

The effect of high broiler litter diets as survival ration on the health of sheep

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ABSTRACT

The use of broiler litter as an emergency feed during droughts and other periods of feed shortages was evaluated in terms of its effect on the health of sheep. Pure broiler litter (*i.e.* excreta plus wood shavings), and litter mixed with 7.5 or 15 % molasses were fed for 83 days to 2-year-old wethers. The addition of molasses to the litter caused a significant increase ($p < 0.01$) in feed intake and final body mass. Various parameters such as plasma enzyme activity, plasma metabolite concentrations and urine mineral and purine excretion did not differ among treatments. Histological evaluation revealed no liver or kidney pathology. Mild myocardial pathology was observed in all 3 treatment groups. This seems to be related to the presence of the ionophore-based coccidiostat, narasin, which was present in the litter at a concentration of 10 mg/kg. It is suggested that these histological lesions are of little practical significance and would not affect the health of sheep being fed the broiler litter as a survival feed.

Key words: broiler litter, drought feeding, ionophore, myocardial pathology, sheep.

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INTRODUCTION

Drought occurs frequently in southern Africa and devastating veld fires often destroy vast areas of grassland during the dry winter periods. Consequently, livestock farmers often have to resort to emergency feeding measures to sustain their animals, utilising whatever feed is available. Such conditions have prompted farmers to resort to feeding poultry manure/litter¹⁰ as the sole feed for their livestock.

Although poultry manure/litter has been used successfully as a feed supplement in ruminant diets^{6,14}, many potential problems exist. Problems of nutrient imbalances and the effects of potentially toxic components such as pathogens, dietary copper (Cu), medicinal compounds, mycotoxins, ionophores and botulinum toxin^{9,10} will be accentuated when the product is fed as a sole feed. Silanikove and Tiomkin¹⁸ reported liver damage in beef cows when overwintered on high poultry-litter diets in Israel. The

authors attributed the damage to high ruminal ammonia concentrations due to fermentation of the litter in the rumen. In South Africa, this report of liver damage seems to have prompted warnings against the use of poultry litter as a ruminant feed at any rate of inclusion. However, liver damage is not usually observed when poultry litter is fed, except when it contains high concentrations of Cu¹⁷. The objective of the present study was to investigate the effect of high levels of broiler litter on the health of sheep. Since broiler litter contains excessive levels of non-protein nitrogen, improving the dietary nitrogen (N) to energy balance to improve the product as a survival feed was evaluated as well.

MATERIALS AND METHODS

Experimental procedure

Eighteen South African mutton Merino wethers (*ca.* 2 years old and *ca.* 42 kg body mass) were assigned randomly to 3 treatments, namely pure broiler litter and broiler litter mixed with 7.5 or 15.0 % molasses. Sun-dried broiler litter with wood shavings as bedding material was used. The litter was sifted to remove lumps and foreign material. Before the onset of the trial the sheep were vaccinated twice against botulism and dosed with a broad-spectrum anthelmintic.

During a 14-day adaptation period the sheep received broiler litter and grass hay. The sheep were fed individually in slatted-floor feeding pens with free access to water. Feed refusal was measured weekly. The sheep were weighed once a month. Feed samples were collected throughout the trial for chemical analysis. Blood samples collected by venipuncture in heparinised vacutainers and spot urine samples were obtained 4 times during the experimental period. A spot urine sample was collected in a clean plastic bag tied to the stomach of the wether. The urine was preserved in 10 % HCl.

After an average of 83 days the wethers were slaughtered over a period of 10 days. The warm carcasses, livers and kidneys were weighed. Liver, heart and kidney samples were taken and preserved in 10 % buffered formalin for histopathological examination. Liver samples were also dried at 80 °C for Cu analysis. Backfat thickness at the 13th rib was measured on the cold carcass.

