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TITLE OF CASE

Laminitis in dairy goats *(Capra aegagrus hircus*) on a low forage diet

SUMMARY

Dairy goats on high concentrate diets attain high production levels, but at what cost? Here, ongoing lameness problems in a herd offered *ad lib* concentrates and roughages throughout their lifetime were investigated. Five severely affected, chronically lame animals were euthanased and examined post-mortem.

Foot pathology consisted of distortion of the claw shape and irregular fissures over the solar and bulbar horn with the distal phalanx rotated downwards on two claws.

Rumen pH measured between 5.26 and 5.46 with moderate rumen mucosa hyperkeratosis, and ulcerative, mild lymphocytic rumenitis. Feet showed irregular hyperplasia of the epidermal laminae with parakeratotic hyperkeratosis, especially in solar regions. Dense clusters of lymphocytes expanded the dermal laminae.

Based on these findings, chronic laminitis was suspected. Ruminal hyperkeratosis was likely a result of prolonged periods of acidosis. The consequences of feeding a high concentrate ration throughout the life of dairy goats needs more research.

BACKGROUND

The dairy goat (*Capra aegagrus hircus*) industry is continuously looking for ways to improve its product in terms of both the quantity and quality of the produced milk, whilst maintaining high animal welfare standards. The industry in Southern Europe is substantially larger than its British counterpart. Eurostat (www.ec.europa.eu) reported that in 2015 there were 100,000 goats in Britain compared with 4.0, 3.0, 1.2 and 1.0 million in Greece, Spain, France and Italy, respectively. Subsequently, theoretical and practical husbandry knowledge often originates from continental Europe before being implemented in Britain to suit local conditions and facilities.

It has been previously reported that the goat can withstand both acute acidosis and prolonged periods of subacute ruminal acidosis (SARA) without detrimental effect on health and welfare (1, 2). This ability is thought to derive from the goats' natural tendency to browse rather than graze, allowing them to adjust their feeding behaviour around food availability (3). This perceived ability, coupled with the lack of high quality roughages in some areas of Southern Europe, has led to trials being conducted where goats are offered high concentrate rations of up to 85-90% of total dietary intake (4, 5). These trials have indicated an improved animal performance (liveweight gain and milk yield), without any significant impacts on goat health, when compared to low concentrate rations of up to 30% and poor-quality roughages (6). However, other studies have reported this adds no beneficial effect on production when compared to a ration with lower concentrates but consisting of high quality roughages (7). To the best of the authors' knowledge however, there are no published data documenting the impact on production and health of feeding high level concentrate diets for a period longer than 250 days. Most of the relevant studies have used small groups of animals (up to 12 animals per group) rather than an entire herd at different stages of lactation. Indeed, other workers reported noticeable individual variation between the goats studied, indicating that some goats are more resistant to ruminal acidosis-related disease than others (8). The feeding of a high concentrate diet to a whole herd of goats could therefore result in a proportion of the goats being exposed to ruminal acidosis whereas others cope well with the diet.

In cattle, a link between inadequate nutrition, and an increased incidence of lameness has often been reported, especially in the case of SARA (9-11). However, the role of other factors, such as housing conditions, hormonal changes around calving and general inflammatory mediators, may be more significant than previously expected (12-14). In goats, it has been shown that the rumen loses some of its protective function when they are fed diets of 60% barley (*Hordeum vulgare*) for up to six weeks, resulting in a rumen pH below 5.5 (15). However, so far, no link has been made between nutrition in dairy goats and the occurrence of lameness.

CASE PRESENTATION

The case report presented here is part of a preliminary investigation into a possible link between high levels of concentrates feeding and the health status of animals at different stages of production (youngstock, dry period and lactation) using a more suitable cohort of observed animals (farm scale) than has currently been reported. We report on a detailed clinical, gross post-mortem and histopathological examination of goats euthanized for severe lameness problems, to gain a better understanding of links between lameness and gut health possibly driven by the high concentrates feeding regime observed within the studied cohort of animals.

