SPECIES DIFFERENCES BETWEEN RUMINANTS IN SUSCEPTIBILITY TO MOLASSES TOXICITY

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SUMMARY

Rumen thiaminase activities were found to be unimportant in the development of molasses toxicity in beef cattle. Molasses-based diets produced elevated concentrations of propionic acid in both sheep and goats but not in cattle which exhibited high butyric acid concentrations. The importance of glucose supply to animals fed molasses diets is discussed.

INTRODUCTION

The role of molasses as a feed for livestock has changed in recent years from that of a carrier for urea and minerals to the realization that it could provide most of the dietary energy intake of animals (Gulbransen 1984).

Although ad lib diets of molasses/urea or fortified molasses diets (8% urea plus protein meals) are of high dry matter content, such diets provide little in the way of physical stimulation to the rumen. Rumen contractions in animals fed molasses/urea diets are both weak and infrequent (Peron and Preston 1971). Adequate forage supplementation of cattle fed such diets is important in order to stimulate intake of molasses. In practice this is achieved by either offering limited amounts of cut forage daily, or by 9 system of restricted grazing of pasture or legume banks. Elias et al (1969) recommended that in order to obtain maximum intakes of molasses the daily intake of fresh forage should not exceed 3.5% of the animal's liveweight or 0.5-0.7% of liveweight on a dry matter basis. Adequate forage supplementation is also important in preventing the nutritional disorder known as molasses toxicity.

MOLASSES TOXICITY

Molasses toxicity was first noted in Cuba with the introduction of large scale feed lotting of cattle using molasses (Preston and Willis 1974). In such situations it was observed that the toxicity was quickly corrected by increasing the level of forage in the diet. Animals given forage under conditions of restricted grazing also showed a much lower incidence of the disease than those given cut forage in troughs where perhaps difficulty of access occurs for individual animals.

The encephalopathy of the disorder is indistinguishable from cerebro-cortical necrosis (CCN) or polioencephalomalacia which is caused by a deficiency of thiamin (Edwin et al 1979). Experimentally, symptoms of molasses toxicity usually appear 6 to 8 days after removal of forage (Loscada et al, 1971; Rowe et al. 1979). At first there is refusal to eat molasses and an increase in the shivering reflex. Subsequently, excessive salivation occurs and a tendency to wander around in circles. If untreated the animal becomes comatose and quickly dies.

The cause of the necrosis in the central nervous system has been attributed to a decrease in energy supply to the tissues (caused by a deficiency of thiamine, (Edwin et aJ.1979) or by an absolute deficiency of glucose (Losada and Preston 1973). One central feature associated with the disorder however is the need for adequate fibre intake. The reduced rumen turnover rate exhibited by animals fed molasses, would be exacerbated by insufficient fibre intakes (Rowe et al. 1979) and thus causes a reduction in flow of protein and the micronutrients to the

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post-ruminal tract. It has also been suggested that the extremely slow rumen turnover rate may favour the growth of micro-organisms which produce thiaminase enzymes (Mella et al. 1976). However, evidence for their involvement is still inconclusive (Rowe et al. 1980).

In a recent experiment a total of 32 steers (ca. 300 + 10 kg liveweight) were housed in pens (2 animals/pen) under feedlot conditions and fed a diet of ad lib molasses/urea (3% w/w) with varying amounts of chaffed pangola (Digitaria decumbens) hay or barley straw. Each pair of steers received daily either the pangola or barley straw at one of the following treatment levels, 0.2, 0.4, 0.6 or 0.8% of bodyweight. The steers were fed their respective diets for 6 weeks after which time samples of rumen liquor were removed by stomach tube for immediate assay of thiaminase activity (Boyd 1985), and volatile fatty acid analysis.

Rumen thiaminase activities were not significantly different between treatments (Table 1), but two of the steers fed the lowest quantities of pangola hay (0.2% of bodyweight) developed symptans of molasses toxicity and one animal died. However, rumen thiaminase activities in these animals were 4.42 x 10^{-6} and 3.29 x 10^{-6} umol of thiazole formed/ml/min respectively similar to levels recorded in steer fed the highest levels of forage supplements..

TABLE 1 Effects of increasing levels of roughage on thiaminase activity and VFA concentrations and proportions in the rumen liquor of steers fed molasses/urea based diets.

	Barley straw					Pangola grass hay				
Forage level (% LWT)	0.2	0.4	0.6	0.8	SEM	0.2	0.4	0.6	0.8	SEM
Thiaminase activity (umol x 10^{-6} thiazole	4.6*	4.2	4.6	8.0	6.4	8.6	3.1	4.3	3.8	4.0
VFA Concentrations (mMoles/l)	134	110	141	126	10.4	126	141	123	126	15.4
Molar proportions:										
Acetate	0.50	0.54	0.60	0.57	0.05	0.50	0.45	0.56	0.57	0.04
Propionate	0.15	0.16	0.13	0.14	0.02	0.14	0.12	0.14	0.16	0.03
Butyrate	0.35	0.30	0.27	0.29	0.03	0.36	0.33	0.30	0.27	0.04

* Values are means of four animals and three sampling periods/animal.