Laboratory analysis

The concentrations of haemoglobin (Hb) in whole blood, glucose and bilirubin in plasma, and the activities of the following plasma enzymes: aspartate aminotransferase (AST, EC 2.6.1.1), creatine kinase (CK, EC 2.7.3.2), alanine aminotransferase (ALT, EC 2.6.1.2), δ -glutamyltransferase (δ GT, EC 2.3.2.2) and sorbitol dehydrogenase (SDH, EC 1.1.1.14) were measured using Boehringer Mannheim analytical kits (Boehringer Mannheim GmbH Diagnostica, Germany). An analytical kit was used to determine the concentration of β -hydroxybutyrate in plasma (Sigma Diagnostics). Packed cell volume (PCV) in whole blood was determined by microhaematocrit. The method described by De Villiers *et al.*⁸ was used to determine free fatty acid concentration in plasma, that of Tietz¹⁹ for plasma albumin and the Bertelot method² for plasma urea N concentrations. Total allantoin concentration in urine was measured⁵ to estimate urinary purine concentration. The creatinine concentration in urine was read on an auto-analyser using the alkaline picrate method.

Atomic absorption spectrophotometry

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was used to determine the calcium (Ca), Cu, magnesium (Mg), sodium (Na), potassium (K), manganese (Mn) and zinc (Zn) concentrations in the respective samples. The fluorometric method of Koh and Benson¹⁵ was used to determine selenium concentrations. Crude protein, ash, phosphorus (P), ether extract, neutral detergent fibre and acid detergent fibre concentrations in each sample were obtained using standard laboratory techniques. The litter was screened with thin-layer chromatography for the presence of ionophore antibiotics and quantified colorimetrically¹¹.

Pathology

Tissues were fixed in 10 % formalin, sectioned and stained with haematoxylin and eosin according to standard methods. Myocardial lesions were scored according to a histological classification system applied by Bastianello *et al.*³.

Statistical analysis

Data were subjected to analysis of variance and correlation coefficients were calculated using Minitab Statistical Software (Minitab, State College, Pennsylvania).

RESULTS

The dry matter (DM) content of the pure litter was 923 g/kg, and the addition of molasses decreased it to 917 and 870 g/kg for the 92.5 and 85 % litter treatments respectively. The chemical composition of the experimental diets is presented in Table 1. The crude protein concentration of the pure litter was 182 g/kg DM. The inclusion of molasses decreased the crude protein concentration to 169 and 168 g/kg DM for the 92.5 % and 85 % broiler litter diets respectively. The Cu concentration in the diets varied between 22 and 27 mg/kg DM. The Ca:P ratio in all the diets was approximately 2:1.

The sheep in the 15 % molasses group consumed significantly more feed ($p < 0.01$) than the sheep on the 7.5 % molasses and the pure litter groups (Table 2). This was associated with significantly higher ($p < 0.01$) final body mass of the sheep in the high molasses treatment. In the 15 % molasses group, intakes reached an optimum after 3 weeks on the diet while those on the other 2 diets reached optimum intake at about 6–7 weeks. Carcass mass and fat cover at the last rib area of the carcass tended to be higher in the molasses-supplemented groups than in the control, but differences were not significant (Table 2).

Throughout the trial the concentrations of free fatty acid, β -hydroxybutyrate and

Table 1: Chemical composition of broiler litter and litter with molasses.

Broiler litter (%):	100	92.5	85
Molasses (%):	0	7.5	15
Dry matter (g/kg)	923	917	870
Ash (g/kg DM)	97	110	124
Crude protein (g/kg DM)	182	169	168
Ether extract (g/kg DM)	11.2	9.4	8.2
NDF ^a (g/kg DM)	540	520	460
ADF ^b (g/kg DM)	395	355	334
Calcium (g/kg DM)	15.0	16.0	16.0
Phosphorus (g/kg DM)	8.1	8.4	7.4
Sodium (g/kg DM)	2.90	2.75	3.35
Potassium (g/kg DM)	9.9	11.9	12.7
Magnesium (g/kg DM)	4.71	4.79	4.77
Copper (mg/kg DM)	22	27	22
Manganese (mg/kg DM)	214	241	231
Selenium (mg/kg DM)	0.68	0.66	0.66

^aNDF = neutral detergent fibre.

^bADF = Acid detergent fibre.

Table 2: Average feed intake, body mass gain, body mass at slaughter, fresh liver, kidney and carcass mass and fat thickness at the last rib area of the carcass (mean \pm SD).

