Short-and long-term effects of intestinal parasitism on growth in sheep

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ABSTRACT

Forty-eight animals were allocated to 1 of 3 treatments (C = continuous growth; I = infected with 4000 larvae/d of *Trichostongylus colubriformis*; P= individually paired with I animals for change in live weight) in a serial slaughter experiment examining the effects of intestinal parasitism on bone and muscle in young growing sheep (25 to 45kg). Both I and P groups were nutiritionally rehabilitated after the I group had undergone an 83 d period of infection. While I and P groups underwent similar changes in live weight during the period of infection, feed intake of the P group became progessively less than that of the I group. By the end of infection I animals were consuming approximately 30% more feed than P animals.

During the period of infection both I and P groups lost live weight. Of the body chemical components, fat was depleted to the greatest degree (48% loss) followed by protein (23% loss). While bone displayed growth in external linear dimensions (length = +4%; diameter = +6%) there were significant reductions in protein (22%) and cortex width (33%). In contrast muscle showed decreases in all chemical components and in diameter. This greater effect on muscle was exaggerated in the I group. Infection led to greater reductions in muscle protein (45% *v* 34% (I *v* P)) and muscle diameter (19% *v* 8%). There were no appreciable differences between the I and P groups for changes in bone variables. While specific effects of infection on growth of muscle were found, change in most variables was largely attributable to the general restriction in growth consequent upon undernutrition.

Subsequent to the period of parasitic infection, compensatory growth occurred such that muscle recovered relative to bone. There was some evidence that full recovery of muscle relative to bone was delayed in animals recovering from a parasitic infection.

Keywords Bone growth; muscle growth; parasitism; *Trichostrongylus colubriformis*; protein.

INTRODUCTION

Gastro-intestinal parasitism reduces voluntary feed intake (Sykes, 1983) and protein (Poppi, *et al.*, 1986) and mineral (Coop *et al.*, 1976) deposition in lambs. The latter authors speculated that changes in the epiphyseal cartilage may be irreversible and permanently retard skeletal growth.

The effects of undernutrition *per se* on growth is well documented (Wilson and Osbourn, 1960; Allden, 1970; O'Donovan, 1984) and permanent retardation of growth is not considered a feature though it has been reported (Gunn, 1977; Allden, 1979; Suttie *et al.*, 1984). In part this may reflect the short term nature of many trials. The possiblity that undernutrition as a consequence of parasitic infection may have different effects has not been investigated.

This experiment was designed to test the hypotheses that restricted growth associated with intestinal parasitism induced by *Trichostrongylus colubriformis* does not differ from that induced by simple undernutrition and that following cessation of infection animals fulfill their potential for growth and normal tissue relationships are regained.

MATERIALS AND METHODS

Forty-eight female sheep (Coopworth x South Suffolk; mean live weight 26.7kg ± 0.7) were selected (time = day1) 4 weeks after weaning at approximately 84 days of age and randomly allocated to 3 treatment groups in a stratified manner based on live weight (LW).

A control (C) group (n=18) was offered high feed allowances at pasture in order to promote rapid uninterrupted growth. Animals in this treatment were slaughtered in groups (n= 16) at live weights of approximately 25kg (day 1), 35kg (day 83) and 45kg (day 143).

The remaining animals were penned individually indoors and offered meadow hay that had been hammermilled (50mm plate aperture). An infected (I) group (n=18) were offered feed *ad libitum* and dosed at 3 to 4 day intervals with *Trichostrongylus colubriformis* larvae (to result in 4000 larvae/animal/d) from day 1 to day 68. On day 83 they were dosed with anthelmintic to remove infection and allowed *ad libitum* access to pasture feed. Groups (n=6) were slaughtered at the end of infection (day 83; approximate 24kg LW) and at live

weights of 35kg (day 176) and 45kg (day 260).

Animals in a third group (n=12) were individually paired with an I animal of similar live weight and offered the same feed at a level to maintain the same LW as their infected pair-mate (group=P). Groups (n=6) of P animals were slaughtered at the end of infection (day 83) and at the same time as the final group of the I treatment (day 260).

Subsequent to day 83, all 3 treatment groups were run together outdoors at pasture. Anthelmintic was administered to C and P animals at fortnightly intervals and to I animals after 83d (fenbendazole, Panacur, Coopers Animal Health NZ Ltd.; ivermectin, Ivomec, MSDAgvet NZ Ltd.). All animals had access to water and mineral salt block throughout the trial.

Feed dry matter offered and refused by individual animals in the P and I treatments was measured at weekly intervals between days 1 and 83. Live weight was measured weekly and appropriate adjustments made to the quantity of feed offered to P animals.

