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SALINOMYCIN EFFECT ON THE PREVENTION OF RUMINAL LACTIC ACIDOSIS IN SHEEP

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ABSTRACT

The objective of this study was to assess the effectiveness of salinomycin against the lactic acidosis induced in sheep, by analyzing its effects on the clinical picture, and the physico-chemical characteristics of the ruminal fluid. We used 14 crossbred Santa Ines sheep, weighing 30 Kg. They were rumen-fistulated and subdivided into two groups of 7 animals each: the control group and the group that received the drug in the diet at a concentration of 30 mg/Kg of food for 42 days. We established the clinical and laboratory values of the ruminal samples during this phase of the experiment. At the end of the adaptation period, both groups were challenged in a process of sucrose-induced lactic acidosis, at a dose of 10 g/Kg body weight. The clinical and laboratory observations were accomplished at intervals of 4h, 8h, 12h, 16h, 24h, 32h e 48h post-induction (PI). Control and treated sheep showed clinical signs of ruminal acidosis within eight hours after induction, associated with laboratory alterations with varied intensity between the studied groups. The magnitude of the process was minimized and the time of clinical recovery was shortened in the animals that received salinomycin in relation to the control group.

KEYWORDS: fermentation disorder; ionophores; small ruminants.

EFEITO DA SALINOMICINA NA PREVENÇÃO DA ACIDOSE LÁCTICA RUMINAL EXPERIMENTAL EM OVINOS

RESUMO

Este trabalho teve por objetivo estudar a eficácia da salinomicina na acidose láctica induzida em ovinos, analisando os seus efeitos sobre o quadro clínico e as características físico-químicas do fluido ruminal. Para tal, foram utilizados 14 animais ovinos da raça Santa Inês, com peso médio de 30 Kg, fistulados, subdivididos em dois grupos de 07 animais, sendo um o controle e o outro o que recebeu o antibiótico, na concentração de 30 mg/Kg ao dia na dieta, durante 42 dias. Nessa etapa, os padrões clínicos e laboratoriais das amostras ruminais foram estabelecidos. Ao final do período de adaptação, os dois grupos foram desafiados a um processo de acidose láctica induzida com sacarose, na dose de 10 g/ Kg de peso vivo. As observações clínicas e laboratoriais foram realizadas nos intervalos de 4h, 8h, 12h, 16h, 24h 32h e 48h pós indução (PI). Os ovinos do grupo controle e os que receberam o ionóforo apresentaram manifestações clínicas da acidose lática ruminal 8 horas após a indução, associadas às alterações laboratoriais, com intensidade variada entre os grupos estudados. Nos

animais que receberam a salinomicina, verificou-se que a magnitude do processo foi minimizada e, com isso, abreviou-se o tempo de recuperação clínica em relação ao grupo controle.

PALAVRAS-CHAVE: Distúrbios fermentativos; ionóforos; pequenos ruminantes.

INTRODUCTION

The national sheep industry has grown in recent years, due to the significant increase in agriculture and livestock production, where large investments are being applied. The sheep herd in Brazil is estimated at 17,662,201 animals, of which 10,110,352 are in the Northeast (IBGE, 2012). The economic and social impact that this type of activity represents for the region, especially in meat production, is of great importance.

Despite of the sheep industry growth, there are some critical factors to its operation, as inadequacies in the management, breeding, health, nutrition, among others. Regarding nutrition, the need for an intensive production model, in order to obtain short-term excessive weight gain, has changed eating habits, which can cause digestive and metabolic disorders related to the different types of diets used. Among them, we highlight the ruminal lactic acidosis, which is an obstacle for sheep industry, limiting growth due to economic losses (MACKIE et al., 1978; BARROS et al., 1999; LEAN et al., 2007).

Lactic acidosis is a metabolic disorder whose evolution, in most cases, is acute, caused by sudden ingestion of grain or other carbohydrate-rich foods, highly fermentable in large quantities. The disease is characterized by appetite loss, depression and death. It is also known for ruminal overload, acute indigestion, acute compression of the rumen and indigestion for carbohydrates (AFONSO et al., 2000; MIRANDA NETO et al., 2005; WALKER, 2006).

