

## Pathophysiology of infection with *Ostertagia ostertagi* in cattle

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### ABSTRACT

Fox, M.T., 1993. Pathophysiology of infection with *Ostertagia ostertagi* in cattle. *Vet. Parasitol.*, 46: 143–158.

Infection with the abomasal nematode, *Ostertagia ostertagi*, is an important cause of impaired productivity in young cattle in temperate parts of the world. Such losses have been associated with marked changes in feed intake, gastrointestinal function, protein, energy and mineral metabolism, and in body composition. The reduction in feed intake is an important factor in the pathogenesis of infection and may account for a large part of the difference in weight gain between ad libitum fed control and infected calves. Despite the obvious importance of inappetence, only recently has an association been made between reduced intake, altered gut motility and elevated levels of certain gastrointestinal hormones, such as gastrin. It has been suggested that the elevated gastrin levels accompanying abomasal parasitism may impair reticulo-ruminal motility and slow down abomasal emptying, leading to a stasis of ingesta and a reduction in feed intake. The rise in blood gastrin levels may also be partly responsible for the marked hyperplasia of the fundic mucosa seen in abomasal infections. Pronounced changes in protein metabolism have also been associated with *Ostertagia* infection. Radioisotopic studies have demonstrated increased losses of albumin into the gastrointestinal tract which are accompanied by an increase in the rate of synthesis in the liver. Dietary protein breakdown in the abomasum is also likely to be impaired, although there is evidence of a compensatory increase in protein digestion in the lower gut of parasitised calves. Increased losses of albumin are not always accompanied by increases in faecal nitrogen, suggesting that albumin is broken down and recycled as ammonia. Radioisotopic studies in animals with intestinal nematode infections have demonstrated a marked reduction in muscle protein synthesis and an increase in protein synthesis in gastrointestinal tissue. Such changes in the balance of protein synthesis are likely to be brought about by alterations in the balance of certain metabolic hormones. Marked changes in energy metabolism also accompany *Ostertagia* infection. Parasitised calves exhibit a marked increase in non-esterified fatty acid levels, resulting from the mobilisation of adipose tissue, and a reduction in digestive efficiency of energy, probably associated with the increase in cycling of protein through the gastrointestinal tract and the compensatory increases in protein synthesis. Mineral metabolism may also be affected although relatively little work has been conducted in cattle. Changes in body composition reflect a reduction in deposition of muscle protein and fat, and an increase in bone content and water retention.

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## INTRODUCTION

Infection with the abomasal nematode, *Ostertagia ostertagi*, is an important cause of impaired production in cattle in temperate parts of the world. Experimental studies have demonstrated reductions in liveweight gain of between 20% (Taylor et al., 1989) and 100% (Fox et al., 1989a) in animals exposed to different levels of infection. Similarly, a wealth of data has been collected from field studies in which the weight gains of anthelmintic treated and untreated calves grazing *Ostertagia*-contaminated pastures have been compared (e.g. Jacobs et al., 1987).

The effect of gastrointestinal parasitism on production in adult cattle has also received considerable attention. Early studies in the United States reported significant increases in milk yield following anthelmintic treatment of clinically healthy cows around calving (Bliss and Todd, 1973, 1976). Later investigations, however, gave conflicting results. Some workers reported an increase in yield (McBeath et al., 1979; Bliss et al., 1982) while others failed to demonstrate any significant effect of treatment on milk production, despite using large numbers of animals (Baker, 1979; Michel et al., 1982; Fox and Jacobs, 1984). Two recent studies, in which an increase in milk production was demonstrated following treatment of cows during the dry period, also established significant correlations between the production response to treatment and either management practice or mean herd *Ostertagia* antibody titre (Bissett et al., 1987; Ploeger et al., 1989). Such relationships might be used as a basis for deciding whether or not to treat dairy cows. Detailed studies on the specific effects of *Ostertagia* infection on reproductive performance in cattle have yet to be reported although one such study has recently been described in sheep. Infection with *Ostertagia circumcincta* did not, however, appear to exert any significant effect on a range of reproductive parameters in immune ewes. The ovulation rate was, nevertheless, consistently lower in the parasitised animals (Jeffcoate et al., 1988).

