

INTER-RELATIONSHIPS BETWEEN GASTROINTESTINAL HELMINTH INFECTION,
NUTRITION AND IMPAIRED PRODUCTIVITY IN THE RUMINANT

J.W. STEEL*

I. INTRODUCTION

It is well recognised that, because of the incomplete development of immunological control mechanisms, young calves and lambs are particularly susceptible to acute, heavy infections of gastrointestinal helminths. Rapid and pronounced losses of weight are accompanied by anorexia and frequently by diarrhoea and, if animals are not treated with anthelmintics, death occurs. In older animals symptoms of acute disease are more rarely seen and the principal manifestation of parasite exposure may be a reduced rate of liveweight gain and, in sheep, depressed wool production. In this situation the syndrome is analogous to the effects of undernutrition and it is not surprising that in recent years a considerable amount of research has been directed towards an understanding of the inter-relationships between gastrointestinal parasitism, impaired productivity and the nutrient economy of the ruminant.

In this paper I shall attempt to give a broad overview of recent advances in our understanding of the principal physiological and biochemical disturbances which accompany gastrointestinal helminth infection in sheep and cattle. Particular emphasis is placed on those changes which have nutritional implications. More comprehensive accounts of the pathophysiology of helminth infections are given by Symons (1969) and Soulsby (1976).

II. FOOD CONSUMPTION

Reduced food consumption has usually, but not invariably, been recorded following infection with most of the commonly occurring parasitic helminths of sheep and cattle. Inappetance appears to be independent of the site of infection, whether this be the abomasum (Haemonchus spp.; Ostertagia spp.; Trichostrongylus axei), the small intestine (Trichostrongylus colubriformis; Nematodirus spp.; Cooperia spp.), the large intestine (Oesophagostomum spp.; Chabertia ovina) or the bile ducts (Fasciola hepatica). Recent studies with weaner lambs given a good quality ration (19% crude protein) and continuously dosed for 24 weeks with either T.colubriformis or O.circumcincta have shown that the degree of anorexia is dependent upon the number of infective larvae ingested (Symons, Steel and Jones, unpublished; see Figs. 1a,b). Three additional points emerge from this data; firstly, there is a threshold level of exposure below which there is no significant depression in feed intake. Secondly, appetite returns to normal despite continued exposure; this may be related to the development of resistance. Thirdly, the magnitude of effect is dependent on the species of parasite; clearly, O.circumcincta has a considerably less deleterious effect on feed intake than T.colubriformis when compared on a larval intake basis.

Obviously, reduced food consumption must impair productivity simply by reducing the overall availability of nutrients for metabolic

* C.S.I.R.O. McMaster Animal Health Laboratory, Private Bag 1, Glebe
N.S.W. 2037.