Animals within each slaughter group were slaughtered on the same day. Animals were shorn prior to slaughter. Carcass and non-carcass body sub-

TABLE 1 Comparison of means for the treatment groups slaughtered at approximately 25kg LW. C animals were slaughtered on day 1, while I and P animals were slaughtered on day 83. A pooled standard error of the mean (SEM) is presented. Comparisons with the C mean are made by unpaired *t*-test while comparison of I and P means is by paired *t*-test. Change in variables is expressed for I and P means as the percentage difference from the C mean.

Tissue	Means			SEM	*t*-tests			% change	
	C	I	P		C *v* I	C *v* P	I *v* P	I	P
Gravimetric data									
Pre-slaughter LW	25.0	21.1	20.9	1.5	†	†	NS	-15.6	-16.4
GIT empty	2.60	2.34	1.95	0.16	NS	*	**	-10.0	-25.0
Liver	0.522	0.393	0.314	0.022	**	***	†	-24.7	-39.8
CS fat	2.98	1.56	1.37	0.27	*	***	NS	-47.7	-54.0
CS protein	1.91	1.35	1.49	0.08	***	**	*	-29.3	-22.0
NC fat	1.055	0.644	0.636	0.101	*	**	NS	-39.0	-39.7
NC protein	1.49	1.21	1.19	0.06	*	**	NS	-18.8	-20.1
FEM total	95.3	94.1	97.2	3.8	NS	NS	NS	-1.3	2.0
FEM fat	13.3	25.8	29.2	2.6	**	**	NS	94.0	119.5
FEM protein	19.3	14.9	15.3	0.7	***	**	NS	-22.8	-20.7
FEM ash	28.0	24.4	25.3	1.2	†	NS	NS	-12.9	-9.6
ST total	58.1	35.8	42.3	3.9	***	*	NS	-38.4	-27.2
ST fat	2.59	1.10	1.22	0.24	***	**	NS	-57.5	-52.9
ST protein	13.04	7.17	8.56	0.84	***	**	NS	-45.0	-34.4
Geometric data									
FEM length	139.0	144.7	144.3	1.6	*	†	NS	4.1	3.8
FEM diameter	16.4	17.7	17.1	0.4	†	NS	NS	7.9	4.3
FEM cortex width	2.97	1.93	2.05	0.09	***	***	NS	-35.0	-31.0
FEM volume	71.6	74.8	76.7	3.2	NS	NS	NS	4.5	7.1
ST diameter	23.6	19.1	21.8	0.8	**	NS	*	-19.1	-7.6
ST volume	54.3	33.7	39.5	3.7	***	*	NS	-37.9	-27.3
Muscle:bone relationships(ST:FEM)									
weight :weight	0.609	0.382	0.431	0.030	**	**	NS	-37.3	-29.2
protein :protein	0.675	0.475	0.554	0.036	**	*	NS	-29.6	-17.9
diameter :cortex width	7.98	9.89	10.71	0.41	*	***	NS	23.9	34.2

sections, liver and gastro-intestinal tract (GIT) were weighed at slaughter. Carcasses were allowed to attain a state of rigor and both body subsections stored in a frozen state. Subsequently 4 bones and 3 muscles were dissected from the thawed carcass. Only data for weight, volume and various linear dimensions of the femur (FEM) and *M. semitendinosus* (ST) are reported here. For each animal standard procedures were used to determine the chemical composition of the carcass (CS), non-carcass (NC), FEM and ST.

The significance of differences between treatment means was determined by an unpaired *t*-test except for the comparison of I and P treatments at day 83 which was by paired *t*-test (Snedecor and Cochran, 1980). Relative, or allometric, growth of variables was determined by the method of Kermack and Haldane (1950) and differences between allometric coefficients (b from $Y=aX^b$ determined by *t*-test.

RESULTS AND DISCUSSION

The system of feeding to induce similar changes in live weight for I and P groups during the infection period was successful. P animals consumed progressively less feed than I animals such that their intakes were 30% less for the last 5 weeks of infection (50.8 (±2.04) and 62.8 (±4.33) g DM/kg $LW^{0.73}$/d, for P and I respectively). This indicates that infection led to a decrease in the gross efficiency (LW change/feed intake) with which feed was used for change in LW. This is likely to be associated with increased requirements for energy as a result of increased protein synthesis in the alimentary tract (Symons and Jones, 1981; Poppi *et al*, 1986). The restricted growth displayed by I animals could be partly attributable to this but also to reduced appetite, a common feature of such infections (Sykes, 1983).