It is clear, therefore, that *Ostertagia* is of considerable economic importance and the production effects of parasitism are well recognised. There has also been a marked increase in our knowledge of the pathogenesis of infection, particularly in younger animals. Work in calves has shown that infection may result in depressed appetite, impaired gastrointestinal function and alterations in protein, energy and mineral metabolism, and changes in water balance. Such changes lead to alterations in body composition and, as a result, in carcass quality.

## FEED INTAKE

A depression in voluntary food intake is an important feature of infections with gastrointestinal nematodes and is widely recognised as a major factor in

the pathogenesis of such infections (Holmes, 1985). In cattle, the degree of inappetance varies and, as in sheep (Symons, 1985), appears to be dose related. For example, a reduction in intake of up to 77% was recorded by Fox et al. (1989a) in calves experimentally infected with 10 000 *Ostertagia ostertagi* larvae daily, whereas exposure to 2000 larvae of the same strain per day did not appear to affect appetite (Taylor et al., 1989). Quantifying depression in intake in animals exposed to trichostrongyle infection at pasture is, by comparison, more difficult. Bell et al. (1988) were, however, able to demonstrate an 18% depression in appetite in control animals grazing *Ostertagia*-contaminated pasture in comparison with calves protected by a morantel sustained release bolus (MSRB). The difference in intake between the two groups would have been greater had the MSRB-treated calves been totally uninfected and had both groups not lost weight owing to a shortage of grass in mid-summer. The latter is likely to be particularly important as plane of nutrition has been shown to influence the degree of inappetance in sheep infected with *Haemonchus contortus* (Abbott et al., 1986).

The significance of a depression in appetite as a cause of impaired productivity may be assessed by the use of pair-feeding studies in which feed intake of uninfected control animals is restricted to that of their infected pair members. Comparison of data from infected, pair-fed and ad libitum fed control groups thus enables the impaired weight gain due solely to a drop in appetite to be distinguished from that associated with the parasite burden. This technique was used in sheep by Sykes and Coop (1977), who demonstrated that inappetance accounted for over 60% of the difference in weight gain between *Ostertagia*-infected and ad libitum fed control animals. Only recently has pair-feeding been used to assess the relative importance of appetite depression as a cause of lost productivity in *Ostertagia*-infected cattle. Fox et al. (1989a) found that nearly 73% of the difference in weight gain between ad libitum fed controls and calves exposed to a trickle infection was due solely to the depression in appetite. This type of study does not, of course, take account of the changes in body composition that may accompany *Ostertagia* infection (Entrocasso et al., 1986a; Bell et al., 1990).

Despite the obvious importance of inappetance in parasitised animals, relatively little is known of the mechanisms by which parasites induce a depression in food intake. Two comprehensive reviews highlighted the limited state of our current knowledge on this topic (Crompton, 1984; Symons, 1985). Until recently, numerous explanations had been proposed, although none of these were entirely satisfactory. A number of authors have in the past suggested that pain may be an important cause (Andrews, 1939; Gibson, 1955), though this is extremely difficult to assess. Whether the alterations in abomasal pH that accompany *Ostertagia* infection are associated with a reduction in appetite is not known, although Holmes (1985) suggested that the resultant changes in protein digestion and in the availability of amino acids may be

important as absorption of the latter has been shown to influence feed intake (Leng, 1981).