GLUCOSE REQUIREMENT OF RUMINANTS FED MOLASSES DIETS

On grain based diets (particularly sorghum and maize) a substantial proportion of dietary starch may escape microbial degradation (Armstrong 1974) and in animals fed such diets the rumen fermentation and propionic acid is characterised by high concentrations of propionic acid. The sugars in molasses however are readily fermented in the rumen, leaving no' glucose available to the animal from post ruminal carbohydrate digestion and propionic acid concentrations in the rumen of molasses fed animals are usually low (Table 1) which exacerbates any glucose insufficiency.

Gaytan et al. (1977) successfully prevented molasses toxicity in cattle by orally supplying glycerol (400 g/l/d) and attributed the protective response to the additional availability of glucose from post ruminal digestion of the glycerol. However, Rowe et al. (1979) found that glucose entry rates in cattle fed molasses diets were not affected by removal of forage, yet symptoms of molasses toxicity were evident in the animals. Rumen fermentation patterns in the animals used by Rowe et al. (1979) were somewhat atypical of those normally observed in molasses-fed animals showing elevated proportion of acetic acid (0.68) and very low butyric acid levels (0.15).

Although cattle easily succumb to molasses toxicity, the condition has not been recorded in small ruminants fed ad lib molasses.

In a recent experiment (Peiris unpublished information), cattle, sheep and goats (4 animals/treatment) were offered feed lot diets based on cereal grain (cracked sorghum) or molasses/urea with restricted amounts of forage (0.6% of bodyweight) for 4 weeks, after which time, samples of rumen liquid were removed by stomach tube. Elevated concentrations of propionic acid were recorded in the rumen liquid from sheep and goats fed the molasses diets, whereas cattle exhibited high levels of butyric acid (Table 2). Both sheep and goats fed the grain-based diets, produced fermentation patterns with low butyrate concentrations whereas cattle exhibited relatively high levels of butyrate. In a second experiment (Blinks 1985) four rumen fistulated goats were fed the following sequence of dietary regimes:

(i) pangola grass hay (ad lib)
(ii) molasses/urea (ad lib)
plus 100g of pangola grass daily
(iii) molasses/urea (ad lib) without any forage supplements.

TABLE 2 Intakes of molasses and differences in rumen VFA proportions in cattle, sheep and goats fed ad lib diets of sorghum grain or molasses/urea plus restricted forage supplements.

	Grain based diet			Molasses/urea diet		
	Cattle	Sheep	Goats	Cattle	Sheep	Goats
Daily DMI of molasses $g/W^{.75}$	113^a	91^b	73^c	96^b	74^c	66^c
VFA concentration (umols/L)	192	32	36	143	55	56
Proportion of						
acetic acid	0.55^b	0.66^c	0.64^c	0.47^a	0.61^c	0.62^c
propionic acid	0.23^a	0.22^a	0.25^a	0.20^a	0.33^b	0.30^b
butyric acid	0.22^b	0.08^a	0.09^a	0.33^c	0.06^a	0.06^a
Other	-	0.04	0.02	-	-	0.02

Values with different superscripts are significantly different ($P<0.05$).

Each dietary regime lasted 21 days, and during the final three days of each period rumen thiaminase activities and rumen volatile fatty acid concentrations were determined in each animal.

The results presented in Table 3 show that rumen thiaminase activities were not different between dietary treatments and unlike the situation in cattle, the levels of propionic acid in rumen fluid were significantly elevated by the molasses diet. Butyric acid concentrations remained low throughout the experiment. At no time during the last treatment (21 days on molasses/urea without forage supplements) did the goats present symptoms of molasses toxicity.

TABLE 3 Rumen parameters in goats fed an ad lib forage diet on an ad lib molasses diet with or without forage.

	Treatments			
	Pangola ad lib	Molasses ad lib + 100 g forage	Molasses ad lib	L.S.R.
* Rumen thiaminase activity (umolx10^6) thiazole formed/min/ml	0.36	0.14	0.23	0.589
VFA concentration (m moles/l)	84.2	133.1	95.9	58.1
Proportions of				
Acetate	0.714^b	0.546^a	0.504^a	0.130
Propionate	0.200^a	0.366^b	0.386^b	0.080
Butyrate	0.082	0.082	0.049	0.070

* Mean of three sampling periods. Values with different superscripts are significantly different ($P < 0.05$).

Interestingly, in an experiment comparing the effects of molasses and sucrose solutions on rumen metabolism in sheep, Godoy-Montanez et al. (1984) found that the rate of sugar intake significantly affected rumen fermentation patterns. Rapid intake of sucrose solutions produced elevated butyric acid concentrations whereas the slow rate of molasses consumption produced propionate-rich fermentations.

It would seem therefore that glucose deficiency is the main cause of molasses toxicity. The greater quantities of rumen propionic acid produced in small ruminants (perhaps as a direct result of slower intake of the molasses) renders these animals less susceptible to molasses toxicity. In the absence of high rumen propionate levels in cattle fed molasses diets, there is a greater need to maintain adequate rumen turnover rates in order to supply sufficient quantities of gluconeoginic amino acids.

In this regard Preston (1972) reported that supplements of ground dried lucerne significantly reduced the incidence of molasses toxicity in cattle and the presence of supplemental protein of low rumen degradability in fortified molasses mixtures may also protect the animal from toxicity. Gulbransen (1984) reported feeding beef cows up to 6 kg of fortified molasses for 26 weeks without forage with no signs of molasses toxicity occurring. The reduced intake of fortified molasses caused by the high levels of urea (8%) incorporated into such mixtures may also result in high rumen propionic acid levels.

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