Similar decreases in the weights of the internal organs and body chemical components occurred (Table 1). Fat showed the greatest proportional change, followed by protein and water with ash showing the least change. While bone weight showed negligible change, substantial changes occurred in bone chemical components. Weight of fat increased in association with decreases in protein and ash contents. At the same time linear dimensions of bone increased with the notable exception of femur cortical width which decreased markedly. The latter effect has also been demonstrated by Pratt and McCance (1964). Muscle weight was reduced due to considerable losses of all chemical components.

TABLE 2 Allometric growth coefficients for C and I treatment groups. For each treatment, growth was considered in 2 phases, early and late, by combining the 25kg and 35kg LW slaughter groups (early) and the 35kg and 45kg LW slaughter groups (late). Comparisons were made of coefficients for treatments within phases and for phases within treatments using the *t*-test.

X variable	Y variable	C Early	C Late	I Early	I Late	Time period C	Time period I	Treatment Early	Treatment Late
Gravimetric data									
EB fat-free	GIT	1.311	1.610	1.114	1.552	†	***	*	NS
EB fat-free	Liver	0.911	1.725	1.218	0.781	***	*	***	***
CS protein	NC protein	0.958	1.478	0.683	1.161	*	**	**	NS
FEM protein	FEM ash	1.419	1.091	1.171	0.965	*	*	*	NS
ST protein	ST water	1.005	0.994	0.974	0.999	NS	***	***	NS
Geometric data									
FEM length	FEM diameter	1.335	1.153	1.398	1.161	NS	NS	NS	NS
FEM length	FEM cortex width	1.374	1.622	4.634	2.583	NS	***	***	**
FEM length	FEM volume$^{1/3}$	0.825	0.703	1.096	0.938	NS	NS	NS	NS
ST length	ST diameter	1.504	0.781	2.742	2.035	**	**	***	***
ST length	ST volume$^{1/3}$	1.017	0.853	2.102	1.184	NS	***	***	NS
Muscle:bone relationships									
FEM weight	ST weight	1.183	1.928	2.913	1.316	***	***	***	*
FEM protein	ST protein	1.222	1.452	2.187	1.089	NS	***	***	NS
FEM diameter	ST diameter	1.138	1.128	3.936	1.846	NS	***	***	*
FEM cortex width	ST diameter	1.105	0.866	1.187	0.830	NS	NS	NS	NS

Diameter and volume of muscle decreased. Muscle was generally affected to a greater extent than bone though bone cortical width was reduced to a greater degree than muscle diameter. The data support the findings and suggestions of Young (1988) and Young and Sykes (1985) that weight alone does not adequately describe the functional relationship between bone and muscle. The changes outlined here were representative of those occurring in other bones and muscles dissected from the same animals (M.J. Young and A.R. Sykes, unpublished).

Specific effects of parasitism, other than those induced by reductions in feed intake and the overall efficiency with which feed was used can be examined through comparison of I and P data at day 83. (Table 1). I animals had heavier internal organs (liver + 25%, GIT + 20%) which may be associated with the increased protein turnover associated with infection (Symons and Jones, 1981; Poppi *et al*, 1986) and inflammation at the site of infection. Infection may lead to a relatively higher metabolic rate which should lead to increases in the size of these metabolically active organs (Ferrell *et al.*, 1986). Protein was preferentially depleted from the carcass muscle tissue of parasitized animals (45% loss of ST protein *cf.* 34% for P treatment). While this effect was not statistically significant for this muscle, similar effects in 2 other muscles were (M.J. Young and A.R. Sykes, unpublished). There were no specific effects of parasitism on bone variables since both I and P groups showed similar changes (protein, -23% and -21% (I and , respectively); cortical width,

TABLE 3 Comparison of means for the treatment groups slaughtered at approximately 45kg LW. C animals were slaughtered on day 134, while I and P animals were slaughtered on day 260. A pooled standard error of the mean (SEM) is presented. Comparisons of means was made by unpaired *t*-test. change in variables is expressed for I and P means as the percentage difference from the C mean.

