantal geproduceerde lammeren en het aantal lammeren geboren per 100 ooien.

In overeenstemming met de bevindingen van Ogwu *et al* (1986) in rundvee, Elhassan, (1987) en Reynolds en Ekwuruke (1988) in schapen, aborteerden geïnfecteerde ooien meer gedurende het derde trimester. Deze auteurs geven aan dat de chronische of sub-klinische trypanosomiasis een negatief effect heeft op de reproduktie en de fertiliteit, zoals anoestrus, mislukte bevruchting en slechte libido. Deze afwijkingen hebben een directe invloed op de produktiviteitsindex[5] (PI) beschreven door Bosman *et al* (1988) en de reproduktie index (RI) beschreven door

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[5] PI = kg gespeend lam/lammerende ooien per jaar

Burfening *et al* (1993). Volgens de produktiviteit indices beschreven door deze onderzoekers, heeft de incidentie van parasitaire ziekten bij de teelt van kleine herkauwers een vermenigvuldigingseffect, omdat de twee indices (PI en RI) directe determinanten zijn van vruchtbaarheid (proliferatie[6]) (Gabina, 1988). Dit impliceert dat factoren die beïnvloed worden door parasitisme, determinanten zijn van produktiviteit en dien ten gevolge verantwoordelijk zijn voor de vermindering in de expressie van het genetisch potentieel en de optimale produktie per vee eenheid. Zoals in deze dissertatie is aangetoond stoppen de gevolgen van een infectie niet bij de morbiditeit maar wordt ook de mortaliteit beïnvloed (Anene *et al*, 1991). In het algemeen bevestigen de bevindingen van deze dissertatie de opmerking van Smith *et al* (1988) dat het probleem van vee in de tropen niet is gelegen in onvoldoende aantallen, maar in een te lage produktiviteit.

De pathogenese en de klinische bevindingen die vermeld worden in dit proefschrift, zijn gelijk aan de veelvuldig gedane veld waarnemingen onder een natuurlijke infectiedruk. De sub-klinische infectie houdt de gastheer in een ogenschijnlijk gezonde staat, maar de negatieve effecten van de infectie op de produktiviteit zijn aanzienlijk. Een sub-klinische infectie is belangrijker voor de produktiviteit dan een klinische infectie. Deze laatste is uniek ofwel gemakkelijk te herkennen en te behandelen, of ze resulteert in sterfte. In sub-klinische infecties zoals veel voorkomende bij sommige rassen met wisselende graad van resistentie kan het effect op de produktiviteit en economie zeer ernstig zijn.

Concluderende kan gesteld worden, dat gebaseerd op de bevindingen van deze dissertatie voer-beperkte, gezonde dieren, zich fysiologisch aanpassen aan een veranderde voeropname door de lichaamstemperatuur en de onderhoudsbehoeften te reduceren. Ook de lichaamstemperatuur blijkt positief gecorreleerd te zijn met de verteerbare organische stofopname. De relatie tussen deze laatste en stikstofretentie wordt niet beïnvloed door de infectie of kunstmatige voederrestrictie.

De vrijwillige voeropname was lager in de acute fase van de infectie in drachtige, niet-drachtige en droogstaande geparasiteerde ooien. Dit leidde tot een verminderde stikstofretentie en produktiviteit. De verminderde voeropname werd

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[6] Proliferatie = aantal lammeren geboren per 100 ooien.

echter niet waargenomen gedurende een lage tot middelmatige fascioliasis infectie in volwassen ooien. Bij die dieren was ook de digestie capaciteit niet aangetast. Wel werd een verminderde stikstofretentie en produktiviteit gemeten.

De infectie van drachtige dieren resulteerde in verminderde reproduktie en lage produktiviteit. Melkopbrengst en -samenstelling van niet op melkproduktie gefokte schapen bleven voldoende gedurende de trypanosomiasis infectie om een normale groei van de lammeren te waarborgen.

VOORUITZICHTEN

Dit onderzoek heeft aangetoond dat anorexia en de compensatoire rol van de lipolyse een belangrijke rol spelen bij de verminderde produktie van dieren met trypanosomiasis. Verder is gevonden dat het verbruik van nutriënten versterkt werd door de aanwezigheid van koorts tijdens trypanosomiasis. Een prominent kenmerk van trypanosomiasis en fascioliasis is de verminderde stikstofretentie die wordt veroorzaakt door de anorexia en/of de verhoogde verliezen van stikstof in de urine.

Toekomstig onderzoek zal zich moeten richten op strategieën op het gebied van de regulatie van voeropname tijdens infectie (trypanosomiasis) en op het effect van hogere besmettingsniveaus gedurende fascioliasis. Er zal tenslotte additioneel onderzoek gedaan moeten worden op het gebied van de fysiologische mechanismen die verbonden zijn met het stikstof metabolisme gedurende pathologische omstandigheden.

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CURRICULUM VITAE

Olurotimi Oseyemi Akinbamijo was born on September 4th, 1960 in Ondo, Nigeria. He was educated at the Egbado College Ilaro and Anglican Grammar School Otan-Aiyegbaju where he graduated with First Division of the School Certificate examination conducted by the West African Examination Council (WAEC) in June 1978. In September 1978, he was admitted to the Federal School of Arts and Science, Ondo for the Advanced General Certificate of Education (GCE - A level). During the harmattan semester of 1979, he gained admission in to the Faculty of Agriculture of the University of Ife (now Obafemi Awolowo University) Ile - Ife, Nigeria. In the rain semester of 1984, He graduated from the University with Bachelor of Agriculture with honours. After completing his National service, the author commenced graduate studies in Obafemi Awolowo University Ile-Ife and conducted his post-graduate research under the auspices of the West African Dwarf Goat Research Project. In 1988, he was awarded the degree of Master of Science (Animal nutrition major).

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