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Claw lesions and rumen liquor profile in dairy cattle on high grain diet

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ABSTRACT

The present study is conducted to investigate how long the effect of feeding high grains is sustained in the rumen and how this changed or adapted rumen environment is going to affect the hoof health in approximately 10 weeks. Crossbred cows (18) from university dairy farm were divided into 2 groups keeping equal number of animals with same parity in both. Animals in the feeding group were fed with high grain diets for 75 days. The pH, sodium, potassium and different fractions of total volatile fatty acids (TVFA) of rumen liquor were studied every fortnight. All the animals were evaluated for foot lesions before the start of experiment and then at the end of feeding trial. A significant decrease in rumen liquor pH and sodium concentration was observed in the feeding group animals with the progress of time. TVFA concentration increased in the feeding group with rise in acetate, propionate and butyrate levels at the end of the trial. High prevalence of heel erosions, white line haemorrhages, white line fissures and overgrown soles were observed in the feeding group animals at the end of trial along with an increase in rear leg view index. It was concluded that constant feeding of non-structural carbohydrates may increase total volatile fatty acids and frequency of claw lesions in crossbred dairy cattle.

Key words: Dairy cattle, Foot lesions, Grain diet, Lameness, Rumen liquor, Rumen profile, Volatile fatty acids

Excessive feeding of high grain diet causes excessive production of total volatile fatty acids (TVFA) in the rumen, which leads to reduction in rumen motility (Furll *et al.* 1993) as well as ruminal pH (Krause and Oetzel 2006). The shift in the microbial population of the rumen is mainly responsible for acidosis. With the increase in carbohydrate intake, gram positive microbes responsible for digesting this nutrient increase. These gram positive bacteria *Streptococcus bovis* and *Allisonella histaminiformans* produce histamine, lactic acid, endotoxins and other volatile fatty acids (VFA) (Nocek 1997, Garner *et al.* 2002) and cause a decrease in rumen pH (Suber *et al.* 1979). Bacterial endotoxins and histamine were observed to inhibit rumen motility (Hoeben *et al.* 2000). Ruminal pH in dairy cattle with ruminal acidosis is usually closer to a pH range of 5.5 to 5.6 (Keunen *et al.* 2002). Surveys suggested that incidence of sub acute ruminal acidosis was between 19% and 26% in early and mid-lactation dairy cows respectively (Plaizier *et al.* 2008). Causes of sub acute ruminal acidosis include feeding excessive amounts of non-structural carbohydrates and highly fermentable forages and insufficient dietary coarse fiber. Consequences of sub acute ruminal acidosis include feed intake depression, reduced fiber digestion, milk fat depression (Hendry *et al.* 1997) diarrhoea, laminitis, liver abscesses, increased production of bacterial endotoxins and inflammation characterized by increase in acute phase proteins (Plaizier *et al.* 2008). Bacterial endotoxins when absorbed into circulation, affect the digital capillaries and lead to edema, ischemia and disruption of dermo-epidermal junction (Shearer 1998) and ultimately result in different types of lesions.

Though the effect of high grain diet has been investigated in previous studies but usually it is done in controlled experiments. The present study was conducted to investigate how long the effect of feeding high grains is sustained in the rumen and how this changed or adapted rumen environment is going to affect the hoof health in approximately 10 weeks. Ten weeks is a period, which is usually considered as time elapsed for the degenerative changes at the dermo-epidermal junction to reach the sole surface (Shearer 1998).

