THE INFLUENCE OF MONOSACCHARIDE TYPES (D-GLUCOSE, D-MANNOSE AND D-GALACTOSE) IN INDUCING LACTIC ACIDOSIS IN GOATS.

by

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DEDICATION

This work is dedicated to my dear wife, Ladi, and my two daughters, Mary and Kuceli. It is also dedicated to my mother.

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SUMMARY

Three goats weighing 40 kg each were given D-glucose, D-mannose and D-galactose intraruminally in increasing doses in separate experiments. The purpose was to arrive at a dose of each of the monosaccharides that could induce lactic acidosie. The induction and severity of lactic acidosis was assessed by the clinical signs and the quantitative changes in each of rumen fluid and blood parameters. It was found that 10 gm/kg body weight of D-glucose and 15 g/kg each of D-mannose and D-galactose induced lactic acidosis. The acidosis induced by D-galactose at 15 gm/kg was not as severe as that induced by D-glucose and D-mannose at the doses indicated. Clinical signs observed correlated with high blood D(-) lactic acid concentration above 5 mg%. Blood D(-) and L(+) lactic acids and phosphorue levels increased with all the acidotic doses of the three monosaccharides. There was an increase in Ca, K, PCV, total protein, glucose in two of the three goats. There was a decrease in blood and rumen pH, blood PO_2, PCO_2, HCO_3^- serum carbon dioxide and BUN in all the acidotic doses of the monosaccharides. Blood Na, alkaline phosphatase, albumin, creatinine, SDH showed variable results in all the three goats.

INTRODUCTION

Numerous metabolites are produced in the rumen of ruminant animals as the result of fermentation by microorganisms which populate the rumen. Lactic acid is one of these metabolites. It has been known to contain a racemic mixture (58), (126) referred to as L(+) and D(-) lactic acids. The (+) and (-) refer to the rotation of a plane of polarized light. The (D) and (L) referred to structural relationship with the isomer of glyceric acid. The L(+) form does not accumulate under normal pH of 6.7-8.0 because it is readily converted to volatile fatty acids. The D(-) form which is formed after readily fermentable carbohydrate are consumed in excess may be metabolized through various routes (61). The most important route of metabolism is its absorption into blood (43). The absorption is favored under low rumen pH (43). The D(-) form has been known to be the cause of the acidosis (43), (19). The major route of elimination of blood D(-) lactic acid appears to be elimination by the urinary system (28).

The production of lactic acid depends on the type of diet which influences the type and number of the microorganisms (90). Complete replacement of rumen microflora from gram negative to gram positive has been reported. This was attributable to reduced rumen pH which favours the growth of lactic acid forming bacteria such as lactobacilli (118) and streptococcus bovis (62).

Feeds rich in fermentable carbohydrates consumed in excess have been known to cause lactic acidosis. Some of the names given to this condition by various workers were: acute impaction (89) rumen intoxication

(51), rumen overload (70) grain engorgement (5) and D-lactic acidosis (43), (28).

Under practical conditions, ruminants can gain access to readily fermentable carbohydrates and eat lethal doses especially when they are too hungry or too greedy (110).

Induced lactic acidosis has been produced experimentally using grains (62), fruits (99), pure chemicals (41), root crops (107) and vegetables (96). Clinical signs most commonly observed are diarrhea, anorexia, prostration, dehydration and distended rumen. Among the physiological changes reported are increase in blood lactic acid (118), fall in rumen and blood pH (118), disturbance of acid base balance and increase in blood glucose (70), (28) and phosphorus (70).

The objectives of this study are:

1) To compare the doses of each monosaccharide that induce lactic acidosis. The induction and severity will be assessed by clinical signs and quantitative changes in blood and rumen levels of L(+) and D(-) lactic acids, blood and rumen fluid pH, PCV, and total protein levels.

2) Monosaccharides are the substrates in the fermentative processes which produce lactic acidosis. It is known from in-vitro work that rumen microflora have different rates and patterns of fermenting different monosaccharides. This work will compare the patterns and rates of fermentation of these monosaccharides. Such knowledge will provide guide lines for feeding diets that contain naturally occurring polysaccharides composed of these monosaccharides, so lactic acidosis can be avoided.

3) To measure and evaluate quantitative changes in the following blood parameters:

a) K, Na, Cl, PCO_2, HCO_3^-, PO_2, BUN, pH and creatinine as factors directly or indirectly involved in acid base balance.

b) SGPT, and SDH to assess any liver damage.

c) P, Ca and alkaline phosphatase to obtain additional information.

d) Serum glucose - to assess extent of glucose accumulation and the contribution of each monosaccharide to blood glucose.

REVIEW OF LITERATURE

The biochemical and clinical entity of lactic acidosis was first described in 1949 (118). As early as 1863, Reiset (101) stated that if ruminants were not accustomed to a grain diet, they often suffered acute digestive disturbances which sometimes resulted in death within twenty four hours.

Acute indigestion in ruminants had been induced experimentally (36). The signs of acute indigestion reported were associated with toxic factors and not with lactic acid. Phillipson (96); Phillipson and MacAnnally, (97) and Elsden (46), demonstrated an increase in rumen lactic acid levels but did not relate the signs in the engorged animals with lactic acid, the earliest definitive work on grain engorgement which related the signs of acidosis to lactic acid was by Turner and Hodgetts (118). Research from that time until the present has been concerned with events taking place in the rumen and the signs of the syndrome.

The lactic acid produced in the rumen has been shown to be a racemic mixture (58), (127), (18), (41), (118).

The major portion of elevated blood lactate encountered in acute indigestion is the D-isomer (127), (43) and hence considered to be the cause of the acidosis (19), (43) and (127). Following absorption, the D-isomer was known to be metabolized at a slower rate than the L-isomer (19), (58), (119), (42), (43), (87), (29), (22) and (23).

There are several pathways by which lactic acid is metabolized. Conversion to pyruvic acid is catalyzed by an isomer specific

dehydrogenase. The activity of the dehydrogenase specific for the D-isomer of lactic acid has been shown to be low in mammalian cells (11), (117). Another pathway is the conversion to volatile fatty acids (65). Gases are also produced during these reactions. Somerville (111) found that labelled carbon from lactate appeared in various amino acids isolated from Peptostreptococcus elsdenii. Propanediol and methyl-glyoxal pathways are other potential pathways for lactate in the rumen which have not been explored (44). Interconversion between the two isomers also occurs (61).

The terms "acid indigestion" (19), (45), "rumen intoxication" (51), "rumen overload" (70), "grain engorgement" (5), "acute impaction" (89), "lactic acidosis" (44) and "D-lactic acidosis" (43), (28) are some of the names reported by the various workers to the lactic acidosis, the term used in this experiment.

Irregardless of the name used, large doses of fermentable carbohydrates promote the growth of lactic acid producing bacteria which dominate the ruminal environment (118), (110). The end product of fermentation of carbohydrates by these bacteria is lactic acid. This acid raises the level of ruminal acidity which results in inhibition (123) or destruction (110) of other microorganisms and is detrimental to the host (44).

Historically Turner and Hodgetts reported on "Toxic doses of Wheat" in 1-11th Annual Report of the Commonwealth Scientific and Industrial Research Organization Bulletin (1949-1959). The 1949 report concerned a sheep given 75-80 g/kg body weight of cracked wheat intraruminally. They found that rumen microflora predominantly changed to lactobacilli. There was a large production of lactic acid, rumen pH fell to about 4.2

and blood lactate rose to as high as 200 mg/100 ml. The blood pH fell to 6.6 and there was a fall in alkali reserve. The form of lactic acid produced in the rumen was predominantly the D-form. They first demonstrated that lactic acid could be absorbed into the blood stream from the reticulorumen. The animals they used died of intoxication, depletion of alkali reserve and haemoconcentration.

In 1950 they pointed out an important observation. Higher doses of wheat were required to induce the disease in animals on high plane of nutrition than those that are undernourished. Several animals were reported to have developed lameness. Blood clearance of lactate was very long compared to the volatile fatty acids. They also confirmed that low rumen pH favoured absorption of the lactic acid. The combination of longer blood clearance and increased absorption at low pH could be the reasons why lactic acid accumulates and why acidosis follows. They suggested that the high level of the D-isomer could be a factor in the acidosis produced. This was later confirmed (19), (45). While this work was in progress in Australia, Douherty and Cello (39) were working on apple engorgement in New York. Turner and Hodgett's works were not exposed to the scientific world otherwise Douherty and Cello would not have repeated almost the same work. Douherty and Cellos' first work on apple engorgement (39) was based on earlier claims by clinicians that the signs of apple engorgement was due to alcohol formed in the rumen. They reported fall in rumen pH but alcohol was found only in traces. This was confirmed by Allison (6) who demonstrated that the rate of metabolism was slow and alcohol was not an important intermediate in the rumen under normal conditions. The levels of alcohol in overfed animals

could not have caused the signs usually seen. The near neutral pH of the rumen and low hydrogen ion concentration limits the formation of alcohol. In subsequent experiments with wheat, rye, corn and starch, Dorherty and Cello demonstrated that rumen contents of the animals used contained more toxic materials. Intravenous injection of the toxic substance(s) depressed blood pressure of anaesthetized dogs and goats, inhibited rumen motility in unanaesthetized sheep and stimulated respiration in intact animals (36). They identified histamine but histamine did not produce all the physiological changes characteristic of the ingested toxin. They were unable to isolate the individual toxic substance(s). Reports on toxic substances were also given by Turner and Hodgetts in their 1952 report and Douherty and Cello (38). The work of these two groups of workers (Turner and Hodgetts in Australia, Douherty and Cello in USA) formed the basis for subsequent work by others.

Other important findings were noted by Turner and Hodgetts in their third report in 1951 (118). They found that aureomycin, given with a lethal dose of wheat prevented lactic acidosis. The lactobacillary type of fermentation was circumvented by the antibiotic. This evidence showed that lactic acidemia and acidosis was a direct result of absorption from the reticulo-rumen. Absorption was suggested to exceed the rate of blood clearance. They also reported that urine excretion was depressed and there was a fall in blood volume, but failed to conclude whether the fall in blood volume was a direct consequence of excessive consumption of wheat. It was not determined whether the fall in blood volume was osmotic or due to rumen injury.

A fourth report in 1952 (118) indicated the fall in blood volume

to be osmotic in nature. Subcutaneous or intraruminal infusion of glucose in sheep produced the severe fall in blood volume. At this stage, they concluded that the fall in blood and rumen pH, the rise in blood lactate and fall in blood bicarbonate were due in part to direct absorption of enterogenous lactic acid. They also considered hypoxia due to fall in blood volume. Rumen atony was reported in their work in 1954 (118) which they thought was due to low pH. The haemoconcentration they observed in their earlier work was confirmed in 1955 (118) to be due to fall in plasma volume and a great increase in red cell volume. The great increase in red blood corpuscles was confirmed in their 1956 (118) work to be partly due to splenic contraction. When histamine was given intravenously, there was splenic contraction which they attributed to indirect release of catecholamines. The fall in plasma volume was due to water efflux from body fluids to the rumen. The role of the spleen was a compensatory one and this compensatory contribution is about 30%. Subsequent fall in blood volume was due to fall in plasma volume. They suggested that the total pool of lactic acid produced during acidosis came from two sources - the enterogenous and endogenous lactic acids produced from stagnant and histotoxic hypoxia. Their work on histamine in 1957 (118) indicated a lack of evidence that histamine plays a significant role in lactic acidosis. The report showed that the concentration in the rumen never exceeded 3.3 ug/ml. Blood concentration of histamine did not rise and the antihistamine Phenergan had no effect.

The work reported in 1958 showed another important finding. Salivary secretion virtually ceased 6 hrs after engorgement. The role of saliva in buffering rumen acids has been documented (13).

The conclusion reached by Turner and Hodgets in their works was that the condition was due to osmotic dehydration and alimentary lactic acidosis. Lactic acid was incriminated as the sole cause of the disease. Other workers have incriminated lactic acid as the sole cause (18), (104), (43), (114), (62), (66), (86), (17).

Mullinax (91) and Douherty (38) showed that toxic factors (amines, alcohol, bacterial endotoxins) beside the formation of large amounts of lactic acid may contribute to signs of grain engorgement.

Several factors lead to the accumulation of lactic acid. Certain feeds, particularly the silages (130), (124) and brewers grains (93) contain lactic acid in large quantity. Ingestion of these feeds directly increases the level of rumen lactic acid. The major route of lactic acid accumulation is via microbial fermentation of precursors of lactic acid in the diet. These yield lactic acid as end or intermediary product (44). Such precursors have been listed (44). The degree of accumulation and hence of hazard depends on the content of the precursors. It is on the basis of this that various workers have experimentally induced lactic acidosis by using toxic doses of feeds: Various doses of wheat (62), (31), (51), (4), (5), (105), (2) (70), (89), (73), barley (51), (118), (70), (85), (89), (31), (52), (73), unripe green corn standing in the field (44), oats (26), rye (62), milo (119), sorghum (89), mollasses (44), cabbage (96), brewers grains (93), (77), sulfur deficient purified diets (126), kiawe beans (1), potatoes (77), fruits including grapes (99), peaches (63), pears (63), (2), (44), apples (63), (44), (39), lactic acid (26), (114), (73), starch (100), (76), (28), glucose (62), (120), (70), (60), (64), sucrose (74), (32), (112), lactose (75), root crops of mangolds (96), (107), fodder beet (94), (129) and

sugar beet leaves with beat tops attached (34) have been used to induce lactic acidosis.

Under normal conditions the glycolytic pathway is the route of carbohydrate fermentation and the precursor of lactic acid is pyruvic acid which is readily converted to volatile fatty acids (55). Lactic acid can accumulate in various ways by altering events in the rumen. Firstly, lactic acid producing-bacteria can increase when fermentable carbohydrates are fed. The rate of production therefore exceeds the rate of utilization (61). Secondly, the rate of pyruvate production can exceed the rate of conversion to fatty acide. The excess pyruvate then acts as hydrogen acceptor for the reoxidation of reduced nitotinamide adenine dinucleotide (NADH) generated in the glycolytic reactions (61). Thirdly, Huber (61) suggested that the enzymes involved in pyruvate dissimilation may be pH sensitive, so at low pH the enzymes may be inactivated. This can result in accumulation of pyruvate with subsequent conversion to lactic acid. Fourthly, it was reported that fermentation of some lactic acid producing organisms has been known to proceed faster in bicarbonate buffer than phosphate buffers. At very low pH, bicarbonate in the rumen has been known to be completely depleted (61). This could result in excess accumulation of lactic acid.

Total lactic acid has been known to increase in rumen from 1-1,500 mg/100 ml (119), (124) and in blood from 4.5 to 90 mg/100 ml (56), (43) following a large consumption of large amount of grains. Studies by Phillipson (96) and Phillipson and McAnnally (97) showed that lactic acid accumulated rapidly and transiently in the rumen fluid of sheep following ingestion of feeds of mangold and cabbage or introduction of

100 gm of various sugars into the rumen. They found that the concentration of lactate never exceeded 50 m M/L. Elsden (46) showed that ruminal fluid taken from sheep fed poor quality hay fermented glucose at a much slower rate than those taken from sheep fed good quality hay. Phillipson (98) observed very high concentrations of lactic acid in the ingesta of lambs fed large amounts of flaked maize. Levels of 72.5 m M/L were reported. There was no sickness reported. Gall et al (50) found lactic acid in the rumens of animals on high grain rations but only traces in animals on high roughage diets. Scarisbrick (107) observed an accumulation of 90 m M/L of lactic acid in the ingesta of sheep that over-indulged on mangolds. Waldo and Schultz (121) found no lactic acid in the rumens of steers prior to feeding. On giving various diets of grain, glucose and feeds rich in silages, the lactic acid levels rose and declined rapidly. Balch and Rowland (12) found only traces of lactic acid in the rumens of cattle fed a variety of diets. Lactic acid appeared in appreciable concentrations only when the animals were fed large amount of flaked maize. Briggs et al (21) found levels of 80 m M/L on diets of high levels of soluble carbohydrates. Reid et al (100) reported levels of 150 m M/L when feeding wheaten starch to sheep. Annison et al (7) observed moderate decrease of less than 80 m M/L of ruminal lactic acid when stall fed sheep were turned on pasture. Bond (19) reported the accumulation of lactic acid of up to 327 m M/L in ruminal ingesta of sheep overfed on readily fermentable carbohydrate.