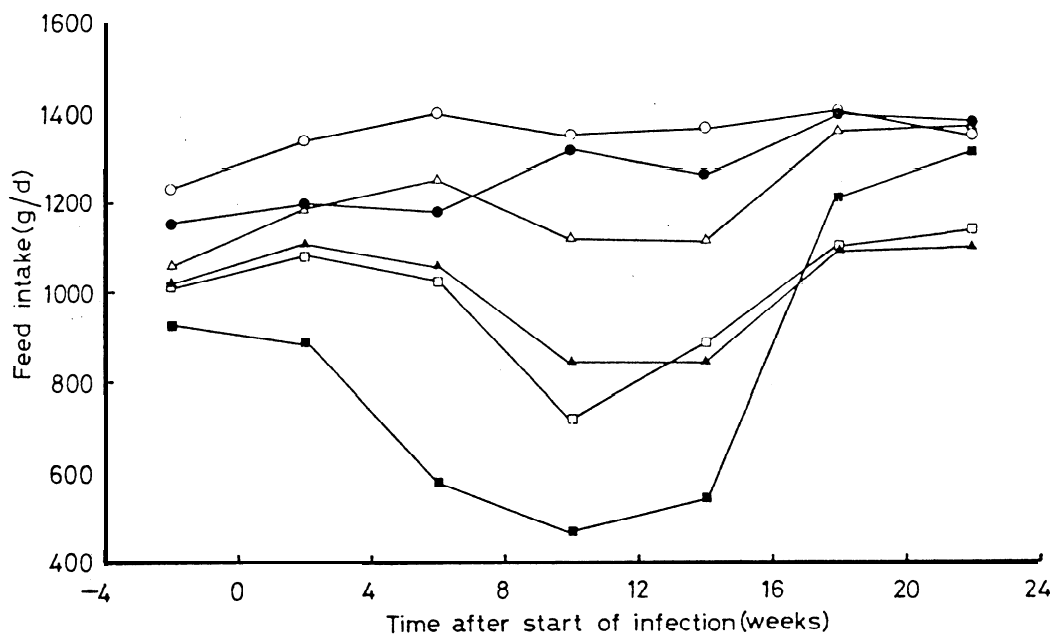


Fig. 1a. Mean daily feed intake over four-week periods in lambs continuously infected with *T.colubriformis* larvae at weekly dose rates of: (○) 0; (●) 300; (△) 900; (▲) 3,000; (□) 9,000; (■) 30,000.

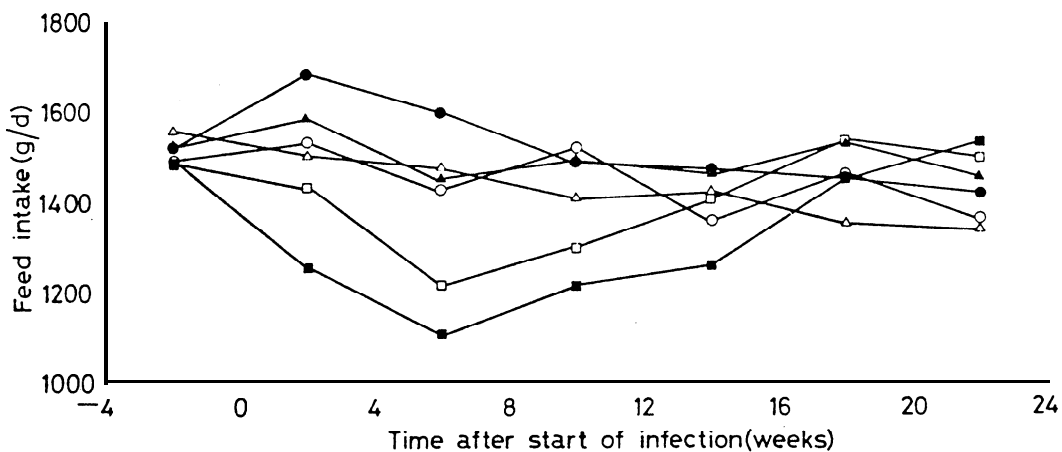


Fig. 1b. Mean daily feed intake over four-week periods in lambs continuously infected with *O.circumcincta* larvae at weekly dose rates of: (○) 0; (●) 1,200; (△) 3,750; (▲) 12,000; (□) 37,500; (■) 120,000.

processes. However, comparisons between infected and pair-fed, uninfected animals have shown that this does not wholly account for the reduced liveweight gain of lambs infected with T.colubriformis (Sykes and Coop 1976) and O.circumcincta (Sykes and Coop 1977) or in calves infected with Oe.radiatum (Bremner 1961). Similarly, clean wool production may be reduced by as much as 60% in sheep infected with T.colubriformis compared with worm-free animals on similar feed intakes (Carter, Franklin and Gordon 1946; Barger, Southcott and Williams 1973).

It has been suggested that factors such as pain and inflammatory reactions at the site of infection may be involved in appetite suppression of parasitised animals. To date there is little information on the underlying mechanisms, but recent studies have indicated that the gastrointestinal hormone cholecystokinin may be a mediator of feed intake regulation in sheep infected with T.colubriformis (L.E.A. Symons, pers. comm.).