Currently, the most promising line of research in this area is the association between certain gut peptides and inappetance in parasitised ruminants (Titchen, 1982). Recent work has demonstrated that the depression in appetite seen in *Ostertagia*-infected calves is closely associated with an elevation in blood gastrin levels and a reduction in the rate of passage of ingesta (Fox et al., 1989a,b). The authors suggested that the hypergastrinaemia may be partly responsible for the drop in feed intake. While one of the more important actions of gastrin is to stimulate acid and pepsin secretion by the abomasum (McLeay and Titchen, 1970), the peptide also affects the contractility of smooth muscle, inhibiting reticulo-ruminal motility (Carr et al., 1970) and slowing down gastric emptying (Bell et al., 1977). This would result in a stasis of ingesta in the reticulo-rumen and abomasum and lead to a reduction in feed intake (Campling and Freer, 1966). A similar relationship between appetite, reticulo-ruminal motility and blood gastrin levels was established by Grovum (1981) who recorded a significant depression in intake when the synthetic analogue, pentagastrin, was infused into the jugular veins of sheep. Subsequent work by Fox et al. (1989c) demonstrated a 40% depression in appetite in worm-free calves when endogenous blood gastrin concentrations were raised indirectly, by the human gastric acid secretion inhibitor omeprazole, to values comparable with those seen in parasitised calves. If gastrin is to become accepted as an important factor in the depression of appetite in cases of abomasal parasitism, its action must be blocked, possibly by immunoneutralisation, with a view to restoring appetite towards normal (Poppi et al., 1990).

#### GASTROINTESTINAL FUNCTION

Although reduced feed intake has a marked effect on the productivity of parasitised ruminants, studies using pair-feeding techniques have shown that inappetance is not the only consequence of infection and that such animals often exhibit impaired utilisation of nutrients relative to pair-fed animals on the same feed intake (Holmes, 1987). Such changes may be accompanied by alterations in gut motility, in gastrointestinal secretions and in digestion and absorption.

##### *Gastrointestinal motility*

The effect of nematode infections on gut motility and rate of passage of ingesta has been studied in relatively few experiments. One recent investigation examined the rate of passage of chromium-mordanted hay in calves exposed to a trickle infection with *O. ostertagi* (Fox et al., 1989a). The authors

found that rate of passage was markedly affected, not only by the parasite burden but also by the reduction in feed intake. Infected calves exhibited a 74% reduction in rate of passage and pair-fed controls exhibited a 50% reduction in comparison with ad libitum-fed control animals. Such studies, however, only record overall changes in the rate of passage of ingesta and may therefore mask differences in response to the parasite infection in different compartments of the gut.

Changes in gut motility in different parts of the gut, associated with abomasal parasitism, have been investigated in sheep infected with *Trichostrongylus axei* (Bueno et al., 1975) and *H. contortus* (Bueno et al., 1982). Both experiments revealed marked disturbances in gastrointestinal motility which, like the study in cattle, were partly associated with a depression in feed intake and partly with the parasites themselves. A marked disruption of the migrating myoelectric complex and severe inhibition of the reticulo-rumen and abomasum were recorded which Gregory (1985) suggested could be associated with alterations in circulating levels of gastrointestinal hormones.

### *Gastrointestinal secretions*

Marked changes in gastrointestinal secretions accompany *Ostertagia* infections in ruminants including a reduction in gastric acid secretion and an increase in circulating pepsinogen and gastrin levels. The reduction in acid output is brought about by the replacement of functional parietal cells with those of reduced activity, rather than a direct response to parasite secretions (Murray et al., 1970; McKellar et al., 1990). The effect of such a change on abomasal digestion is difficult to assess as will become apparent later.

The elevation in blood pepsinogen levels in parasite-naïve animals was originally thought to result from the combined effects of a rise in abomasal pH, the accumulation of non-activated pepsinogen in the gastric glands and an increase in mucosal permeability (Murray et al., 1970). A similar change in permeability was also thought to be responsible for the pepsinogen response to infection in immune cattle and caused by an allergic reaction to recently ingested larvae (Armour et al., 1979). However, Stringfellow and Madden (1979) were unable to show any differences in mucosal permeability between infected and worm-free calves, when using horseradish peroxidase as a marker, and reported ultrastructural evidence of direct secretion into the blood. More recently, Fox et al. (1989c) recorded elevated blood pepsinogen values in association with a hypergastrinaemia in worm-free calves. It appears, therefore, that the mechanisms by which pepsinogen levels become elevated are more complicated than originally envisaged and may be multifactorial in origin (McKellar et al., 1986). The zymogen is, nevertheless, a valuable tool in the diagnosis of abomasal parasitism and attention has focused recently on the different forms of pepsinogen occurring in the blood

of calves with *Ostertagia* infection (Eckersall et al., 1987). Unfortunately, from the diagnostic point of view at least, the same forms appear to predominate in cattle irrespective of their previous experience of the parasite or the method of infection, i.e. oral infection with infective larvae or direct transplantation of adult worms into the abomasum (McKellar et al., 1988).

