GLUCOSE METABOLISM IN CATTLE ON MOLASSES BASED DIETS: STUDIES ON MOLASSES TOXICITY[1]

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A total of 7 bulls (200 kg live weight) wee used to study the dynamics of glucose metabolism on molasses-based diets or molasses-based diets from which the forage component had been removed in order to induce molasses toxicity. Glucose entry rates were measured by isotope dilution procedures. The 4 animals without forage all showed symptoms of molasses toxicity. Glucose entry rates did not appear to differ between the intoxicated and control animals and showed values within a range expected of animals growing at 500 g tally. This was surprising since the animals had been off feed for at least one day. The most severely intoxicated animals showed hyperglycaemia indicating a possible inhibition of glucose utilisation. The data do not support the hypothesis that molasses toxicity is simply due to the lack of glucose precursors.

Key words: Cattle, molasses, isotope dilution, molasses toxicity, glucose metabolism

Molasses toxicity is a disease syndrome affecting cattle fed high levels of liquid molasses and restricted forage (for review see Preston and Willis 1974) and is of economic importance in many tropical countries. The clinical symptoms of the disease are in coordination of the limbs and head; animals slaughtered in the terminal stages of the disease showed a necrosis of the brain (Verdure and Zamora 19703. The condition is unresponsive to thiamine and still occurs when animals are fed or injected with thiamine (Losada et al 1971), thus differentiating the condition from cerebro-cortical necrosis resulting from thiaminases produced by microorganisms in the digestive tract (Edwin & Lewis 1971) and occurring in grain and pasture fed animals.

The disease can be induced by withdrawing forage from the ration, Under these circumstances, intake of molasses falls and the rumen fermentation pattern is characterised "by very low levels of propionate and high levels of butyrate (Losada and Preston 1973). The low rumen propionate levels in association with a central nervous system disorder suggests that since glucose is essential for brain metabolism (Setchell 1961), glucose availability may be impaired in these animals. There is some support for this hypothesis in that Gayton et al (1977) showed that by giving 400 ml of glycerol daily (a glucose precursor) the condition was prevented even when the forage was withheld.

In order to examine the suggestion that lack of availability of glucose is involved in molasses toxicity, we have examined glucose entry rates in animals in which molasses toxicity has been induced.

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Materials and Methods

Animals: A total of 7 Zebu bulls weighing between 196 and 250 live kg were used in this study. The animals had free access to molasses containing 2.5% urea and were given 500 g daily of groundnut meal. All animals also received raw bagasse at the level of 1% of live weight daily.

Experimental Procedure: In four of the animals the roughage source (bagasse) was removed from the diet of animals with the intent to induce molasses toxicity. As soon as clinical symptoms of molasses toxicity were observed both the control and experimental animals were cannulated in one jugular vein and 5 hr later when the symptoms were acute, glucose entry rates were measured using the procedures described by Ferreiro et al (1978) and Ravelo et al (1978),

Results

While all animals were receiving the roughage daily the intakes of molasses were in the range 4 - 6 kg/d. Intake fell progressively in the group which had their roughage withheld. The intoxicated animals had not eaten for 24 hr prior to measurement of glucose entry rate. The 3 control animals also had been fasted for 6 hr prior to the experiment as they went off feed immediately after implantation of cannulas.

The parameters of glucose metabolism for the 2 groups of animals are given in table 1. Glucose entry rates showed a tendency (P = .21) to be higher in the animals with molasses toxicity.

The two most intoxicated animals showed a high and consistent hyperglycaemia while all other animals (affected and control animals) had glucose levels in the normal range.

Table 1:
Total glucose entry rates in Zebu bulls given molasses based diets. Animals 40, 41, 42 and 43 suffering put molasses toxicity. All control animals were fasted for 4 hr prior to experiment

Animal no.	Body weight (kg)	Symptoms of molasses toxicity	Plasma glucose (mg/100ml)	Glucose entry rate		Pool size		$t^{1/2}$ (min)	Space	
				(Mg/min)	(mg/kg^{75}/min)	(g)	(mg/kg)		(litres)	(% LW)
40	249	+++	102	330	5.1	49	197	101	48	19
41	259	+++	109	321	5.0	43	166	92	39	15
42	229	+	67	255	4.3	30	131	81	44	19
43	232	++	80	430	7.2	65	279	105	81	34
46	196	-	73	249	4.8	28	143	77	38	19
47	213	-	60	172	3.1	16	75	63	26	12
48	191	-	80	309	6.0	61	319	137	77	40

Discussion

It must be stressed that the data presented here are of a limited nature in terms of the number of animals employed, That the control animals had ceased to feed following cannulation also makes interpretation of the data difficult. Nevertheless some interesting observations were obtained which should be valuable for the planning of other work in this area.

The one noteworthy finding was the apparently high glucose entry rate in animals which had clinical symptoms of molasses toxicity. These animals had been off feed for at least 24 hr end had consumed only a proportion of their molasses ration in the 3 - 4 days prior to the experiment. These values are only slightly less than reported for animals growing at 500 g/d on molasses diets (474 mg/min) (Smith et al 1978). In contrast control animals which had been off feed for only 4hr and which previously had been consuming considerable quantities of molasses (plus roughage) had a tendency to show lower glucose entry rates that those which were intoxicated, The two animals which showed the most severe symptoms of molasses toxicity were hyperglycaemic, However, their glucose entry rates were similar to the other intoxicated animals which did not show the hyperglycaemia, These latter findings appear to indicate an inhibition of glucose utilization in severely intoxicated animals.

Conclusion

These data, limited though they are, do not support the hypothesis that molasses toxicity is simply due to a lack of glucose precursors. It therefore appears that other factors are involved possibly of hormonal nature.

References

Ferreiro H M, Priego A, Lopez J, Preston T R & Leng R A 1978 Glucose metabolism in cattle given sugar cane based diets supplemented with varying quantities of rice polishings Brit J Nutr (submitted)

Edwin E E & Lewis G 1971 Diseases of dairy cattle. Thiamine deficiency with particular reference to cerebro-cortical necrosis-a review and discussion J Dairy Res 38:79

Gaytan T, Zamora F & Shimada A S 1977 Glycerol as a protective agent against molasses intoxication in beef cattle Cuban J Agric Sci 11:29-37

Losada H & Preston T R 1973 Molasses toxicity and cerebro-cortical necrosis Cuban J Agric Sci 7:169

Losada H, Dixon F & Preston T R 1971 Thiamine and molasses toxicity: 1. effects with roughage free diets Rev Cubana Cienc Agric 5:369-378

Preston T R & Willis M B 1974 Intensive beef production (2nd Ed) Pergamon Press: Oxford

Ravelo G, Fernandez A, Bobadilla M, MacLeod N A, Preston T R & Leng R A 1978 Glucose metabolism in cattle on sugar cane based diets: a comparison of supplements of rice polishings and cassava root meal Trop Anim Trop 3:12-18

Setchell B P 1961 Cerebral metabolise in the sheep I Normal sheep Biochem J 72:265-275

Smith G H, Kempton T J & Leng R A 1978 Glucose metabolism and growth in castle (to be submitted to Brit J Nutr)

Verdura T & Zamora I 1970 Cerebro_cortical necrosis in beef cattle fed high levels of molasses in Cuba.Rev Cubana Cienc Agric (Eng.ed) 4:209

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