MATERIALS AND METHODS

Animals and feeding: Holstein Fresian-Sahiwal crossbred cows (18) with total milk yield per lactation between 4,000–5,000 kg (control group: 4,750.16 kg; feeding group 4,753.91 kg) were selected on the basis of first and second parity from university dairy farm. Animals were segregated into 2 groups keeping proportionate number of animals of the same parity in both groups. Six animals were fed on routine diet i.e. control in concentrate forage ratio 50:50 and 12 animals were fed on concentrate

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ration having high amount of nonstructural carbohydrates according to NRC feeding standards. The composition of concentrate mixture in control group was (maize 30, *bajra* 5, mustard cake 20, soybean meal 5, full fat roasted soya 4, groundnut cake 4, wheat bran 10, rice polish 5, mollases 4, deoiled rice polish 8 dcp 1, bioplex 0.1, limestone power 0.5, mineral mixture 2 and salt 1 parts) whereas in high grain diet, wheat (30 parts) and refined wheat flour (5 parts) was taken; rest of all the feed ingredients were same as in control diet. The chemical composition of the control and high grain diet is presented in (Table 1). The rumen liquor was collected every fortnightly starting from 0th day on 15th, 30th, 45th, 60th and 75th day and analysed for pH, total volatile fatty acids, and sodium and potassium concentrations.

Table 1. Chemical composition of concentrate mixture, % DMB

Component	High grain diet	Control diet
CP	19.26	19.56
EE	4.6	4.5
ADF	14.8	17.1
NDF	31.8	33.2
ASH	7.6	7.2
OM	92.4	92.8
Hemicellulose	17	16.1

Lameness scoring and rear leg view conformation: On the day of sampling, each animal was allowed to walk on the flat surface for up to 30 m and gait of each animal was examined on a 5 point scale. Score 0 was assigned for 'No gait abnormality on walking', 1 for 'Mild lameness, with slight gait asymmetry at walk', 2 for 'moderate lameness, with moderate and regular gait asymmetry', 3 for 'severe lameness, with obvious gait asymmetry or severe symmetric abnormality' and 4 for 'recumbent or non-ambulatory animals'.

The rear leg view was recorded in squarely standing dairy cows by observing the position of hocks i.e. narrow hocks (cowhock rear leg view) or straight hocks (normal rear leg view) (Randhawa *et al* 2008).

Rumen liquor biochemical profile

Sampling: Rumen liquor samples were taken with the help of rumenocentesis (Duffield *et al*. 2004) by using 16 gauge 4 inch long needle from the left flank and collected in the 10 ml sample collection vials. Rumen liquor samples were evaluated for pH with the help of digital pH meter. Biochemical analysis of rumen liquor was carried out after filtering it through a double layer muslin cloth after centrifugation especially for estimation of sodium and potassium. The samples were stored at –20°C till further analysis.

Total volatile fatty acids of rumen liquor (mMol/L): The rumen liquor sample was taken in a centrifuge tube and 0.5 ml of metaphosphoric acid was added to it, the tube was incubated at room temperature for 25 min and then centrifuged at 2,500 rpm for 15 min and supernatant was collected. One microlitre of supernatant was taken for the estimation of total volatile fatty acids by using gas liquid chromatography (GLC) method.

Rumen liquor Na^+ and K^+: Rumen liquor sodium and potassium concentration (mEq/L) was estimated by colorimetric method using diagnostic kits (Table 2).

Table 2. Comparison of rumen liquor pH, sodium and potassium concentrations in control and feeding groups at different time intervals (mean±SE)

pH	Day	Control group (6)	Feeding group (6)
	0	7.21±0.15	6.96±0.05^{b}
	15	7.18±0.10	6.61±0.12^{a}
	30	7.20±0.08	6.75±0.09^{ab}
	45	7.15±0.07	6.98±0.14^{b}
	60	7.23±0.04^{1}	6.95±0.13$^{ab, 2}$
	75	7.28±0.07^{1}	6.95±0.07$^{ab, 2}$
Sodium (mEq/L)	0	177.19±4.80	185.61±2.41^{c}
	15	175.84±2.90	169.76±5.31^{b}
	30	172.20±3.00	167.19±3.98^{b}
	45	175.41±4.07	163.96±2.50^{ab}
	60	174.16±1.99^{1}	156.61±2.92$^{a, 2}$
	75	177.10±2.25^{1}	155.54±1.69$^{a, 2}$
Potassium (mEq/L)	0	21.01±0.30^{a}	21.73±0.18^{a}
	15	21.36±0.27^{ab}	20.03±0.56^{b}
	30	21.22±0.26^{a}	20.96±0.41^{a}
	45	21.41±0.17^{ab}	20.94±0.36^{a}
	60	22.08±0.17b^{c}	20.73±0.22^{a}
	75	22.28±0.22^{c}	20.34±0.11^{a}

Figures between columns with different superscripts are significantly different at 5% level, figures within columns with different superscripts are significantly different at 5% level.