The marked rise in abomasal pH appears to be the main stimulus for the hypergastrinaemia seen in sheep and cattle infected with *Ostertagia* (Anderson et al., 1981; Fox et al., 1987). However, work in sheep (Anderson et al., 1981) and, more recently, in cattle (McKellar et al., 1987) suggests that the parasites or their secretions may also exert a mild stimulatory effect on gastrin secretion in the absence of any change in pH. The rise in blood gastrin in *Ostertagia*-infected calves appears to be due to a marked increase in synthesis of the peptide, as judged by increases in gastrin gene expression, coupled with a reduction in storage in the pyloric mucosa. Surprisingly perhaps, this is accompanied by a reduction in the number of gastrin-producing G cells. Whether a reduction in turnover of the peptide also contributes to the elevation in blood levels is at present unknown. Chromatographic separation of the different molecular forms of gastrin in the blood has revealed that the hypergastrinaemia consists of largely big gastrin (G-34, containing 34 amino acids) rather than little gastrin (G-17) which is the predominant form in the blood of worm-free calves (M.T. Fox, P. Shivalkar, A. Carroll, U.E. Uche, A. Purewal, D.E. Jacobs and C. Vaillant, unpublished data, 1992). This supports observations made in *Ostertagia*-infected sheep (Anderson et al., 1988) and may reflect incomplete post-translational processing of the precursor molecule, progastrin, the longer half-life of G-34 or an increase in contribution of duodenal gastrin, which contains a greater proportion of G-34 (Reynolds et al., 1991). The marked hypergastrinaemia, associated with abomasal parasitism, has made assay of the peptide a useful diagnostic tool although, like pepsinogen, it is not specific for *Ostertagia* infection (Fox et al., 1987; Fox et al., 1988; Snider et al., 1988). Of greater significance, however, is the possible relationship between elevated blood gastrin, impaired gut motility and depressed appetite referred to earlier. In addition to stimulating gastric acid secretion and impairing smooth muscle contractility in the wall of the reticulo-rumen and abomasum, gastrin also has a trophic action on the parietal cell region of the abomasum. Anderson et al. (1988) and Snider et al. (1988) suggested that this may be partly responsible for the hyperplasia of the fundic mucosa seen in *Ostertagia* infections. An increase in blood levels of another gut peptide with trophic effects, enteroglucagon, in sheep with *H. contortus* (Nichols et al., 1985), adds further interest to the role of these peptides in the pathogenesis of infection.

### *Digestion and absorption*

*Ostertagia* infections are associated with marked elevations in gastric pH which might be expected to impair abomasal digestion and, as a result, the

absorption of nutrients. A few studies have indeed shown that the digestion of dietary nitrogen is impaired, although precise interpretation of these data is difficult because these studies do not allow for significant passage of endogenous nitrogen into the gastrointestinal tract of parasitised animals. Fewer difficulties arise with the evaluation of results from balance studies that include pair-fed control animals though even these fail to allow direct measurement of digestion and absorption in different parts of the gut. Such studies should ideally be conducted in animals with indwelling gastrointestinal canulae. The results of such studies are referred to later; in general they indicate that in abomasal infections impaired digestion and absorption are not important causes of poor utilisation of nutrients in parasitised animals (Holmes, 1987).