III. LOSS OF BLOOD AND PLASMA INTO THE GUT

Elevated loss of blood and/or plasma at the site of infection has a critical bearing on the pathogenesis of impaired productivity caused by gastrointestinal helminths. In sheep infected with either of the haematophagic parasites, H.contortus and F.hepatica, approximately 20 ml of red blood cells may be lost daily into the gut, compared with less than 1 ml/d in uninfected sheep (Dargie 1973, 1975). As a consequence of this loss the daily catabolism of haemoglobin has been estimated to increase in sheep infected with 2000 H.contortus by 15 g over and above the normal 3 g/d attributable to red cell breakdown; increased amounts of iron are also lost into the abomasum and may not be completely reabsorbed (Dargie 1973). Thus, the continuous loss of red cells in chronic haemonchosis culminates in iron deficiency and exhaustion of the erythropoietic system with consequent severe anaemia. As might be expected, the blood-sucking activities of these parasites also result in an increased loss of plasma into the gut, but this is substantially greater than that attributable to ingestion of blood alone (see Table 1).

Infection with non-haematophagic parasites similarly results in an elevated loss of plasma (Table 1). This effect is apparently due to the breakdown of junctional complexes between the cells of the mucosal epithelium at the site of infection which thereby become more permeable to macromolecules (Murray *et al*, 1971). At total protein concentrations observed in plasma during continuous infection of lambs with T.colubriformis it was estimated that up to 12 g/d of protein was lost into the gut (Symons, Steel and Jones, unpublished; see Fig. 2). It is also clear that both the magnitude of this loss and its rate of development is dependent upon the level of exposure to infective larvae. Furthermore, as animals become immune to infection plasma protein leakage returns to normal values (Fig. 2).

Increased desquamation of mucosal epithelial cells may further exacerbate the loss of nitrogen into the gut. Although no quantitative estimates are available in sheep and cattle, increased turnover and loss into the gut of intestinal epithelial cells has been demonstrated in small intestinal nematode infections of the rat. Histological evidence of the proliferation of mucous cells at the site of nematode infections suggests that mucoprotein secretion may also be elevated but again no quantitative data are available.

TABLE 1. Gastrointestinal plasma leakage in helminth infections of sheep and cattle

Host	Parasite	Site of infection	Plasma leakage (ml/d)		Reference
			Infected	Control	
<u>Sheep</u>					
	<u>H. contortus</u>	abomasum	273	38	Dargie (1975)
	<u>O. circumcincta</u>	abomasum	92	29	Holmes & MacLean (1971)
	<u>T. colubriformis</u>	small intestine	100-300	28	Barker (1973)
	<u>C. ovina</u>	large intestine	115	46	Herd (1971)
	<u>F. hepatica</u>	bile ducts	174	22	Dargie (1975)
<u>Cattle</u>					
	<u>Oe. radiatum</u>	large intestine	972	144	Bremner (1969a)

