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Effects of high concentrate:forage ratio diets containing monensin on the management of ruminal acidosis in Gezhel lambs

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ABSTRACT

A total 20 male Ghezel lambs were fed diets containing different concentration of monensin (0, 10, 20 and 30 mg/kg DM) in a completely randomized design. Diets were offered ad libitum in the form of total mixed ration. Feed conversion ratio (FCR) improved significantly by supplementation of diets containing high amount of concentrate with monensin ($p < 0.01$). Rumen liquor pH was significantly ($p < 0.01$) affected by monensin and the level of concentrate in the ration (6.38 for 30 mg monensin/kg DM vs. 5.72 for 0 mg monensin/kg DM). Treatments had significant effect on the rumen reduction potential (RP) and total acidity ($p < 0.01$) with higher rate (342.50 Sec., and 16.39) for 0 mg monensin/kg DM and lower (271.25 Sec., and 15.26) for 10 mg monensin/kg DM and control diet, respectively. Monensin did not have a remarkable effect on sedimentation and flotation (SAF) and blood metabolites. The results showed that diets containing monensin may reduce the incidence of laminitis in lambs fed high concentrate: forage ratio due to the increased rumen pH and acidosis management.

1. Introduction

Laminitis is an inflammation of the sensitive layer of tissue in the foot (laminae) that occurs in acute, subclinical, and chronic forms (Bergsten, 1994; Greenough and Vermunt, 1991; Mackie et al., 1984; Nordlund and Garrett, 1994). Many predisposing factors are associated with the occurrence of laminitis including farm management, housing, genetics, breeding, and nutrition (Vermunt and Greenough, 1994; Ossent and Lischer, 1994). Although the etiology of laminitis is multifactorial, nutrition is considered as an important risk factor (Pilachai, 2013; Vermunt, 2000; Bergsten, 2003). Laminitis, polioencephalomalacia, and liver abscesses often accompany acidosis (Owens et al., 1998). The potential consequences of subacute ruminal acidosis (SARA) like laminitis, milk fat depression, poor body condition and others have been reviewed thoroughly (Kleen et al., 2003; Plaizier et al., 2008; Enemark, 2008). Laminitis is regularly mentioned as resulting from acidosis as there is a correlation between ration type, ruminal fermentation and hoof lesions (Bergsten, 1994; Nocek, 1997). Rumen acidosis is considered to be a major predisposing cause of laminitis (French, 2009; Nocek, 1997; NRC, 2001); presumably mediates its destructive effects through various vasoactive substances (like histamine) that are released into the blood stream with the development of rumen acidosis (French,

