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Non-infectious intestinal conditions of sheep

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Abstract
Enteric disorders are common in sheep and have major economic consequences attributable to dags, production losses and mortalities. The purpose of this paper is to provide a review of non-infectious intestinal conditions in sheep with an emphasis on the role of nutrition and the effects of various nutritional factors. The principles of treatment and prevention are discussed, but not reviewed in detail. Non-infectious causes of scouring in sheep are generally not well understood. In particular, the role of various nutritional factors in determining faecal consistency (in both the presence and absence of infectious agents) warrants further investigation. Nutritional manipulation may provide options for management and prevention of scouring in sheep.

Introduction

Enteric disorders are common in sheep and have major economic consequences attributable to dags, production losses and mortalities. The intestinal tract is responsible for the digestion and absorption of nutrients and water from ingesta and the excretion of wastes in the form of faeces. Conditions that affect the intestinal tract generally result in illthrift or diarrhoea due to the reduced capacity to absorb nutrients and/or reabsorb sufficient water from ingesta for normal faecal pellet formation.

When investigating cases of intestinal disease, infectious causes must be ruled out. Infectious causes of scouring are normally diagnosed by identifying the organism involved and/or associated pathological signs. In general, non-infectious causes of scouring are problematic to diagnose, particularly where diagnostic tests are not available or where the relevance of such findings may be questionable. The implication of this is that where an infectious agent cannot be identified, investigations can be unrewarding in terms of providing an aetiological diagnosis.

Many non-infectious causes of intestinal disease in sheep are not well understood. The purpose of this paper is to provide a review of intestinal conditions in sheep with non-infectious aetiologies with an emphasis on the role of nutrition. The principles of treatment and prevention are discussed, but not reviewed in detail.

Trace element and mineral imbalances

Scouring and illthrift may be seen in association with deficiencies or toxicities of trace elements.

Selenium

A variety of disease syndromes have been associated with selenium deficiency with the most important being degenerative myopathy, or white muscle disease. Scouring and illthrift may be seen in conjunction with reduced wool production, however this is not a consistent finding in flocks with confirmed selenium deficiency. Scouring and reduced wool production are described as "selenium responsive" as they are not proven symptoms of deficiency and so caution should be taken when considering selenium deficiency as a primary cause of scouring. Clinical signs are more common in weaners and in sheep grazing lush green feed.[1]

The pathogenesis of scouring with selenium deficiency is not well described, although it has been suggested that it may be related with a depressed immune state and resulting increased susceptibility to internal parasitism.[2]

Soils deficient in selenium have been well demarcated in Australia. Factors which predispose the development of deficiency include clover dominant pastures, rainfall in excess of 500mm per annum, acidic basalt/granite soils and heavy application of sulfur-fortified superphosphate or gypsum.[2]

Scouring may also be a minor clinical finding with selenium toxicity following accidental overdosing. Other clinical signs include dyspnoea, bloat and abdominal pain and death due to respiratory failure.

Cobalt

Cobalt is necessary for the production of vitamin B_{12} by rumen microbes. Scouring only occurs with chronic cobalt deficiency.[2] More common clinical signs include depressed appetite, ill thrift, anaemia, weepy eyes, reduced wool production and infertility. Liver damage, photosensitization and neurological signs may also occur.[2,3]

Copper

Scouring is a minor clinical sign with copper deficiency and is more common in cattle than in sheep. Other signs associated with chronic copper deficiency include enzootic ataxia, loss of wool crimp, pigment and tensile strength, brittle bones anaemia, ill thrift and scouring.[2,3]

The pathogenesis of scouring is probably associated with impaired tissue oxidation and interference with intermediary metabolism and is more commonly seen where copper deficiency is secondary to excessive molybdenum intake.[2]

Copper deficient soils are seen most commonly on sandy coastal soils, sandy loams and peaty swamps. Availability of dietary copper is reduced by liming of pastures and high intakes of molybdenum, sulphur, zinc, iron, cadmium and calcium.[2]