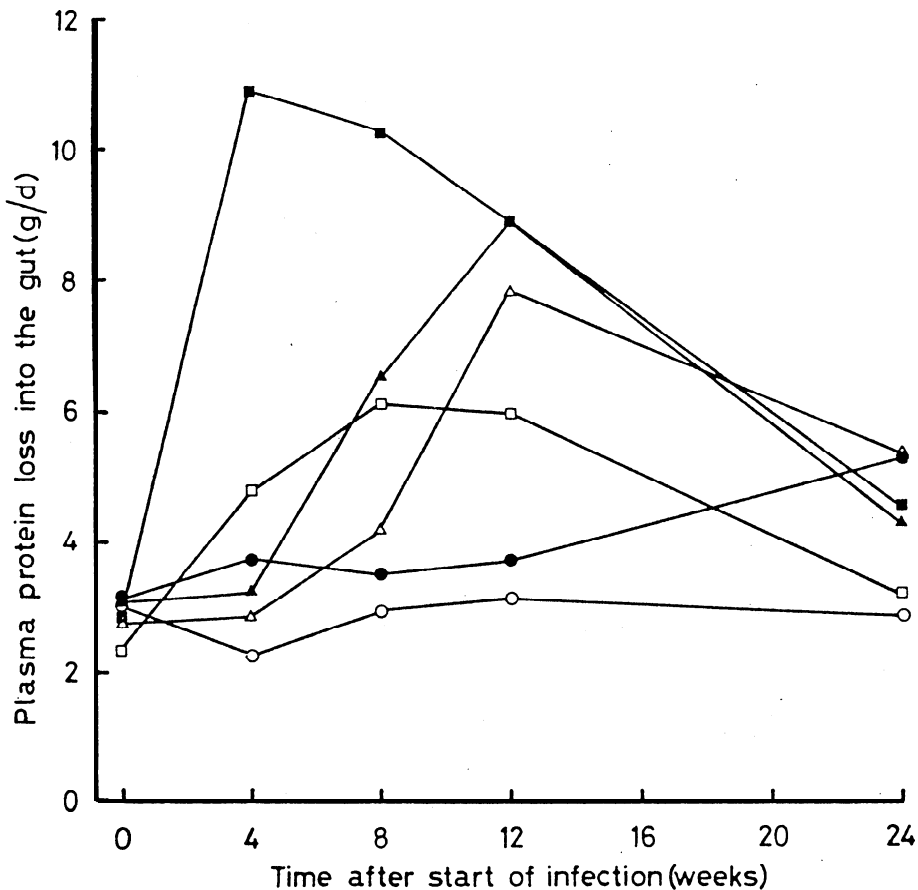


FIG. 2. Plasma protein loss into the gut of lambs continuously infected with T. colubriformis larvae at weekly dose rates of: (○) 0; (●) 300; (△) 900; (▲) 3,000; (□) 9,000; (■) 30,000.

Clearly, the extent to which increased nitrogen loss into the parasitised gut influence the nutritional economy of sheep and cattle, will, in part, be determined by the capacity for reabsorption. The potential for reabsorption must be dependent on the site of infection since the small intestine is the major site of amino acid absorption. In this context, it is interesting to note that in calves infected with Oe. radiatum the loss of plasma protein into the large intestine exceeded that in pair-fed, worm-free animals by 40 g/d; a concurrent determination of nitrogen balance demonstrated that increased faecal nitrogen excretion in infected calves was equivalent to this protein leakage (Bremner 1969b).

IV. GASTROINTESTINAL FUNCTION

Helminth infection is commonly accompanied by pronounced macro- and microscopic changes to the gastrointestinal tract. These, sometimes associated with distension of the small intestine with fluid and ingesta and diarrhoea, have led to the belief that malabsorption is a major cause of poor body and wool growth. However, detailed studies over the past 15-20 years have firmly established that malabsorption per se does not play a major role in the aetiology of production loss in small-intestinal nematode infections (see review by Symons 1976). It is clear that while reduced digestion and absorption of sugars, amino acids and fatty acids may occur at the site of infection in the upper small intestine, compensatory digestion and absorption occurs at sites distal to infection, with the result that overall absorption by the small intestine is unimpaired (Barger et al. 1973; Symons 1976). Nevertheless, measurements of the flow of digesta and its constituents through the gastrointestinal tract of cannulated sheep consuming a normal forage diet, have demonstrated that the partition of nutrient uptake between the stomach, small and large intestines may be substantially altered following infection with T. colubriformis (see Steel 1974). In these sheep volatile fatty acid production in the rumen was depressed by up to 30%, (Steel 1972) and the quantities of organic matter and cell-wall constituents passing to the intestine was increased. However, compensatory increase in digestion in the large intestine resulted in only a marginal increase in faecal output of the latter constituents.

In animals exhibiting marked inappetance, the flow of non-ammonia nitrogen (NAN) from the ileum was similar to that in uninfected sheep on a similar nitrogen intake. With increasing food consumption, NAN flow from the ileum increased at a faster rate in infected sheep such that at normal intakes, the flow of NAN at this site was 2 g/d, or 25%, higher than in the controls. Faecal nitrogen output showed a similar relationship with feed intake (see Steel 1974). Paradoxically, it appeared that there was a greater effect of infection on net uptake of nitrogen by both the small intestine and the whole tract in animals judged, by loss of appetite, to be less severely affected.

In similar studies in sheep infected with O. circumcincta (Steel 1975), the pattern of NAN movement through the gastrointestinal tract was also markedly affected (see Fig. 3). Compared with worm-free controls on similar intakes, infection increased NAN flow from the abomasum and ileum and nitrogen output in faeces by a mean 5.1, 1.1 and 0.5 g/d respectively.