The organisms involved in the production and utilization of lactic acid have been identified and characterized by many workers. Generally, there was a shift from gram negative to gram positive organisms when

ruminants were fed readily fermentable carbohydrates. Turner and Hodgetts (118) reported the complete replacement of rumen flora by lactobacilli. Hungate et al (62) isolated streptococcus bovis from the rumen of sheep. Mann et al (20) noted that lactobacilli predominated in rumens of animals fed high concentrate diet and suggested that they might be more potent acid producers than streptococcus bovis. Robinson et al (103) studied fractionated washed suspension of rumen bacteria and found that the rate of lactate accumulation was dependent upon pH, level of glucose and maltose or cellobiose present. Jensen et al (67) isolated numerous strains of lactobacilli from ovine and bovine ruminal ingesta. They studied two species, one of which produced a preponderance of D-lactic acid. Perry and Briggs (95) found very large numbers of lactobacilli in the rumens of young calves. Krogh (74), (75), (76), (77), (78) studied the chronological sequence of changes in microbial population of the rumens of sheep when sucrose, lactose, and starch were given in repeated doses. The lactobacilli were for the most part amylolytic as were the streptococci. Kistner et al (72) stated that diet is the most significant variable in inducing changes in microbial population. The organisms involved in the utilization of lactic acid have also been identified. A species of propionibacterium which fermented lactate (46) was isolated. Johns (68) (69) isolated veillonella gazogenes which fermented lactate. The two organisms fermented lactate through succinate by carbon dioxide fixation. Gutierrez (54) isolated a disulphovibrio organism that utilized lactate. Elsden et al (47) isolated a Gram - positive coccus which metabolized D-lactate to short-chain fatty acids. This organism was called "LC" and later named Pepto

streptococcus elsdenii.

Work done with goats are scarce and some are not related directly to lactic acidosis. Tsuda (116) performed some absorptive experiments using typical metabolites contained in the rumen, among which are lactic acid and glucose. He found out that for glucose, as the concentration increased, the degree of absorption decreased. Absolute amount of absorption was much higher in more concentrated solution. For lactic acid using 0.154 m isotonic lithium lactate solution, 30-50 percent of lactate was absorbed within four hours. He found that in the rumen contents, 30 mg of lactic acid/100 ml was always present. Shinozaki (108) studied the effects of V.F.A. and lactic acid on rumen motility. He reported that 0.2 m lactic acid did not influence so much rumen motility on comparative basis, formic acid in the same concentration severely affected rumen motility. Mowry et al (90) reported some work done on the effects of diets on the infusoria of goat rumen. They reported that grain mixture with fodder increased infusoria population and the feeding of grains alone resulted in lowered rumen pH with subsequent reduction in infusoria population as the amount of grains was increased. The decrease in numbers they said, was due to pH. They reported also that based on daily microbial counts, seven to nine days were required for normal infusoria to get established to normal when changing from one diet to the other. Castillejos, J. E. (28) induced moderate to acute lactic acidosis in goats with 11 gm and 18 gm of pure corn starch per kg. body weight respectively. Clinical signs were observed when blood D(-) lactic acid was 5 mg/100 ml or more.

MATERIALS AND METHODS

Animals

Three mature goats each weighing 40 kg were housed in individual stalls in the animal quarters of the laboratory in Veterinary Medical Science Building. They were fed twice daily on a maintainance ration of alfalfa hay. Water was available at all times. Each goat was surgically adapted with a permanent rumen fistula and a temporary indwelling canula in the jugular vein during experimental use. The jugular canulae were kept open by injecting heparinized saline after each sampling.

Supplies

The monosaccharides used in the experimental procedures were 1) D-glucose[1], 2) D-galactose[2] and 3) D-mannose[2]. Nicotinamide adenine dinucleitide[2], L^+ lactic dehydrogenase[2], D-lactic dehydrogenase[2], L^+ lactic[2] acid standard and D-lactic[2] acid standard were other reagents used. Appropriate dilutions of the L^+ lactic acid and D-lactic acid were made using double distilled water.

Procedure

D-glucose was administered intraruminally at dosage levels of 5 and 10 gm/kg body weight. D-mannose and D-galactose were administered at dosage levels of 5, 10 and 15 gm/kg body weight in separate experiments.

The rumen fistulae were used for the administration of the monosa-

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[1]Calbiochem, San Diego, California, 92112.

[2]Sigma Chemical Company, St. Louis, Missouri 63178, P. O. Box 14508

ccharides and sampling of rumen contents. Food was withheld 10-15 hrs before each experiment. Each goat was dosed with 5, 10 or 15 gm/kg body weight of each of the monosaccharides at 6 am on the sampling day. Samples of rumen contents and blood were taken one hour before dosing (zero hour) and at hourly intervals up to ten hours after dosing. An interval of seven days was allowed between experiments when using the lower doses (5 and 10 gm/kg body weight) and fourteen days for the higher doses (15 gm/kg body weight) to allow rumen microflora to adjust to normal.

Sampling

A few milliliters (mls) of blood which contained the heparinized saline were rejected. After this, blood was collected in the following manner:

1) Nine mls of blood was drawn with a 10 ml syringe and emptied into a 10 ml test tube containing 1 ml of anticoagulant (approx. 4% sodium fluoride and 4% potassium oxalate), and 80 mg of mercuric chloride for ammonia determination. The tube was sealed airtight and the contents mixed on a vortex mixer.
2) Another 9 mls of blood was withdrawn without using anticoagulant. This was allowed to settle at room temperature for thirty minutes before refrigeration. The serum obtained from it was analyzed on a Sequential Multiple Analyzer[3] (SMA).
3) Another 5 mls of blood was drawn with a monojet syringe and emptied into a 10 ml test tube containing one drop of heparin and 0.02 mg

[3]Sequential Multiple Analyzer. 12/60 Technicon Instruments Corporation, 2829 Blystone Lane, Dallas, Texas 75220.

of sodium fluoride as anticoagulant. This was centrifuged at 8000 rpm and the plasma was freed of its protein using 1 ml of plasma and 3 mls of 4.5% trichloroacetic acid. This was used for analysis of L^+ and D-lactic acids.

4) 1.5 mls of blood was taken anaerobically into a syringe containing one drop of heparin. The syringe was sealed by bending the needles and inserting it into its cover. The syringe was placed into an ice-bath and was analyzed within 15 minutes for blood pH, PCO_2, PO_2, total CO_2 and HCO_3^- on a corning gas analyzer.[4] The remaining blood was used for determining packed cell volume (PCV).

Rumen fluid was sampled for L^+ and D-lactic acids, pH and osmolarity. A 5 mls of rumen sample was withdrawn and 0.04 mg of mercuric chloride added to stop microbial fermentation. It was centrifuged at 8000 rpm, freed of its protein by using the same procedure as for blood lactic acid determination. Rumen pH was determined on pH meter[5] and osmolarity was determined on advanced osmometer.[6]

Analytical Procedure

The above samples were treated as follows: Blood and rumen fluid lactic acids were determined, using the method of Sigma Technical Bulletin 825-UV, 165. Blood and rumen ammonia were determined by Conway

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[4]Corning Blood Gas Analyzer, Scientific Products, Kansas City, Missouri.

[5]Beckman Instruments Inc., Scientific Instrument Division, 4901 Main Street, Suite LL-103, Kansas City, Missouri 64112.

[6]Advanced Instruments Inc., 55 Kenneth Street/617, Decatur 2-8200, Newton Highlands 61, Massachusetts 02161.

microdiffusion method. Whole blood pH, PCO_2, PO_2, and HCO_3^- and total carbon dioxide were determined by using blood gas analyzer.[4]

Serum creatinine, glucose, phosphorus, albumen, total protein, serum glutamic pyruvic transaminase (SGPT) chloride and sorbitol dehydrogenase were determined on sequential Multiple Analyzer[4] (SMA). Rumen Osmolarity was determined by advanced osmometer[3] and the rumen pH by using pH meter[2]. The PCV was analyzed using microcapillary haematocrit method.

The induction and severity of lactic acidosis was assessed by the clinical signs and quantitative change in each of the parameters listed above.

Results

Lactic acidosis was induced with 15 gm/kg body weight of both D-mannose and D-galactose and 10 gm/kg body weight of D-glucose. The acidosis induced by the galactose was not as severe as that induced by D-glucose and D-mannose as evaluated by the clinical signs observed.

General Observations

The goats exhibited clinical signs within the 10 hr observation period. The time of onset differed in each animal according to the monosaccharide administered in the doses that induced lactic acidosis. In one goat given the acidotic dose of D-glucose (10 gm.kg body weight)

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[2]Sigma Chemical Company, St. Louis, Missouri 63178, P.O. Box 14508.

[3]Sequential Multiple Analyzer. 12/60 Technicon Instruments Corporation, 2829 Blystone Lane, Dallas, Texas 75220.

[4]Corning Blood Gas Analyzer, Scientific Products, Kansas City, Miseouri.

the signs began 5 hrs after feeding. The feces became putty-like, leading to profuse diarrhea. Muscular twitching of the hind legs were noticed at about the same time with change in fecal consistency. Muscular twitchings were also noticed in both the other goats given D-mannose (15 gm/kg body weight) and D-galactose (15 gm/kg body wt.). The onset of clinical signs was noticed at about the 7th hour after dosing the acidotic dose of D-mannose. Incoordination of the left front leg of this goat was noticed. All the three goats exhibited the following signs: anorexia, increased rate of breathing, reduced rumen motility as examined by auscultation; dehydration and depression. All the doses did not produce acute ruminal acidosis that led to the death of the animals.

Physiological Changes

Intraruminal parameters: These included the pH, osmolarity, L(+) and D(-) lactic acids. Values obtained for these parameters are recorded in Tables (1-5 and Appendix I), Figures (1, 3, 5, 7a, 8a and 9a). In all goats, the rumen contents had a discernable odor. Froth was noticed in the rumen fluids of the two goats given acidotic doses of D-mannose and D-glucose. The rumen contents became more fluid post feeding.

The pH of the rumen fluids of the goats given acidotic doses of the monosaccharides showed a significant decrease. The pattern of rumen pH in the lower doses was that of decrease in early hours followed by increase in the later part of the observation periods. In the goat given acidotic dose of D-glucose, L(+) lactate ranged from 0.55 mg % at zero hour to a peak value of 28.8 mg % that was recorded at 1 hr post

Tables and Figures

Table # 1

Values of L(+) and D(-) lactic acids (mg %) of rumen fluid and blood from intraruminal administration of various doses of D-glucose.

Time of Sampling	Rumen Fluid				Blood			
	5 gm/kg. bod. wt.		10		5 gm/kg. bod. wt.		10	
	L^+	D^-	L^+	D^-	L^+	D^-	L^+	D^-
0 hr	0.99	0.2	0.55	0.38	0.83	1.77	3.0	2.47
1 hr	0.87	12.37	28.8	10.4	23.0	0.66	2.8	13.87
2 hr	0.47	22.7	15.0	20.1	13.0	0.59	2.6	12.75
3 hr	0.79	15.48	1.83	16.2	5.9	1.11	3.7	17.76
4 hr	0.27	9.88	0.6	20.6	8.9	2.65	2.9	10.92
5 hr	2.88	12.13	2.21	18.3	9.2	0.07	2.5	22.74
6 hr	1.1	5.06	2.46	18.4	17.3	1.25	2.2	8.64
7 hr	1.6	2.1	4.8	15.9	15.3	1.18	2.3	5.9
8 hr	1.0	2.41	2.1	16.6	13.9	1.18	2.3	8.19
9 hr	1 6	5.83	3.3	16.4	7.1	0.29	2.9	10.1
10 hr	1.6	1.01	4.2	16.0	7.0	0.07	2.0	11.37

Table # 2

Values of L(+) and D(-) lactic acids (mg %) of rumen fluid and blood from intraruminal administration of various doses of D-mannose.

Sampling Time	Rumen Fluid						Blood					
	5 gm/kg bod. wt.		10 gm/kg bod. wt.		15 g/kg bod. wt.		5 gm/kg bod wt.		10 g/kg bod.wt.		15 g/kg bod.wt.	
	L^+	D^-	L^+	D^-	L^+	D^-	L^+	D^-	L^+	D^-	L^+	D^-
0 hr	0	0	0.05	0	0	0	1.06	-	3.1	0	1.2	-
1 hr	0.1	0.02	12.0	5.7	49.5	6.6	2.96	-	9.3	0	21.6	-
2 hr	0.45	0.03	0.52	1.2	24.6	7.7	2.92	-	8.9	0	27.6	-
3 hr	0	0	0.81	0	12.9	16.86	2.67	-	11.9	0.3	-	-
4 hr	6.17	0	-	0	2.43	14.1	2.68	-	-	0	24.3	-
5 hr	0	0	0.09	0	5.8	20.3	1.48	-	7.2	2.1	-	-
6 hr	0.14	0	0.49	0	6.3	7.2	2.89	-	8.8	0.63	32.1	-
7 hr	0.35	0	0.33	0	8.4	-	2.48	-	6.2	0.54	-	-
8 hr	0.76	0	1.44	0	18.9	18.6	1.63	-	10.1	0.54	27.3	-
9 hr	0.24	0	4.68	1.5	39.6	11.6	1.87	-	8.8	0.63	-	-
10 hr	0.24	0	5.2	1.2	65.4	17.92	0	-	8.9	1.08	31.5	

Table # 3

Values of L(+) and D(-) lactic acids (mg %) of rumen fluid and blood from intraruminal administration of various doses of D-galactose.

Sampling Time	Rumen Fluid						Blood					
	5 gm/kg bod.wt.		10		15		5		10		15	
	L^+	D^-	L^+	D^-	L^+	D^-	L^+	D^-	L^+	D^-	L^+	D^-
0 hr	0.23	0	0.33	1.6	0.5	3.02	2.1	0	8.7	0	1.3	1.38
1 hr	36.9	3.9	1.53	3.3	0.36	3.9	15.4	0	16.2	0.07	4.9	1.38
2 hr	33.8	4.5	1.56	2.4	2.43	6.8	14.1	0	9.44	0.29	4.4	2.8
3 hr	32.2	5.1	1.87	4.8	1.35	7.1	10.9	0	15.3	-	5.1	2.4
4 hr	24.2	6.2	2.22	4.5	2.04	-	7.5	0	11.65	0.22	5.5	4.9
5 hr	15.8	3.3	1.65	5.4	0.72	9.0	15.1	0.13	10.0	2.5	5.5	8.64
6 hr	10.3	0	1.23	6.3	1.56	8.6	13.9	0.21	8.55	-	5.1	9.3
7 hr	5.2	0	1.23	6.0	1.98	11.9	12.9	0	12.1	-	6.2	9.5
8 hr	11.7	0	2.13	5.2	2.07	16.8	12.9	0	12.3	1.5	5.9	5.07
9 hr	10.8	0	1.5	13.5	2.25	11.4	14.2	0.63	9.6	1.7	7.2	9.9
10 hr	1.3	0	0.78	15.3	2.25	14.6	11.5	1.3	17.1	2.04	8.5	12.6

Table # 4.

Values of rumen fluid pH from intraruminal administration of various doses of monosaccharides.

Sampling time	D-mannose (gm/kg. bod. wt.)			D-glucose (gm/kg. bod. wt.)		D-galactose (gm/kg. bod. wt.)		
	5	10	15	5	10	5	10	15
0 hr	6.2	7.2	6.3	7.1	6.3	7.25	6.75	6.9
1 hr	5.94	7.0	6.15	7.0	5.75	6.55	6.7	6.85
2 hr	5.5	6.8	5.85	6.8	5.6	6.2	6.15	6.45
3 hr	5.85	6.5	5.7	6.7	5.5	5.95	6.0	6.4
4 hr	5.6	-	5.6	6.8	5.4	5.75	5.95	6.2
5 hr	5.75	6.45	5.5	6.6	5.35	5.15	5.65	6.0
6 hr	5.95	6.18	5.5	6.65	5.35	5.25	5.6	5.9
7 hr	6.15	6.0	5.5	6.7	5.25	5.2	5.5	5.6
8 hr	6.15	5.8	5.4	6.7	5.3	5.65	5.6	5.5
9 hr	6.3	5.7	5.35	6.45	5.25	5.85	5.7	5.3
10 hr	6.4	5.6	5.3	6.1	5.2	-	5.6	5.25

Table # 5

Values of rumen fluid osmolarity (m Osmoles/L) from intraruminal administration of various doses of monosaccharides.