Scouring may also be a minor clinical finding with copper toxicity which results in gastroenteritis, abomasal ulceration, pulmonary oedema and deaths.[3,4]

Diagnosis of trace element deficiencies and toxicities

Selenium is necessary for the production of glutathione peroxidase and cobalt is necessary for the production of vitamin B_{12}. Measurements of these enzymes at a flock level are generally used to diagnose selenium and cobalt deficiencies. Blood is collected into lithium heparin tubes (which are also used for plasma copper analysis). Whole blood is preferable to plasma which only gives an indication of current selenium intake.

Signs of copper deficiency develop when the copper-dependent enzymes are depleted which takes about a month after plasma levels have decreased.[2] At least 5 sheep should be sampled so as to provide an assessment of selenium, copper and cobalt status of the flock.

Tissue levels of selenium and copper may be assessed on fresh frozen post-mortem material. Liver is preferred for diagnosis of deficiencies whilst kidney and liver are the tissues of choice where toxicity is suspected. Samples should be kept frozen or on ice prior to analysis. Post mortem samples should include fixed liver samples for histopathology. Other causes of diarrhoea should be ruled out and a response to treatment trial considered to confirm cases where a deficiency is suspected.

Treatment and prevention of trace element deficiencies

Injectable preparations, intraruminal bullets, drenches, licks and supplements are used in the treatment of clinical cases of trace element deficiencies. Vitamin B_{12} injections can be used to treat clinical cases of cobalt deficiency. Top dressing paddocks with appropriate fertilizers and intrauminal bullets are generally used for prevention. Toxicity can occur with overdoses of copper and selenium in particular, so care must be taken that a deficiency is correctly diagnosed before treatment and that doses are correct.

Toxins

Plants

Many plants have been implicated as causing gastroenteritis and diarrhoea in grazing livestock – Hungerford lists 70 although many of these are only anecdotally linked with scouring.[5] Extensive lists of toxic plants are also available in Everist.[6] Toxic plants generally vary between districts and so local knowledge is valuable. In addition, the toxicity of the plant may vary throughout the year and as such seasonal patterns may be important.

Diagnosis of plant toxicosis

Diagnosis of suspected plant toxicities is based on animals having access to plants and having actually eaten the plant, botanical identification of gastrointestinal gut contents, consistent clinical and pathological findings and controlled feeding experiments.[2]

Treatment and prevention of plant toxicosis

Treatment will depend largely on the plant ingested and its' mode of toxicity. As a rule, removal of access to the offending plant and provision of alternative feed and water may aid recovery and prevent the development of new cases.

Nitrates

Certain plants are capable of accumulating nitrates including oats (*Avena satavia*), pigweed (*Portulaca* spp) and mintweed (*Salvia reflexa*). Nitrates may be lethal at more than 1.5% dry matter (DM) due to the production of methemoglobinaemia resulting in hypoxia and death. Clinical signs of methaemaglobinaemia develop when 20-40% of haemaglobin has been converted and include dyspnoea, cyanosis, rapid pulse, collapse, coma and terminal clonic convulsions. Nitrate poisoning can also occur due to contaminated water at levels over 0.12% via biological runoff, industrial effluent and fertilizer.[7]

Sub-lethal ingestion of nitrates at 0.5 and 1.5% DM may have a direct caustic effect on gut mucosa resulting in gastro-intestinal irritation.[1,6] Clinical signs include diarrhoea and possibly abortions. Some adaptation may occur with chronic exposure to nitrates.[7]

Nitrate accumulation is affected by a wide range of factors. Factors which increase accumulation are those which disrupt the balance between nitrate uptake from the soil and utilisation by the plant. Such factors include plant species, high nitrogen content of soil (natural or due to recent fertilization), appropriate soil conditions (acid soils and molybdenum deficient soils), drought, freezing or disease stress, low light and soil temperature, adequate moisture supply in early growth stages and herbicide treatment (eg 2,4-D). Nitrate accumulates preferentially in stalk or rough portion of plant and less so in leaves and flowers.[7]

Little information is available in the literature about sub-clinical exposure and its relative importance as a cause of scouring in sheep has not been defined.