The most acceptable interpretation of the changes in digesta nitrogen movement in both trichostrongylosis and ostertagosis is that

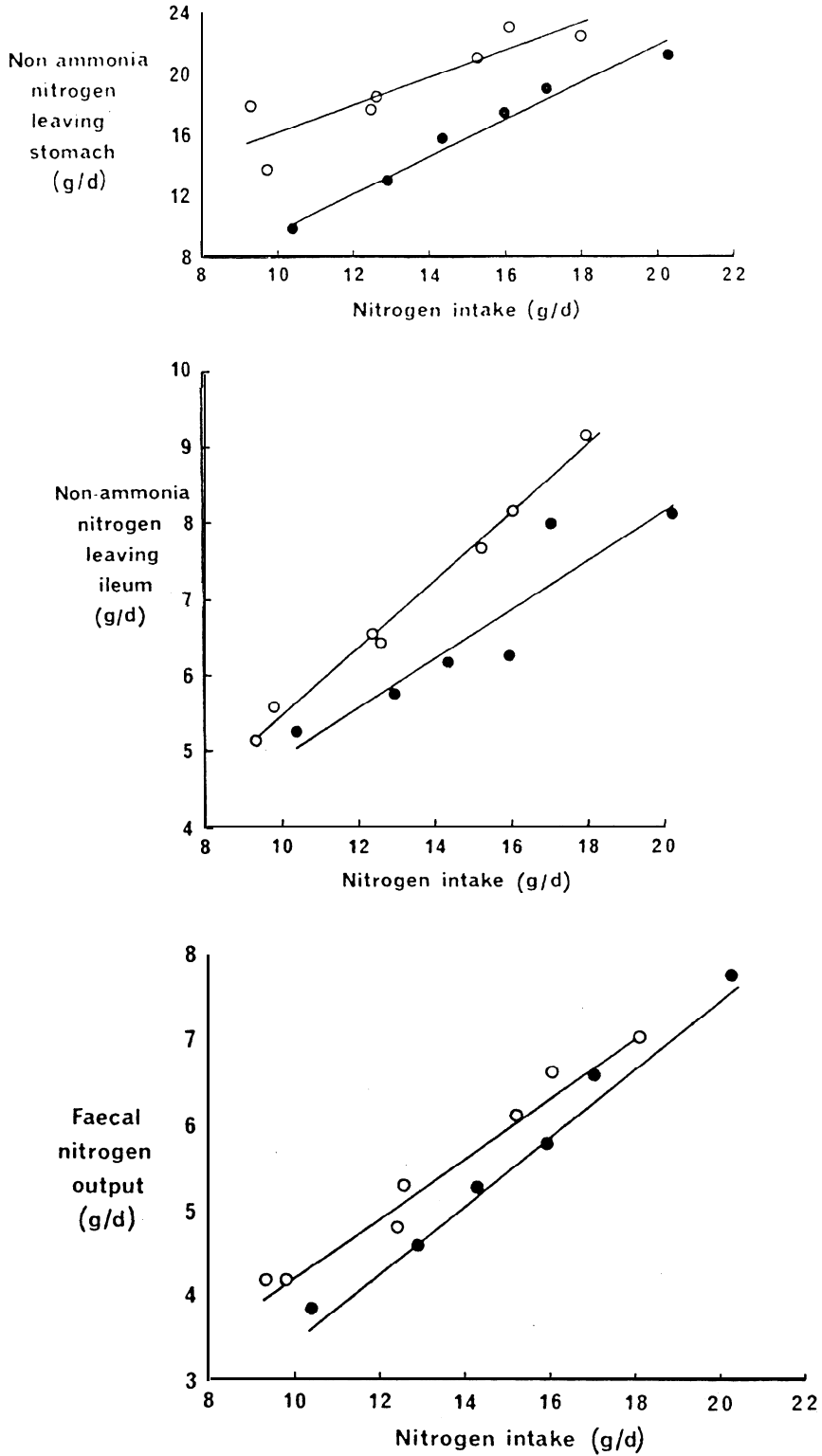


FIG. 3. Effect of *O. circumcincta* infection on nitrogen movement through the gastrointestinal tract of sheep: (O) infected; (●) worm-free.

they are mainly due to increased endogenous nitrogen loss at the site of infection rather than reduced digestion and absorption per se. Clearly, in ostertagosis the majority (80%) of the endogenous nitrogen lost into the abomasum is reabsorbed during passage through the small intestine. In trichostrongylosis it appears that incomplete reabsorption of endogenous nitrogen from the small intestine occurs at higher nitrogen intakes, probably when the capacity for reabsorption is exceeded.