Sampling time	D-mannose (gm/kg. bod. wt.)			D-glucose (gm/kg. bod. wt.)		D-galactose (gm/kg. bod. wt.)		
	5	10	15	5	10	5	10	15
0 hr	320	229	257	254	203	258	210	303
1 hr	369	364	525	359	359	423	391	320
2 hr	370	418	363	358	326	377	378	-
3 hr	329	448	409	337	263	352	395	-
4 hr	321	-	391	312	251	339	339	-
5 hr	300	415	279	296	215	321	298	-
6 hr	276	398	270	274	188	313	270	752
7 hr	239	350	252	272	217	290	227	592
8 hr	242	339	233	278	165	279	226	464
9 hr	237	329	226	249	158	277	199	
10 hr	227	299	-	256	139	232	198	387

Fig. # 1

Time course changes in rumen fluid L(+), D(*) lactic acids and pH from intraruminal administration of acidotic dose of D-glucose.

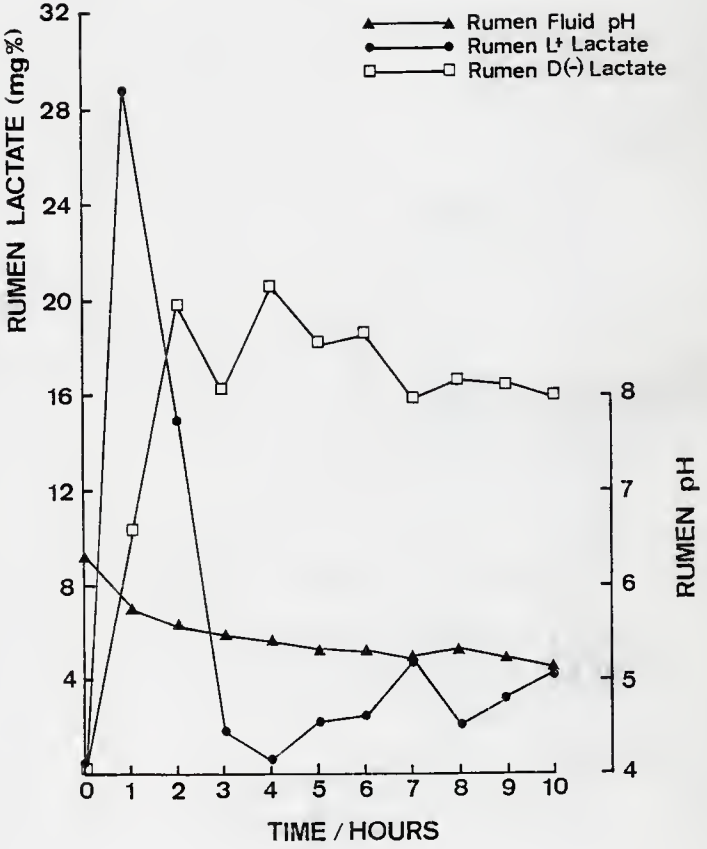

Rumen Fluid pH
Rumen L+ Lactate
Rumen D(-) Lactate
RUMEN LACTATE (mg%)
32
28
24
20
16
12
8
4
RUMEN pH
8
7
6
5
4
0 1 2 3 4 5 6 7 8 9 10
TIME / HOURS

Fig. # 2

Time course changes in blood L(+), D(-) lactic acids and pH from intraruminal administration of acidotic dose of D-glucose.

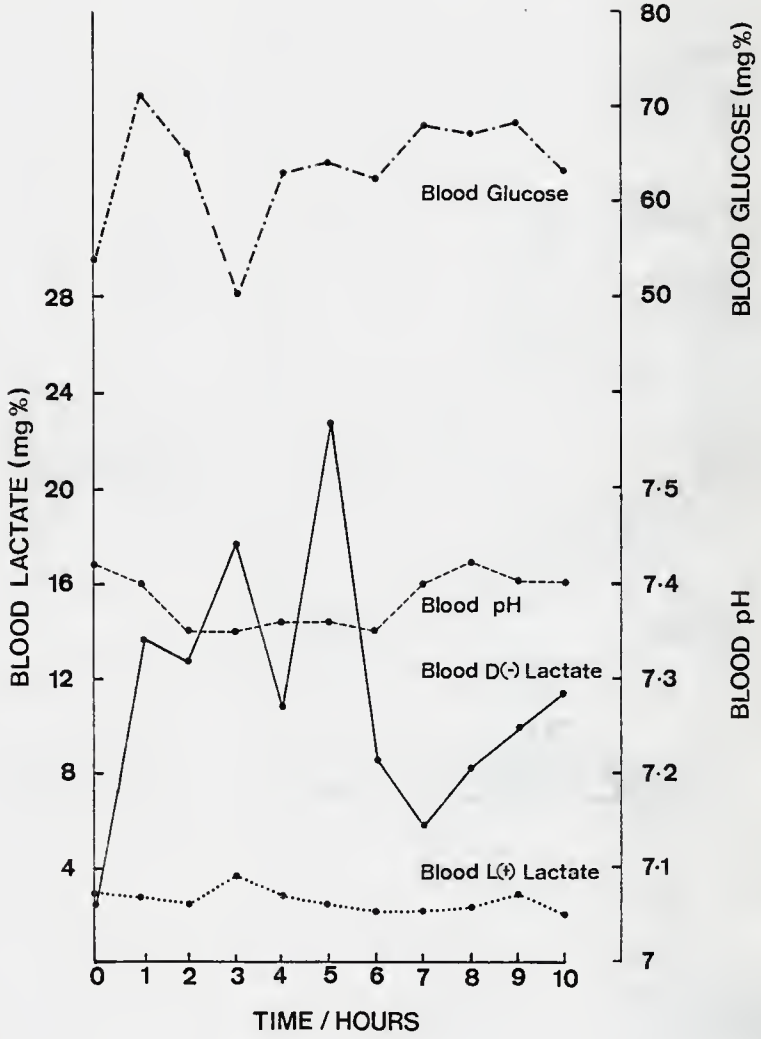

BLOOD LACTATE (mg %)
28
24
20
16
12
8
4
BLOOD GLUCOSE (mg %)
80
70
60
50
BLOOD pH
7·5
7·4
7·3
7·2
7·1
7
Blood Glucose
Blood pH
Blood D(-) Lactate
Blood L(+) Lactate
0 1 2 3 4 5 6 7 8 9 10
TIME / HOURS

Fig. # 3

Time course changes in rumen L(+), D(-) lactic acids and pH from intraruminal administration of acidotic dose of D-mannose.

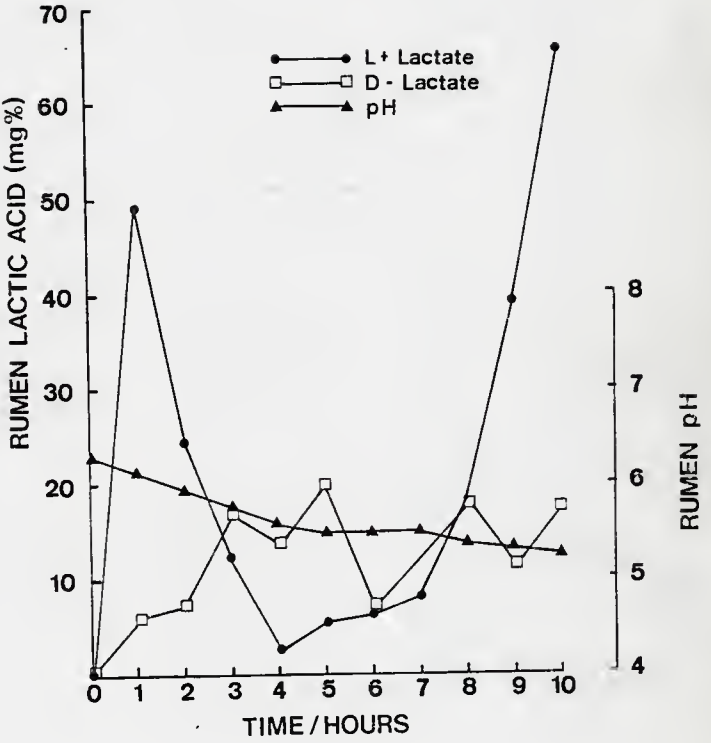

L+ Lactate
D - Lactate
pH
RUMEN LACTIC ACID (mg%)
RUMEN pH
TIME / HOURS
70
60
50
40
30
20
10
8
7
6
5
4
0
1
2
3
4
5
6
7
8
9
10

Fig. # 4

Time course changes in blood L(+), D(-) lactic acids and pH from intraruminal administration of acidotic dose of D-mannose.

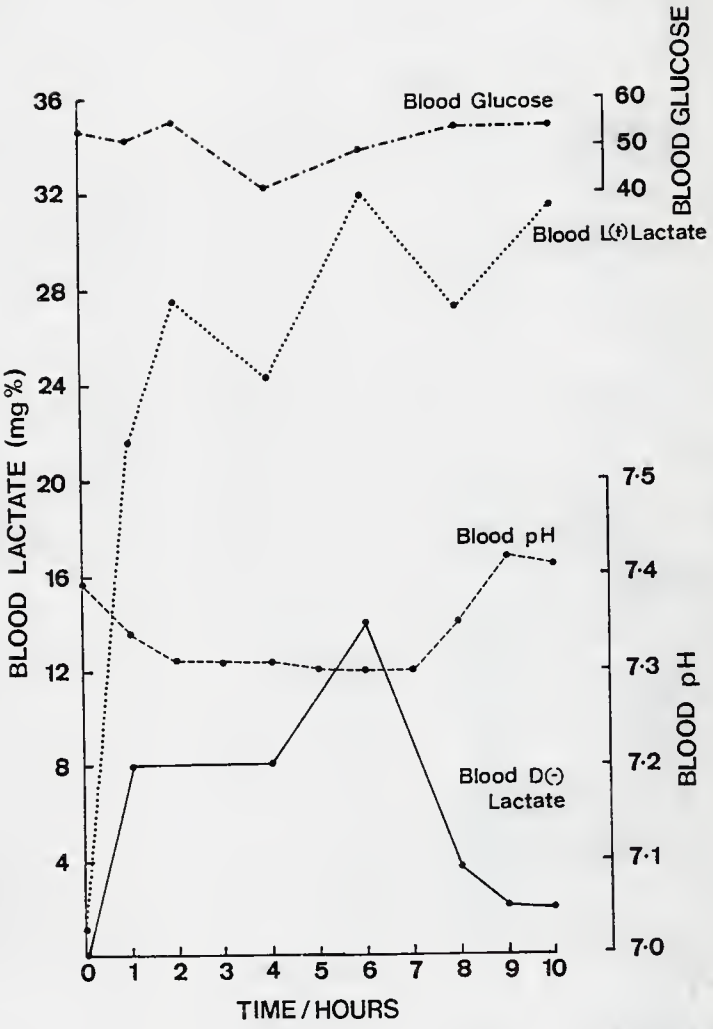

Blood Glucose
Blood L(+)Lactate
Blood pH
Blood D(-) Lactate
BLOOD LACTATE (mg %)
BLOOD GLUCOSE
BLOOD pH
TIME / HOURS
36
32
28
24
20
16
12
8
4
60
50
40
7·5
7·4
7·3
7·2
7·1
7·0
0 1 2 3 4 5 6 7 8 9 10

Fig. # 5

Time course changes in rumen L(+), D(-) lactic acids and pH from intraruminal administration of acidotic dose of D-galactose.

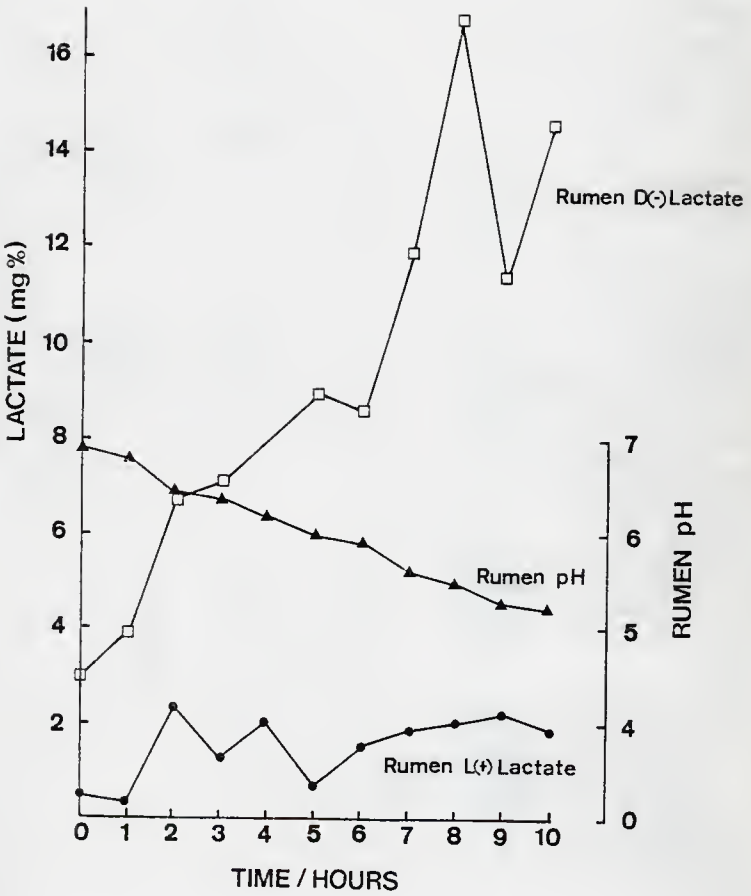

LACTATE (mg %)
16
14
12
10
8
6
4
2
Rumen D(-) Lactate
Rumen pH
Rumen L(+) Lactate
RUMEN pH
7
6
5
4
0
0 1 2 3 4 5 6 7 8 9 10
TIME / HOURS

Fig. # 6

Time course changes in blood L(+), D(-) lactic acids and pH from intraruminal administration of acidotic dose of D-galactose.

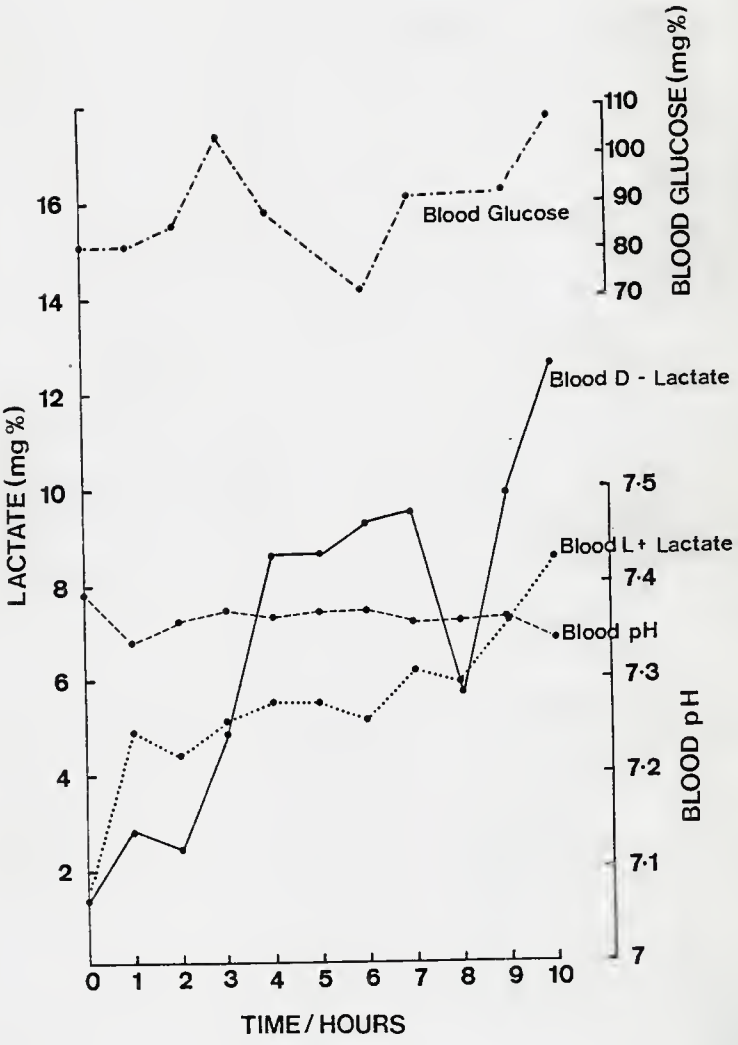

Blood Glucose
BLOOD GLUCOSE (mg%)
110
100
90
80
70
16
14
12
10
8
6
4
2
LACTATE (mg %)
Blood D - Lactate
Blood L+ Lactate
Blood pH
7·5
7·4
7·3
7·2
7·1
7
BLOOD pH
0 1 2 3 4 5 6 7 8 9 10
TIME / HOURS

Fig. # 7a

Time course changes in rumen ammonia-N from intraruminal administration of various doses of D-glucose.

