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Nitrogen transactions in the digestive tract of lambs exposed to the intestinal parasite, *Trichostrongylus colubriformis*

BY D. P. POPPI*, J. C. MACRAE†, A. BREWER AND R. L. COOP‡

Rowett Research Institute, Bucksburn, Aberdeen AB2 9SB

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1. Ten 5-month-old lambs (29 (SE 1·2) kg), reared parasite-free and prepared with rumen duodenal and ileal cannulas, were paired and given rations of Ruminant Diet AA6 (90 g/kg live weight$^{0.75}$) by means of continuous feeders. From 6 months of age one of each pair was dosed daily with 2500 *Trichostrongylus colubriformis* larvae for 14 weeks. Untreated animals received the amount of ration consumed by their infected pair-mates the previous day.

2. During three periods, ((1) the week before and the first 2 weeks of dosing with infected larvae, (2) during weeks 5–7 and (3) during weeks 11–13 of dosing) all lambs underwent a series of experiments to determine their nitrogen balance, the amounts of N leaving the small intestine, the amount of $^{51}CrCl_3$-labelled plasma protein leaking into the small intestines, and the disappearance of ^{35}S-labelled bacteria from the small intestine.

3. The infection caused varying degrees of feed refusal in all infected animals. As a result the values for N balance and for the flow of N at the ileum during the latter two periods were regressed against dry-matter intakes for each group in each period.

4. The infection caused a reduction ($P < 0·05$) in N retention and increased ($P < 0·05$) flow of N at the ileum. The increase in N flow at the ileum of infected lambs was greater ($P < 0·01$) at weeks 11–13 of dosing (infected – control 3·6 g N/d (standard error of difference (SED) 0·57), $P < 0·01$) than at weeks 5–7 of dosing (infected – control 1·5 g N/d (SED 0·57), $P < 0·05$).

5. There were no between-treatment or between-period differences in the disappearance of ^{35}S-labelled bacteria from the small intestines of infected or control lambs, but the infection did cause an increase in plasma N leakage during both periods. During weeks 5–7 and 11–13, plasma N leakage in infected lambs was 1·1 g N/d ($P < 0·01$) and 1·7 g N/d ($P = 0·056$) respectively higher than that in the control lambs.

6. A proportion of the endogenous secretions which enter the small intestine is likely to be resorbed before the ileum. It was calculated that to account for the extra non-ammonia-N (NAN) flow at the ileum up to 3–5 g NAN/d during weeks 5–7 of dosing and 15–20 g N/d during weeks 11–13 of dosing could have entered the small intestine as mucin and sloughed cells.

7. The results seem to indicate that the nutritional penalty associated with the development of resistance to infection is greater than that associated with the primary infection.

Lambs infected with the small intestine roundworm, *Trichostrongylus colubriformis* (TC), excrete more urinary nitrogen and exhibit a poorer N retention than do pair-fed control lambs (Sykes & Coop, 1976*a*; Steel *et al.* 1980). The roundworms, TC, lodge in the proximal section of the small intestine where marked histological changes in the mucosa are then observed (Sykes & Coop, 1976*b*). Such changes in cellular structure might affect the digestion and absorption of nutrients, including protein. Steel (1974) observed an increase in non-ammonia-nitrogen (NAN) flow from the ileum of infected lambs and Sykes & Coop (1976*a*) postulated that if this occurred then the extra urinary N excreted in infected animals could be the result of this protein being fermented in the large intestine. Extra ileal NAN could arise from a reduction in the availability of amino acids entering the small intestine or enhanced endogenous secretions. Symons & Jones (1970) reported that the true digestibility and absorption of protein (^{14}C-labelled *Chlorella* protein) in the small intestine was not affected by TC parasitism, whereas Steel *et al.* (1980) observed an increased leakage of plasma protein into the gastrointestinal tract of infected lambs.

Present addresses: * Department of Animal Science, Lincoln College, Canterbury, New Zealand. ‡ Moredun Research Institute, Gilmerton Road, Edinburgh EH1 7JH.

† For reprints.

The present study was designed to examine the effect of repeated daily doses of TC larvae on the movement of NAN within the small intestine. In particular, we wished to determine whether extra NAN flowed past the ileum of infected lambs and, if so, whether this resulted from a reduced digestion and absorption of protein from the small intestine or an increased endogenous secretion of N into the small intestine or both. A preliminary report of this work has appeared (Poppi *et al.* 1981).