Claw lesions

All the 18 animals were evaluated for the presence of any kind of foot lesion before and after paring a layer of approximately 1mm of horn from the weight bearing surface (sole) after restraining them in trimming chute at the start of study and after 75 days of feeding trial. Heel erosions, sole haemorrhages, sole avulsions and white line haemorrhages were identified according to their severity (Randhawa *et al.* 2008). The rest of the foot lesions viz. sole ulcers, white line fissures, toe haemorrhages, abaxial wall overgrowth, abaxial wall degeneration, under run soles, double soles, overgrown hooves, overgrown soles, cork screw hooves and slipper foot were categorized as 1 when present and 0 when absent.

Statistical analysis

The data were analysed and evaluated statistically using Kruskal Wallis test of analysis of variance or H-test to analyse the effect of high grain diet on severity of foot lesions, locomotion score and rear leg view index in crossbred cows and Student's t-test distribution

(independent t-test) was used to analyse the effect of feeding of high grain diet on pH, sodium, potassium and total volatile fatty acid concentration between and within groups analysis using SPSS version 17 software.

RESULTS AND DISCUSSION

A significant difference between control and feeding groups was observed in pH at 60th day (P<0.05) and 75th day (P<0.01) of feeding of high grain diet (Table 2). Ruminal pH ranges from 6.61 to 6.98 with overall mean 6.8±0.09 in feeding group animals and from 7.15 to 7.28 with overall mean pH value of 7.2±0.08 in control group animals in the present study. Increased production of total volatile fatty acids in the feeding group animals might have caused a decrease in rumen liquor pH which has also been associated with increased osmolality of the ruminal contents and decreased feed intake (Slyter 1976, Carter and Grovum 1990). The resulting endotoxemia due to death of gram negative bacteria (Gozho 2005) might be responsible for the increased odds of lameness (Manson and Leaver 1987,1988) and other health problems resulting in decreased production (Krause and Oetzel 2006). Increase in the number of heel erosions, white line haemorrhages and fissures and overgrown soles in the present study was in full agreement with these findings.

A nonsignificant difference in ruminal pH was observed in both the groups within group comparison over the period of trial. It is well documented that ruminal pH is mainly affected by the amount of fermentable carbohydrate in each meal and the changes of 0.5–1.0 pH unit within a 24 h period are common (Dado and Allen 1993, Nocek *et al.* 2002). Since acid production in ruminants varies too much from meal to meal, therefore they possess highly developed system to maintain ruminal pH within a physiological range of about 5.5–7.0. The non-significant change in ruminal pH in feeding group animals over the period of trial in the present study clearly demonstrated the distinct property of ruminants to maintain pH within the physiological range.

A nonsignificant difference in rumen liquor sodium concentration was observed between control and feeding group at 0th, 15th , 30th and 45th day except 60th and 75th day (P<0.05) where significant variations were witnessed (Table 2). Within the group, though no significant difference was observed in control group but a significant decrease in rumen liquor sodium concentration was observed in the feeding group from the start of the trial to the end of study. A nonsignificant difference in the rumen liquor potassium concentration was observed within group and between group comparison in both control and feeding groups (Table 2).

First and foremost reason for decrease in the concentration of rumen liquor sodium level from the start of feeding to the end in feeding group animals might be increased osmolality of the ruminal contents (Carter and Grovum 1990) during the feeding of high grain diet. Secondly, it could be due to dilution of the rumen contents due to influx of water from the intravascular and extravascular compartments (Shinosaki and Nakabayashi 1974). On the other hand, Choudhuri *et al.* (1980) observed a decrease in sodium as well as potassium levels and an increase in calcium, magnesium and inorganic phosphorus levels. Though increased salivation during ruminal acidosis (Nokata *et al.* 1977) caused an increase in sodium concentration but the increased inflow of water into rumen might have masked this effect leading to decrease in sodium concentration in rumen liquor. Gianesella (2008) also associated peri-partum sodium concentration <139 mmol/l and prepartum potassium concentrations >4.7 mmol/l with subacute ruminal acidosis (SARA).