The above results suggest that the presence of T.colubriformis in the small intestine may impede reabsorption of endogenous nitrogen lost into the abomasum infected with O.circumcincta, while adding to total endogenous nitrogen loss. Thus they emphasise the need for caution when applying the results of experimental infections with individual parasites to the field situation in which sheep normally acquire a number of parasite species simultaneously. In the latter instance, effects due to one species may be exacerbated by the presence of another. Clearly, further investigation is warranted to determine whether the combined effect of O.circumcincta and T.colubriformis on nitrogen uptake from the small intestine is greater than the sum of effects of the individual infections.

Net uptake of calcium and phosphorus by the gastrointestinal tract has been shown to be impaired in young growing lambs infected with T.colubriformis (Sykes and Coop 1976) and O.circumcincta (Sykes and coop 1977). Studies of the movement of mineral elements through the gastrointestinal tract in mature sheep suggest that net uptake of calcium and phosphorus in the small intestine is reduced following infection with T.colubriformis and no compensatory increase in absorption occurs in the large intestine (Steel and Hennessy, unpublished). In O.circumcincta infections there was little change in the pattern of movement of calcium and phosphorus through the gastrointestinal tract. Substantial increases in the amounts of sodium, potassium and chloride flowing from the abomasum were recorded in O.circumcincta infections and of sodium and potassium at the ileum in T.colubriformis infections. However, in both infections complete reabsorption of these minerals occurred and faecal excretion was unchanged.

V. KINETICS OF PLASMA PROTEIN METABOLISM

The development of a varying degree of hypoalbuminaemia in experimental and field infections with helminths has been extensively documented. Total protein concentration may remain relatively constant due to elevation of globulin levels. Radiotracer techniques have clearly demonstrated in a number of helminth infections of sheep and cattle that the development of hypoalbuminaemia is associated with a substantial increase in the turnover rate of the plasma albumin pool (see Table 2). The size of the plasma pool decreases, but irreversible loss, which equals synthetic rate of albumin under steady state conditions, is generally increased. The increased turnover of albumin has been shown to be closely related in time with increased loss of plasma into the gastrointestinal tract.

There is a limited amount of evidence which indicates that the rate of synthesis of plasma immunoglobulin-G is substantially increased, particularly on exposure of resistant sheep to challenge infection (Cripps and Steel 1978). However, this response may not be associated with plasma leakage.

TABLE 2. Albumin metabolism in helminth infections of sheep and cattle

Host	Parasite	*Group	Plasma conc. (g/100ml)	Plasma pool size (g/kg)	Turn-over rate (%/d)	Irreversible loss (mg/d/kg)	Reference
<u>Sheep</u>							
	<u>H. contortus</u>	I	1.87	0.93	13.8	128	Dargie (1975)
		C	2.63	1.17	8.3	97	
	<u>O. circumcincta</u>	I	2.53	1.27	14.2	180	Holmes &
		C	3.38	1.46	6.2	91	MacLean (1971)
	<u>T. colubriformis</u>	I	2.48	1.89	14.6	275	Symons, Steel &
		C	3.58	2.13	7.6	158	Jones (unpub.)
	<u>F. hepatica</u>	I	1.66	0.80	14.0	112	Dargie (1975)
		C	2.54	1.08	5.4	58	
<u>Cattle</u>							
	<u>O. ostertagi</u>	I	1.44	0.77	10.4	80	Halliday,
		C	2.67	0.98	6.7	65	Mulligan & Dalton (1968)