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2009). Histamine is a potent vasodilator that increases capillary permeability (Brent, 1976). The association of histamine fits well with the nutritional theory of laminitis development (Nocek, 1997). The ruminal production and accumulation of histamine are generally associated with low pH (Dain et al., 1955; Van Der Horst, 1961; Irwin et al., 1979). Feedlots or herds with high incidence of locomotory problems such as laminitis, foot rot, and claw lesions might be more likely to experience ruminal acidosis (González et al., 2012). In recent studies by Pilachai et al. (2012), the feeding of high amounts of starch rich concentrates (i.e. 10.5 kg DM containing 40% starch) in combination with a low roughage intake (i.e. 1.5 kg DM) resulted in rumen acidosis and cases of subacute laminitis. In lambs, an overload of corn syrup induced lactic acidosis and acute laminitis (Pierson and Jensen, 1975). Morrow et al. (1973) showed that an intraruminal administration of a high dose of lactic acid (0.35% of body weight) induced laminitis in lambs. Thus, management of ruminal acidosis is critical in preventing laminitis. The influence of carbohydrate on ruminal pH is the critical link among nutrition, acidosis and laminitis. Relatively few studies have evaluated the direct influence of carbohydrate per se on the incidence of laminitis. The incidence of laminitis can be controlled through good nutrition management (high fiber diets), by using good feeding and management practices (Nocek, 1997). Acidosis control includes feed additives that inhibit microbial strains which produce lactate. This mode of action stimulates activity of lactate-using bacteria or starch-engulfing ruminal protozoa which in turn reduces meal size. Feeding management (such as forage to concentrate ratio, forage and grain particle size and bunk management) and non-nutritive additives (i.e. ionophores, malate, direct-fed microbials and buffers) may reduce ruminal acidosis and other health related problems (Owens et al., 1998). Rumen modifier products such as monensin, lasalocid, tylosine and virginamycine have been used successfully to control the risk of acidosis (Vermunt, 2006). Despite the efforts in order to ban antibiotics, monensin is still used by animal nutritionists working for most feedlots worldwide (Gomes et al., 2009). Monensin is an ionophore that has been extensively used for more than 30 years in beef feedlot diets to improve feed efficiency and prevent acidosis (Gonzalez-Momita et al., 2009). Other results doubt the effectiveness of monensin in SARA prevention (Mutsvanga et al., 2003). Monensin selectively inhibits Gram-positive bacteria, thereby impacting ruminant metabolism by increasing efficiency of energy metabolism, improving nitrogen metabolism, and reducing bloat and lactic acidosis risk (Schelling, 1984). The major claims for dietary monensin inclusion are to improve feed efficiency by increasing weight gain and, in most studies, by decreasing feed intake (Frumholtz, 1991). Gastaldello Junior et al. (2013) observed that concentrations of acetate and propionate in feedlot lambs fed high grain diets were lower ($p < 0.05$) and there were no differences in ruminal pH, water intake and ruminal acetate: propionate, and ruminal butyrate concentration when the monensin was added. The use of additives in sheep production is not a common practice even when these modifiers may reduce metabolic disorders and improve gain or feed efficiency. Nutritional factors may potentially moderate the incidence and severity of lameness by contributing to the occurrence of subclinical laminitis, which, in this situation, is likely to be associated with subacute rumen acidosis (Vermunt, 2006). Horton et al. (1980) reported that Plasma glucose was not affected by either dietary barley level or monensin, whereas plasma urea nitrogen increased ($p < 0.001$) with higher levels of barley. Monensin, and live yeast supplementations significantly decreased ($p < 0.05$) the plasma urea-N concentration while plasma β-hydroxybutyrate (BHBA), glucose and other blood parameters were unaffected (Ding et al., 2008). Ruminal pH was higher ($p < 0.05$) in lambs fed monensin and decreased linearly ($p < .05$) with increasing levels of barley in the diet (Horton et al., 1980). Our hypothesis was that ruminal pH would be higher in lambs fed high grain diets containing monensin. By doing this study we worked toward two objectives:

A. Investigating the effects of monensin on rumen fermentation pattern of feedlot Ghezel lambs fed high grain diets and
B. Demonstrating the link among nutrition, acidosis and laminitis.

2. Materials and methods

2.1. Animals, diets and sampling procedures

20 male Ghezel lambs (mean live weight 24.5 ± 4.9 kg) were allocated to individual cages for evaluation of different levels of monensin on Ghezel lambs performance by step-up feeding program. The experiment was carried out over 8 weeks by completely randomized design with five treatments (control diet and rations contain 0, 10, 20 and 30 mg monensin/kg DMI) and 4 replications. After 2 weeks of adaptation and acclimatization period, animals fed by experimental diets for 6 weeks. At the start of experiment, Forage (F):Concentrate (C) ratio was 45:55 that was fixed for control treatment till the end of experiment. However, we changed the ratio every week. While Forage was decreased by 5% per week, concentrate was increased 5% weekly. Therefore, at the end of experiment F:C ratio was 20:80 with different levels of monensin. Diets were composed of alfalfa hay, wheat straw, barley, soy bean meal, vitamin–mineral supplement and salt. Diets were offered ad libitum to all animals 2 times a day with free access to water and salt. Daily feed intake for individual lambs was estimated from difference between offered feed and refusals. The live weights of lambs were recorded on the first day of each week of the study before morning feeding. Blood samples were collected from 4 lambs of each treatment by jugular venipuncture into a heparinzed evacuated tube 4 h after morning feeding at the start and last week of experiment. The samples were analyzed for glucose, urea, and total protein of serum. Rumen fluid was collected via ruminal tube before morning feeding and squeezed through 4 layer of cheesecloth and analyzed for pH, total acidity, reduction potential (RP), and sedimentation and flotation (SAF).

2.2. Statistical analysis

Data were analyzed with an analysis of variance of a completely randomized design with 5 treatments and 4 replications. Treatment effects on DM intake, weight gain, feed efficiencies, rumen parameters and blood metabolites were analyzed using the GLM procedure of a completely randomized design of SAS software. The Significant difference between means was determined by Duncan's Range test. Initial live weight was used as a covariable and covariance analysis was conducted on live weight gain.