Rumen liquor acetic acid and propionic acid concentration increased towards the end of trial in feeding group animals with no significant difference in isobutyric acid concentration. The between group comparison of control and feeding group animals has no significant difference in rumen liquor acetic acid, propionic acid and isobutyric acid concentrations (Table 3). A significant difference in rumen liquor butyric acid and isovaleric acid concentrations was observed between control and feeding group animals at 15 days (P<0.05) and valeric acid concentration at 45th day (P<0.01) during feeding of high grain diet. A significant increase in butyric acid and valeric acid concentration was observed within feeding group animals towards the end of trial, though there was no change in isovaleric acid concentration (Table 3).

Overall a significant increase in total volatile fatty acid (TVFA) concentration was observed from the start to the end of experiment in feeding group animals as compared to control group animals. This could be due to rapid and complete fermentation of high grain diet by the rumen microbes. Dairy cattle cannot tolerate diets proportionately high in concentrates as beef feedlot diets. Krause and Oetzel (2005) suggested that elevated total VFA concentration was the main cause of low ruminal pH. Acetate to propionate ratio in the present study remained between 2.62 to 3.05. This could be due to fact that sampling in the present study was done every fortnightly which was sufficient period for the rumen environment to stabilize after change of diet. In most of the previous studies, diurnal variation between the various fractions of volatile fatty acids was seen (Khorasani and Kennelly 2001) and the effect of high grain diet was not observed after feeding for long periods as in present study. The increased production of TVFA could be the result of the establishment of an acid-tolerant lactobacilli population (Therion *et al.* 1982). In sub-acute ruminal acidosis, the reason for drop in rumen liquor pH below 5.6 is the accumulation of VFA, which is a combination of overproduction and possibly due to decreased absorption. Lactic acid is produced during sub acute acidosis, it does not accumulate because lactate-fermenting bacteria remain active (Goad *et al.* 1998) and rapidly metabolize it to VFA. *S ruminantium* sub sp *lactilytica* metabolizes lactate to mainly succinate and propionate. *M elsdenii*, a predominant ruminal lactate-using organism under acidotic conditions produces butyrate, caproate, and valerate in the presence

Table 3. Comparison of rumen liquor volatile fatty acid concentrations (mMol/L) in control and feeding groups at different time intervals (mean±SE)