Diagnosis of nitrate toxicity - Diagnosis depends on grazing history, clinical and pathological signs and determination of methaemaglobin levels in the blood.

Post mortem samples may exhibit signs of non-specific gastroenteritis. Chocolate brown blood can be seen where methaemaglobin levels exceed 30%. Mucous membranes may also be discoloured. Methaemaglobin levels can be measured in ocular fluid and serum. Blood measurements are problematic as methaemaglobin can be converted to haemaglobin and vice versa post mortem or in transit. This makes confirmation of suspected toxicities, particularly sub-clinical disease, problematic. Laboratory staff should be contacted for protocols for collection, transport and storage as appropriate prior to collection.

Treatment and prevention of nitrate toxicity - Removal from suspected nitrate containing pastures will alleviate signs and help prevent new cases Animals should be removed from the offending pasture and given an alternative feed source. Clinical cases of methaemaglobinaemia respond to intravenous methylene blue injection, although most reports of this treatment involve cattle rather than sheep.[7]

Prevention involves proper harvest and management of potential nitrate accumulating plants and gradual introduction of stock to potentially-high nitrate forages.

Cape weed

Capeweed (*Arctotheca calendula*) is commonly implicated by producers as a cause of diarrhoea. There is little scientific data available to suggest whether scouring is indeed associated with cape weed in the absence of other disease agents and the pathological processes associated with cape weed ingestion are not known. In a pen trial, sheep fed *ad lib* Capeweed for one week had a similar faecal consistency to control animals fed lucerne/oaten hay.[8]

During rapid growth phases, cape weed may accumulate nitrates as high as 2.0-4.4% dry matter.[9] The nitrate content of the cape weed fed by Pethick and Chapman was not investigated and so the possibility remains that capeweed may, under certain circumstances, result sub-lethal nitrate toxicity and gastrointestinal irritation.[8] Clearly, further work is required to determine the relative importance of capeweed in causing gastroenteritis in otherwise 'normal' sheep.

Fungal Endophyte

The most common syndrome associated with rye grass endophyte is ryegrass staggers. Work in New Zealand has shown that hyperthermia and diarrhoea may be common manifestations of disease. Lambs grazing rye grass pasture infected with endophyte (*Acremonium lolii*) have increased incidence of scouring and breech strike compared with lambs on non-infected pastures.[10-12] Another study by Pownell concluded that ryegrass endophyte metabolites other than lolitrem B located in

the leaf of the plant were associated with increased faecal moisture.[13] It has been suggested that some metabolites arising from the *Acremonium* fungus other than ergovaline, lolitrem B and peramine may be responsible scouring and dags.[14]
The pathogenesis of scouring in response to ryegrass endophyte is unknown. It has been suggested that it may act by increasing nutrient content of ingesta reaching the hind gut and thus an increase in fermentation at this site.[14]

Thus, ryegrass endophyte may play a role in scouring in areas of Australia where ryegrass is a dominant species, although further work is necessary to define the toxin(s) involved and the pathophysiological response to endophyte that may result in diarrhoea and dags.

Chemical poisonings
Arsenic, lead, phosphorus, salt, mercury, zinc, organophosphates and paraquat have been associated with scouring in sheep.[2] Diagnosis is based on clinical signs and pathological findings, including toxicology on selected tissues. Treatment is specific to the toxin and mode of action. Prevention involves removal of access to toxic substances.