Several published papers focus on the practice of preventive measures for the control of rumen acidosis, varying from providing adequate diet to the use of buffering and antibiotics, including ionophores; however, there are few reports of its use in animals (McGUFFEY et al., 2001; AFONSO et al., 2002a; MIRANDA NETO et al., 2011). Therefore, the purpose of this study was to evaluate clinical and laboratory efficacy of the use of salinomycin in prevention of ruminal lactic acidosis induced in sheep.

MATERIAL AND METHODS

The work was conducted at Bovine Clinic - UFRPE, from April to September 2007. We used 14 male and female, clinically healthy, Santa Inês sheep, with average weight of 30 kg. The animals underwent surgery for implantation of permanent ruminal cannulas according to the technique described by MUZZI et al. (2009). We determined a post-operative interval of four weeks for complete recovery and adaptation of animals. During this period and throughout the experimental phase, the sheep were fed diet based on soybean meal (150 g per animal), offered twice a day at 8:00 a.m. and 4:00 p.m., as well as elephant grass *(Pennisetum purpureum)* and Tifton *(Cynodom sp),* common salt and mineral water *ad libitum*.

We divided the animals into two groups of seven animals: one control group and another that received salinomycin[1] directly into the rumen via the fistula, at a daily dose of 30 mg/kg of diet per animal during 42 days (MERCHEN & BERGER, 1985). After recovery from surgery, two days before induction, we evaluated the clinical parameters and collected ruminal fluid of the animals. We performed the physical examination and sample collection for laboratory tests, in order to establish mean values for the physiological pattern (control moment - 0h) for the variables studied. After the initial adaptation period, we maintained antibiotic application and induced acidosis by providing 10g sucrose / kg body weight as substrate through the ruminal fistula, at 8:00 a.m., before the morning feeding (DELAK & ADAMIC, 1959). Clinical observations during the experiment and the collection of rumen fluid samples were carried out at intervals of 4h, 8h, 12h, 16h, 24h, 32h, and 48h post-induction (PI), to observe the emergence of clinical and laboratory changes indicative of lactic acidosis, according to the recommendations by KEZAR & CHURCH (1979) and RADOSTITS et al. (2007).

We collected the ruminal content samples via a vacuum suction pump coupled to a flexible plastic tube inserted through the rumen cannula. We collected approximately 200 ml of ruminal fluid from each sample, in glass flasks, to be processed in the laboratory. The tests were started no more than 15 minutes after collection.

We performed the analysis of the physical aspects – color, odor, consistency, and pH – as well

[1] Salocin 120 – Intervet

as proof of reduction of methylene blue (RMB), titratable acidity (TA) and chloride content tests according to the techniques described by VIEIRA et al. (2007).

The observation of the percentage of living animals, density and motility of infusoria and evaluation of the bacterial flora were made by direct examination on slides, using the model proposed by DIRKSEN (1993). For the infusoria count, we used the technique recommended by DEHORITY (1977).

We analyzed the values obtained throughout time, comparing the effects of the seven PI moments with control time, and between groups. We used the variables – heart rate, respiratory rate, rectal temperature and chloride content of rumen fluid –, the statistical method of analysis of variance, contrasting the means by Tukey's method and considering the calculated F-statistics significant at 5%. For the analysis of the variables – percentage of living protozoa, counting, density, motility, pH, proof of methylene blue reduction and total acidity of the ruminal fluid –, we obtained the median as a measure of central tendency and used Friedman' s nonparametric analytical method for dependent samples and Mann Whitney' s test for independent samples, using X^2 and calculating the dms for alpha equal to 0.05, in order to determine whether there were significant differences between values (SAMPAIO, 2010).