MATERIALS AND METHODS

The animals used, diets fed and experimental procedures adopted were the same as those described previously when the effects of infection on calcium and phosphorus metabolism of these animals were reported (Poppi *et al.* 1985). Briefly, the details were as follows.

Animals

Ten 5-month-old Suffolk × Finn Dorset castrate lambs (average weight 29 kg) were each cannulated at the rumen, proximal duodenum and terminal ileum with simple 'T'-shaped cannulas (Hecker, 1974). From 6 months of age they were paired on the basis of their live weight and one of each pair was given orally a daily dose of 2500 larvae of TC suspended in 10 ml water for 14 weeks.

Feeding

Initially all animals consumed a daily ration of Ruminant Diet AA6 (details of the source, composition and nutritive value of Ruminant Diet AA6 have been reported by Wainman *et al.* (1975); the N content of this particular batch was 24·9 g/kg dry matter (DM)) at the level of 90 g/kg metabolic body size ($W^{0.75}$). When, from week 3 of dosing, the infected lambs started to leave feed, their respective pair-mates were offered the quantity consumed by the parasitized lamb on the previous day.

The animals were housed in metabolism cages and exposed to continuous artificial light for the duration of the experiment. Food was supplied from continuous-belt feeders.

Experimental procedures

The experiment was of 15 weeks duration and consisted of three main experimental periods: (1) the week preceding and the first 2 weeks of dosing (i.e. weeks 0–2), (2) weeks 5–7 of dosing, and (3) weeks 11–13 of dosing. The experimental procedures during each of these periods are summarized in Table 1. In addition, live weight, leucocyte counts and faecal egg counts were monitored at regular intervals over the experimental period.

Amounts of N leaving the small intestine

The technique used for measuring the flow of digesta at the ileum has been described in detail by Poppi *et al.* (1985). Basically 10 μCi ^{103}Ru-P/d and 40 μCi ^{51}CrEDTA/d were infused continuously into the rumen of all sheep. Digesta samples (three four-hourly samples, 75 g/sample) were obtained from the ileum on days 5 and 7 of the infusion. Composite 24 h samples were prepared by bulking these individual samples on an equal weight basis within days. Chemical analysis of the composite samples was performed as well as measurement of radioactivity. Flow-rates of N and NAN were calculated as described later.

True digestibility of ^{35}S-labelled bacteria. The technique was similar to that described by Salter & Smith (1977). The ^{35}S-labelled bacteria were prepared from two adult sheep offered a ration of 1 kg Ruminant Diet AA6/d in two equal feeds. $Na_2{}^{35}SO_4$ (1 mCi) was infused continuously, into the rumen of each sheep, over a 4 d period. On the 4th day of infusion and 2–3 h after the morning feed approximately 2–3 litres rumen fluid were obtained from

Table 1. *Chronological sequence of experimental procedures*

Day of experiment	Procedure
1	Inject ^{35}S-labelled bacteria plus ^{51}CrEDTA into duodenum. Collect ileal digesta when ^{51}Cr detected
3	Start continuous infusion of ^{103}Ru–P plus ^{51}CrEDTA. Also start 5 d faecal and urine collections
8	Ileal collection
9	Duodenal collection
10	Ileal collection
11	Duodenal collection. Infusion stopped after last sample
12, 13, 14, 15	Faecal samples taken at regular intervals to measure outflow rate of ^{103}Ru and ^{51}Cr from rumen
16	$^{51}CrCl_3$ injection into jugular vein
17, 18, 19, 20, 21	Jugular blood and faeces sampled daily to determine plasma leakage from ratio, ^{51}Cr in jugular blood: ^{51}Cr in faeces

each animal by suction. This was strained through gauze before centrifugation (2200 ***g*** for 2 min). The supernatant fraction and the top residue layer were removed and examined microscopically. Bacteria but no protozoa were present. This fraction was then centrifuged at 28000 ***g*** for 15–20 min. The supernatant fraction was discarded and the residue washed by resuspension in isotonic saline (9 g sodium chloride/l) followed by recentrifugation. This washing procedure was repeated twice more. The final residue was frozen, freeze-dried and stored at −20° for 1–2 d before injection into the duodenum of the test animals.