Salt poisoning
Salt poisoning is a syndrome of sodium-water imbalance. Toxic effects are associated with excess salt concentration in tissue resulting in a high osmotic gradient drawing water into the tissue. Excess salt cannot be excreted by the kidneys without the excretion of water, so salt poisoning occurs with concurrent relative deprivation of water. Excess sodium ions can inhibit cellular enzymes, including those associated with the glycolytic pathway. This can have a profound effect on nervous tissue where glycolysis is the major energetic pathway.[15]

The source of salt can generally be attributed to feed sources with inherently high salt content, supplements, such as licks, and water with high salt content. Sheep can tolerate reasonably high levels of salt in their diet so long as adequate fresh water is available.[15] A relative restriction of access to fresh water must be present for the development of salt toxicity. Salt can be concentrated in troughs as water evaporates. Salt poisoning should be considered when sheep have access to supplements, are forced to drink salt water or given access to salt or water after being on a restricted supply for some time. Lambs and weaners are most susceptible to the development of clinical signs.

Clinical signs may be associated with central nervous system (shivering, staggers, convulsions and death) and the gastrointestinal tract (diarrhoea and colic). Diagnosis is based on a history of water deprivation or heavily salt contaminated water supply and the testing of feed or water supply. Safe levels are 7000 mg/L for breeding ewes, lambs and weaners and 10,500 to 14,000 mg/L for dry adult sheep. Water with less than 3000 mg/L is recommended for continual use by all livestock.

Treatment is aimed at decreasing tissue salt concentration without the development of excess tissue hydration which runs the risk of overhydrating nervous tissue. Treatment with isotonic glucose solutions may be of some assistance in diluting excess sodium chloride and aiding the reversal of glycolytic inhibition. Prevention is achieved by providing access to adequate amount of drinking water with safe salt levels.

Red Gut
This is a relatively rare condition in Australia. Sheep grazing very lush green pasture may develop acute abdominal pain, collapse and death. It is suggested that the cause of death is from arrested mesenteric blood flow following intestinal torsion.[16] The mechanism by which torsion develops is not well documented. No specific treatment is available other than possibly surgical correction of the torsion if detected very early.

Sand
Ingestion of sand can result in gastroenteritis and scouring or, alternatively impaction of gut contents. Large amounts of sand may be ingested when grazing very close to the ground, such as on badly eroded paddocks or under drought conditions. Diagnosis is based upon post-mortem findings and no specific treatment is available, although mineral oil, epsom salts, psylluim and fluid therapy are used in the treatment of equine impaction colic due to sand. Prevention involves providing adequate feed to prevent scavenging and avoiding feeding concentrates on bare sand.

Nutritional diarrhoea
'Nutritional diarrhoea' is a diagnosis of exclusion that often made when sheep grazing lush green feed develop

diarrhoea that cannot be attributed to any other factor. Typically, affected sheep are bright and maintaining or gaining condition.

The pathophysiology of nutritional scours is not well studied. Glastonbury suggested that the cause of this syndrome was related to the high water and soluble sugar content of lush pasture increasing gastrointestinal flow rate resulting in higher sugar concentrations in the caecum and colon. It was proposed this in turn caused affected animals to develop an osmotic diarrhoea.[2]

Water absorption in the large intestine is dependent on an osmotic gradient. Cation absorption, particularly sodium, plays an important role in water absorption.[14] Water content of faeces will increase when this osmotic gradient is poorly established due to rapid passage of digesta through the large intestine or malabsorption in the small or large intestine. High levels of intake result in rapid passage through the rumen and small intestine with insufficient time for adequate digestion and absorption of nutrients, resulting in digesta with relatively high osmolarity and the potential for loose faeces.[14]

The production of volatile fatty acids (VFA) in the rumen and hindgut may also affect the osmolarity of ingesta. Where production of VFA exceeds absorption, this can increase the osmotic load of ingesta in the hindgut. This syndrome is well documented in pigs.

However, little information is available to describe the mechanisms by which lush green feed may affect flow rate and faecal consistency and which nutritional components of the green feed are responsible for any such changes. In addition, little information is available that might explain why scouring occurs in particular individuals, mobs or classes of sheep more so than others when grazing under apparently similar conditions, either within a single property or district.