#### PROTEIN METABOLISM

The effect of gastrointestinal parasitism on protein digestion and metabolism has been extensively reviewed (Dargie, 1980; Sykes, 1982; Steel and Symons, 1982; Poppi, 1986; Parkins and Holmes, 1989; Poppi et al., 1990). In abomasal infections, protein intake is likely to be depressed and gastric digestion impaired as a result of the increase in abomasal pH. However, quantifying the depression in protein breakdown in the stomach is difficult. Interpretation of apparent nitrogen digestibility data, based on the analysis of feed and faecal samples, is hampered by the loss of endogenous nitrogen, largely plasma proteins, exfoliated epithelial cells and mucus, into the abomasum and compensatory increases in protein digestion and absorption lower down the intestine. Although methods to quantify the loss of exfoliated epithelial cells and mucus are not yet available (Holmes, 1987), it is possible to measure blood protein losses using radioisotopic techniques. Taylor et al. (1989) recorded significant losses of labelled albumin into the abomasum of *Ostertagia*-infected calves, but found no significant increase in faecal nitrogen loss. The authors suggested that albumin lost into the gut lumen may have been degraded by bacteria in the large intestine and reabsorbed as ammonia. This would then be transported to the liver, converted to urea and either used for resynthesis of amino acids or excreted in the urine (Vernon and Peaker, 1983). Increased losses of urinary nitrogen have been recorded in *Ostertagia*-infected calves by Parkins et al. (1982) and Taylor et al. (1989).

Radioisotopic studies have also demonstrated compensatory increases in the synthesis of liver proteins, including albumin, in sheep with gastrointestinal infections (reviewed by Symons, 1985). Where such infections are heavy, the increase in albumin synthesis may not match the loss, resulting in lowered blood protein levels (Mulligan et al., 1963). There is, however, likely to be a delay in the appearance of a hypoalbuminaemia because of the relatively long half-life of albumin (Mackie and Fell, 1971; Fox et al., 1989b). Increases in

protein synthesis have also been recorded in gastrointestinal tissue of animals with intestinal infections, although so far, such observations have been restricted to guinea pigs infected with *Trichostrongylus colubriformis* (Symons and Jones, 1983). It is important to establish whether similar changes occur in ruminants, as these sites of increased protein synthesis may be an important cause of impaired nutrient utilisation in parasitised sheep and cattle (Holmes, 1987). Radioisotopic techniques have also been used to monitor changes in muscle protein synthesis in parasitised ruminants. Symons and his colleagues used L-[ $^{14}\text{C}$ ]-leucine and L-[ $^{14}\text{C}$ ]-tyrosine in a series of experiments to demonstrate significant reductions in the level of protein synthesis in the muscle of sheep infected with *T. colubriformis*. Comparison of infected and pair-fed control data in these experiments indicated that while anorexia may contribute to impaired muscle protein synthesis, it was not the only factor responsible. As a result of these studies, Symons (1985) concluded that there is a net movement of amino acid nitrogen from muscle to the liver and gastrointestinal tract, which decreases its availability for growth and milk production in parasitised animals.

Relatively little attention has been focused on the mechanisms underlying these changes in protein synthesis. Symons (1985) suggested that they may be hormonal in origin and subsequent work has tended to favour this hypothesis. Fox et al. (1987) demonstrated significant reductions in circulating insulin levels in calves exposed to a single large *Ostertagia* infection, which in the long term would have impaired muscle protein synthesis and reduced protein catabolism in the liver (Murray et al., 1990). Similar results were obtained in calves exposed to a trickle challenge which, by comparison with pair-fed control data, could be largely ascribed to a depression in feed intake (Fox et al., 1989b). The mechanism responsible for the depression in blood insulin remains a matter for speculation, but may be related to the rate of protein breakdown in the intestine. This is known to be closely correlated with plasma insulin levels in sheep (Bassett et al., 1971). Changes in corticosteroid and thyroxine levels have also been implicated as possible causes of altered protein metabolism in parasitised sheep (Pritchard et al., 1974). However, Fox et al. (1987) were unable to detect any difference in circulating corticosteroid levels in *Ostertagia*-infected calves, although they did record a depression in total thyroxine which they associated with fluctuations in feed intake. This change was unlikely to be important as levels of free (or unbound) thyroxine, the metabolically active form of the hormone, were not significantly affected.