This work was carried out as part of a clinical investigation by Langford Veterinary Services Farm Animal Practice (University of Bristol), as requested by the farmer, due to ongoing severe lameness issues on the farm. The farm was located in the South-West of England and consisted of 320 lactating dairy goats and approximately 80 hoggets and 70 kids. The breeds on the farm were Toggenburg, British Alpine and British Saanen. During these previous investigations, it also became clear that two different types of lameness were seen. The first cause of lameness were severely infected lesions on the sole of the claws. During previous investigations of this problem, the involvement of treponeme bacteria was established by PCR on swabs taken from the lesions. *Dichelobacter nodosus*, the causative agent of footrot, had not been found by PCR (16, 17). The second cause of lameness was thought to be caused by a specific abnormal hoof conformation. The feet would have one normally formed claw and one elongated, 'boxlike' claw. Although no lesions were presented on the claws, the animals were seen to be severely lame on these legs.

Previously, measures taken to prevent and treat infections with *treponeme spp*., such as hygiene measures, isolating affected animals and treatment with antibiotics, did not improve the situation on the farm as much as expected. Therefore, it was decided to perform post-mortem examinations to further investigate the aetiology of the lameness caused by the abnormal hoof conformation.

INVESTIGATIONS

Husbandry and nutrition

All goats, including youngstock, were housed indoors in straw yards all year round. The adult goats were housed in three different groups, according to stage of lactation. The youngstock were housed in two different groups, one group of kids born that year and one group of hoggets. All animals were given the same diet from weaning onwards, consisting of *ad lib* concentrates with unlimited access to roughages, regardless of their age, stage of lactation or pregnancy.

The concentrates were offered in open troughs, running the full length of each yard and were accessible from both sides, ensuring enough spaces for each goat to eat at the same time. The farmer filled up the concentrates each morning, ensuring the feeding was truly *ad lib*. Goats were therefore never seen gorging or waiting for fresh concentrates.

Tables 1 and 2 show the formulation of the concentrates used on farm, which were designed to provide high levels of protein and rapidly digestible carbohydrate. No analysis of the diet was made at the time because of the costs involved.

The roughages were offered on large racks, two in each yard. These were filled with new bales once the old ones were finished. Roughages consisted of either perennial ryegrass (*Lolium perenne*) hay or perennial ryegrass silage from individually wrapped bales, depending on what was available. The quality of these roughages could vary considerably between bales as well as between silage or hay cuts throughout the year. As the amount of roughages would be low at times, the feeding space limited and the quality and type of roughages varied, it was considered the intake of roughages would vary more than the intake of concentrates.

The diet was formulated by the farms' nutritionist, based on the assumption that the goats would self-regulate concentrate intake. High yielding animals were expected to consume up to 70% of their diet from concentrates and 30% of roughages whereas low yielding animals were expected to eat considerably less. The true intake of each individual however remained unclear as it was impossible to measure in the farm setting.

Table 1 Feed ingredients used

Feeds and Energy	kg fresh weight unless indicated
Soya (*Glycine max*) hulls	25.00
Extracted rape (*Brassica napus*) meal	22.00
Barley (*Hordeum vulgare*)	13.50
Palm kernel *(Elaeis guineensis*) exp.	12.50
Wheatfeed	10.00
Molasses	7.00
Soya (hi pro)	5.00
Megalac	1.00
Fat 100%	1.00
Limestone	1.30
Cal Mag	0.20
Trace Elements	1.00
Salt (Sodium Chloride)	0.50

Metabolisable Enery (Ingredients)	12.3 MJ/kg
Metabolisable Energy (Analysis)	12.0 MJ/kg

Table 2
Feed made up to contain (Groenevelt and others 2015b)

DM[a] (%)	87.8
Crude Protein (% as fed)	18.2
Starch (% as fed)	10.7
Sugar (% as fed)	7.9
Oil (% as fed)	4.9
NCGD[b] (% DM)	75.7
NDF[c] (% DM)	39.3

a Dry Matter
b Neutral detergent Cellulase and Gammanase Detergent
c Neutral Detergent Fibre

Clinical inspection
The whole lactating herd was observed by veterinarian MG during one milking session. During milking, the number of goats with an abnormal hoof conformation were scored. While exiting the parlour, the animals were locomotion scored, on a flat surface in a straight line. Lameness scoring according to Anzuino et al. (2010) was used.
With regards to abnormal hoof conformation, 25% of animals had at least one leg that showed one normal claw and one 'box-like' claw (Fig 1). Lameness scoring revealed 62% of lactating goats were scored as 'lame' (LS>0) (Fig. 2).

