SOME ASPECTS OF MOLASSES TOXICITY

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SUMMARY

Molasses toxicity is the most important disease syndrome associated with high molasses fattening systems which are currently under investigation at the Faculty of Agriculture, University of Dar es Salaam. The components of the high molasses system are outlined and possible causal factors leading to the onset of molasses toxicity are reviewed. It is suggested that adequate roughage intake appears to be the crucial factor in preventing molasses toxicity. Management recommendations for minimising the risk of molasses toxicity are made. It is stressed that molasses toxicity is not yet fully understood and that adaptation of the high molasses system in Tanzania must take into account local factors which may predispose to this condition.

INTRODUCTION

One of the research projects in the Animal Science Department of the Faculty of Agriculture at Morogoro is an investigation of the local potential for the use of high levels of molasses for fattening beef cattle. A first experiment in the project has been completed (Fielding and Kyomo, 1979). In the course of this experiment, involving thirty steers averaging 400 kg liveweight, two cases of a condition known as "molasses toxicity" occured (1). As the molasses fattening system holds considerable potential for the increased utilisation of locally produced molasses, the purpose of this paper is to review some of the most important aspects of the condition.

MOLASSES FATTENING SYSTEM

The high level molasses fattening system consists essentially of the following components:

(i) Ad. lib. molasses with urea added at the rate of 2.5-3% of the molasses dry matter.

(ii) A protein supplement, preferably of animal origin.

(iii) Limited amounts of forage, either as restricted grazing, 4-5 hours per day, or as freshly cut forage, 1.5-2.5% of liveweight per day.

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The system was originally developed in Cuba by Dr. T.R. Preston and is now used in commercial feedlots in many countries. Depending upon their weight, animals may consume molasses at the rate of up to 8-9 kg per day. A major advantage of the system is that it can utilize urea as a cheap source of nitrogen for the synthesis of microbial protein in the rumen without any risks of urea toxicity (Preston, 1972).

MOLASSES TOXICITY

Molasses toxicity is the most important disease syndrome associated with high molasses feeding (Preston, 1972), and was first reported by Preston, Willis and Elias (1967). The symptoms have been described by Verdura and Zamora (1970), as being similar to those of polyencephalomalacia or cerebro-cortical necrosis. Morbidity is generally low (Preston, 1972). At the feedlot level, symptoms may include signs of apparent drunkness, staggering and lack of co-ordination. Affected animals may be seen with their fore limbs crossed and leaning or pressing against fences or other objects. Vision may also be affected. There may be paralysis of the mandibular and throat muscles so as to prevent chewing and swallowing. Animals may also become prostrate and comatose. Recovery from the condition can occur, althogh the rate and degree of recovery is not yet fully documented.

Molasses toxicity occurs with varying degrees of intensity. In Mauritius the present author has observed cases which have fully recovered following removal of the molasses and provision of forage only. Ugarte and Preston (1974) have also reported favourable responses to the withdrawal of the molasses and in another instance (1975b) reported no mortality following four cases of toxicity.

In the Morogoro experiment both cases progressed very quickly, within a few hours, to the stage of headpressing and blindness and there was thus no opportunity to curtail the molasses and feed only forage. Despite symptomatic treatment one animal died after seven days without showing signs of any recovery and the second, whilst making a partial recovery did not become fit enough to return to the feedlot (Fielding and Kyomo, 1979).

When molasses toxicity first occured in Cuba it was thought to be caused by an overconsumption of molasses, and hence the term "molasses toxicity". It was thought that such overconsumption lead to potassium toxicity as this mineral is found in relatively high levels in final molasses (2) which is the type of molasses normally used for feeding (Preston, 1970). However, cases of molasses toxicity have since been demonstrated on low potassium molasses such as high-test molasses (3) and although mineral imbalance may be a factor in molasses toxicity it is not now thought to be the primary cause (Losada and Preston, 1974b).

(2) Final molasses: the sugar cane juice from which the economic maximum of sugar has been extracted.

(3) High-test molasses: purified, unextracted cane juice which has been partially inverted with acid or by enzymic action to avoid crystallisation of the sucrose when the product is concentrated.

A second explanatory theory involved the B vitamin thiamine which, it was thought was becoming effectively deficient on high molasses diets. This theory was developed because cerebro-cortical necrosis, which can occur on high energy diets not based on molasses, may respond to thiamine therapy (Jensen and Mackey, 1971). However, Preston (1972) reported the use of prophylatic thiamine to be completely ineffective in reducing the incidence of molasses toxicity. Thus although molasses toxicity may terminate in cerebro-cortical necrosis its primary cause appears not to involve a thiamine deficiency.