Acetic acid	Day	Control group (n=6)	Feeding group (n=6)
	0	84.5±4.7^{ab}	89.5±2.8^{a}
	15	95.5±2.7^{b}	89.5±3.7^{a}
	30	80.6±2.0^{a}	89.9±3.0^{a}
	45	85.8±3.4^{ab}	100.7±4.3^{b}
	60	93.6±3.9^{b}	104.0±4.0^{b}
	75	88.3±3.8^{ab}	120.9±2.9^{c}
Propionic acid	0	25.3±3.6^{ab}	30.1±1.9^{ab}
	15	19.0±1.9^{a}	27.1±1.4^{a}
	30	19.6±1.9^{a}	34.1±1.5^{bc}
	45	26.4±2.9^{ab}	33.0±2.3^{bc}
	60	30.4±2.8^{b}	37.4±2.5^{cd}
	75	27.7±3.0^{ab}	40.0±2.1^{d}
Isobutyric acid	0	1.7±0.1	1.7±0.0^{a}
	15	1.9±0.0	1.1±0.2^{a}
	30	2.1±0.6	2.1±0.6^{b}
	45	1.7±0.1	2.5±0.1^{a}
	60	2.4±0.5	2.6±0.1^{a}
	75	2.3±0.5	1.9±0.0^{a}
Butyric acid	0	17.2±1.1^{b}	18.6±0.6^{a}
	15	11.3±0.2$^{a, 1}$	15.5±0.9$^{a, 2}$
	30	15.9±1.0^{b}	16.4±0.7^{a}
	45	19.3±1.2^{b}	16.6±1.2^{a}
	60	17.2±1.7^{b}	24.1±2.8^{b}
	75	19.0±1.2^{b}	25.8±1.1^{b}
Valeric acid	0	1.9±0.1^{a}	1.9±0.2^{ab}
	15	2.1±0.1^{ab}	1.5±0.2^{a}
	30	2.3±0.1^{ab}	2.0±0.2^{ab}
	45	2.2±0.1$^{ab, 1}$	2.8±0.5$^{bc, 2}$
	60	2.3±0.2^{ab}	2.0±0.1^{ab}
	75	2.7±0.2^{b}	3.4±0.2^{c}
Isovaleric acid	0	2.2±0.1	3.1±0.2
	15	2.1±0.1^{1}	4.1±0.4^{2}
	30	2.2±0.0	2.4±0.2
	45	2.6±0.3	2.0±0.1
	60	2.4±0.3	3.2±0.9
	75	2.6±0.2	3.1±0.2
Total volatile fatty acid (TVFA)	0	132.8±9.0^{ab}	144.8±4.9^{ab}
	15	132.3±2.5^{ab}	136.4±5.1^{a}
	30	122.9±4.0^{a}	146.7±4.0^{ab}
	45	139.9±7.1^{ab}	155.8±7.2^{b}
	60	148.6±6.1^{b}	172.4±6.1^{c}
	75	142.6±6.9^{ab}	195.2±5.8^{d}

Figures between columns with different superscripts are significantly different at 5% level, figures within columns with different superscripts are significantly different at 5% level.

of glucose, with a concurrent reduction in propionate (Marounek *et al.* 1989). Sub-acute ruminal acidosis results in decreased rumen liquor pH due to accumulation of volatile fatty acids and insufficient rumen buffering (Plaizier *et al.* 2008).

There was no significant difference in locomotion score at different time intervals in both the groups. A significant difference was observed in rear leg view index ($x^2 = 13.438$; $P<0.01$) (Table 4) within group comparison of feeding group animals. The primary motive of our study was to see the impact of high grain diet on the severity of foot lesions which was only possible if we continue feeding of this diet for at least 10 weeks, the time required for changes at dermis level to reach to the solar surface (Shearer 1998). A significant difference was observed in heel erosions, white line haemorrhages (Fig 1), white line fissures (Fig. 2) and overgrown soles (Fig. 3) between control and feeding group animals in the present study (Table 5). It is known that any nutritional practice that decreases the rumen pH below 5.5 can result in chronic subclinical laminitis (Olson 1997). Though the rumen pH was not observed below 5.5 at any time interval in the present study, but a comparative low pH of rumen liquor was observed in the feeding group animals. Moreover, a corresponding increase was also observed in TVFA concentration in the feeding group that could be a possible reason for ruminal acidosis which might be responsible for more number of heel erosions, white line disease lesions (Figs 1, 2) and overgrown soles (Fig. 3) in the feeding group. During acidotic conditions, many vasoactive substances are released which disturb the blood circulation in the feet (Nocek 1997, Shearer 1998). A reduction in systemic pH during acidosis activates a vasoactive mechanism that increases total blood flow to the hoof (Nocek 1997, Oetzel and Nordlund 1998).