Clinical examination of individually lame animals indicated two causes of lameness. The first being infected lesions on the sole of the claw, confirmed in previous investigations to contain *treponeme spp*. and no *Dichelobacter nodosus* (17). Approximately 60% of lameness was caused by these lesions. A more detailed description of those lesions is described by Groenevelt and other (2015b). The other cause of lameness seen was the abnormal hoof conformation as described above, without any lesions visible on the claw itself. Approximately 40% of lameness was caused by this abnormal hoof conformation.

The youngstock was not scored individually but inspected in their pens. No lameness was seen in the young animals up to kidding.

Pathological findings

At the time of this investigation, herd lameness had become increasingly severe where 30 animals were judged to be so severely affected with such a poor prognosis to recovery that it was advised that these animals be culled on welfare grounds. Of those 30 animals, five animals were selected for further post mortem investigation.

The animals, aged between three and six years, were selected based on cause of lameness ensuring that both the infected lesions and the abnormal hoof conformation were represented (Table 3). They were humanely euthanased on farm using intravenous boluses of sodium pentobarbital (Euthatal, Merial Animal Health Ltd.).

The animals were transported to the University of Bristol, School of Veterinary Sciences where the post mortems were carried out by a Veterinary Pathologist. Rumen pH was measured within 4 hours of death ranged between 5.26 and 5.46. The gross pathology findings were similar in all lame feet. There was marked distortion of the overall shape of the claws, with most of the animals showing marked overgrowth of the dorsal horn wall. There were marked irregular fissures throughout the solar and bulbar horn, measuring up to 20mm deep. Marked focal downward rotation of the distal phalanx was seen with marked distal phalangeal distortion and the phalangeal palmar and plantar bony edges displaying marked irregularity, interpreted as osteolysis (Fig. 3). Other significant gross lesions included the presence of focal, severe pleural adhesions in one animal, with multifocal, mild to, moderate cranioventral lung lobe consolidation in three animals. No other gross lesions were detected.

Histopathological changes were similar in all lame animals. Rumen mucosa showed moderate surface hyperkeratosis, moderate lamina proprial lymphoplasmacytic infiltrates and multifocal ruminal epithelial erosion and ulceration, thinning and sloughing, with occasional superficial mucosal and intraepithelial neutrophilic infiltrates present (Fig. 4). Although autolysis-induced artefacts such as epithelial separation were noted, the presence of both eroded and ulcerated mucosa and intraepithelial inflammatory infiltrates in the affected regions made a diagnosis of rumenitis much more likely.

The chief microscopic hoof lesion showed irregular hyperplasia of the epidermal laminae (Fig. 5a). The epidermal laminae exhibited marked multifocal to coalescing hyperkeratosis, most prominent in the solar region (Fig. 5a, b). Lymphocytes and plasma cells predominated these populations. Arterial intimal and medial proliferation of moderate to marked irregular collagen was occasionally noted in the solar and bulbar dermis (Fig. 5c). There were occasional multifocal mild perivascular accumulations of macrophages, often containing hemosiderin. There was multifocal extensive chronic marked perioplic, coronary, solar and bulbar dermal granulation tissue, this was most prominent in the solar region (Fig. 3). Lung lesions in four out of the five animals were consistent with chronic, mild to moderate, bronchointerstitial pneumonia, the cause of which was not determined.