RESULTS

The administration of 10g of sucrose per kg of body weight through the rumen caused a mild rumen acidosis in sheep, triggering clinical manifestations varying in intensity. During physical examination, we observed signs of loss of appetite, anorexia, lethargy, tachycardia (Figure 1), abdominal distension, hypomotility and/or rumen atony, and lack of rumination. Feces did not become diarrheic, but presented grouped to pasty aspect. We observed variation in the degree of dehydration, but only in some animals, which is not clinically relevant. We did not verify cases of bloat. These signals become evident from 8h PI, however, in most sheep, we obsereved the recovery to clinical picture from 32h PI. There was no statistical difference (P> 0.05) within the groups or between them.

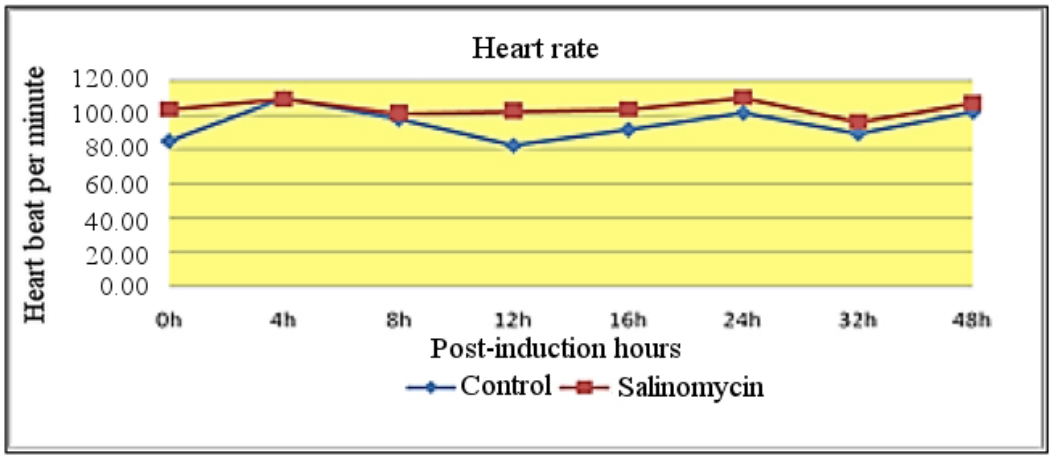

Figure 1. Mean values of heart rate (bpm) of sheep in control and salinomycin groups, with rumen lactic acidosis induced by sucrose (10g/kg body weight), at the analyzed moments.

Changes in color, odor and consistency of the ruminal fluid of sheep with rumen acidosis began from 4h PI and showed greater intensity at 8h PI.

The color changed from olive green or brown to yellow, and remained altered until 16 PI. Thereafter, the fluid began to return to normal color, and was reestablished in the control group at 32h PI. The recovery time of this variable was shorter in the salinomycin group, 24h PI for most of the animals.

The aromatic odor first became sweet, and then acid between 8h and 16h PI in the salinomycin group, and it was still altered at 32h PI in most animals of the control group. Full recovery was seen only 48h PI. It is noteworthy that we detected putrid odor only in one animal of the control group at 16h PI.

The consistency was also altered, becoming watery, between 8h and 16h PI. However, the recovery was observed from 24 hours PI in both groups.

The induction caused a progressive pH reduction in the ruminal fluid in both control and

salinomycin groups, mainly at 8 PI; we found values of 6.06 and 6.07, respectively, with significant difference ($P < 0.05$) compared to control time (0 h). By the evolution of the fermentation process, we verified that the pH values rose, reaching values close to normal. We observed no significant differences ($P > 0.05$) between groups at different times of observation. However, we found that the animals of salinomycin group presented earlier recovery, at the moments following observation, compared to control group (Figure 2).