Fig. # 7b

Time course changes in blood ammonia-N from intraruminal administration of various doses of D-glucose.

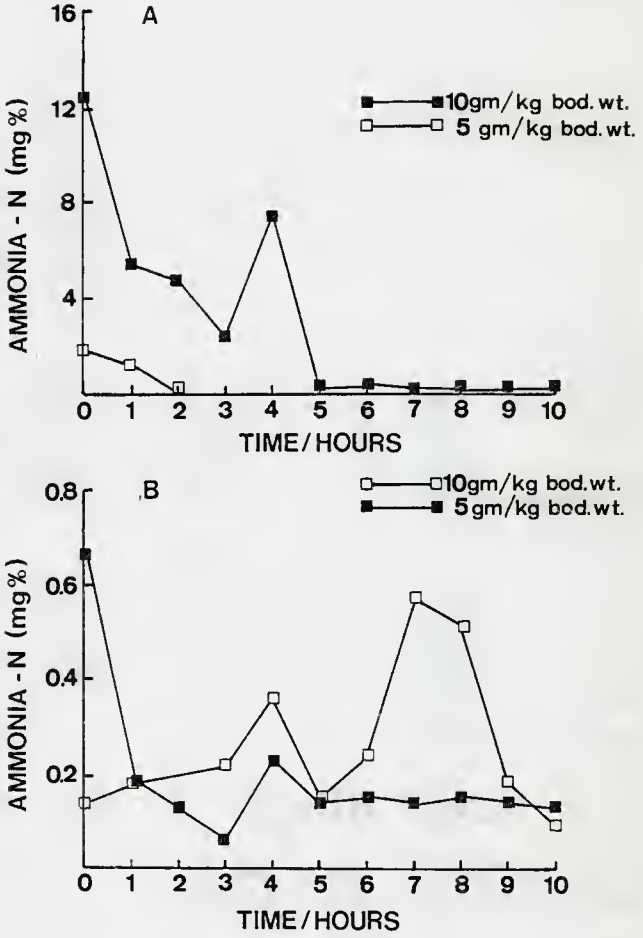

A
16
12
8
4
AMMONIA - N (mg %)
10gm/kg bod.wt.
5 gm/kg bod.wt.
0 1 2 3 4 5 6 7 8 9 10
TIME/HOURS
B
0.8
0.6
0.4
0.2
AMMONIA - N (mg %)
10gm/kg bod.wt.
5gm/kg bod.wt.
0 1 2 3 4 5 6 7 8 9 10
TIME/HOURS

Fig. # 8a

Time course changes of rumen ammonia-N from intraruminal administration of various doses of D-mannose.

Fig. # 8b

Time course changes in blood ammonia-N from intraruminal administration various doses of D-mannose.

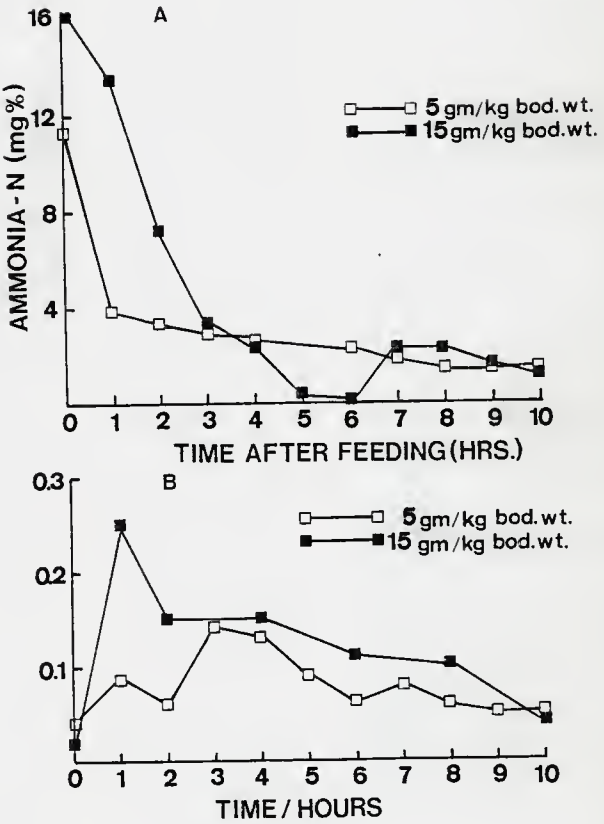

A
16
12
8
4
AMMONIA - N (mg%)
5 gm/kg bod. wt.
15 gm/kg bod. wt.
0 1 2 3 4 5 6 7 8 9 10
TIME AFTER FEEDING (HRS.)
B
0.3
0.2
0.1
5 gm/kg bod. wt.
15 gm/kg bod. wt.
0 1 2 3 4 5 6 7 8 9 10
TIME / HOURS

Fig. # 9a

Time course changes of rumen ammonia-N from intraruminal administration of various doses of D-galactose.

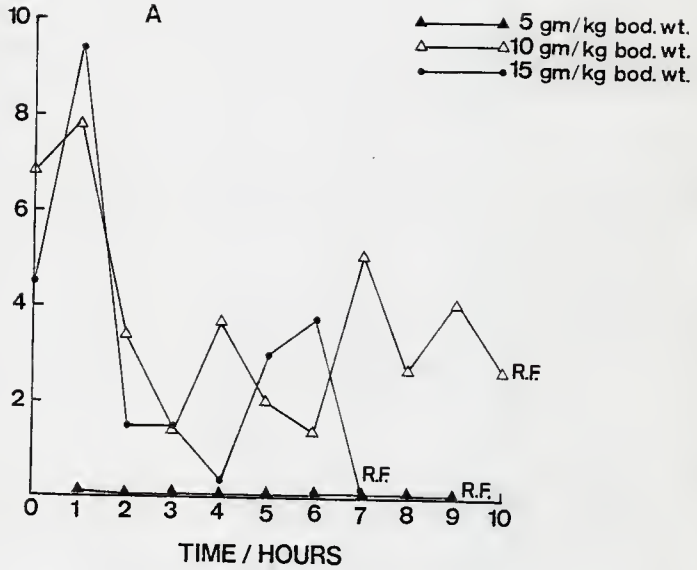

A
5 gm/kg bod. wt.
10 gm/kg bod. wt.
15 gm/kg bod. wt.
10
8
6
4
2
0
1
2
3
4
5
6
7
8
9
10
R.F.
R.F.
R.F.
TIME / HOURS

Fig. # 9b

Time course changes in blood ammonia-N from intraruminal administration of various doses of D-galactose.

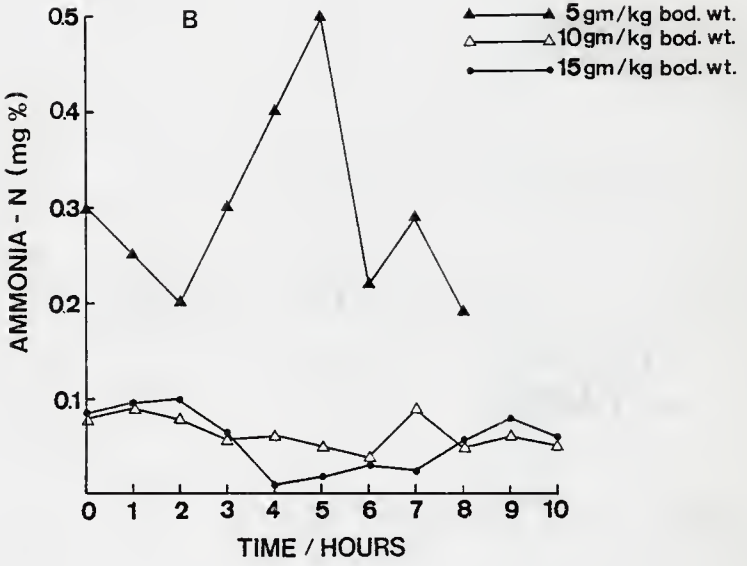

B
5 gm/kg bod. wt.
10 gm/kg bod. wt.
15 gm/kg bod. wt.
AMMONIA - N (mg %)
0.5
0.4
0.3
0.2
0.1
0 1 2 3 4 5 6 7 8 9 10
TIME / HOURS

Table # 6

Values of blood pH from intraruminal administration of various doses of monosaccharides.

Sampling time	D-mannose (gm/kg. bod. wt.)			D-glucose (gm/kg. bod. wt.)		D-galactose (gm/kg. bod. wt.)		
	5	10	15	5	10	5	10	15
0 hr	7.4	7.42	7.39	7.4	7.42	7.32	7.4	7.39
1 hr	7.42	7.42	7.34	7.42	7.4	7.47	7.43	7.34
2 hr	7.4	7.42	7.31	7.43	7.35	7.43	7.43	7.36
3 hr	7.44	-	7.31	7.42	7.35	7.4	7.44	7.37
4 hr	7.41	7.43	-	7.41	7.36	7.42	7.44	7.38
5 hr	7.43	-	7.34	7.42	7.36	7.44	7.41	7.37
6 hr	7.48	7.4	7.35	7.47	7.35	7.45	7.44	7.37
7 hr	7.43	-	7.34	7.43	7.4	7.57	7.49	7.36
8 hr	7.42	7.41	7.35	7.46	7.42	7.5	7.57	7.36
9 hr	7.41	-	7.42	7.45	7.44	7.48	7.5	7.39
10 hr	7.41	7.44	7.41	7.44	7.45	7.52	7.49	7.40

Table # 7

Values of blood packed cell volume (PCV) from intraruminal administration of various doses of monosaccharides.

Sampling time	D-mannose (gm/kg. bod. wt.)			D-glucose (gm/kg. bod. wt.)		D-galactose (gm/kg. bod. wt.)		
	5	10	15	5	10	5	10	15
0 hr	28.0	21.5	25.0	28.5	22.5	39.0	27	28.5
1 hr	27.5	20.5	24.4	23.0	21.0	33.5	28.5	28.8
2 hr	25.5	23.5	23.8	23.25	21.0	33.0	25	25.0
3 hr	24.5	23.4	-	19.8	20.5	32.5	23	25.4
4 hr	25.8	-	24.0	22.5	21.0	31.5	25.5	25.0
5 hr	26.5	22.0	-	22.0	17.5	30.8	23.5	25.5
6 hr	23.5	20.0	24.5	20.5	19.5	28.2	20.5	23.0
7 hr	23.5	21.5	-	19.5	19.5	29.5	24.5	24.5
8 hr	24.0	21.5	25.5	20	21.0	28.4	20.5	26.5
9 hr	25.0	20.0	-	19.0	20.0	26.4	20.0	-
10 hr	23.0	19.5	29.0	19.0	18.0	25.0	23.5	-

Table # 8

Values of bicarbonate levels (m Eq/L) from intraruminal administration of various doses of monosaccharides.

Sampling time	D-mannose (gm/kg. bod. wt.)			D-glucose (gm/kg. bod. wt.)		D-galactose (gm/kg. bod. wt.)		
	5	10	15	5	10	5	10	15
0 hr	29.5	21.8	21.5	-	-		37.1	32.9
1 hr	30.5	26.8	20.6	-	-		36.2	28.9
2 hr	29.1	24.3	21.6	-	36.9		38.8	31.2
3 hr	30.6	-	22.8	-	38.7		31.0	31.3
4 hr	28.1	25.6	-	-	32.6		32.5	31.3
5 hr	28.5	-	27.0	-	31.3		31.0	30.4
6 hr	29.5	26.5	26.6	-	33.2		29.6	31.8
7 hr	30.0	27.6	27.6	-	31.9		26.7	29.9
8 hr	28.7	27.6	25.4	-	31.4		25.1	30.0
9 hr	28.3	-	28.3	-	39.1		31.0	33.0
10 hr	29.1	26.6	26.4	-	35.4		32.8	31.6

Table # 9

Values of blood pCO_2 (mm Hg) from intraruminal administration of various doses of monosaccharides.

Sampling time	D-mannose (gm/kg. bod. wt.)			D-glucose (gm/kg. bod. wt.)		D-galactose (gm/kg. bod. wt.)		
	5	10	15	5	10	5	10	15
0 hr	49.5	36.7	45.8	41.9	42.0	51.4	50.9	46.3
1 hr	48.7	34.7	42.2	44.3	41.0	50.5	51.1	45
2 hr	48.6	43.4	40.5	39.4	40.1	43.8	43.0	47.4
3 hr	46.3	46.6	-	44.5	39.8	45.8	48.0	40.0
4 hr	45.5	-	36.3	44.6	41.2	48.8	44.9	39.2
5 hr	44.4	50.2	-	44.5	42	43.2	49.6	36.6
6 hr	41.3	49.4	35.2	40.1	40.3	44.1	46.2	35.5
7 hr	46.6	51.6	-	48.3	36.9	41.9	46.8	34.0
8 hr	45.4	48.0	40.5	46.5	41.0	37.8	46.4	39.6
9 hr	45.7	45.3	-	45.6	42.0	42.3	47.3	44.7
10 hr	47.1	43.0	41.3	46.8	42.0	41.2	47.8	47.2

Table # 10

Values of blood total carbon dioxide (mg Eq/L) from intraruminal administration of various doses of monosaccharides.

Sampling time	D-mannose (gm/kg. bod. wt.)			D-glucose (gm/kg. bod. wt.)		D-galactose (gm/kg. bod. wt.)		
	5	10	15	5	10	5	10	15
0 hr	30.8	27.2	43.8	37.8	35.8	-	38.8	33.8
1 hr	31.9	22.0	38.9	38.7	31.1	-	41.6	31.4
2 hr	30.6	22.8	40.4	40.7	33.0	-	42.8	33.6
3 hr	31.9	24	-	44.6	33.0	-	46.7	34.3
4 hr	29.6	-	38.3	43.5	32.8	-	45.0	37.4
5 hr	29.8	28.6	-	45.6	33.3	-	45.2	33.3
6 hr	30.9	28.4	38.7	43.5	32.6	-	45.8	32.9
7 hr	31.6	29.2	-	43.5	30.7	-	43.6	31.7
8 hr	30.1	27.1	39.4	41.0	31.7	-	45.3	34.8
9 hr	29.9	29.9	-	37.5	31.0	-	45.4	33.2
10 hr	30.8	27.9	34.6	-	32.7	-	45.7	36.6

Table # 11

Values of serum carbon dioxide (m Eq/L) from intraruminal administration of various doses of monosaccharides.

Sampling time	D-mannose (gm/kg. bod. wt.)			D-glucose (gm/kg. bod. wt.)		D-galactose (gm/kg. bod. wt.)		
	5	10	15	5	10	5	10	15
0 hr	-	23	28	-	26	30	29	26
1 hr	-	24	27	-	29	23	33	25
2 hr	-	21	28	-	34	23	33	27
3 hr	-	-	-	-	31	26	35	32
4 hr	-	21	35	-	30	-	36	32
5 hr	-	27	-	-	32	-	35	-
6 hr	-	24	30	-	31	24	39	34
7 hr	-	29	-	-	32	-	35	31
8 hr	-	29	36	-	34	27	37	-
9 hr	-	30	-	-	27	26	38	28
10 hr	-	28	33	-	28	26	37	40

Table # 12

Values of blood glucose (mg %) from intraruminal administration of various doses of monosaccharides.