Before this, however, predigestion of the ^{35}S-labelled bacteria was considered necessary to simulate abomasal action. The procedure adopted was that of Mathers & Miller (1980). The bulked, freeze-dried bacteria (approximately 50 g DM) were suspended in 600 ml saline plus pepsin to a final concentration of approximately 500 Anson units/ml (Ben Ghedalia *et al.* 1978). The pH was adjusted to 2·75 by addition of hydrochloric acid. The mixture was then incubated with gentle agitation at 39° for 2 h.

A small subsample (approximately 10 ml) was removed as a control standard and the remainder was mixed with approximately 600 μCi ^{51}CrEDTA. Twelve samples, each approximately 50 ml, were taken by syringe. Two were retained as standards and the others were slowly injected (approximately 10 ml/min) into the duodenum via the fistula of each of the ten experimental sheep.

Starting 105 min after injection, successive 50 g samples of digesta were obtained from the ileal cannulas. Records were kept of the time at the start and end of each collection and samples were assayed immediately for ^{51}Cr. Those samples without ^{51}Cr were discarded and after the first appearance of ^{51}Cr (113–275 min after injection, depending on intake of DM by sheep) two to four samples were taken over the next 3 h and ^{51}Cr content (counts/min per g digesta) determined. The samples were then frozen and analysed later for ^{35}S content.

The ^{35}S content of the ileal digesta and the two standard infusates were determined on triplicate samples by the technique of Mahin & Lofberg (1966) except that sample size was increased to 0·5 g digesta (the other reagents were increased accordingly; 0·5 ml perchloric

acid and 1 ml hydrogen peroxide) and a 10 h digestion procedure in an oven set at 65° was used to alleviate incomplete recovery of ^{35}S found over the shorter digestion periods. This final solution was mixed with 20 ml NE260 (Nuclear Enterprises Ltd, Edinburgh) and β-emission determined, with suitable allowance being made for ^{51}Cr radiation.

The ratio ^{35}S:^{51}Cr in the infusate and in the ileal digesta samples was determined and true digestion and absorption of ^{35}S-labelled bacteria determined as:

$$\frac{\text{ratio in infusate} - \text{ratio in ileal digesta}}{\text{ratio in infusate}}.$$

It was assumed that all the ^{35}S in the infusate was in an organic form and that negligible recycling occurred over the period of collection.

Plasma protein loss into the digestive tract. The technique of Van Tongeren & Majoor (1966) as modified by Holmes & MacLean (1971) was used. Basically 300 μCi $^{51}CrCl_3$ contained in 10 ml sterile saline was injected into the jugular vein and the radioactivity and protein concentration of the plasma determined in samples of blood withdrawn by vacupuncture at 09.00 hours each morning for the following 5 d. The total daily amounts of ^{51}Cr excreted in the faeces were determined over the same period. The plasma lost into the digestive tract (ml/d) was determined as the total daily excretion of ^{51}Cr (counts/min) in the faeces divided by the plasma radioactivity (μCi/ml) at the beginning of the 24 h period and this was converted to protein loss (g/d) based on the plasma protein concentration on each day.

Determination of radioactivity. ^{51}Cr and ^{103}Ru were assayed on a Tracerlab T-52 scintillation detector (Mechelen, Belgium). ^{35}S activity was counted on a Packard β-counter.

N analyses. Digesta and digesta supernatant fraction obtained after centrifugation (12000 *g* for 20 min at 0–4°) were analysed for N content by a Kjeldahl procedure (Davidson *et al.* 1970) and NH_3-N (supernatant fraction only) by an autoanalyser technique (Fawcett & Scott, 1960).

Data compilation analyses

Calculation of ileal NAN flow-rates. The flows of NAN at the ileum were calculated by the two-phase-marker procedure described by MacRae *et al.* (1979). Although this procedure is less rigorous than the dual-phase-marker technique of Faichney (1975), recent comparisons made with sheep given a ration of chopped dried grass in the experiments described by MacRae *et al.* (1985) indicate that flow-rates of N at the ileum calculated by the present two-phase-marker procedure were only 1·8 (standard error of difference (SED) 0·66, *n* 15) % higher than those calculated using the Faichney procedure.