Table 4. Within group comparison of locomotion score and rear leg view index in control and feeding group animals between start and end of experiment

	Control group		Feeding group	
	Test statistic	P-value	Test statistic	P-value
Locomotion score	0.177	0.915	1.217	0.544
Rear leg view index	0.000	1.000	13.438	0.001**

**Significant at 1% level.

Table 5. Within group comparison of different hoof lesions in control and feeding group animals between start and end of experiment

Lesions	Control group		Feeding group	
	Test statistic	P-value	Test statistic	P-value
Heel erosions	3.823	0.051	10.052	0.002**
Sole haemorrhages	0.017	0.895	0.330	0.566
Sole avulsions	3.811	0.051	3.242	0.072
White line haemorrhages	0.025	0.874	5.191	0.023*
White line fissures	0.415	0.519	4.488	0.034*
Under running soles	3.643	0.056	3.107	0.078
Overgrown soles	2.349	0.125	5.717	0.017*
Overgrown hooves	0.000	1.000	0.620	0.431
Sole ulcers	0.000	1.000	0.500	0.480

*Significant at 5% level of significance, **significant at 1% level of significance.

Figs 1–3. **1.** White line haemorrhage in a cow in feeding group; **2.** White line fissure in a cow in feeding group; **3.** Overgrown soles in a cow in feeding group.

Alternatively, histamine may be absorbed through rumen epithelium damaged during acidosis. Histamine is an inflammatory agent and vasoactive substance, and may increase blood pressure and damage blood vessel walls causing inflammation and haemorrhaging within the hoof. It is well established that grain feeding increases the formation of histamine in the rumen (Garner *et al.* 2002). Another cause of more number of lesions may be endotoxins produced due to death of microbes in the acidotic rumen environment. These endotoxins may get absorbed into the circulation and affect digital capillaries or vessels leading to ischemic hypoxic changes (Boosman *et al*. 1989) responsible for dermo- epidermal disruption (Shearer 1998) and subsequent claw lesions (Cook *et al.* 2004, Nordlund *et al.* 2004). It was also reported that laminitis does not occur exclusively under acidotic conditions, but acidosis is noted as a major predisposing factor (Olson 1997, Shearer 1998).

The impact of acidosis on laminitis may also be mediated by matrix metalloproteinases in a manner similar to the effects of parturition. At parturition, hormonal changes affect connective tissue metabolism within the hoof by elongating collagen fibers and loosening the connective tissues (Tarlton *et al*. 2002). In the case of laminitis, metalloproteinases are thought to be activated by exotoxins released by bacteria. Once activated, these metalloproteinases degrade key components of the corium (Mungall *et al.* 2001). On the other hand, some authors (Momcilovic *et al.* 2000, Donovan *et al.* 2004) reviewed that acidosis in cattle does not always result in laminitis, thus other factors appear to alter the susceptibility of cattle to acidosis induced laminitis. Cook *et al.* (2004) observed that animal's environment may exacerbate the effects of acidosis on laminitis.

There was no significant difference in locomotion score at different time intervals in both the groups. One reason for this may be that feeding of high grains was done for a very short period. Many authors have reported that non-lame cows have hoof lesions (Manske *et al*. 2002, O'Callaghan *et al.* 2003). Despite the presence of lesions in the hoofs of feeding group animals, they were not showing any degree of clinical lameness. This may be due to the reason that all the lesions were not able to cause clinical lameness in dairy animals. This might be the reason that though there were more numbers of heel erosions, white line lesions and overgrown soles in the feeding group animals, they could not cause clinical lameness. Zahid *et al*. (2014) made a similar observation and reported that sole ulcers and white line fissures were the only lesions responsible for clinical lameness in crossbred dairy cows. A significant difference was observed in rear leg view index ($x^2 = 13.438^*$; $P<0.01$) within group comparison of feeding group animals. It might be due to increase in number of lesions in claws of animals after feeding of high grain diet (Peterse *et al.* 1984) which might not cause change in locomotion score but definitely change the standing stance of the rear legs. With the increase in number of lesions, the placement of foot on the ground surface may get altered to relieve pressure on the digit having foot lesions which may be responsible for inward knuckling of hocks or cow hock rear leg view in the feeding group animals.

It was concluded that feeding of high grain diet in cattle definitely caused a change in the rumen environment with decrease in rumen liquor pH and sodium levels and increase in total volatile fatty acids. The continuous presence of acidic environment in the rumen could be a possible reason for increased frequency of claw lesions in the crossbred cattle on high grain diet.

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