Sampling time	D-mannose (gm/kg. bod. wt.)			D-glucose (gm.kg. bod. wt.)		D-galactose (gm/kg. bod. wt.)		
	5	10	15	5	10	5	10	15
0 hr	26	43	53	54	54	83	69	81
1 hr	33	62	51	51	71	111	60	81
2 hr	38	51	55	67	65	97	64	85
3 hr	43	50	-	69	50	95	76	104
4 hr	55	51	41	75	63	87	64	88
5 hr	60	58	-	67	64	54	80	-
6 hr	66	56	49	77	63	52	88	71
7 hr	54	69	-	64	68	34	53	91
8 hr	64	69	54	68	67	43	65	-
9 hr	81	67	-	68	68	38	75	92
10 hr	71	74	53	64	63	53	88	108

Table # 13

Values of serum glutamic pyruvic transaminase (SGPT in mU/ml) from intraruminal administration of various doses of monosaccharides.

Sampling time	D-mannose (gm/kg. bod. wt.)			D-glucose (gm/kg. bod. wt.)		D-galactose (gm/kg. bod. wt.)		
	5	10	15	5	10	5	10	15
0 hr	1.0	0	5	-	0	0	0	-
1 hr	-	0	2	-	0	0	0	-
2 hr	-	0	2	-	0	0	0	-
3 hr	-	0	-	-	0	0	0	1
4 hr	-	0	2	-	0	2	0	-
5 hr	-	0	-	-	0	0	0	-
6 hr	-	0	3	-	0	0	0	-
7 hr	-	0	-	-	0	0	0	-
8 hr	-	0	0	-	0	0	0	-
9 hr	-	0	-	-	0	0	0	-
10 hr	-	0	0	-	0	0	0	-

Table # 14

Values of blood PO_2 from intraruminal administration of various doses of monosaccharides.

Sampling time	D-mannose (gm/kg. bod. wt.)			D-glucose (gm/kg. bod. wt.)		D-galactose (gm/kg. bod. wt.)		
	5	10	15	5	10	5	10	15
0 hr	37.5	36.8	23.8	33.2	30.3	27.0	22.4	25.9
1 hr	34.8	33.2	26.1	36.0	37.6	24.1	28.6	23.0
2 hr	32.5	30.8	27.6	37.8	31.4	28.1	30.1	30.8
3 hr	31.0	31.4	26.1	34.2	31.1	31.8	31.1	29.8
4 hr	31.3	-	26.8	32.2	28.4	27.4	31.0	28.8
5 hr	29.5	29.6	25.6	31.7	29.8	29.9	29.7	29.2
6 hr	21.0	29.9	24.3	33.0	28.3	31.9	30.2	29.8
7 hr	30.1	27.1	25.6	35.1	27.4	35.4	29.4	27 8
8 hr	29.2	37.8	24.1	34.1	25.4	31.4	27.7	23.9
9 hr	29.1	31.0	23.5	34.0	26.2	32.3	27.9	24.5
10 hr	29.1	31.0	23.5	34.0	26.2	32.3	27.9	24.5

Table # 15

Values of serum total protein (gm %) from intraruminal administration of various doses of monosaccharides.

Sampling time	D-mannose (gm/kg. bod. wt.)			D-glucose (gm/kg. bod. wt.)		D-galactose (gm/kg. bod. wt.)		
	5	10	15	5	10	5	10	15
0 hr	8.0	8.7	7.5	8.6	7.4	9.1	7.5	8.3
1 hr	8.5	8.0	7.7	8.1	7.3	9.3	7.4	8.3
2 hr	7.6	8.4	7.8	7.7	7.5	9.1	7.5	7.8
3 hr	7.7	8.6	8.0	-	7.6	9.1	7.6	7.9
4 hr	7.7	7.9	8.1	-	7.9	8.4	7.3	7.9
5 hr	7.9	7.9	8.1	7.7	7.8	7.9	7.0	-
6 hr	7.6	8.1	8.2	7.8	7.5	8.7	7.1	7.6
7 hr	7.2	7.9	8.2	7.7	7.8	8.3	7.4	7.6
8 hr	7.0	7.9	8.1	7.9	7.9	8.4	7.2	7.5
9 hr	7.9	7.7	8.2	6.8	7.3	8.5	7.0	8.0
10 hr	7.2	7.7	8.1	7.7	7.4	8.7	7.4	7.5

Table # 16

Values of serum albumin (gm %) from intraruminal administration of various doses of monosaccharides.

Sampling time	D-mannose (gm/kg. bod. wt.)			D-glucose (gm/kg. bod. wt.)		D-galactose (gm/kg. bod. wt.)		
	5	10	15	5	10	5	10	15
0 hr	3.4	3.5	3.5	3.4	3.0	3.9	3.3	3.7
1 hr	3.6	3.1	3.6	6.0	3.1	3.9	3.3	3.7
2 hr	3.3	3.3	3.6	5.7	3.2	3.9	3.3	3.5
3 hr	3.3	3.5	-	5.7	3.0	4.0	3.3	3.5
4 hr	3.4	3.3	3.5	-	3.2	3.6	3.2	3.5
5 hr	3.4	3.4	-	3.0	3.1	3.6	3.1	-
6 hr	3.3	3.3	3.5	3.0	3.2	3.8	3.1	3.5
7 hr	3.1	3.5	-	2.9	3.0	3.5	3.2	3.3
8 hr	3.1	3.5	3.5	3.0	3.2	3.8	3.1	-
9 hr	3.1	3.3	-	2.9	3.1	3.7	3.0	3.5
10 hr	3.0	3.3	3.6	2.9	3.2	3.7	3.2	3.4

Table # 17

Values of serum sodium (m Eq/L) from intraruminal administration of various doses of monosaccharides.

Sampling time	D-mannose (gm/kg. bod. wt.)			D-glucose (gm/kg. bod. wt.)		D-galactose (gm/kg. bod. wt.)		
	5	10	15	5	10	5	10	15
0 hr	-	141	155	147	154	154	145	162
1 hr	-	140	156	149	147	158	143	-
2 hr	-	143	158	151	150	157	144	168
3 hr	-	-	-	152	153	163	149	159
4 hr	-	148	161	144	155	-	151	168
5 hr	-	149	-	143	154	-	163	-
6 hr	-	152	168	144	156	161	162	155
7 hr	-	152	-	143	155	-	163	-
8 hr	-	152	161	142	156	157	162	-
9 hr	-	151	-	140	145	158	160	158
10 hr	-	150	159	130	155	159	160	161

Table # 18

Values of serum potassium (m Eq/L) from intraruminal administration of various doses of monosaccharides.

Sampling time	D-mannose (gm/kg. bod. wt.)			D-glucose (gm/kg. bod. wt.)		D-galactose (gm/kg. bod. wt.)		
	5	10	15	5	10	5	10	15
0 hr	-	5.2	3.7	4.9	4.9	6.3	4.0	6.0
1 hr	-	4.4	4.0	4.6	5.1	5.8	3.9	-
2 hr	-	5.0	4.1	4.1	5.1	5.3	3.9	6.1
3 hr	-	-	4.2	4.3	4.8	5.1	4.2	5.1
4 hr	-	5.3	4.3	4.0	5.4	-	3.7	5.4
5 hr	-	4.9	4.4	4.0	5.2	-	3.7	-
6 hr	-	4.9	4.3	4.1	5.0	5.8	3.5	4.8
7 hr	-	4.6	4.1	4.2	5.6	-	3.7	-
8 hr	-	4.6	3.9	3.9	5.6	5.5	3.4	-
9 hr	-	5.0	3.9	4.1	6.1	5.4	3.2	5.8
10 hr	-	4.8	4.0	4.2	5.0	5.8	3.6	4.7

Table # 19

Values of serum chloride (m Eq/L) from intraruminal administration of various doses of monosaccharides.

Sampling time	D-mannose (gm/kg. bod. wt.)			D-glucose (gm/kg. bod. wt.)		D-galactose (gm/kg. bod. wt.)		
	5	10	15	5	10	5	10	15
0 hr	108	102	109	-	108	105	112	-
1 hr	-	108	112	-	108	110	105	-
2 hr	107	104	114	-	106	110	107	-
3 hr	106	104	114	-	106	-	108	112
4 hr	-	-	116	-	107	-	109	-
5 hr	108	104	-	-	108	-	111	-
6 hr	-	104	121	-	107	111	109	112
7 hr	106	106	121	-	109	-	110	-
8 hr	106	106	120	-	107	109	112	-
9 hr	111	106	120	-	114	112	115	-
10 hr	104	112	119	-	110	114	-	110

Fig. # 10

Time course changes in serum alkaline phosphatase from intraruminal administration of various doses of D-glucose.

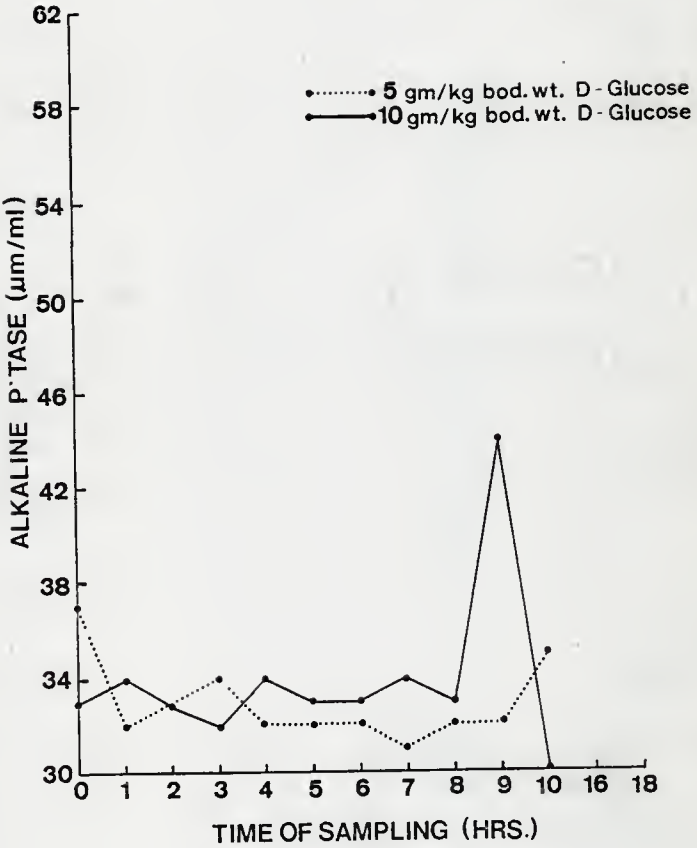

5 gm/kg bod. wt. D - Glucose
10 gm/kg bod. wt. D - Glucose
ALKALINE P'TASE (µm/ml)
62
58
54
50
46
42
38
34
30
0
1
2
3
4
5
6
7
8
9
10
16
18
TIME OF SAMPLING (HRS.)

Fig. # 11

Time course changes in serum alkaline phosphatase from intraruminal administration of various doses of D-mannose.

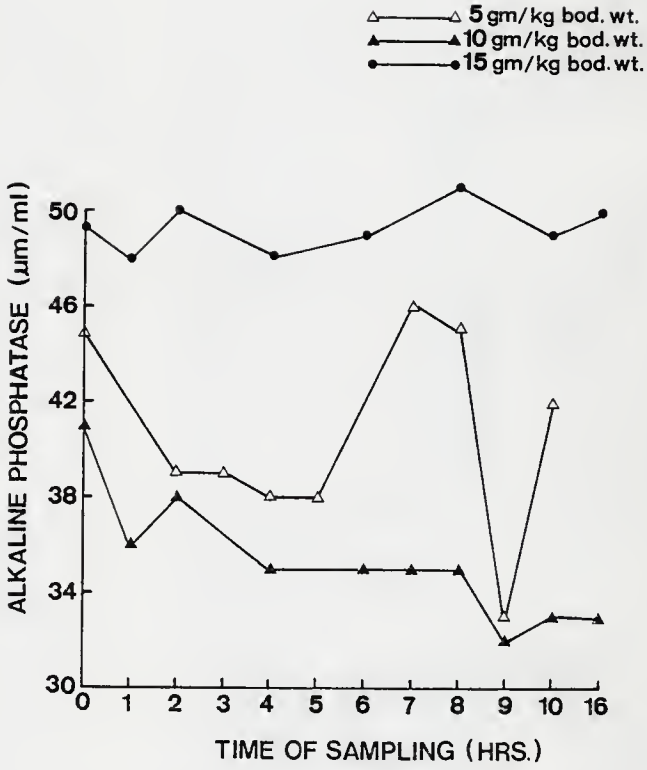

5 gm/kg bod. wt.
10 gm/kg bod. wt.
15 gm/kg bod. wt.
ALKALINE PHOSPHATASE (µm/ml)
50
46
42
38
34
30
0 1 2 3 4 5 6 7 8 9 10 16
TIME OF SAMPLING (HRS.)

Fig. # 12

Time course changes of serum alkaline phosphatase from intraruminal administration of various doses of D-galactose.

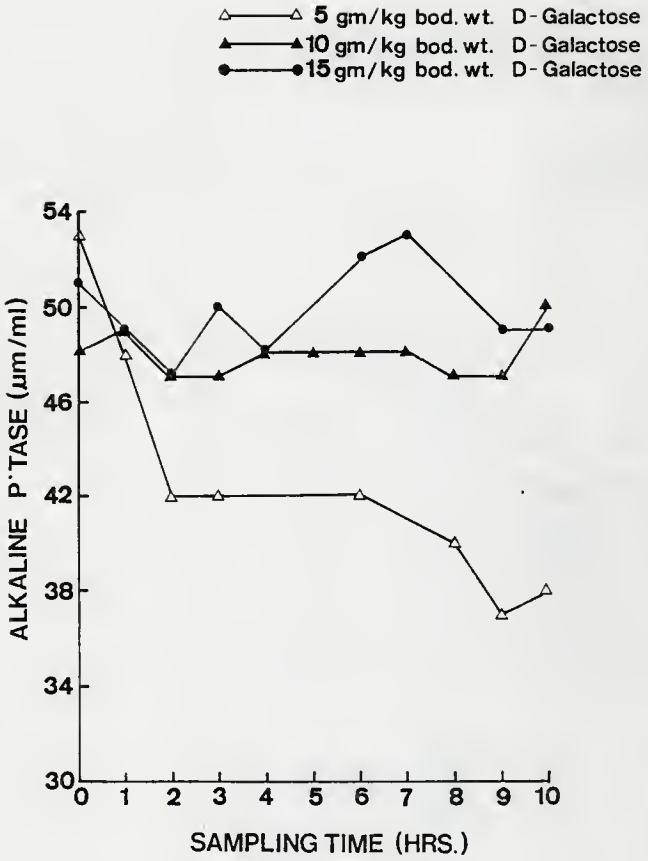

5 gm/kg bod. wt. D-Galactose
10 gm/kg bod. wt. D-Galactose
15 gm/kg bod. wt. D-Galactose
ALKALINE P'TASE (µm/ml)
54
50
46
42
38
34
30
0 1 2 3 4 5 6 7 8 9 10
SAMPLING TIME (HRS.)

Fig. # 13

Time course changes in serum creatinine from intraruminal administration of non-acidotic doses of various doses of monosaccharides.