Calculation of DM intake and marker infusion rate on day of ileal sampling. Where animals are given *ad lib.* access to feed and the actual intakes vary from day-to-day it is necessary to calculate a 'DM intake which relates to the day of ileal collection'. Similarly, if there is any day-to-day variability in the output of the infusion pumps which administer the ^{103}Ru and ^{51}Cr markers it is necessary to calculate an 'infusion rate' which relates to the day of the collection. In this experiment the daily intakes for each duodenal and ileal collection and the daily infusion rates of ^{103}Ru and ^{51}Cr, where day-to-day variability of the pumps made this necessary, were calculated from the weighted mean of intake and infusion rates on the day of collection and the previous 3 d by the procedure used by MacRae *et al.* (1985). Briefly, when all duodenal and ileal collections had been made the infusion pumps were stopped and the faecal samples were taken at regular intervals to determine the fractional outflow rate of ^{103}Ru and ^{51}Cr from the rumen of each sheep (Grovum & Williams, 1973). A 'DM intake' for each day of collection was then calculated from the equation:

$$\sum_{i=1}^{4} A_i e^{-mt_i} \div \sum_{i=1}^{4} e^{-mt_i},$$

where m is the fractional outflow rate (/h) of ^{103}Ru from the rumen of each sheep; A_1, A_2, A_3 and A_4 are the actual daily intakes of the sheep on the day of collection, the previous day, 2 d previously and 3 d previously respectively, and t_1, t_2, t_3 and t_4 are 0, 24, 48 and 72 h respectively. 'Infusion rates' for ^{103}Ru were calculated from the same equation by substituting infusion rates for A_1–A_4. Infusion rates of ^{51}Cr were calculated similarly with m = fractional outflow rate of ^{51}Cr.

Statistical methods

A range of statistical techniques have been used in the analysis of the results according to the objectives of the analysis and the nature of the data. From weeks 3–4 of dosing there were considerable differences in the voluntary intakes of infected lambs and so for the N balance and flow measurements (Table 2, Figs. 1 and 2) values from periods 5–7 weeks and 11–13 weeks were subjected to covariance analysis on DM intake, partitioned into between- and within-animal strata of variation with independent estimation of covariate regressions in each strata. It was intended that the covariance would account for between-pair differences in the between-animal variation but in the cases of N retention and N leaving the small intestine there was some evidence of pair-effects over and above that explained by the covariance and in these cases a term for pair-effects was included in the analysis. SED between control and infected animals within either period were based on a combination of between- and within-animal variation and since estimation of missing values was also involved in the analysis the values given are approximate.

The digestion of ^{35}S-labelled bacteria (Table 3) was subjected to a one way analysis of variance within each observation period and the SEDs are based upon the pooled within-group variation.

The plasma leakage values (Table 4) exhibited differences in variation between the two treatment groups and differences between treatment means within each period were assessed by means of the non-parametric two-sample Mann–Whitney U test. Tempo changes in the infected animals were assessed by t tests on the within-animal differences.

RESULTS

Feed intake and clinical observations

Dosing with larvae caused varying degrees of inappetence after the first 3–4 weeks. Details of the intakes of the individual lambs and the excretion of parasite eggs in faeces of infected animals have been reported by Poppi *et al.* (1985). Total worm counts at post mortem, i.e. week 16 of dosing, ranged from 0 to 10800 live worms in the upper third of the small intestine of infected animals.

N digestion

There were no between-treatment differences in any of the variables examined during the first experimental period (i.e. weeks 0–2) and as these values were not significantly different from those presented for control animals in periods 2 and 3 they are not presented separately here.

Figs. 1 and 2 illustrate the observed N balances and ileal N flow rates respectively for infected and control animals during period 2 (weeks 5–7 of dosing) and period 3 (weeks 11–13 of dosing). Figs. 1 and 2 include an analysis of variance carried out at the adjusted treatment mean DM intake (i.e. 730 g DM/d). Similar analyses which identify differences in faecal and urinary excretion of N and the flows of NAN at the ileum are given in Table 2.

Infected lambs retained less N ($P < 0{\cdot}05$; see Fig. 1) and had higher ileal flows of N ($P < 0{\cdot}01$; see Fig. 2) than did control lambs. The flow of NAN at the ileum of infected