Table 3 Animal details

Goat (breed)	Feet with infected lesions	Feet with abnormal hoof conformation	Other pathological lesions
765 (Sannen)	right front and hind - medial claw	not applicable	bronchointerstitial pneumonia
84 (Saanen)	right hind - both claws	right front	No other findings
130 (Toggenburg)	both hind - both claws	not applicable	bronchointerstitial pneumonia
96 (Saanen)	left front – both claws, right front – lateral claw	right front – medial claw	bronchointerstitial pneumonia
174 (Toggenburg)	right hind - both claws	right front – medial claw	bronchointerstitial pneumonia

DIFFERENTIAL DIAGNOSIS

Initial differential diagnosis for the foot lameness seen in the goats were either footrot, treponeme infection or a combination of those two together and secondary bacterial infection of a primary claw horn lesion such as white line disease or solar ulcer. Before the pathology was performed, differential diagnosis for the abnormal hoof conformation were thought to be natural occurring deformities, horn overgrowth due to a lack of trimming or incorrect trimming and deformed horn growth because of inflammation. Based on the pathology findings a diagnosis of chronic laminitis was made.

TREATMENT

Treatment and prevention

The advancement of the lesions seen in the feet of the euthanased animals confirmed the correct advice that the 30 severely affected animals should be culled. Treatment for those advanced cases was considered unsuccessful and it was therefore decided to focus instead on prevention of new cases of chronic lameness. Hygiene measures to prevent the spread of *treponeme spp*. had already been put in place. Therefore, new prevention measures focused on the ration. The farmer was unable to change the ration to a more roughage based system. To try and avoid the suspected changes in rumen pH in the goats, it was advised that the roughages always be of consistently good quality and readily available to encourage a greater proportion of their ration to include structural fibre which would help in rumen buffering and gut health throughout lactation. It was also advised to change to more appropriate concentrates for the non-lactating does and youngstock to suit their nutritional needs.

OUTCOME AND FOLLOW-UP

Due to the problems being of a chronic nature it was too early to comment on the full outcome at the time of writing. After strict culling of all chronically lame animals, lameness levels were forced down and very few new cases were noted in the subsequent months after the implementations were made.

DISCUSSION

This paper reports on the clinical, gross and microscopic pathological findings in a herd of goats offered an *ad lib* concentrates and roughages diet, from a commercial dairy farm in the South-West of England. Based on the histological findings, it is likely that chronic laminitis had played a role in the aetiology of lameness in this herd. Although the direct link between nutrition and laminitis in cattle is still unclear (19, 20) and little information is available for goats, in this case the feeding regime was suspected to be of importance, due to the ruminal hyperkeratosis observed in all five examined goats.

A feeding regime, consisting of *ad lib* access to both concentrates and roughages from weaning through to pregnancy, lactation, dry period and subsequent lactations is used on many dairy goat farms in Britain. Several papers report on the positive effect on milk production of feeding a high concentrate diet compared to a high roughages diet (21). However, in efficiency terms this effect seems to reach a plateau at around 50% concentrates, as goats fed higher ratios gain more body weight (BW) with no further increase in milk yield (22). Indeed, four of the five goats examined had high levels of intra-abdominal fat, indicating that at least some of the ration offered was not metabolically partitioned for lactation but rather to adipose tissue. As with dairy cattle, a high body condition score around gestation can increase disease before, during and after parturition, such as dystocia, ketosis and hepatic lipidosis (2, 23). Although not recorded in detail, these conditions were all seen on the farm.

The use of an *ad lib* concentrate/roughage feeding system relies heavily on the ability of the goats to adjust their own feed intake and selection (24). Although the feed appeared to contain sufficient levels of neutral detergent fibre (NDF), it was predominantly in the form of high density short fibre from soya hulls (ca. 70% NDF) and palm kernel (ca. 65% NDF) and little structural-functional fibre to stimulate saliva production and buffering

capacity in the rumen. These NDF sources are typically highly digestible and when fed with roughage, which provide functional fibre (e.g. straw), can form a balanced feed to regulate rumen pH and provide high quality digestible carbohydrate for maximal rumen microbial protein formation. However, the provision of high density short fibre NDF should not be formulated as a replacement of forage NDF and free access to poor quality silage or hay (high DM content hay ca. 10%, poor quality silage <30% DM) in the current study may have resulted in a lack of self-regulation by some goats, increasing incidence of SARA. Learned aversion following prior exposure to poor quality silage would likely see a low intake of forage NDF in favour of higher concentrates intake and therefore a greater propensity to develop low rumen pH (25). In addition, it has been reported that there is variation within goats to cope with ruminal acidosis based on chewing behaviour to buffer rumen pH (26). Unfortunately, no full feed analysis was carried out at the time of the post-mortem examination and the only information available was that of the concentrates' constituents.