The nutritional factors that control faecal consistency in many monogastric species are well studied, both in the presence and absence of disease agents. Little literature is available is available on the impact of dietary components on the faecal consistency of sheep and whether nutrition interacts with infectious agents to determine the outcome of disease conditions.

Excess Protein

Excess protein in green feed is often cited as a potential cause for 'nutritional scours' on sheep grazing lush green feed. However, excess protein has not been proven to be a primary cause of gastrointestinal disease in sheep.

There is a potential on lush green feed for a protein-energy imbalance which may alter rumen and hindgut microbial population to favour proteolytic species. This may result in mucosal pathology attributed to the production of ammonia and biogenic amines in other species, including pigs. The role of these in sheep are not well documented.

High levels of protein in pasture will also result in increased ammonia concentration in the rumen. This can have adverse effects on rumen microbial population and there is potential for systemic subclinical and clinical ammonia intoxication where the crude protein in the diet exceeds 14%.

Level of feeding affects the extent of protein escape from rumen degradation. As intake increases relative to maintenance, so will rumen protein escape. If rumen undegraded protein passes through to hindgut, there is potential for osmotic diarrhoea. However, this may not occur in field situations, as the feeding of supplements high in protein (eg. lupins), and rumen undegradable protein (eg. canola meal), do not routinely cause scouring.

Excess soluble carbohydrate and acidosis

Rumen and hindgut acidosis occurs in sheep where soluble carbohydrate fermentation causes an alteration in the microbial population and pH in the rumen or hind gut. This is the topic of a paper by James Rowe presented in these proceedings and so will not be discussed in detail here.

There is potential for the development of sub-clinical or clinical acidosis to develop when grazing very lush pasture with high levels soluble carbohydrate, particularly where there are corresponding low levels of effective fibre (NDF) in the diet.

Fibre and soluble non starch polysaccharides

Ruminal pH is stabilised by a proper balance of slowly and rapidly fermentable carbohydrates (both fibre and

non-fibre carbohydrates) and adequate physical fibre (measured as particle length) to stimulate chewing activity and saliva production.[17] Physical fibre helps to stabilise rumen pH by stimulating the production of saliva which acts as a buffer to the acidity resulting from carbohydrate fermentation. Saliva is a highly effective buffer, being rich in sodium, potassium, bicarbonate and phosphate.[18] Fresh grass does not stimulate saliva production as much as coarse, fibrous feeds, such as standing dry feed and hay. Data from cattle shows that the grazing of fresh grass results in the production of 0.94 ml/g feed compared with 3.24 ml/g when grazing dried grass and 3.63 ml/g with hay.[19] This difference in saliva production and rumen buffering may contribute to the development of low ruminal pH and subclinical acidosis.

Large fibre particles may also act as a sieve in the rumen preventing larger particles from passing out the rumen.[20] As such, fibre length may play some role in the determination of particle size, digestibility and osmolarity of ingesta as it passes through the small and large intestines.

Structural polysaccharides in plant cells can also be grouped into insoluble structural polysaccharides (including cellulose and hemicellulose) and soluble structural polysaccharides (including pectins and β-glucans). Soluble structural polysaccharides are thought to be readily fermented in the rumen[21] although literature on this is difficult to source. In non-ruminants, particularly chickens and pigs, soluble non-starch polysaccharides (solNSP's) have been shown to have an antinutritive effect attributable to the formation of viscous ingesta in the small intestine.

Recent work suggests that digesta viscosity *per se* does not affect the nutrient absorption and endogenous nitrogen flow within the small intestine of pigs. Other properties of complex dietary fibre, digesta passage rate or bacterial activity probably contribute to observed changes.[22]

These also clear interactions between solNSP and disease agents affecting the expression or outcome of disease. In pigs, diets high in solNSP result in the expression of diarrhoea in response to infection with *E. coli* and spirochaetes.[23,24] Interactions between nutritional components and the expression of disease have not been studied in sheep.