* I = infected; C = control

VI. NUTRIENT UTILISATION

Although the pattern of digestion and absorption is altered in infected sheep, these changes do not necessarily cause an overall change in uptake or digestibility of dietary constituents. On the other hand, utilisation of nutrients for growth and wool production is usually impaired. Elevated plasma urea levels, increases in the rate of production of urea and rate of urinary urea excretion indicate a detrimental effect on the nitrogen economy of sheep infected with T. colubriformis not attributable to reduced food consumption alone (Roseby and Leng 1974). Increased urinary nitrogen excretion has also been demonstrated with infections of H. contortus (Dargie 1973) and O. circumcincta (Parkins, Holmes and Bremner 1973; Sykes and Coop 1977). Depression of nitrogen retention may markedly reduce deposition of carcass protein in growing animals, even at low levels of infection. For example, the protein content of the gain in empty body weight in young lambs infected with 2,500 T. colubriformis larvae daily for 14 weeks was 80 g/kg compared with 112 g/kg and 124 g/kg in pair-fed and ad lib.-fed; uninfected lambs respectively- (Sykes and Coop 1976). In the same experiment the calculated gross efficiency of utilisation of metabolisable energy (ME) for growth was 13% in infected lambs compared with 24% and 21.6% in the uninfected groups. Conversely, in growing lambs infected with 4,000 O. circumcincta larvae daily the depression in protein deposition could be accounted for by reduced food consumption alone (Sykes and Coop 1977). However, there was again a profound effect of parasitism on energy utilisation; the gross efficiency of utilisation of

ME for growth was 14%, 19% and 20% in infected, pair-fed and ad lib. groups respectively.

Studies of amino acid incorporation into protein in sheep infected with T.colubriformis showed that most of the reduced rate of muscle protein synthesis was due to depressed food consumption (Symons and Jones 1975). Incorporation of amino acid into protein by wool follicles was also severely depressed in the infected sheep. Although liver protein synthesis was apparently elevated, evidence was equivocal. Previous studies in the guinea pig infected with T.colubriformis showed that amino acid uptake by liver ribosomes responsible for synthesis of circulating plasma proteins, was stimulated in response to increased albumin turnover rate and loss into the intestine (Symons, Jones and Steel 1974). Preliminary studies of the partition of nutrient utilisation have shown that in guinea pigs infected with T.colubriformis a greater proportion of injected ^{14}C -leucine was taken up by the gut and liver than in uninfected animals (Symons and Jones, unpublished).

Reduced retention of calcium and phosphorus is associated with severe reduction in the deposition of these elements in the skeleton and reduced bone growth in young lambs infected with either T.colubriformis (Sykes, Coop and Angus 1975; Sykes and Coop 1976) or O.circumcincta (Sykes, Coop and Angus 1977; Sykes and Coop 1977). Although these changes may stem primarily from mineral deficiency due to reduced uptake by the gastrointestinal tract, it is possible that reduced availability of protein and energy for bone synthesis may also limit skeletal development. As Sykes *et al.* (1977) emphasise, reduced skeletal growth in the young animal has long-term implications for production since skeletal size, in part, determines the capacity of the growing animal to accumulate muscle.

Further quantitative studies of the utilisation of nutrients by the tissues of sheep and cattle infected with gastrointestinal parasites are clearly needed to fully understand the pathogenesis of production loss. However, there is now a substantial weight of evidence which indicates that anorexia and changes in the patterns of digestion and metabolism impair utilisation of nitrogen and energy for synthesis of muscle and wool, due probably to reduced availability of nutrients at these sites. Increased amino acid and energy requirements by other tissues, particularly the gut and liver, to synthesise proteins being turned over at an increased rate seems to be the major factor limiting nutrient availability for muscle and wool protein synthesis. This view has been substantiated by preliminary experiments at this laboratory (Steel and Hennessy, unpublished) which indicate that the requirement for sulphur-containing amino acids is increased by about 50% in sheep infected with T.colubriformis. Since these amino acids are essential for wool protein synthesis, this observation helps to explain the disproportionate effect of trichostrongylosis on wool growth.

VII. EFFECTS OF PLANE OF NUTRITION

From the preceding observations it is reasonable to surmise that plane of nutrition would be a major determinant of the host's response to parasitism. Such an effect could be mediated in two ways. Firstly, nutritional status could influence the immunological competence of the host with consequent effects on the establishment and subsequent development of a parasite population. Secondly, through its influence on the host's ability to repair damaged tissues and maintain metabolic

processes at higher levels associated with the presence of an established parasite burden, nutritional status would be expected to influence the pathogenicity of an infection.