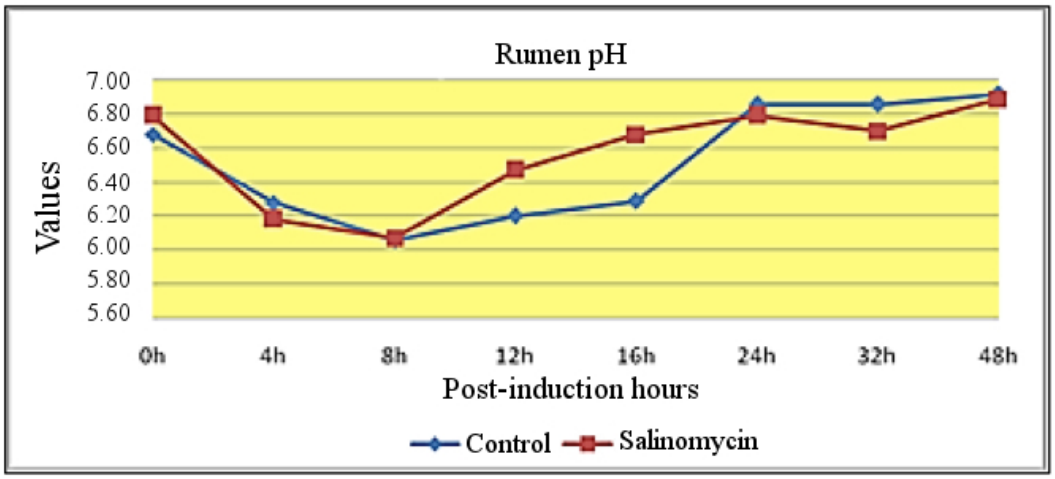

Figure 2. Median values of the pH of the rumen fluid of sheep in control and salinomycin groups with rumen lactic acidosis induced by sucrose (10g/kg body weight), at the analyzed moments.

Rumen acidosis caused changes in the values of the titratable acidity of the ruminal fluid in relation to the values at 0h and, during this clinical manifestation, a significant increase ($P < 0.05$) in this variable occurred from 4h PI, reaching the maximum value of 48° UC at 4h PI in the salinomycin group and at 8h PI in the control group (Figure 3). We did not verify significant differences ($P > 0.05$) between groups for this variable along the analyzed moments.

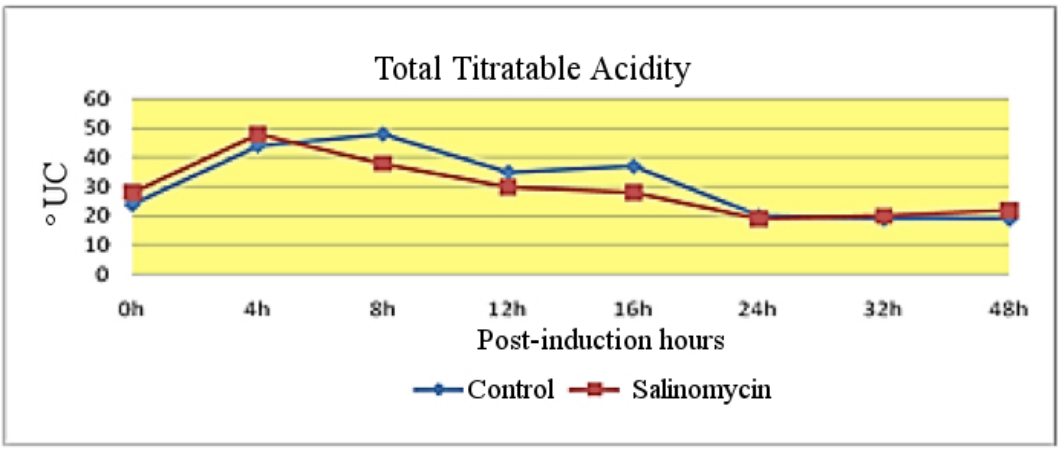

Figure 3. Median values of the total titratable acidity of the ruminal fluid of sheep in control and salinomycin groups with rumen lactic acidosis induced by sucrose (10g/kg body weight), at the analyzed moments

The chloride content in the ruminal fluid of sheep with lactic acidosis decreased throughout the observation period. We found out the lowest values of 17.78 and 19.08 mEq / L 12h PI for salinomycin

and control groups, respectively. There was no significant difference (P> 0.05) between groups at different times of observation. At the end, this variable values returned to the baseline in both groups.