Tissue	Means C	Means I	Means P	SEM	*t*-tests C *v* I	*t*-tests C *v* P	*t*-tests I *v* P	% change I	% change P
Gravimetric data									
Pre-slaughter LW	46.7	47.2	49.0	1.2	NS	NS	NS	1.1	4.9
GIT empty	5.55	5.97	6.58	0.26	NS	*	NS	7.6	18.6
Liver	0.983	0.848	0.809	0.035	*	*	NS	-13.7	-17.7
CS fat	9.69	9.81	11.58	0.50	NS	*	*	1.2	19.5
CS protein	3.44	3.46	3.69	0.08	NS	†	NS	0.6	7.3
NC fat	3.16	3.75	4.39	0.26	NS	*	†	18.7	38.9
NC protein	2.66	2.51	2.42	0.12	NS	NS	NS	-5.6	-9.0
FEM total	139.4	148.4	138.8	4.0	†	NS	NS	6.5	-0.4
FEM fat	31.5	38.8	35.4	2.4	*	NS	NS	23.2	12.4
FEM protein	28.3	28.0	26.9	0.8	NS	NS	NS	-1.1	-4.9
FEM ash	49.8	49.8	48.6	1.8	NS	NS	NS	0.0	-2.4
ST total	105.5	119.0	126.2	4.8	†	*	NS	12.8	19.6
ST fat	6.99	7.22	7.84	0.53	NS	NS	NS	3.3	12.2
ST protein	23.3	25.6	27.4	1.1	NS	*	NS	9.9	17.6
Geometric data									
FEM length	161.5	163.2	164.4	2.1	NS	NS	NS	1.1	1.8
FEM diameter	19.4	20.9	19.6	0.4	*	NS	*	7.7	1.0
FEM cortex width	3.33	3.30	3.59	0.08	NS	NS	*	-0.9	7.8
FEM volume	99.7	110.1	100.9	3.2	*	NS	NS	10.4	1.2
ST diameter	29.1	33.2	33.0	0.8	*	*	NS	14.1	13.4
ST volume	99.1	111.8	118.5	4.5	†	*	NS	12.8	19.6
Muscle:bone relationships (ST:FEM)									
weight :weight	0.756	0.801	0.909	0.020	NS	***	**	6.0	20.2
protein :protein	0.826	0.913	1.015	0.032	†	**	†	10.5	22.9
diameter:cortex width	8.70	10.04	9.22	0.28	*	NS	†	15.4	6.0

-33% and -31%). Clearly muscle was specifically affected by parasitism but changes in bone paralleled those induced by simple undernutrition. Although specific effects of parasitism did occur, a greater part of changes in the body and tissues associated with infection could be simply attributed to the restriction in growth consequent upon the reduction in feed intake.

Following administration of anthelmintic at day 83, compensatory growth occurred. Only a few variables displayed compensatory growth, defined classically as higher than normal rates of growth in *absolute terms* (Wilson and Osbourn, 1960), notably bone cortical width and muscle diameter (rates of growth between the first and last slaughter groups for C, I and P treatments respectively, were 2.7, 7.7 and 8.7 μm/d for bone cortical width and 41.4, 80.0 and 63 μm/d for muscle diameter). Other variables displayed compensatory growth in relative terms, whereby those variables most retarded by restricted growth showed greater than normal growth relative to those variables least retarded by restricted growth (Table 2). Thus during early recovery compensatory growth in relative terms, was displayed by the carcass, liver, body fat, body protein and tissue protein, while muscle protein displayed preferential repletion relative to bone protein. Overall greater compensatory growth effects were displayed by those variables most retarded by restricted growth (Table 2 *cf.* Table 1) and this largely occurred during early recovery. Similar findings have been reported by Drew and Reid (1975a, b) and Butler-Hogg (1984).

By the final slaughter point, values for body components of both I and P sheep were similar to those of C sheep indicating that recovery was largely complete (Table 3). Higher fat weights in P animals were attributed to the greater live weight of these animals. I and P animals did however have lower liver weights. These are not readily explainable and may reflect a lower plane of nutrition immediately prior to the slaughter of the final group in these treatments since it is known that this tissue is very sensitive to level of nutrition (Ferrell *et al.*, 1986).

There was no direct evidence of any persistent treatment effects on bone which is contrary to the expectations of Coop *et al.* (1976) . This further suggests that the effect of parasitism is mediated through general effects on nutrition. Where studies of undernutrition have led to permanent reductions in body size they were characterized by recovery growth occurring in less than ideal nutritional conditions, whereas animals in this study were offered high quality feed in generous quantities. However, greater development of muscle relative to bone in the P treatment at day 260 indicated that recovery in I animals was not complete (muscle weight: bone weight, 0.801 and 0.909 for I and P respectively; muscle protein: bone protein, 0.913 and 1.015). Whether full recovery was possible could not be assessed from these data.

CONCLUSIONS

Parasitism acted to reduce animal performance largely through effects on appetite and on the efficiency with which feed was used for change in LW. Specific effects of parasitism on the carcass were small and restricted to muscle. While the data provide little evidence for permanent effects of parasitism on growth of bone or muscle there were indications that recovery is not as rapid as might be expected.

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