Despite the marked changes in protein digestion and metabolism that accompany abomasal parasitism, there appears to be little or no effect on protein absorption per se. It should therefore be possible to meet the increased nutrient demand by improving the protein content of the diet (Poppi et al.,



1990). Support for this hypothesis has been provided by Abbott et al. (1986, 1988) who increased protein deposition and liveweight gain in sheep infected with *H. contortus* to levels similar to those in control animals by increasing intestinal protein supply.

Such an approach is, however, unlikely to be successful in animals with concurrent abomasal and intestinal infections. Compensatory digestion and an increase in absorption of nutrients by the small intestine will necessarily depend upon the integrity of this organ. Concurrent infections with *O. ostertagi* and *Cooperia oncophora* in calves have indeed shown that production losses are more pronounced than would have been expected from the combined effects of monospecific infections alone. For example, Parkins et al. (1990) recorded an increase in establishment of *Ostertagia* in mixed infections with *Cooperia*, an increase in the magnitude and duration of the pepsinogen response and an impairment in digestive efficiency of crude protein and in nitrogen retention, not normally affected in monospecific infections.

#### ENERGY METABOLISM

One of the main factors limiting the availability of energy for growth and maintenance in parasitised animals is undoubtedly reduced feed intake (Holmes, 1987). Cattle are, nevertheless, able to compensate to a limited extent for this reduction in intake by mobilisation of adipose tissue. Fox et al. (1989b) demonstrated a marked rise in blood non-esterified fatty acid levels which would have been used as an additional source of energy during a period of reduced intake (DiMarco et al., 1981). Hormonal changes, such as an increase in the growth hormone:insulin ratio exhibited by both infected and pair-fed control groups, were probably responsible for the increase in lipolysis (Murray et al., 1990).

Reductions in the efficiency of energy utilisation (Sykes and Coop, 1977; Randall and Gibbs, 1981; MacRae et al., 1982; Entrocasso et al., 1986b; Parkins et al., 1990) and in energy retention, whether shown by carcass analysis (Sykes and Coop, 1976, 1977; Coop et al., 1982; Stevenson 1989) or calorimetric measurement (Randall and Gibbs, 1981; Stevenson, 1989), have been demonstrated in parasitised ruminants. Increases in liver and gastrointestinal protein synthesis, coupled with the cycling of large amounts of protein through the digestive tract and increases in methane production and urinary urea excretion, are likely to account for most of these losses (Holmes, 1987; Stevenson, 1989).

#### MINERAL METABOLISM

Marked changes in mineral metabolism have been associated with *Ostertagia* infections, although the majority of studies have been conducted in sheep. Sykes and Coop (1977) demonstrated a 65% depression in the mineralisation of bone in lambs exposed to a trickle infection with *O. circumcincta*

in comparison with pair-fed controls. Sykes et al. (1977) attributed this change to parasite-induced deficiencies in protein and energy rather than a reduction in uptake. This view was supported by Wilson and Field (1983) who found that rates of calcium and phosphorus absorption in parasitised lambs were not impaired. In contrast, intestinal parasitism has been shown to affect mineral uptake and, in particular, that of phosphorus. Lambs infected with *T. colubriformis* exhibit impaired phosphorus absorption, increased losses of endogenous calcium and phosphorus, lower plasma phosphorus and, as a result, reduced bone formation (Sykes and Coop, 1976; Coop and Field, 1983; Wilson and Field, 1983; Poppi et al., 1985; Bown et al., 1989).

In cattle, there is very little evidence for impaired mineral metabolism associated with abomasal parasitism. Waymack and Torbert (1969) recorded changes in blood phosphorus levels and a reduction in phosphorus retention in animals given a single infection with *Ostertagia*, while Fox et al. (1989b) reported a hypocalcaemia in calves exposed to a trickle challenge. The latter was surprising in view of the marked changes in mineral metabolism that may accompany *Ostertagia* infection in sheep without any significant change in blood calcium (Sykes and Coop, 1977). The drop in calcium levels was probably related to either the associated hypoalbuminaemia (as at least 40% of calcium is transported in the blood bound to albumin) (Swenson, 1984) or a reduction in calcium absorption in the upper intestine. Absorption is facilitated by high intestinal protein levels and acidic conditions (Murray et al., 1990), both of which are likely to be adversely affected by *Ostertagia* infection.