The values obtained in RMB revealed, after ruminal lactic acidosis induction, an increase in the reduction time of the test, showing that it was not significant in the salinomycin group at 8h PI and in the control group at 16h PI, compared to the control time (0h). However, we observed that the recovery of the bacterial flora activity occurred earlier in animals that received salinomycin than in the control group (from 12h IP).

After acidosis induction, we found quantitative change in the count of infusoria in ruminal fluid of sheep. We verified the occurrence of a significant decline (P <0.05) in the number of infusoria at the first moment, at 4h PI, in the control group, which remained throughout the observation, when compared to the initial time (0h), whereas salinomycin group showed significant changes (P <0.05) between 12h and 16h PI compared to the initial moment. The comparison between the two groups at different times of observation showed no significant difference (P> .05). Before induction, all animals presented a higher prevalence of small infusoria, around 60%, and the remainder was made up by medium and large infusoria. It is noteworthy that, during acidosis phase, the infusoria population showed some alterations, with a predominance of small infusoria (80%), which is more sensitive to changes in the rumen environment, compared to medium and large ones.

The results observed in the percentage of living infusoria showed more accentuated decrease at 8h and 12h PI, with no viable infusoria in any group, and with significant changes (P <0.05) throughout the observation time, compared to control time (0h). There was no significant difference (P> 0.05) between groups at different times of observation. However, there was gradual recovery of this feature in rumen fluid from 16h PI in the salinomycin group compared to control, which presented infusoria gradual restoration from 48h PI.

After acidosis induction, there was a significant decrease (P <0.05) in the micro-fauna density, which reached minimum values from 8h PI until the last moment of observation in the control group and from 8h until 12h in the salinomycin group. It is noteworthy that there was no absence of infusoria, which were only scarce, with the density considered as one. There was no significant difference (P> 0.05) between groups at different times of observation.

With the onset of the fermentative disorder, we observed a decrease in motility, with significant differences (P <0.05) at all times in the control group when compared to the 0h time; however, salinomycin group showed significant difference (P < 0.05) only at 8h PI. There was no difference (P> 0.05) between the groups over time.

Although the induction caused a clinical picture of mild acidosis, it changed the morpho-tinctorial pattern of the bacterial population of the ruminal fluid in animals of both groups, and the most evident change in the salinomycin group was the predominance of Gram-positive bacteria (60-70%) at 8h IP, when we observed cocci and coccobacilli structures, while in the control group this manifestation was more intense and longer, observed from 8h to 16h PI. By the end of the observation period, both groups showed recovery of the patterns compared to the control time (0h).

DISCUSSION

We observed that the clinical signs in the animals with induced acidosis in this experiment were very similar to those described in other models, where different kinds of substrates and animal species were used. The typical clinical findings of rumen lactic acidosis occurred at some moments, but in general, the animals' appetite reduced with the decrease in ruminal dynamics, dehydration, polyuria, and mild tachycardia in a few animals; feces changed its appearance to pasty; there was frequent water intake, and mild or absent abdominal distension; in other words, lactic acidosis occurred with little intensity. Such manifestations coincided with the decrease in pH of the rumen fluid, especially when values were at their lowest point, which was also observed by MUIR et al. (1980), CRICHLOW (1989), OWENS et al. (1998), METKARI et al. (2001), MIRANDA NETO et al. (2005), NAGARAJA & LECHTENBERG (2007), ALMEIDA et al. (2008) and COMMUN et al. (2009), who reported the decrease due to the increase in lactic acid concentration and ruminal osmolarity of the medium in relation to the bloodstream, triggering the described clinical alterations.

Regarding appetite, the control group showed a greater number of animals with decreased and/or absent appetite from 8h to 32h PI, while in salinomycin group the appetite was impared in fewer animals and only between 12h and 32h PI. This demonstrates the favorable aspect of the use of salinomycin in the treated group in relation to the deleterious effects of lactic acidosis when compared to animals that did not receive the ionophore, agreeing with reports by KEZAR & CHURCH

(1979), AFONSO et al. (2002b), MIRANDA NETO et al. (2005) and LEAN et al. (2007), who correlated the recovery of normal intake of foods with a favorable rumen environment, where pH is greater than six, the level of lactic acid is not detectable or its concentration is low, and the concentrations of volatile fatty acids (AGV) present values above 50 mM.