The rumen pH of each lame animal, measured four hours post-mortem, was below 5.5. Part of the difficulty in establishing an association between ruminal acidosis and laminitis in cattle is due to the difficulty in establishing a definitive rumen pH range (13). Ruminal epithelial hyperkeratosis, thinning and ulceration was demonstrated histologically in all the lame animals from this study, this is consistent with experimentally induced caprine ruminal acidosis (27). Prolonged ruminal acidosis has been reported as a cause of similar rumen lesions in cattle (28). A connection between starch content in feed rations and the incidence of bovine laminitis (29, 30) has been demonstrated. In goats, high grain diet feeding over a 7-week period can cause ruminal epithelial cell and intercellular junction damage (27) while chronic ruminal acidosis has been shown to have a detrimental effect on caprine ruminal epithelium's protective abilities, leaving it more permeable to microorganisms and endotoxins (15).

Gross haemorrhage was present in the plantar and palmar region of the distal phalanx dermis of the sole, wall and coronary regions. Infiltrating leukocytes were found in the dermis of all affected goats. Leukocytes still likely play a role in the physiopathology of injuries associated with naturally acquired laminitis and possibly are present as a response to antigen exposure following the loss of integrity of the overlying dermo-epidermal junction.

For future investigations, fresh unfixed tissue PCR, immunohistochemistry or in situ hybridization to establish the presence of *treponeme spp.* in claws with similar appearance to the ones presented in this case should clarify if there may be an infectious component to the etiology of these lesions as well.

Bronchointerstitial pneumonia was detected in four out of the five goats; while a link between pneumonia-induced sepsis and laminitis has been shown in horses (31), the relatively moderate nature of the lung lesions and the lack of pathological changes noted in other organs would make a diagnosis of sepsis-induced laminitis less likely. Further work is required on the effects of sepsis-induced disease in goats.

In this study, chronic cases of laminitis were confirmed as a cause of lameness in several goats on a farm feeding a high concentrate diet throughout the life of the animal. In addition, it was evident that overfeeding of concentrates and possibly lack of assumed self-selection of forage was putting animals at risk of metabolic disease, probably resulting in SARA, ruminal hyperkeratosis and excess fat deposition. Further research is needed to investigate the full consequences of using a high concentrate ration on a commercial dairy goat farm through all production cycles versus the development of rationed diets for

rearing, lactation, dry periods and transition which match the metabolic requirements of the animals, as with current dairy cow rations.

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LEARNING POINTS/TAKE HOME MESSAGES

- Laminitis must be considered as a cause of lameness in dairy goat herds fed a high concentrate diet
- A high concentrate diet can cause ruminitis and ruminal hyperkeratosis in dairy goats
- Other possible negative effects of feeding a high concentrate diet through all lactation stages are unknown and more research needs to be done.

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FIGURE/VIDEO CAPTIONS

Figure legend
Figure 1
Foot of goat with one normal left claw and elongated and boxlike deformed right claw.
Figure 2
Results of lameness scoring (LS) (n=317) according to protocol described by Anzuino and others 2010.
Figure 3
Longitudinal section animal 96 left fore exhibiting P3 osteolysis and granulation tissue, inset solar aspect of left fore.
Figure 4
Section of rumen mucosa showing ulcerative rumenitis, Bar = 50µm.
Figure 5a
Composite section subgross, with selected area (box) showing dermis, basal cells and solar horn. Bar = 5cm.
Figure 5b
The chief microscopic lesion was marked irregular hyperplasia of the epidermal laminae, from area within white box. Bar = 100µm.
Figure 5c
Moderate to marked, perivascular to band-like, inflammatory cell infiltration was noted in the sub-epidermal superficial and deep dermis. Bar = 50µm.

OWNER'S PERSPECTIVE

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