The interaction between trace elements and abomasal parasitism has only recently been the subject of detailed investigation. MacPherson et al. (1987) demonstrated an impairment in functional immunity in calves on a cobalt-deficient diet given an experimental *Ostertagia* infection. These animals had higher faecal egg counts and shorter prepatent periods than calves on a cobalt-sufficient diet, but higher blood pepsinogen values. This, the authors postulated, was probably due to amplified immune reactions occurring at the gut wall. Bang et al. (1990a) demonstrated a marked reduction in uptake of copper by *Ostertagia*-infected sheep which they thought was probably caused by the parasite-induced elevation of abomasal pH interfering with copper solubilization. Bang et al. (1990b) also observed a marked reduction in the parasite burdens of *Haemonchus* and *Ostertagia*-infected sheep given copper supplementation. While the anthelmintic properties of copper have long been recognised (Hall and Foster, 1918; Gordon, 1939), Poppi et al. (1990) urged caution in the interpretation of weight gain responses to copper treatment simply as alleviation of copper deficiency, particularly in areas where *Haemonchus* and *Ostertagia* are significant species.

#### BODY COMPOSITION

Change in body weight is probably the most widely used criterion for judging the effect of gastrointestinal parasitism on the host. However, such changes

do not take account of alterations in body composition which may be of considerable importance in determining carcass quality. Entrocasso et al. (1986a) and Bell et al. (1990) demonstrated that the carcasses of cattle exposed to natural mixed trichostrongyle infections had poorer killing-out percentages, reduced muscle and fat deposition and increased bone content, in comparison with animals that had been protected by prophylactic anthelmintic treatment. Increases in water retention in *Ostertagia*-infected cattle have also been observed (Halliday et al., 1965; Entrocasso et al., 1986a; Taylor et al., 1989), despite increases in the amounts of water being excreted in both urine and faeces (Parkins et al., 1982). Such changes in water retention clearly demonstrate that tissue loss attributable to parasitic infections cannot be reliably determined from changes in body weight alone (Holmes, 1986).

Some of the technical difficulties associated with body composition studies in ruminants, such as the need to restrict analysis to indicator joints or whole body homogenisation, have recently been overcome by the use of radioisotopic dilution techniques. These enable sequential *in vivo* measurements of total body water and exchangeable sodium and potassium to be used as indicators of body composition changes in cattle exposed to parasitic infection at pasture. One such study employing these techniques demonstrated significant increases in total body water and exchangeable potassium, indicating an increase in body protein, in animals protected by prophylactic anthelmintic therapy (J.M. Maclean, K. Bairden, P.H. Holmes, W. Mulligan and P.N. McWilliam, unpublished data, 1992).

## CONCLUSIONS

A considerable body of information now exists on the pathogenesis of infection with *Ostertagia*, although a number of important aspects still require further investigation. For example, the precise role of gastrin as a cause of inappetance in cases of abomasal parasitism requires clarification, as does the extent to which appetite is affected in immune adult cattle exposed to challenge. Alternative explanations will have to be sought to explain the drop in feed intake associated with cases of intestinal parasitism in which blood gastrin levels are not normally elevated (Titchen, 1982). Recent work with *T. colubriformis* in sheep suggests that central satiety signals are associated with inappetance rather than changes in peripheral peptide levels (Dynes et al., 1990). Further work is also required to identify the factors that influence the partitioning of protein and energy between the different body compartments. Systemically activated mediators may well play a role in addition to the hormonal changes that have been identified already. Clearly, the continued use of a multi-disciplinary approach to the study of the pathophysiology of *Ostertagia* infection is required in view of the wide range of effects that the parasite has on the host (Parkins and Holmes, 1989).

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