As for the time of clinical recovery, the animals of the ionophore group began returning to normal at an earlier time, when compared to the control group that required a longer period of time to recover, similar to the observations by BEED & FARLIN (1977), AFONSO et al. (2000) and MIRANDA NETO et al. (2011), except for the time interval from the onset of clinical manifestations regarding the maximum peaks of low rates of ruminal pH and lactate after induction, probably due to the experimental model, variation of the species used, and different types of induction.

We observed accentuated changes in the physical characteristics of ruminal fluid during rumen acidosis, such as color, becoming a little milky to milky, aqueous consistency and slightly sweetish to acid odor. These findings are consistent with those reported by some authors that relate the changes with the decrease in pH in the rumen, caused by excessive rise in the concentration of lactic acid and VFA, which increases the osmolarity of the medium, making it hypertonic in relation to plasma, causing greater flow of water from the intracellular and extracellular compartments into the digestive tract, especially the rumen (JUHÁSZ & SZEGEDI, 1968; DOUGHERTY et al., 1975; BRAUN et al., 2010). These changes began from 8h PI and were similar to the manifestations observed in goats and sheep with rumen acidosis studied by HUBER (1971), CAO et al. (1987), LEAN et al. (2007) and ALMEIDA et al. (2008). The restoration of these characteristics followed the recovery of pH values prior to induction.

As for the decrease in rumen pH values at the beginning of the process, due to the administration of sucrose and its rapid fermentation, JUHÁSZ & SZEGEDI (1968), OWENS et al. (1998) and BROSSARD et al. (2003) attributed these findings to changes in the rumen micro-flora, wherein the Gram-negative bacteria, sensitive to the acidity of the medium, are replaced by Gram-positive bacteria, particularly *S. bovis* and *Lactobacillus sp.*, which are the main producers of lactic acid, under the forms of L (+) and D (-), which is considered a strong acid, by having a very low p*K*a .

Although there was no significant difference between the groups, we verified that the salinomycin group had earlier recovery compared with the control group, as also reported by NAGARAJA et al. (1981) and NAGARAJA et al. (1982), who used lasalocid or monensin to prevent lactic acidosis and observed a decrease in the AGV as well as in lactate D (-) and L (+), raising the pH of the medium, which was the reason for its prevention, justifying the action by the effectiveness of the antibiotic on the population of Gram-positive bacteria, producing lactic acid, and favoring Gram-negative flora (GOAD et al., 1998; McGUFFEY et al., 2001; GUO et al. 2010). By improving the rumen environment, especially for pH, and thus favoring the return of appetite, buffering and restoration of the microbial population were also improved, facilitating clinical recovery.

The total acidity values were high in induced animals, according to the findings of DIRKSEN (1993), who reported normal rates ranging from 8 to 26 °UC, and values reaching 70 or more units in case of rumen acidosis, depending on the degree of hyperacidity of the medium. This change reflects the reduction in rumen pH at the first few moments due to the increased level of lactic acid present in the ruminal fluid (AFONSO et al., 2002b). Over time, pH increased, most likely due to decrease in lactate level, faster in the group of sheep treated with the antibiotic, than in the control group. This is due to the ability of ionophore compounds to modulate acidosis effects and change rumen fermentation pattern, reducing the damage caused by the intensity and duration of low rumen pH, and selectively inhibit Gram-positive bacteria in the rumen, which are greater lactic acid producers (NAGARAJA et al. 1981; McGUFFEY et al. 2001; BUTAYE et al. 2003).