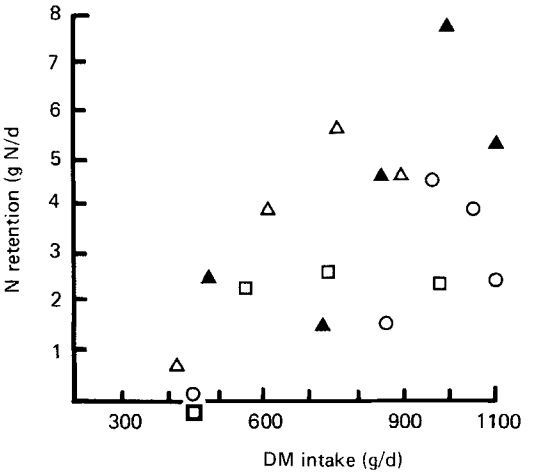

Fig. 1. Relations between nitrogen balance and dry matter intake (DMI) in *Trichostrongylus colubriformis*-infected lambs during weeks 5–7 of dosing (○) and weeks 11–13 of dosing (□) and in pair-fed control lambs during the corresponding periods (△, weeks 5–7; ▲, weeks 11–13). Between-animal covariance analysis on DMI: treatment means at mean DMI of 727 g/d, N balance of control group 3·60 g N/d, of infected group 1·80 g N/d, SED (3 df), 0·35 ($P < 0·05$); covariate regression coefficient 0·0088 (SE 0·0021). Periods and period × treatment interactions were not significant based on within-animal variation.

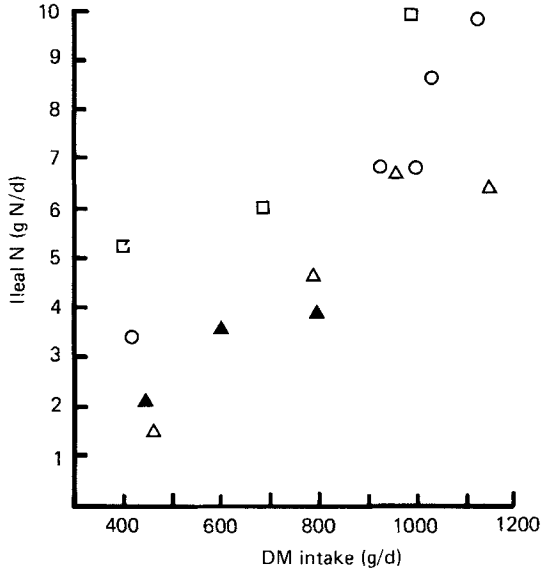

Fig. 2. Relations between nitrogen leaving the small intestine and dry matter intake (DMI) in *Trichostrongylus colubriformis*-infected lambs during weeks 5–7 of dosing (○) and weeks 11–13 of dosing (□) and in pair-fed control lambs during the corresponding periods (△, weeks 5–7; ▲, weeks 11–13). Covariance analysis on DMI: adjusted means at DMI of 731 g/d:

	Control	Infected
5–7 weeks of dosing	5·83	7·39*
11–13 weeks of dosing	6·44	10·02†

SE of difference (SED) of comparisons across columns (approximately 5 df) 0·57, SED of comparisons within columns (3 df) 0·40. * Infected value was significantly greater than control value at 5–7 weeks ($P < 0·05$). † Infected value at 11–13 weeks was significantly higher than all other means ($P < 0·01$).

Table 2. *Differences between* Trichostrongylus colubriformis-*infected and control lambs in the excretion of nitrogen (g/d) in faeces and urine and the flow of nitrogenous components past the terminal ileum*

(The values obtained are differences of adjusted treatment means after covariance on dry matter intake)

Period of infection (weeks)	Faecal N	Urinary N	Total ileal N	Total ileal NAN
5–7	1·20	1·09	1·51*	0·42
11–13	0·38	0·97	3·58**	2·42*
Approximate SE	0·805	0·805	0·570	0·521

NAN, non-ammonia-nitrogen.
* $P < 0·05$, ** $P < 0·001$.

Table 3. *True digestion and absorption of* ^{35}*S-labelled bacteria in the small intestine of* Trichostrongylus colubriformis-*infected lambs*

Period of infection (weeks)...	Pre-infection	5–7	11–13
n...	5	5	4
Parasite	0·71	0·71	0·66
Control	0·71	0·71	0·69
SE of difference	0·035	0·044	0·014

Table 4. *Plasma protein-nitrogen (g/d) losses into the digestive tract of* Trichostrongylus colubriformis-*infected lambs*

Period of infection (weeks)...	Pre-infection	5–6	11–12
n...	5	5	4
Control	0·53	0·53	0·54
Parasite	0·69	1·63	2·22
SE of difference	0·122	0·430	0·497
Difference relative to pre-infection	—	0·93*	1·48*
SE	—	0·32	0·32

* $P < 0·05$.

lambs was not significantly higher than that for control lambs during weeks 5–7 of dosing (difference 0·4 g N/d; SED 0·52) but was significantly higher ($P < 0·05$) during weeks 11–13 of dosing (difference 2·4 g N/d; SED 0·52) (see Table 2).