Preston (1972) reported a further hypothesis for explaining the onset of molasses of toxicity as follows: - for whatever reason the animals fails to eat sufficient forage, this leads to a fall in molasses intake, possibility due to reduced rumen motility, which in turn causes a rise in rumen pH and reduces proportions of proprionic acid in the rumen; as proprionic acid is an important source of glucose for the brain, which can only use glucose as an energy source, a state of cerebral hypoglycaemia developed which triggers the onset of the molasses toxicity syndrome. This hypothesis is supported by Lora, Ravelo, Preston and Leng (1977), who in addition suggest that a hormonal imbalance may be induced, which interfers with the uptake of glucose. Lora et. al. (1977) liken the molasses toxicity syndrome to that of pregnancy toxaemia. Experiments on the artificial induction of molasses toxicity have underlined the critical role of forage as the condition has been most easily induced by the abrupt withdrawal of the forage component in the system (Preston, 1972; Losada and Preston, 1974b). In a large experiment involving over 600 animals Losada and Preston (1974a) showed that the incidence of toxicity increased markedly with reduced frequency of feeding forage i.e. every second day as opposed to every day and with high stocking density in the feedlot pens, a factor which would be expected to interfere with adequate forage intake by all the animals.

Thus molasses "toxicity" may not be a toxicity but rather a deficiency, not of molasses but of dietary dry matter capable of yielding glucose precursors on fermentation on the rumen. Such dietary dry matter may be provided by adequate forage and possibly by additional starch supplements such as rice polishings which promote the production of proprionic acid in the rumen (Preston, 1972). In the Morogoro experiment all the animals received 500 gms of rice polishings and at least 0.7 kg of plant protein per animal per day. If non molasses/starch supplements are capable of preventing molasses toxicity then these levels are apparently not adequate. However, Preston (1972) reported umpublished findings where animals were kept on a molasses/urea diet completely without roughage but supplemented with 1 kg of ground maize per day and fish meal; without the ground maize the diet led to a hundred percent molasses toxicity while with the addition of the ground maize the animals continued for four months without any case of toxicity. No mention was made of the performance achieved by the animals in this experiment, and it is an extreme diet not for practical application, although it provides an interesting aspect of the toxicity problem.

However, providing the roughage is of the appropriate type and quantity there is apparently no need for starch or no forage protein with reference to the prevention of molasses toxicity, as there are many reports of successful molasses/urea use with forage as the only supplement in addition to minerals and sult (Ugarte and Preston, 1975a; Molina and Preston, 1975; Alvarez, Wilson and Preston, 1977; Fernandez, Macleod and Preston, 1977; Gonzalez, 1977).

THE PREVENTIVE MEASURES

Adequate roughage thus appears to be the crucial factor in preventing molasses toxicity. In terms of quantity this appears to be in the range of 1.5-2.5% of live-weight per animal per day: below this level the intake of molasses is reduced, and the conditions for molasses toxicity may develop; above this level molasses intake is reduced, resulting in lowered performance.

Obviously many factors are involved in adequate roughage intake: species, composition, degree of maturity, stocking rate, amount of trough space etc. In particular, legume roughage appears to give good results with the molasses system (Alvarez et. al., 1977). This is probably because legume roughage provides relatively insoluble protein, which to some extent by-passes the rumen, so avoiding degradation. It has in fact been suggested that such by-pass protein is the first limiting nutrient molasses/urea systems (Silvestre, Macleod and Preston, 1977).

From the literature and from the work at Morogoro it is possible to draw up some recommendations for avoiding molasses toxicity:

(i) Feed a balanced diet before the animals enter the feedlot, particularly with reference to minerals and vitamins.

(ii) Introduce the molasses/urea over a two week period before attaining the ad. lib. level.

(iii) Cover the molasses/urea trough so as to avoid the entry of rain water which may lead to fermentation and the production of alcohol which may disturb the rumen fermentation.

(iv) Provide adequate forage in terms of quality and quantity i.e. younger grasses or legumes, sufficient grazing time, 4-5 hours per day or adequate feeding facilities if fed in racks, 1.5-2.5% of liveweight as fresh forage per day. The important point being that every animal should consume adequate forage every day.

(v) Provision of starch supplement, e.g. rice polishings or maize bran, 100-200 gms/100 kg liveweight per day.

(vi) Constant provision of water, minerals and vitamins.

(vii) Avoidance of sudden changes with reference to any dietary component, e.g. change in viscosity of molasses from one batch to another.