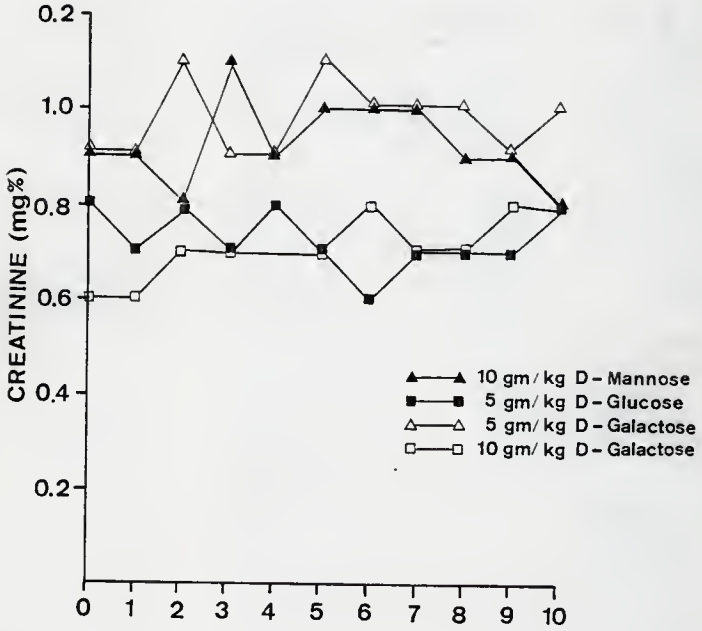

CREATININE (mg%)
0.2
1.0
0.8
0.6
0.4
0.2
0 1 2 3 4 5 6 7 8 9 10
10 gm/kg D-Mannose
5 gm/kg D-Glucose
5 gm/kg D-Galactose
10 gm/kg D-Galactose

Fig. # 14

Time course changes in serum creatinine from intraruminal administration of acidotic doses of monosaccharides.

A 5 gm/kg D-Mannose
B 10 gm/kg D-Glucose
C 15 gm/kg D-Galactose

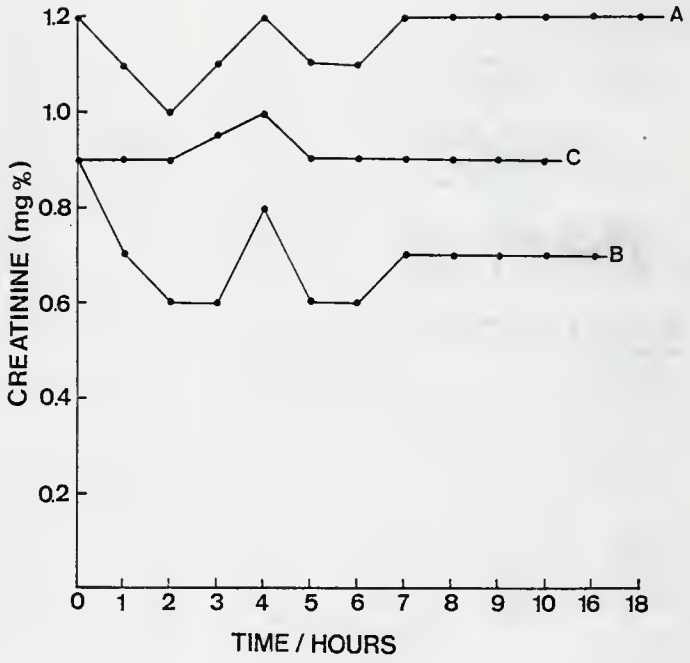


Fig. # 15

Time course changes in serum phosphorus from intraruminal administration of various doses of D-glucose.

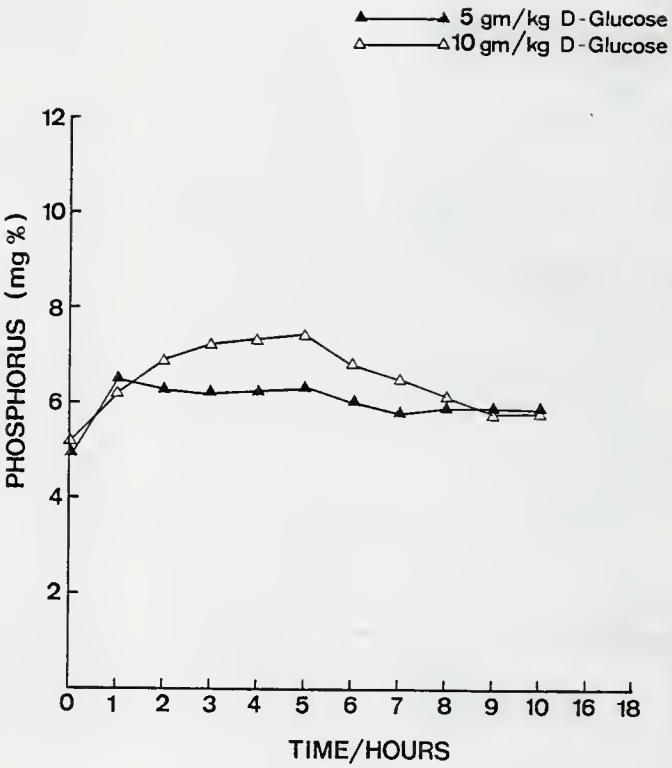

5 gm/kg D-Glucose
10 gm/kg D-Glucose
PHOSPHORUS (mg %)
12
10
8
6
4
2
0 1 2 3 4 5 6 7 8 9 10 16 18
TIME/HOURS

Fig. # 16

Time course changes in serum phosphorus from intraruminal administration of various doses of D-mannose.

△——△ 5 gm/kg bod.wt. D-Mannose
▲——▲ 10 " "
■——■ 15 " "

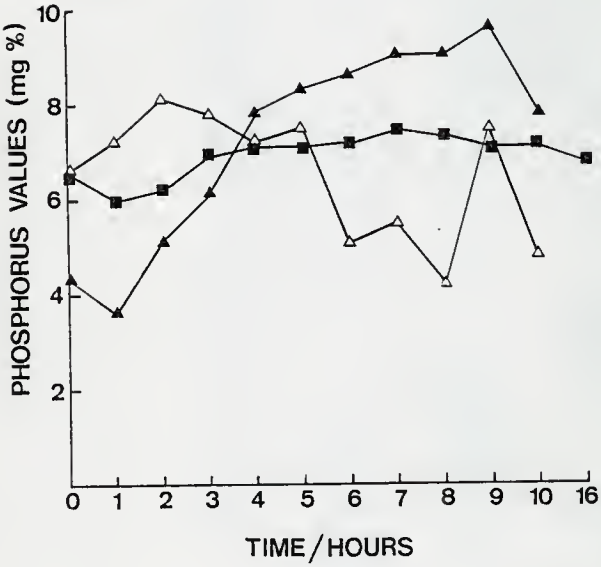


Fig. # 17

Time course changes in serum phosphorus from intraruminal administration of various doses of D-galactose.

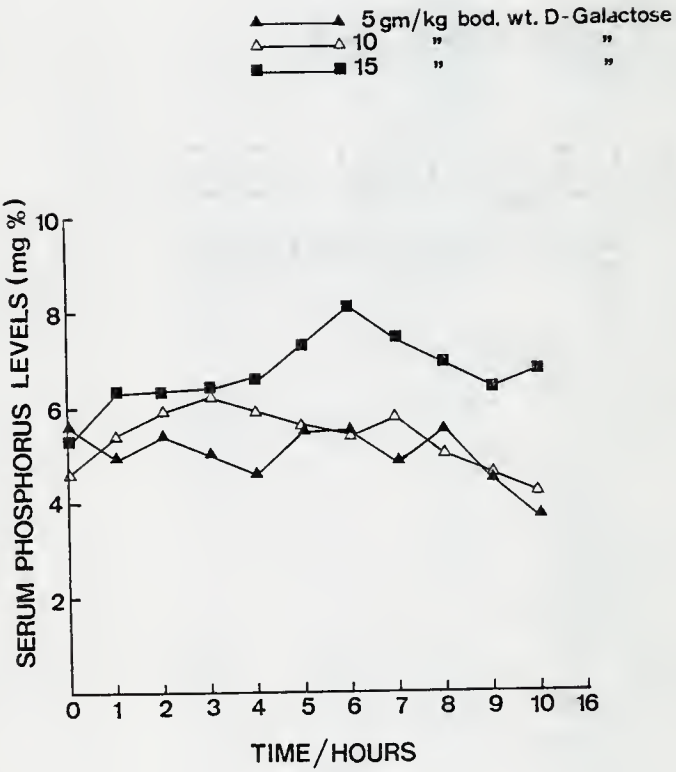

5 gm/kg bod. wt. D-Galactose
10 " "
15 " "
SERUM PHOSPHORUS LEVELS (mg %)
10
8
6
4
2
0 1 2 3 4 5 6 7 8 9 10 16
TIME/HOURS

Fig. # 18

Time course changes in serum calcium from intraruminal administration of various doses of D-glucose.

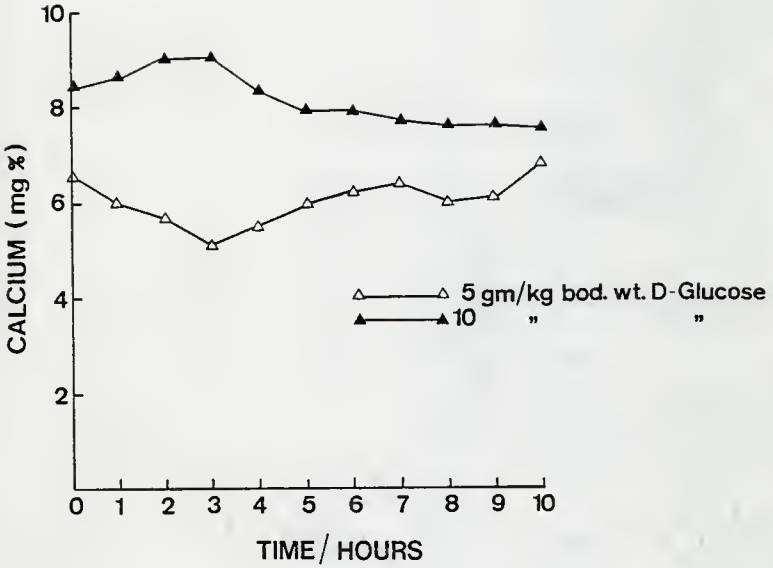

10
8
6
4
2
CALCIUM (mg %)
0 1 2 3 4 5 6 7 8 9 10
TIME / HOURS
5 gm/kg bod. wt. D-Glucose
10 " "

Fig. # 19

Time course changes in serum calcium from intraruminal administration of various doses of D-mannose.

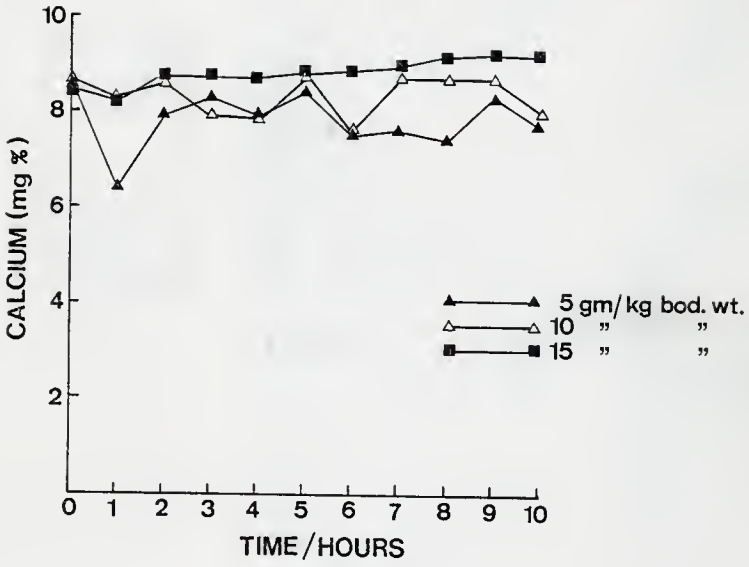

10
8
6
4
2
CALCIUM (mg %)
0 1 2 3 4 5 6 7 8 9 10
TIME/HOURS
5 gm/kg bod. wt.
10 " "
15 " "

Fig. # 20

Time course changes in serum calcium from intraruminal administration of various doses of D-galactose.

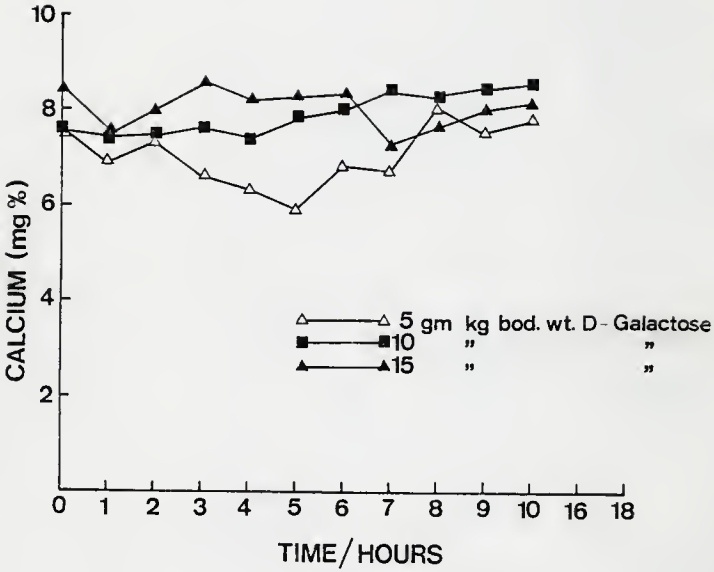

10
8
6
4
2
CALCIUM (mg %)
0 1 2 3 4 5 6 7 8 9 10 16 18
TIME/HOURS
5 gm kg bod. wt. D-Galactose
10 " "
15 " "

Fig. # 21

Time course changes of blood urea nitrogen (BUN) from intraruminal administration of acidotic doses of monosaccharides.

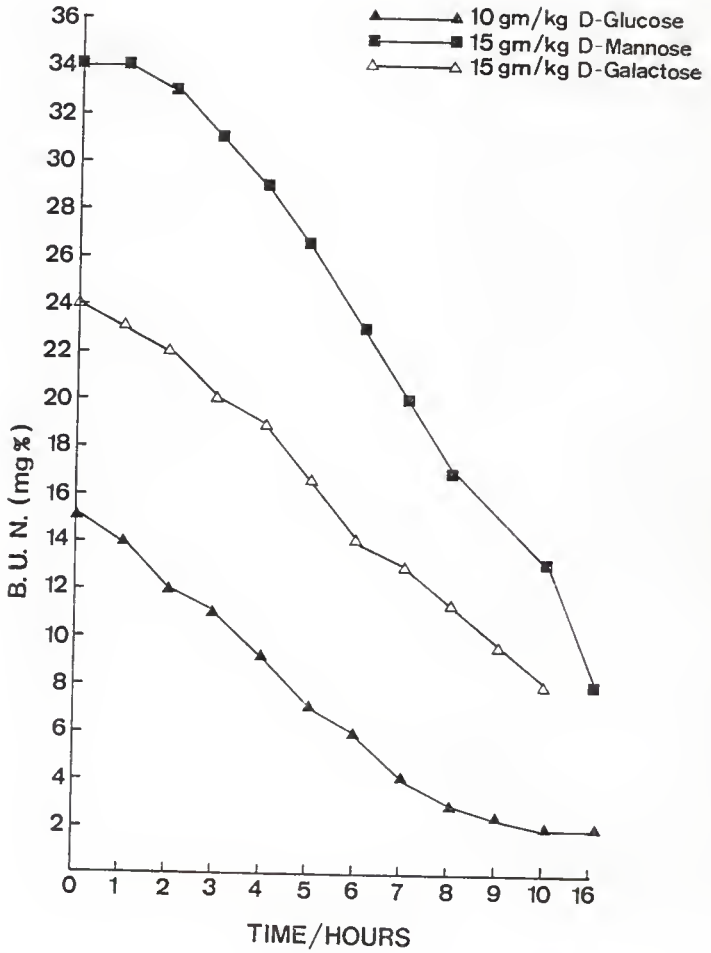

10 gm/kg D-Glucose
15 gm/kg D-Mannose
15 gm/kg D-Galactose
B. U. N. (mg %)
36
34
32
30
28
26
24
22
20
18
16
14
12
10
8
6
4
2
0 1 2 3 4 5 6 7 8 9 10 16
TIME/HOURS

Fig. # 22

Time course changes in serum sorbitol dehydrogenase (SDH) from intraruminal administration of various doses of D-glucose.