At the adjusted treatment mean DM intake (DMI) the N leaving the small intestine of infected lambs represented 127% (weeks 5–7 of dosing) to 155% (weeks 11–13 of dosing) of that leaving the small intestine of control lambs. Corresponding values for NAN were 107 to 137%.

The infection did not appear to alter the disappearance of ^{35}S-labelled bacteria across the small intestine (see Table 3). It did, however, increase the amount of plasma protein which leaked into the tract (see Table 4). Plasma leakage in infected lambs was 0·93 g N/d higher ($P < 0·05$) than before infection by week 6 of dosing and 1·48 g N/d higher ($P < 0·05$) by week 12 of dosing.

Duodenal samples were obtained from control and infected lambs during each phase of the experiment but those collected from infected lambs showed signs of severe contamination with bile contents (i.e. they were bright green) and mucus and so had to be discarded. The pH of these samples was higher (weeks 5–7 of dosing, mean pH 3·4, range 2·4–4·2; weeks 11–13 of dosing, mean pH 4·0, range 2·8–6·5) than in control sheep (mean pH 2·5, range 2·1–2·8).

DISCUSSION

The general response to daily dosing with subclinical levels of larvae observed in the present study, i.e. the varying degree of inappetance (Poppi *et al.* 1985), the rise and then decline in faecal egg counts and the elevation in blood leucocyte counts (mainly eosinophils) (MacRae *et al.* 1982) are broadly in agreement with previous studies (Sykes & Coop, 1976*a*; Roseby, 1977; Steel *et al.* 1980).

N digestion

The findings of increased flows of NAN and NH_3-N at the terminal ileum of infected animals agree with the observations of Steel (1974). The magnitude of these increases were more than sufficient to account for the increased excretion of N in faeces and urine. This extra NAN leaving the small intestine could have resulted from either an overall depression in true digestion and absorption of protein or an increased secretion of endogenous N into the small intestine. Infection did not appear to alter the disappearance of ^{35}S-labelled bacteria across the small intestine (Table 3), a result in accord with Symons & Jones (1970) who used ^{14}C-labelled *Chlorella* protein. Their results were obtained in a number of different animal species but the comparable sheep values were obtained using animals fed at the maintenance level of intake and given massive single (40000) or multiple (60000 in three equal daily amounts) doses of larvae. The present results were obtained from lambs receiving 'trickle-doses' of larvae and consuming a wide range of daily DMI (450–1100 g DM/d) and over a longer period of infection. The values obtained from both infected and control lambs in the present study (0·66–0·71) agree closely with values obtained by Elliott & Little (1977) in mature wethers (0·72 (SE 0·004)), but were lower than those obtained by Salter & Smith (1977) in young calves (20–30 weeks of age). TC larvae affect the proximal section of the small intestine and in this area it might be expected that infection would interfere with digestion and absorption. However, Ben Ghedalia *et al.* (1974) observed that most protein disappears in the mid-portion of the small intestine. Armstrong & Hutton (1975), in reviewing this subject, noted that the optimum pH requirement for enzyme proteolysis was 7·5–8·8 and so areas distal to the site of infection have the potential to degrade protein and absorb amino acids. Recently, Bown *et al.* (1984) have reported that studies with ^{125}I-labelled albumin indicate that most of the protein absorption in control and parasitized animals normally occurs distal to the site of parasite habitation.

If digestion and absorption of amino acids from the small intestine was unimpaired by infection it follows that the extra amount of NAN leaving the small intestines of infected animals at 5–7 weeks of dosing must have come from endogenous protein secretions, either plasma leakage or the addition of mucus and sloughed cells to the digestive tract.

The results on plasma leakage in this experiment agree closely with values obtained previously (Dargie, 1979; Steel *et al.* 1980; i.e. plasma losses in infected animals ranged from 100 to 300 ml/d or 1·6 to 2·2 g N/d and these were three to four times greater than those in control animals). However, Bown *et al.* (1984) have recently reported that ^{125}I-labelled albumin, introduced at the abomasum of the infected lambs, is absorbed from the small intestine with a coefficient of 0·87. If the plasma protein which leaked into the small intestine of the infected lambs in the present study was resorbed to this same high degree then only 0·13 of the amount entering the tract would contribute to the extra NAN flow (i.e.