It is only recently that these questions have been examined in detail, but the limited evidence available suggests that both mechanisms may be important. For example, following a single, primary infection of Oe.columbianum, sheep which had previously been maintained on a ration containing 7% crude protein were found to harbour a larger population of adult worms than sheep given a ration containing 19% crude protein (Dobson and Bawden 1974). Although there was no difference in the number of larvae which became established in sheep on the higher plane of nutrition, more worms were eliminated, larval development was arrested to a greater degree, egg output occurred later and fewer eggs were produced per female worm than on the low-protein diet. From a detailed histological examination of the gut made by Dobson and Bawden (1974) it is apparent that the increased susceptibility of malnourished sheep infected with Oe.columbianum was associated with reduced proliferation of some of the cell types which have been implicated in the host's immune response to gastrointestinal nematode parasites.

Studies with H.contortus in lambs suggest that low-protein diets may be conducive to greater establishment and fecundity of this parasite also (Kates and Wilson 1955; Poeschel and Todd 1969). On the other hand, Berry and Dargie (1976) found no difference in the adult fluke burdens of lambs maintained on diets of different protein content when given the same dose of metacercariae. However, animals on the low-protein (6%) diet developed anaemia, hypoalbuminaemia and lost weight more rapidly and died earlier than sheep on the high-protein (13%) diet. In view of the similar fluke burdens these differences must reflect a greater capacity of sheep on a higher plane of nutrition to withstand the pathogenic effects of the parasite rather than a superior ability to limit the level of infection. Chronically infected animals also develop clinical symptoms of fascioliasis more rapidly when switched from a high to a low protein diet. (Berry and Dargie 1976). Similarly, in field studies with H.contortus, Allonby and Urquhart (1975) found that chronically infected ewes, which had hitherto appeared clinically normal in spite of some evidence of anaemia, lost weight and became severely anaemic with some deaths when pasture quality deteriorated markedly. This effect was noted with relatively low burdens and was not due to an increased larval pick-up from pasture.

More work is necessary before the interaction between parasitism and plane of nutrition is understood, but it is clear that nitrogen intake is an important determinant of the host's response to gastrointestinal helminth infection. In view of the virtually continuous intake of helminths by grazing animals the extent to which nutrient requirements are elevated during parasitic exposure of both susceptible and resistant sheep and cattle is an area of research that deserves high priority.

VIII. CONCLUSIONS

This review has examined those functional disturbances particularly relevant to an understanding of the aetiology of production loss in sheep and cattle with gastrointestinal helminthiasis. Current evidence indicates that helminth infection causes a sequence of metabolic effects producing a syndrome analogous to undernutrition. Apparently the major effect is on protein metabolism with resultant changes in the partition

of nutrient utilisation. This probably stems directly from the increased loss of plasma at the site of infection, which is reflected in an increased rate of catabolism of plasma proteins. In the case of blood-sucking helminths, increased degradation of haemoglobin represents a further protein loss. To maintain normal circulating levels of these proteins parasitised animals must increase their rates of synthesis by diverting a greater proportion of amino acid and energy supplies to this purpose. Although some, if not all, of the protein loss into the gut may be reabsorbed and reutilised, this is clearly wasteful in terms of the nitrogen and energy economy of the host. Repair to damaged gut tissues is probably a further drain on the nutrient supply, but little is known of the quantitative significance of this effect. Alteration in the partition of nutrient supply for different body functions is presumably a major factor limiting protein synthesis by muscle cells and wool follicles. Obviously, these effects will be exacerbated by a depressed food consumption.

Clearly, a full understanding of the aetiology of production loss in helminthiasis of ruminants necessitates further study of the mechanisms which control nutrient utilisation by various tissues. Much work is needed to determine the reasons for appetite loss, one cause of which may be alterations in protein and energy metabolism. Above all, an examination of the increased nutrient requirements of parasitised sheep and cattle is needed. It is conceivable that at lower levels of infection impaired productivity may be alleviated by even minor improvements in the plane of nutrition.

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