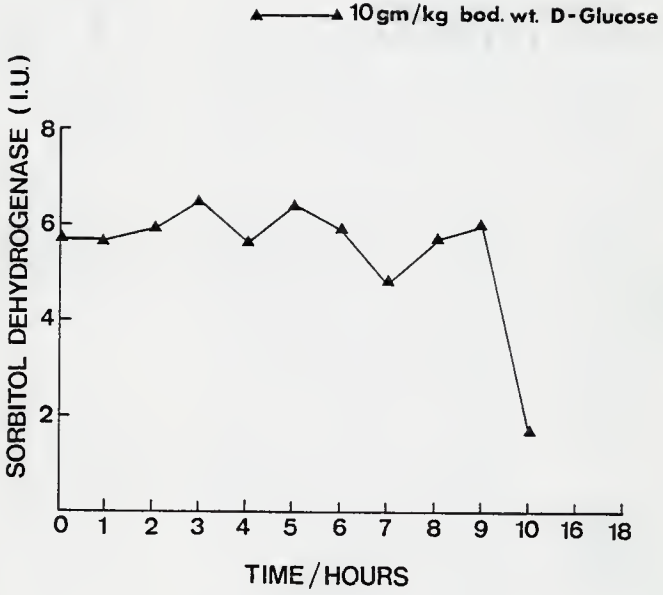

10 gm/kg bod. wt. D-Glucose
SORBITOL DEHYDROGENASE (I.U.)
8
6
4
2
0 1 2 3 4 5 6 7 8 9 10 16 18
TIME / HOURS

Fig. # 23

Time course changes in serum sorbitol dehydrogenase (SDH) from intraruminal administration of various doses of D-mannose.

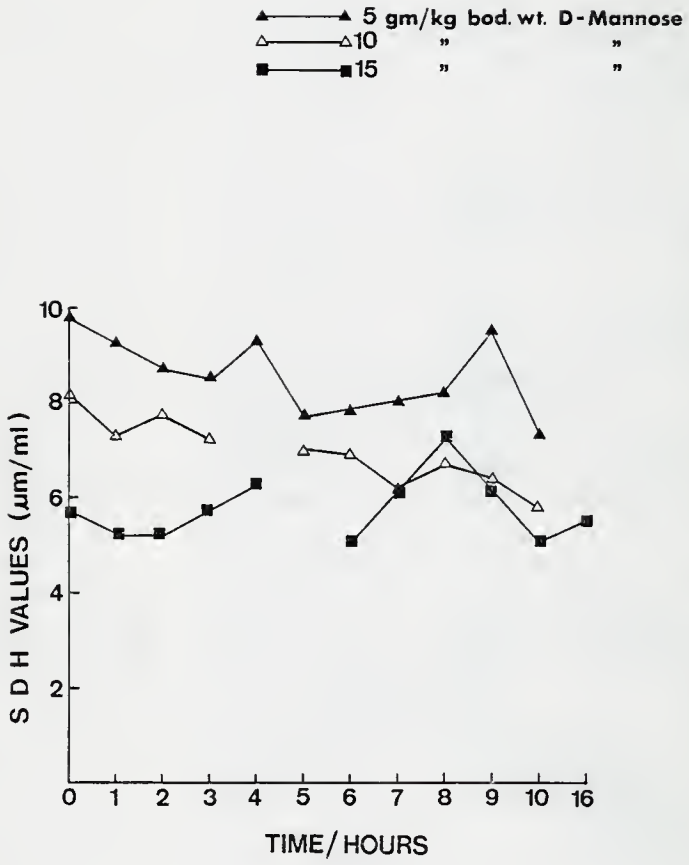
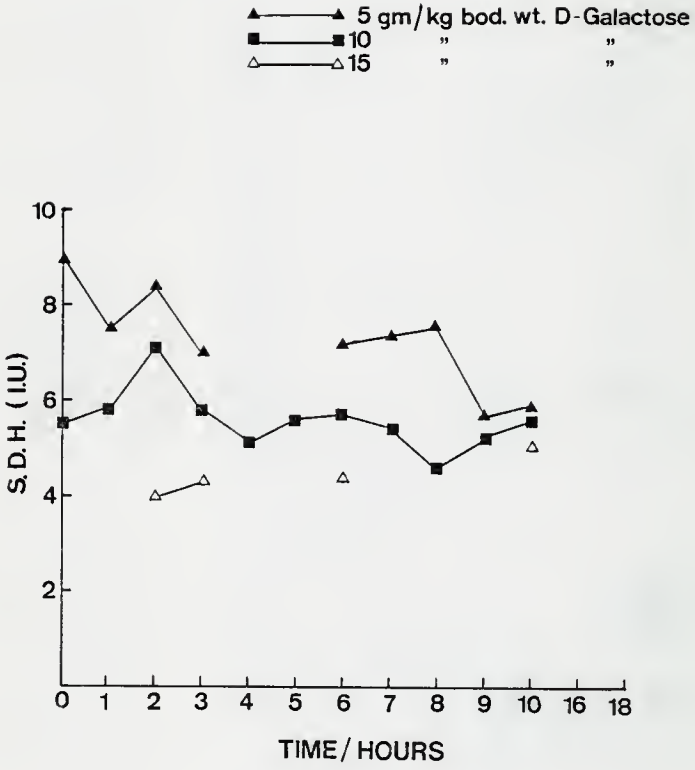

5 gm/kg bod. wt. D-Mannose
10 " "
15 " "
S D H VALUES (µm/ml)
10
8
6
4
2
0 1 2 3 4 5 6 7 8 9 10 16
TIME/HOURS

Fig. # 24

Time course changes in serum sorbitol dehydrogenase (SDH) from intraruminal administration of various doses of D-galactose.


5 gm/kg bod. wt. D-Galactose
10 " "
15 " "
S.D.H. (I.U.)
10
8
6
4
2
0 1 2 3 4 5 6 7 8 9 10 16 18
TIME/ HOURS

dosing. D(-) peak value was 20.6 mg % at 4 hrs post dosing. The lowest rumen pH recorded was 5.2. The clinical signs observed correlated with the peak blood D(-) lactate level. Peak blood glucose was 71 mg %. The level started to decrease throughout the rest of the observation period. The sudden addition of pure glucose to the rumen at such a high dose led to immediate production of D(-) lactic acid whose production is favoured by glucose accumulation and low pH. Substantial amount of rumen D(-) lactate was recorded at 5 gm/kg body weight but not much was absorbed into blood as seen in Table (1). There was high L(+) lactate level in rumen at 10 gm/kg. body weight whereas high blood L(+) lactate at 5 gm/kg. body weight for D-glucose. With the acidotic dose of D-mannose, rumen L(+) lactate was 49.5 mg % at 1 hr, 2.43 mg % at 4 hrs and 65.4 mg % at the 10th hour, the D(-) level rose gradually, reaching a peak value of 20.3 mg % at 5 hr. Lowest pH value recorded was 5.25. Blood L(+) rose steadily up to the 10th hour. The D(-) lactate reached ite peak value of 14.1 mg % at 6 hr post dosing. The blood pH fell steadily up to the 7th hr and rose again. There was no significant rise in blood glucose. Muscular twitchings and incoordination observed at about the 7th hour in this goat were related to the blood D(-) lactate level.

With the acidotic dose of D-galactose, rumen L(+) lactate did not exceed 4 mg %. The D(-) form rose steadily with a peak value of 16.8 mg % at 8 hr after dosing. The lowest pH value recorded was 5.25. Blood D(-) lactate rose steadily with a peak value of 12.6 mg %. The acidotic dose of D-galactose had the lowest level of blood D(-) lactic acid and highest level of blood glucose (Table 12).

Rumen osmolarity increased both postdosing and with increased doses of each monosaccharide.

The rumen fluid ammonia (Fig. 7a and Appendix I) decreased in all the doses of monosaccharides fed. There was a slight increase at 5 hrs and 7 hrs for 5 gm/kg bod. wt. and 15 gm/kg. bod. wt. of D-mannose respectively. For D-galactose the increase wae not noticed at 5 gm/kg dose but at 10 gm/kg and 15 gm/kg dosee. The increase for these dosee were at 7 hrs and 5 hrs respectively. For 10 gm/kg dose of D-glucose the increase was at 4 hrs post dosing. The decrease in ammonia levels were more marked with doses of D-glucose followed by D-galactose and D-mannose.

Values of blood parameters are recorded in Tables (1-3, 6-19), Figures 2, 4, 6, 7b, 8b, 9b, 10-24) and Appendix II-VIII. There was a general increase in blood ammonia (Fig. 7b and Appendix II) above predosing levels for all the doses of the three monosaccharides. The increase persisted with some doses, but there wae a general decrease for the remainder of the observation period, with some reaching predosing levels. The increase in blood ammonia observed did not correlate with rumen levels (Fig. 7a and Appendix I).

Blood pH (Table 6) was lower with acidotic dosage of each monosaccharide. The PCO_2 (Table 7), HCO_3^- (Table 8), PO_2 (Table 14), total carbon dioxide (Table 10) and serum carbon dioxide (Table 11) all decreased with all the doses of the monosaccharides. The decrease in PCO_2 was small and variable. The PCV (Table 7) increased with acidotic dosee of D-glucose and D-galactose and variable with acidotic dose of D-mannose.

The blood glucose levels (Table 12) were above normal range for all

the doses of all the three monosaccharides.

Serum total protein (Table 15) increased and albumin values (Table 16) were variable for acidotic doses except for the D-glucose which showed variable results for the albumin and decrease in total protein for D-galactose. All lower doses showed decrease in protein and normal levels for albumin values.

Serum creatinine (Fig. 13 & 14 and Appendix III) showed relatively constant levels throughout the observation period. With the acidotic doses, however, the results were variable, with normal values for D-galactose and D-mannose and a slight decrease for D-glucose. The decrease was observed 1 hr post dosing until the remaining observation period.

Blood urea nitrogen (BUN) (Fig. 21 and Appendix VII) decreased markedly for acidotic doses of D-glucose and D-galactose to below normal values and remained within normal for D-mannose.

Alkaline phosphatase (Fig. 10, 11 and 12 and Appendix VI), phosphorus (Fig. 15, 16 and 17 and Appendix IV) and calcium (Fig. 18, 19 & 20 and Appendix V) showed variable results. There was a small increase in calcium for acidotic dose of D-glucose between 1-2 hrs post dosing. No increase for acidotic dose of D-mannose from 1-10 hrs post dowing. Alkaline phosphatase showed an increase at 9 hrs with acidotic dose of D-glucose and remained normal for acidotic doses of D-mannose and slight increase for D-galactose. There was an increase in phosphorus levels with all the acidotic doses of the three monosaccharides.

The electrolytes also showed variable results. A slight increase in K for the acidotic doses of D-mannose and D-glucose and no increase

for acidotic dose of D-galactose. There was a slight increase above predosing level for Cl^- in acidotic doses of D-mannose and D-glucose. There was not sufficient data for acidotic doses of D-galactose. Sodium level increased slightly for acidotic dose of D-mannose and remained variable for D-glucose and D-galactose. Sorbitol dehydrogenase (Fig. 22, 23 and 24 and App. VIII) showed variable results. Serum glutamic pyruvic transaminase (SGPT) was demonstrated in the three goats given the doses of 15 gm/kg, 5 gm/kg and 15 gm/kg of D-mannose, D-glucose and D-galactose respectively. The rest of the doses did not show any blood SGPT.

Discussion

Monosaccharides are the substrates used in fermentative processes by the rumen microorganisms. Excessive consumption of monosaccharides may lead to excessive production of lactic acid leading to acidosis. The purpose of this experiment was to administer three sugars in increasing doses until a certain dose of each was reached to induce acidosis. The induction and severity of lactic acidosis was assessed by clinical signs and quantitative changes in both blood and rumen parameters measured.

The values of all the parameters measured at zero hour predosing have been assumed to be normal for the type of ration the goats were maintained on. Hence zero hour values served as controls for the changes observed when the monosaccharides were given. Acidosis caused muscular twitchings and incoordination and lameness, if the acidosis was severe. Lameness has been reported (82), (93), (99), (107). Incoordination has

been reported due to a potassium deficiency (114). The increased respiratory rate has been reported (59) due to compensatory mechanisms caused by the acid depletion of HCO_3^- and increased carbon dioxide, leading to increased PCO_2. The latter stimulates the respiratory center to increaee breathing in order to get rid off carbon dioxide by the lungs.

The distended rumens and rumen stasis have been reported (25) due to increased osmotic pressure resulting in water efflux into the rumen and reduced rumen motility respectively. Dehydration wae due to the movement of water from blood to rumen resulting in hemoconcentration. The decrease in rumen pH is consistent with that reported by earlier workers. Slyter (110) attributed the initial fall in pH to the production of large amounts of volatile fatty acids as well as lactic acid. The VFA have pKa of 4.8 and hence buffer the rumen at pH between 5 and 6. Lactic acid has a pKa of 3.8 and hence has a tenfold greater ionization than VFA, therefore causing the pH to fall as low as 4 in some cases. This is below range of pH that salivary buffers can cope with (13). The pH change reflected the level of lactate in the rumen. The same relationship has been observed by others (119), (107), (129), (4), (12), (25). Fall in rumen fluid pH has also been attributed to the poor b buffering capacity of the readily fermentable carbohydrates (115). The increase in rumen osmolarity observed in this experiment has been reported (43) and (60). According to Huber, lactic acid contributed about 60% of the increase in rumen osmolarity. The rest of the increase was due to other osmotically active constituents. The percentage contribution to increase in osmolarity in this experiment was not determined

since the purpose was to observe the pattern of change. Goats which were for the first time given the doses (5 /kg D-galactose, 5 g/kg of D-glucose and 15 gm/kg of D-mannose) showed higher levels of lactic acid than those given to goats in subsequent experiments, indicating some form of adaptation. D-glucose was more efficient in its ability to increase the lactic acid levels in the rumen. As the dose of each monosaccharide increased, the proportion of D(-) lactate to L(+) lactate became higher. The dilution factor was not accounted for when a liter of double distilled water was used to dissolve the monosaccharides. The presence of a discernable odor has been reported by others as vile smelling (114). Frothy bloat would have been initiated in the two goats given acidotic doses of D-mannose and D-glucose had it not been for the release of gas from the fistula at each sampling. The decrease in rumen ammonia could be due to several factors: a) Increased utilization by rumen microbes in the presence of available energy which was used to support more bacterial numbers, b) escape through fistula at each samplingtime and c) the fact that carbohydrates are not precursors of ammonia. If the decrease was mainly due to increased utilization, the results suggest that the microorganisms utilized D-glucose faster than D-mannose and D-galactose. But from the level of rumen fluid and blood lactic acids, the microorganisms utilized D-galactose more efficiently than D-glucose and D-mannose at acidotic doses.

Ammonia absorption is known to be inhibited at low pH because it is in the ionized form, hence one can expect higher rumen levels, but this wasn't the case. The significant drop between predosing levels and subsequent post dosing levels suggested also that some amount had been

released during sampling. The general increase between 4-8 hrs was true for all animals. The increases at the times indicated for each monosaccharide could correspond to the time at which maximum utilization of energy for bacterial need was reached or due to the pH at which rumen ammonia utilizing bacteria were inhibited. The increase in blood ammonia post dosing could be due to a combination of absorption at higher rumen pHs in the early part of the experiment as well as from endogenous production. Increased ammonia production by the kidney in acidosis can enter systemic blood. Urea in venous blood as well as glutamine could also give rise to ammonia. The subsequent decline observed at the later part of the observation period showed it was being utilized in the body or eliminated. Hills (57) said that defense of extracellular fluid against acidosis took precedence over nitrogen conservation. This defense was automatic and resulted from the control exerted by urinary pH over the passive partitioning of the renal ammonia supply between renal vein and urine. The role of ammonia in the body to conserve cations during acidosis in ruminants has been suggested (114).

The increased PCV observed has been reported (43), (66), (24), (129). The possible cause of the increase has been suggested to be due to plasma concentration due to haemoconcentration and release of red blood cells from the spleen (118), (71). Increased serum total protein has been reported (34).

The values of blood pH, PCO_2, total CO_2, serum CO_2 and bicarbonate showed not marked decrease. The decreases were directly proportional to the amount of lactic acid entering the blood. The standard acid base equation below explains the relationship of these parameters.