However, it seems from the literature that even when such precautions as suggested above are taken, cases of molasses toxicity may still occur, although usually at a low level, e.g. 2-5% (Losada and Preston, 1974a). Thus the risk of cases occurring appears to be the price to be paid for a fattening system capable of achieving high levels of performance i.e. over 1 kg liveweight gain per day.

Molasses toxicity is a metabolic condition which is not yet fully understood and which may involve several causal factors acting alone or together. Adoption of the high level molasses system in Tanzania must first ensure that the characteristics of local molasses, grazing, etc. do not abnormally predispose to the condition. To do so is one of the objectives of the Morogoro project.

REFERENCES

Alvarez, F.J., Wilson, A., Preston, T.R. (1977)
Leucaena leucocephala as a combined source of protein and roughage for steers fattened on molasses/urea.
Trop. Anim. Prod. 2: 288 (Mexico).

Fernandez, A., Macleod, N.A., Preston, T.R. (1977)
Cassava as a combined source of protein and roughage for cattle fed on molasses/urea.
Trop. Anim. Prod. 2: 195 (Mexico).

Fielding, D., Kyomo, M.L. (1979)
Molasses as an energy source for fattening beef cattle:
I. A report on the use of molasses/urea for fattening exotic cross Boran steers and comparing sunflower and cottonseed cake as protein supplements.
Research Report Animal Science Department, Faculty of Agriculture, Forestry and Veterinary Science, University of Dar es Salaam, (in preparation).

Gonzalez, R.A. (1977)
Effect of castration on performance of cattle fed molasses/urea diets with and without supplement. Joint Annual Meeting of the Centro de Investigacion y Experimentacion Ganadera, Chetumal, & Roo, and Centro Superior de Agricultura Tropical Cardenas, Tabasco, Mexico, 2-4 May 1977. Abstr. in Trop. Anim. Prod. 2: 223 (Mexico).

Jensen, R., Mackey, D.R. (1971)
Diseases of feedlot cattle.
Lea and Fiebiger, Philadelphia, U.S.A.

Lora, J., Ravelo, G., Preston, T.R., Leng, R.A. (1977)
Studies on glucose metabolism in cattle affected by molasses toxicity. Joint Annual Meeting of the Centro de Investigacion y Experimentation Ganadera, Chetumal, & Roo, and Centro Superior de Agricultura Tropical Cardenas, Tabasca, Mexico, 2-4 May 1977. Abstr. in Trop. Anim. Prod. 2: 234 (Mexico).

Losada, H., Preston, T.R. (1974a)
Housing density, and forage feeding system on the performance and carcass characteristics of bulls fed molasses/urea diets.
Cuban J. Agric. Sci. 8: 1.

Losada, H., Preston, T.R. (1974b)
Effect of final or high test molasses on molasses toxicity.
Cuban J. Agric. Sci. 8: 11.

Molina, A., Preston, T.R. (1975)
Encapsulated methionine for fattening cattle with molasses/urea diets.
Cuban J. Agric. Sci. 9: 125.

Preston, T.R., Willis, M.B., Elias, A. (1967)
Intensive beef production from sugar-cane, 2: Comparison between normal and invert molasses as a supplement to forage or concentrates.
Rev. cuba. Cienc. agric. (Engl. ed.) 1: 41.

Preston, T.R., Willis, M.B. (1970)
Intensive Beef Production.
Pergamon Press, Oxford, U.K.

Preston, T.R. (1972)
Molasses as an energy for cattle.
World Rev. Ntr. and Diet. 17: 250.

Silvestre, R., Macleod, N.A., Preston, T.R. (1977)
Effect of meat, dried cassava root and groundnut oil in diets based on sugar-cane/urea or molasses/urea.
Trop. Anim. Prod. 2: 151 (Mexico).

Ugarte, J., Preston, T.R. (1974)
Hay or restricted grazing as roughage sources for Holstein steers fed molasses/urea.
Cuban J. Agric. Sci. 8: 151.

Ugarte, J., Preston, T.R. (1975a)
Effect of molasses/urea supplementation on Holstein calves grazing pangola grass.
Cuban J. Agric. Sci. 9: 109.

Ugarte, J., Preston, T.R. (1975b)
Soybean meal as a protein supplement for steers fattening on molasses/urea diets.
Cuban J. Agric. Sci. 9: 119.

Verdura, T., Zamora, I. (1970)
Cerebro-cortical necrosis in Cuba in beef cattle fed high level of molasses.
Rev. cuba. Cienc. agric. (Engl. ed.) 4: 209.