Lipids

Lipid content of sheep feed stuffs is often overlooked, although high dietary inclusions are well documented as reducing digestibility and rumen stability in cattle. Rye grass may contain as much as 6% DM lipid content. Oats are another potential source of lipids.

Digestibilty may be reduced by the formation of a lipid coating on ruminal contents which possibly acts to restrict microbial fermentation. High levels of dietary fat have been shown to cause deficiencies of fat soluble vitamins, particularly vitamins A and E, and the chelation of calcium. The relative importance of lipid content of diet in sheep is largely unknown.

Minerals

Mineral imbalances in lush green feed have been proposed as a potential cause of scouring. Cation content of digesta affects the osmotic gradient and may affect faecal consistency. Sodium and potassium are the main cations in digesta with potassium absorbed primarily in the small intestine and sodium absorbed primarily in the large intestine. Malabsorption of cations due to enteric disease or very rapid flow rates may result in increased digesta osmolarity and therefore water contents of faeces.

Lush green feed often has very high potassium levels (3-4% DM), particularly if there has been a recent history of application of potassium fertilizer.[25] In sheep, little potassium absorption occurs in the large intestine[14], therefore where potassium intake exceeds the capacity of the small intestine for absorption, potassium will contribute to the osmolarity of hind gut digesta. Reduced absorption of other cations may contribute to increased osmolarity and faecal moisture.[14] Magnesium absorption is likely to be inhibited on pastures with very high potassium content.[25]

Mineral imbalances are not a proven cause of scouring in otherwise healthy animals, although may play a role in determining faecal consistency in animals grazing lush green pasture. Further work is needed in this area to determine the pathogenesis and relative importance of mineral imbalances in sheep.

Excess water intake

Excess intake of water, either in feed or due to polydipsia, will increase the amount of urine produced without significantly affecting faecal dry matter or consistency.

A study by Suttle and Field showed that the majority of water is excreted via urine with much smaller quantities excreted via respiration, perspiration and faecal loss.[26] Any excess water ingested in pasture is therefore likely to be excreted as urine and is unlikely to be related to changes in faecal consistency in otherwise healthy sheep on lush green feed.

Conclusions

Non-infectious causes of scouring in sheep are generally not well understood. In particular the role of various nutritional factors in determining faecal consistency (in both the presence and absence of infectious agents) warrants further investigation. Nutritional manipulation may provide options for management and prevention of scouring in sheep.