$H^+ + HCO_3^- \longrightarrow H_2CO_3 \longrightarrow H_2O + CO_2$. The H^+ represents protons available by acids entering the body or introduced into it. The pH of the system would rise and formation of additional bicarbonate would occur if protons were transferred to the bicarbonate. The carbon dioxide would rise and this would exert greater PCO_2. Carbon dioxide would then be free to diffuse off. Lactic acidosis is a metabolic acidosis since it involves the transfer to the extracellular fluid of acids other than carbonic acid. The rate and course of acidosis will depend on the entry rate of the acids. It was suggested (61) that if lactic acid could be introduced into the body in small amounts, compensatory mechanisms would prevent further decrease in pH and other acidosis consequences. So in lactic acidosis, bicarbonate, PCO_2, pH and carbon dioxide content would decrease and the compensatory mechanism would remove the carbon dioxide through hyperventilation. The results of this experiment showed that compensatory mechanisms have played their role.

The decreased PO_2 observed indicated some degree of hypoxia. As lactic acid entered the blood, the ratio of lactate to pyruvate would increase and would in turn increase requirement for oxygen by the tissues. If oxygen requirement was increased, then the level of oxygen in the blood would decrease. This would decrease the PO_2. The increased respiration could eventually compensate the decreased PO_2. Increased blood glucose has been reported (70), (33). The higher blood glucose reported for D-galactose could be due to two reasons: a) the increase could suggest reversed glycolysis. The slower fermentation rate of D-galactose gave more time to produce propionic acid. This acid was absorbed into the blood and gave rise to high blood glucose or

b) galactose could be absorbed directly and raised blood glucose through gluconeogenetic pathways. Duke gave normal blood glucose values to be in the range 45-60 mg % (40).

The drastic reduction in blood urea nitrogen contrasted the report by Dirkson (34) that blood urea increased during acidosis. The decrease could be due to its rapid conversion to ammonia or increased excretion by the kidney. The two alternatives could be used to increase elimination of hydrogen to help maintain normal pH.

The relatively constant value observed for serum creatinine could suggest little or no direct involvement. The slight decrease indicated its possible excretion along with hydrogen by or in the kidney.

The increase in serum calcium observed in this experiment has been reported also. Calcium balance has been documented (3), (10), (49), (53), (128) and consisted of the following: Urinary excretion, gastro intestinal absorption and bone mobilization. These three routes could be acted upon by parathyroid hormone, vit. D and calcitonin, metabolic acidosis could affect any of these routes by acting on the hormones involved. An increase in phosphorus during acidosis was also reported (70), (33). The control of phosphorus is affected by parathyroid hormone during bone resorption, intestinal absorption and kidney excretion. Lemann (80), Litzlow (81) and Relman (102) observed that metabolic acidosis of varied causes led to negative calcium balance which they postulated to be due to bone resorption. This postulate was supported by (14) and (27). The mechanism for bone resorption was not clear. Barzel (15) proposed that lowering the pH per se increased calcium mobilization from bone. The mechanism through which metabolic acidosis increases calcium

mobilization has been investigated in thyroparathyroidectomized rats (16) in which metabolic acidosis directly raised serum calcium concentration and augmented the effect of PTH on bone to raise the serum concentration.

Blood K^+, cl^- and Na^+

It is a well known clinical fact that when plasma K^+ is low, the plasma HCO_3^- is high and vice versa. In acidosis where there is a decrease in bicarbonate, the blood K^+ increases. The increased H^+ from lactic acid passes into the cells from extracellular fluid and K^+ passes out in place of the H^+ to maintain electrical neutrality, hence elevating the concentration of K^+ in ECF as observed in this experiment. This is an attempt to increase the bicarbonate concentration of the ECF in amount equivalent to that of the H^+ entering the cells. The results of the cl^- and Na^+ were variable.

The results of the sorbitol dehydrogenase (SDH) and serum glutamic pyruvic transaminase (SGPT) did not suggest any liver damage. The slight increase in one goat of SDH and the presence of SGPT in the other two goats could suggest possible increase in liver cell membrane permeability.

Conclusion

From the results, lower dose of D-glucose induced lactic acidosis whereas higher doses of D-mannose and D-galactose were required to induce lactic acidosis.

From the faster rates at which rumen fluid lactic acid was produced and rumen fluid ammonia was reduced, it could be concluded that the

microorganisms utilized glucose faster than D-galactose and D-mannose. Based on the amount of lactic acid produced in the rumen, the results showed low amount of lactic acid by D-galactose. This suggest that D-galactose was utilized more efficiently at higher doses. Under practical conditions, this can provide a guideline for feeding diets that contain naturally occurring polysaccharides composed of D-galactose, so lactic acidosis can be avoided.

The results showed decrease in PCO_2, HCO_3^- and pH and indicates that acid base balance has been directly involved during lactic acidosis. These changes were recorded from venous blood, indicating that venous blood is a good index for assessing lactic acidosis.

The results of SGPT and SDH and also AP indicates no liver damage. The degree of acidosis was not sever at these doses to have caused liver damage. There was a greater increase in blood glucose with doses of D-galactose than with D-mannose and D-glucose. The reason for this needs further investigation.

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ABSTRACT

Three goats weighing 40 kg each were given D-glucose, D-mannose and D-galactose intraruminally in increasing doses in separate experiments. The purpose was to arrive at a dose of each of the monosaccharides that could induce lactic acidosis. The induction and severity of lactic acidosis was assessed by the clinical signs and the quantitative changes in each of rumen fluid and blood parameters. It was found that 10 gm/kg body weight of D-glucose and 15 g/kg each of D-mannose and D-galactose induced lactic acidosis. The acidosis induced by D-galactose at 15 gm/kg was not as severe as that induced by D-glucose and D-mannose at the doses indicated. Clinical signs observed correlated with high blood D(-) lactic acid concentration above 5 mg%. Blood D(-) and L(+) lactic acids and phosphorus levels increased with all the acidotic doses of the three monosaccharides. There was an increase in Ca, K, PCV, total protein, glucose in two of the three goats. There was a decrease in blood and rumen pH, blood PO_2, PCO_2, HCO_3^- serum carbon dioxide and BUN in all the acidotic doses of the monosaccharides. Blood Na, alkaline phosphatase, albumin, creatinine SDH showed variable results in all the three goats.

APPENDIX

Appendix # I

Values of rumen ammonia - N (mg %) from intraruminal administration of various doses of monosaccharides.

Sampling time	D-mannose (gm/kg. bod. wt.)			D-glucose (gm/kg. bod. wt.)		D-galactose (gm/kg. bod. wt.)		
	5	10	15	5	10	5	10	15
0 hr	10.5		16.2	1.88	12.53	-	6.77	4.51
1 hr	3.8		13.5	1.13	5.42	0.09	7.79	9.4
2 hr	3.4		7.5	0.08	4.72	0.09	3.39	1.5
3 hr	3.0		3.4	-	2.37	0.09	1.35	1.5
4 hr	2.6		2.3	-	7.45	-	3.73	0.38
5 hr	6.8		0.08	-	0.07	0.09	2.03	3.01
6 hr	2.3		0.01	-	0.07	0.09	1.35	3.76
7 hr	1.9		2.3	-	0.07	0.09	5.08	0.08
8 hr	1.5		2.3	-	0.07	-	2.71	-
9 hr	1.5		1.5	-	0.07	0.85	4.06	-
10 hr	1.5		1.1	-	0.07	0.04	2.71	-

Appendix # II

Values of blood ammonia - N (mg %) from intraruminal administration of various doses of monosaccharides.

Sampling time	D-mannose (gm/kg. bod. wt.)			D-glucose (gm/kg. bod. wt.)		D-galactose (gm/kg. bod. wt.)		
	5	10	15	5	10	5	10	15
0 hr	0.04		0.015	0.14	0.66	0.3	0.075	0.08
1 hr	0.09		0.25	0.18	0.19	-	0.093	0.09
2 hr	0.06		0.15	-	0.13	0.2	0.081	0.1
3 hr	0.14		-	0.21	0.06	-	0.057	0.06
4 hr	0.13		0.15	0.36	0.23	-	0.065	0.01
5 hr	0.09		-	0.15	0.15	0.5	0.048	0.02
6 hr	0.06		0.113	0.24	0.15	0.22	0.038	0.03
7 hr	0.08		-	0.57	0.14	0.29	0.086	0.02
8 hr	0.06		0.10	0.51	0.15	0.19	0.048	0.06
9 hr	0.05		-	0.18	0.14	-	0.058	0.08
10 hr	0.05		0.09	0.09	0.13	-	0.048	0.06

Appendix # III

Values of serum creatinine (mg %) from intraruminal administration of various doses of monosaccharides.

Sampling time	D-mannose (gm/kg. bod. wt.)			D-glucose (gm/kg. bod. wt.)		D-galactose (gm/kg. bod. wt.)		
	5	10	15	5	10	5	10	15
0 hr	-	0.9	1.0	0.8	0.9	0.9	0.6	0.9
1 hr	-	0.9	1.0	0.7	0.7	0.9	0.6	0.9
2 hr	-	0.8	1.0	0.8	0.6	1.1	0.7	0.9
3 hr	-	1.1	-	0.7	0.8	0.9	0.7	1.0
4 hr	-	0.9	1.0	-	0.6	0.9	0.8	0.9
5 hr	-	1.0	-	0.7	0.6	1.1	0.7	-
6 hr	-	1.0	1.1	0.8	0.7	1.0	0.6	0.9
7 hr	-	1.0	-	0.7	0.7	1.0	0.7	0.9
8 hr	-	0.9	1.0	0.7	0.7	1.0	0.7	-
9 hr	-	0.9	-	0.8	0.7	0.9	0.7	0.9
10 hr	-	0.8	1.0	0.8	0.7	1.0	0.8	0.9

Appendix # IV

Values of serum phosphorus (mg %) from intraruminal administration of monosaccharides.

Sampling time	D-mannose (gm/kg. bod. wt.)			D-glucose (gm/kg. bod. wt.)		D-galactose (gm/kg. bod. wt.)		
	5	10	15	5	10	5	10	15
0 hr	6.6	4.3	6.5	4.8	5.2	5.6	4.6	5.3
1 hr	7.2	3.6	6.0	6.5	6.2	4.9	5.4	6.3
2 hr	8.1	5.1	6.2	6.3	6.9	5.4	5.9	6.3
3 hr	7.8	6.1	6.9	6.2	7.2	5.0	6.2	6.4
4 hr	7.2	7.8	7.1	-	7.3	4.6	5.9	6.6
5 hr	7.5	8.3	7.1	6.3	7.4	5.5	5.6	-
6 hr	5.1	8.6	7.2	6.0	6.8	5.5	5.4	8.1
7 hr	5.5	9.0	7.4	5.8	6.5	4.8	5.8	7.4
8 hr	4.2	9.0	7.3	5.9	6.1	5.5	5.0	-
9 hr	7.5	9.6	7.1	5.9	5.8	4.5	4.4	6.4
10 hr	4.8	7.8	7.1	5.9	5.8	3.7	4.2	6.8

Appendix # V

Values of serum calcium (mg %) from intraruminal administration of various doses of monosaccharides.

Sampling time	D-mannose (gm/kg. bod. wt.)			D-glucose (gm/kg. bod. wt.)		D-galactose (gm/kg. bod. wt.)		
	5	10	15	5	10	5	10	15
0 hr	8.6	8.6	7.8	6.6	8.4	7.6	7.6	8.4
1 hr	6.8	8.3	8.1	6.0	8.6	6.9	7.4	7.5
2 hr	7.9	8.6	8.1	5.7	9.0	7.3	7.4	8.0
3 hr	8.3	7.9	8.3	5.1	9.0	6.6	7.6	8.5
4 hr	7.9	7.9	8.3	-	8.3	6.3	7.4	8.2
5 hr	8.3	8.3	8.0	6.0	7.9	5.9	7.8	-
6 hr	7.5	7.5	8.3	6.2	7.9	6.8	8.0	8.3
7 hr	7.6	8.7	8.4	6.4	7.7	6.7	8.4	7.2
8 hr	7.4	8.7	8.3	6.0	7.6	8.0	8.2	-
9 hr	8.3	8.7	8.3	6.1	7.6	7.5	8.4	8.0
10 hr	7.7	8.0	8.3	6.8	7.5	7.8	8.5	8.1

Appendix # VI

Values of alkaline phosphatase (um/ml) from intraruminal administration of various doses of monosaccharides.

Sampling time	D-mannose (gm/kg. bod. wt.)			D-glucose (gm/kg. bod. wt.)		D-galactose (gm/kg. bod. wt.)		
	5	10	15	5	10	5	10	15
0 hr	45	39	49	-	37	53	48	51
1 hr	-	36	48	-	32	48	49	49
2 hr	39	38	50	-	33	42	47	47
3 hr	39	-	-	-	34	42	47	50
4 hr	38	35	48	-	32	-	48	48
5 hr	38	38	-	-	32	-	48	-
6 hr	-	35	49	-	32	42	48	52
7 hr	46	45	-	-	31	-	48	53
8 hr	45	35	51	-	32	40	47	-
9 hr	33	32	-	-	32	37	47	49
10 hr	42	33	49	-	35	38	50	49

Appendix # VII

Values of blood urea nitrogen (B.U.N.) from intraruminal administration of various doses of monosaccharides.

Sampling time	D-mannose (gm/kg. bod. wt.)			D-glucose (gm/kg. bod. wt.)		D-galactose (gm/kg. bod. wt.)		
	5	10	15	5	10	5	10	15
0 hr	27	20	34	-	15	26	25	24
1 hr	-	20	34	-	14	28	26	23
2 hr	26	19	33	-	12	27	26	22
3 hr	24	-	-	-	11	27	26	20
4 hr	23	17	29	-	9	-	25	19
5 hr	21	16	-	-	7	-	22	-
6 hr	-	15	23	-	6	22	18	14
7 hr	19	13	-	-	4	-	16	13
8 hr	18	13	17	-	3	18	13	22
9 hr	6	11	-	-	2	16	12	8
10 hr	18	9	13	-	2	16	10	-

Appendix # VIII

Values of serum sorbitol dehydrogenase (SDH) from intraruminal administration of various doses of monosaccharides.

Sampling time	D-mannose (gm/kg. bod. wt.)			D-glucose (gm/kg. bod. wt.)		D-galactose (gm/kg. bod. wt.)		
	5	10	15	5	10	5	10	15
0 hr	9.8	8.2	5.7	-	5.7	9.0	5.5	-
1 hr	7.3	-	5.2	-	5.7	7.0	5.8	-
2 hr	7.7	8.7	5.2	-	5.9	8.4	7.1	4.0
3 hr	7.2	8.5	-	-	6.5	7.0	5.8	4.3
4 hr	-	9.3	6.3	-	5.6	-	5.1	-
5 hr	7.0	7.7	-	-	6.4	-	5.5	-
6 hr	6.9	-	5.0	-	5.9	7.2	5.6	4.4
7 hr	6.2	8.0	-	-	4.8	-	5.4	-
8 hr	6.7	8.2	7.3	-	5.7	7.5	4.6	-
9 hr	6.4	9.5	-	3.6	6.0	5.7	5.2	-
10 hr	5.8	7.3	5.1	-	1.7	5.9	5.6	5.0

THE INFLUENCE OF MONOSACCHARIDE TYPES (D-GLUCOSE, D-MANNOSE AND D-GALACTOSE) IN INDUCING LACTIC ACIDOSIS IN GOATS

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ABSTRACT

Three goats weighing 40 kg each were given D-glucose, D-mannose and D-galactose intraruminally in increasing doses in separate experiments. The purpose was to arrive at a dose of each of the monosaccharides that could induce lactic acidosis. The induction and severity of lactic acidosis was assessed by the clinical signs and the quantitative changes in each of rumen fluid and blood parameters. It was found that 10 gm/kg body weight of D-glucose and 15 g/kg each of D-mannose and D-galactose induced lactic acidosis. The acidosis induced by D-galactose at 15 gm/kg was not as severe as that induced by D-glucose and D-mannose at the doses indicated. Clinical signs observed correlated with high blood D(-) lactic acid concentration above 5 mg%. Blood D(-) and L(+) lactic acids and phosphorus levels increased with all the acidotic doses of the three monosaccharides. There was an increase in Ca, K, PCV, total protein, glucose in two of the three goats. There was a decrease in blood and rumen pH, blood PO_2, PCO_2, HCO_3^- serum carbon dioxide and BUN in all the acidotic doses of the monosaccharides. Blood Na, alkaline phosphatase, albumin, creatinine SDH showed variable results in all the three goats.