Changes in chloride content occurred in the early stages, probably due to the decrease in pH of the ruminal fluid, noticed from the administration of sucrose. We observed this decrease after 8h PI, coinciding with the observations by HUBER (1971) in sheep and CAO et al. (1987), MIRANDA NETO et al. (2005) and MIRANDA NETO et al. (2011) in goats, with acidosis induced by different substrates. According to these authors, this change is due to increased osmotic gradient, which led to the withdrawal of fluid from the bloodstream into the rumen, causing an exacerbated dilution of the ruminal fluid and, therefore, reducing the concentration of this ion. However, during the experiment, we observed that the chlorides concentration underwent elevation, which is probably related to the lower volume of liquid compared to the dry matter of rumen contents of the animals that showed clinical signs of diarrhea and dehydration. A complicating factor in this occurrence, according to OWENS et al. (1998), is

that high osmolarity of the ruminal fluid during acidosis causes abomasal hypertonicity, also making it distended, reducing the transit of the bolus and hindering the removal of fluid and acids from the rumen. The reflux of abomasal contents, due to its inertia, has been mentioned by BRAUN et al. (1992) as the cause of the increase in chloride concentrations (> 25mmol/l) in ruminal fluid, in 42% of sheep and goats diagnosed with acute ruminal acidosis.

The impairing of the ruminal flora, observed at the reduction of methylene blue test in some animals during the manifestation of acidosis was similar to the report by BASAK et al. (1993) and QUIROZ-ROCHA & BOUDA (2000), justifying this change by the inactivation of the normal flora, whose metabolism is impaired when environmental conditions are adverse. The restoration of the rumen environment in sheep was again crucial for this variable to return to the normal values initially established, which occurred earlier in the salinomycin group. AFONSO et al. (2002b) described a condition that contributes to this process, where the use of monensin inhibits most lactate-producing bacteria of the rumen, but not fermentative bacteria.

Changes in microbial fauna of the ruminal fluid of sheep regarding decreased viability, density, motility and count of infusoria occurred at different times, with varying intensity, showing a greater degree of recovery in the salinomycin group. These changes were reported by AFONSO et al. (2002b), who found, in a study with sheep, that the protozoa population decreased extensively with the increase in acidity in the rumen, leading to defaunation, differing from our findings in relation to the intensity of the fermentation process. According to HUNGATE et al. (1952), protozoa lose their activity when the pH drops to values between 5.5 and 5.0, disintegrating or suffering lysis in the rumen when acidity of the medium increases and the pH reaches values lower than 5.0. AHUJA et al. (1990) also reported that an increase in the osmotic pressure in the medium causes changes in rumen protozoa population. At the end of the experiment, we found that in some of the sheep that received salinomycin, the fauna reappeared and its functions were restored, which is consistent with the findings by BASAK et al. (1993), who reported this event synchronized with the improvement of the rumen pH.

As we observed, the qualitative change in the morfotinctorial pattern of the bacterial population was due to the decrease in pH. Thus, the predominantly Gram-negative flora was partially replaced for some moments by Gram-positive bacteria, as reported by OWENS et al. (1998) and MIRANDA NETO et al. (2005), who stated that, with the progress of the disease, there is a change in the microbial population characterized by rapid growth of lactic acid-producing bacteria, which accumulates, reducing ruminal pH to critical values (pH < 5.0). In this environment, the concentration and activity of many physiologically important bacteria are greatly reduced, causing a predominance of Gram-positive bacteria over Gram-negative.

Return to normal microbial population in the ruminal fluid occurred gradually with subsequent growth of Gram-negative flora, especially small cocos, which prevailed in the medium. These changes occurred earlier in the group that received the ionophore and this is probably due to the fact that salinomycin exerts an inhibitory effect on Gram-positive bacteria, which is consistent with the time interval where the production of lactic acid decreased and pH increased in the ruminal fluid (NAGARAJA et al. 1982; NAGARAJA et al. 1985; AFONSO et al. 2002b).

CONCLUSIONS

The salinomycin provided to sheep in the dose established in this experiment does not prevent ruminal lactic acidosis induced by sucrose. However, the adverse effects of this disturbance are minimized or the severity of acidosis is reduced in intensity and time of development, allowing an earlier recovery of animals that received the ionophore.

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