References

1. Radostits OM, Blood DC, Gay CC, Arundel JH. *Veterinary medicine : a textbook of the diseases of cattle, sheep, pigs, goats and horses*. London: Bailliere Tindall; 1994.
2. Glasonbury JRW. Non-parasitic scours in weaner sheep. In: *Sheep Medicine*. University of Sydney: Post Graduate Committee in Veterinary Science Proc 141; 1990:459-479.
3. Napthine DV. Scouring in Sheep. In: *Sheep Health and Production*. Sydney: Post Graduate Committee in Veterinary Science Proc 110; 1988:563-575.
4. Farquarson BC. *Sheep. Series A: Control and Therapy*. Sydney: University of Sydney Post Graduate Foundation in Veterinary Science; 1992.
5. Hungerford TG. *Diseases of livestock*. 8th ed. McGraw-Hill, Sydney, 1975.
6. Everist SL. *Poisonous plants of Australia*. Angus & Robertson, Sydney, 1974.
7. Galey FD. Feed Related Poisonings I - Nitrate, Gossypol and Botulism. In: *Clinical Toxicology*. University of Sydney: Post Graduate Foundation; 1998:37-42.
8. Pethick DW, Chapman HM. The effects of *Arctotheca calendula* (capeweed) on digestive function of sheep. *Aust Vet J*. 1991;68:361-363.
9. Harris DJ, Rhodes HA. Nitrate and nitrite poisoning in cattle in Victoria. *Aus Vet J*. 1969;45:590-591.
10. Eerens JPJ, Lucas RJ, Easton HS, White JGH. Influence of the ryegrass endophyte (Neotyphodium lolii) in a cool moist environment. II. Sheep production. *New Zealand J Agric Res* 1998;41:191-199.
11. Fletcher LR, Sutherland BL, Fletcher CG, Matthew C. The impact of endophyte on the health and productivity of sheep grazing ryegrass-based pastures. In: *Grassland Research and Practice Series - New Zealand Grassland Association.*: ew Zealand Grassland Association; 1999:11-17.
12. Leathwick DM, Atkinson DS. Dagginess and flystrike in lambs grazed on *Lotus corniculatus* or ryegrass. In: *Proc NZ Soc Anim Prod*; 1995:196-198.
13. Pownall DB, Lucas RJ, Familton AS, Love BG, Hines SE, Fletcher LR. The relationship between staggers and diarrhoea in lambs grazing different components of endophyte-infected ryegrass. *Proc NZ Soc An Prod*. 1993;53:19-22.
14. Wesselink R, Waghorn G, McNabb W. Causes of Dagginess in Sheep. In. Palmerston North: AgResearch; 1995.
15. Raisbeck MF. Feed Related Poisoning. In: *Clinical Toxicology*. Sydney: Post Graduate Foundation in Veterinary Science; 1998:119-125.
16. Barrell GK, Gumbrell RC, Reid TC. Artificial induction of red gut in sheep. *Res Vet Sci*. 1989;46:318-321.
17. Allen MS. Relationship between fermentation acid production and the requirement for physically effective fibre. *J Dairy Sci*. 1997;80:1447-1462.
18. Oetzel GR. Application of forage particle length determination in dairy practice. *Compendium of Continuing Education*. 2001;23:S30-S37.
19. Bailey CB. Saliva secretion and its relative feeding in cattle. 3. The rate of secretion of mixed saliva in the cow during eating with an estimate of the magnitude of the total daily secretion of mixed saliva. *Brit J Nutr* 1961;15:443-451.
20. Nordlund KV, Garrett EF, Oetzel GR. Herd-based rumenocentesis: A clinical approach to the diagnosis of subacute rumen acidosis. *Compendium of Continuing Education*. 1995;18:S48-S56.
21. Annison EF, Lindsay DB, Nolan JV. Digestion and Metabolism. In: Freer M, Dove H, eds. *Sheep*

Nutrition. Wallingford; UK: CAB International; 2002:95-118.

22. Bartelt J, Jadamus A, Wiese F, Swiech E, Buraczewska L, Simon O. Apparent precaecal digestibility of nutrients and level of endogenous nitrogen in digesta of the small intestine of growing pigs as affected by various digesta viscosities. *Arch Tierernahr*. 2002;56:93-107.
23. Pluske JR, Pethick DW, Durmic Z, McDonald DE, Mullan BP, Hampson DJ. Diseases and conditions in pigs, horses and chickens arising from incomplete digestion and absorption of carbohydrates. *Recent Advances in Anim Nutrition in Australia*. 1997:33-41.
24. McDonald DE, Pethick DW, Mullan BP, Hampson DJ. Increasing viscosity of the intestinal contents alters small intestinal structure and intestinal growth, and stimulates proliferation of enterotoxigenic Escherichia coli in newly-weaned pigs. *Brit J Nutrition*. 2001;86:487-498.
25. Pethick DW, Rowe J. Nutritional Diarrhoea in sheep. In: *IWS Workshops: Sustainable Worm Control & Scouring in Sheep*. Parkville; 1998:50-52.
26. Suttle NF, Field AC. Studies on magnesium in ruminant nutrition. 8. Effect of increased intakes of potassium and water on the metabolism of magnesium, phosphorus, sodium, potassium and calcium in sheep. *Brit J Nutr*. 1967;21:819-31.