0·12 g N/d). This would require that the remainder of the extra ileal NAN (0·3 g NAN/d during weeks 5–7; 2·3 g NAN/d during weeks 11–13) arises from other endogenous secretions, perhaps either mucins or sloughed cells, or both. Lindsay *et al.* (1980) suggested that the resorption of total endogenous protein in normal animals may be as high as 0·85–0·90. If, therefore, a similar value were applicable to the infected lambs this would require that during weeks 5–7 and 11–13 of dosing an extra 3–5 g NAN and 14–21 g NAN/d be secreted into the small intestine to account for the extra non-resorbed endogenous component at the ileum. Even with a resorption factor similar to the true digestibility of ^{35}S-labelled bacterial protein (0·7), the increased endogenous secretions would still amount to 1·7 g NAN/d during weeks 5–7 of dosing and 7 g NAN/d during weeks 11–13 of dosing.

Lindsay *et al.* (1980) have suggested that the amount of endogenous protein-N added to the small intestines of normal animals is approximately 15 g N/d. If this is so then parasitism appears to increase endogenous protein secretion losses by 0·38–1·5 depending on the resorption coefficient used. Of this, the major source of endogenous protein loss would appear to be mucus and sloughed epithelial cells.

The source of the extra NH_3-N at the ileum is unknown. It could arise from diffusion or leakage of plasma urea into the intestines, but whether the rate of this endogenous addition is increased as a result of extra deamination by the parasitic larvae or by any secondary bacterial infection, or both, requires further clarification.

The effect of parasitism on the whole animal metabolism

Elevation of the endogenous protein secretions into the small intestines could have serious consequences on the animal. First, if it results in the animal synthesizing more protein to service this component then there will be an extra energy cost associated with that synthesis. Second, if the lambs are in a N-limiting situation then the incomplete resorption of endogenous secretions will reduce N availability for tissue growth. Such a situation may also lead to an induced specific amino acid deficiency.

It is unlikely that more protein was synthesized because two separate experiments carried out in the current series have failed to demonstrate any significant effect on efficiency of utilization of metabolizable energy in lambs infected with TC (see MacRae *et al.* 1982). It is perhaps more likely therefore that extra protein being secreted in the small intestine is derived from a redirection of protein synthesis away from muscle, etc. to the gut rather than any net increase in protein synthesis *per se*. This in itself would be consistent with the observations of Sykes & Coop (1976*a*) who found that there was a 50% reduction in the amount of carcass protein deposited per unit live-weight gain (i.e. 80 g/kg live-weight gain *v.* 120 g/kg live-weight gain in control lambs) in lambs infected with similar numbers of TC larvae.

It is not possible to present any evidence for or against a specific amino acid deficiency in the present experiment. However, as the absorption of exogenous protein is not affected by parasitism then the increased flow of NAN at the ileum presumably represents endogenous amino acids, mostly epithelial cells and mucus protein. Mucus secretions are high in S amino acids, threonine and histidine (Clarke *et al.* 1966; Lindsay *et al.* 1980; Steel & Symons, 1982) and this could result in a loss of these essential amino acids through incomplete resorption.

Nutritional penalty and stage of infection

One surprising aspect of the present study was the increased severity of the nutritional penalty during the period when the lambs were developing their immunological resistance to the infection. From Tables 2 and 4 it can be seen that the infection had a more pronounced effect on N digestion in the third experimental period (i.e. weeks 11–13 of dosing), when

from the clinical observations it appeared that the animal was resisting the larval challenge. Thus NAN flow at the ileum, plasma leakage and the calculated endogenous non-plasma-leaked protein entering the small intestine were all considerably higher during weeks 11–13 of dosing than they were during weeks 5–7 of dosing. It is not possible from this experiment to determine whether the increased nutritional penalty observed over this period was associated with the mechanism of dispelling the remnants of live worms still attached to the small intestine or with the resistance of the continuous larval challenge. However, observations made in a subsequent study which extended the period of larval challenge beyond 14 weeks seem to indicate that the major nutritional penalty is associated more with the former than the latter type of resistance (A. Kimambo and J. C. MacRae, unpublished results).

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