Broiler litter (%):	100	92.5	85
Molasses (%):	0	7.5	15
Feed intake (kg/d)	1.3 ^a \pm 0.08	1.5 ^a \pm 0.15	2.1 ^c \pm 0.07
Final body mass (kg)	44 ^a \pm 3.6	48 ^{ab} \pm 3.6	52 ^b \pm 2.4
Gain/d (g)	26 \pm 23	63 \pm 32	101 \pm 10
Carcass mass (kg)	20 \pm 0.9	22 \pm 1.3	23 \pm 0.9
Liver:			
mass (fresh) (g)	610 \pm 40	592 \pm 41	668 \pm 23
as % body mass	1.4 \pm 0.19	1.2 \pm 0.18	1.3 \pm 0.05
as % carcass mass	3.0 \pm 0.33	2.8 \pm 0.15	2.9 \pm 0.12
Kidney mass (g)	108 \pm 6.0	134 \pm 5.4	134 \pm 6.5
Fat thickness (mm)	1.8 \pm 0.21	2.6 \pm 0.52	2.8 \pm 0.68

*Values within a row with different superscripts differ significantly at $p < 0.01$.

glucose in plasma showed no differences among treatments (Table 3). Plasma urea N concentrations at the last collection of the trial were 298, 286, and 276 mg/l for the 100, 92.5 and 85 % broiler litter treatments respectively, versus a quoted normal range of 80–200 mg/l¹⁴ (Table 3). However, high concentrations were measured from the onset of the trial. The average \pm standard deviation (SD) plasma albumin (35.3 \pm 4.14 g/l), plasma globulin (24.9 \pm 4.79 g/l), bilirubin (30.4 \pm 6.2 g/l), PCV (0.78 \pm 0.048) and haemoglobin (164 \pm 13.9 g/l) concentrations remained relatively constant throughout the trial, showed no treatment effects and were within the normal range for sheep¹³. Copper concentrations in the livers did not differ significantly among treatment groups and were 387, 383 and 338 mg/kg DM for the 100, 92.5 and 85 % broiler litter treatments respectively.

No significant differences in the activity of any of the plasma enzymes were observed between the different treatment groups (Table 3). Activities were within the normal ranges for sheep with the exception of plasma CK, where the activity was higher than the expected normal values. At the final blood collection the CK activities were recorded at 62.9 \pm 10.6, 56.1 \pm 22.6 and 40.0 \pm 3.6 U/l for the pure litter, 92.5 and 85 % litter diets, respectively. However, these values were within a similar range throughout the trial. The activity of plasma AST was randomly distributed and did not show any correlation with pathological lesions in the liver or heart.

The purine:creatinine ($W^{0.75}$)⁻¹ (PD:C) ratios during the trial are presented in Table 4. At 28 d after the onset of the trial these ratios were lower than at other stages, although differences were not

Table 3: Concentration of various plasma metabolites and activity of enzymes in plasma at the last collection (mean ± SD).

Broiler litter (%): Molasses (%):	100 0	92.5 7.5	85 15	Norm ¹³
Free fatty acids (mmol/l)	0.6 ± 0.16	0.35 ± 0.05	0.47 ± 0.08	0.55 ⁸
β-hydroxybutyrate (mmol/l)	0.3 ± 0.03	0.19 ± 0.02	0.21 ± 0.02	0.55
Glucose (mmol/l)	3.55 ± 0.1	3.35 ± 0.1	3.47 ± 0.12	2.8–4.4
Urea nitrogen (mg/l)	293 ± 17	267 ± 14	257 ± 16	80–200
AST ^a (U/l)	58 ± 3.9	56 ± 2.5	61 ± 8.5	61
δ-GT ^b (U/l)	39 ± 1.9	37 ± 4.1	39 ± 3.5	20–50
CK ^c (U/l)	63 ± 10.6	56 ± 22.6	40 ± 3.6	8–13
SDH ^d (U/l)	1.6 ± 0.2	1.8 ± 0.16	1.4 ± 0.19	8–16
ALT ^e (U/l)	16.5 ± 1.94	13.3 ± 3.15	16.5 ± 2.3	30

^aAST = aspartate aminotransferase.

^bδGT = gamma glutamyltransferase.

^cCK = creatine kinase.

^dSDH = sorbitol dehydrogenase.

^eALT = alanine aminotransferase.

Differences between treatments were not significant.

Table 4: Purine:creatinine ratio ($W^{0.75}$)⁻¹ in urine from spot samples taken during the trial (mean ± SD).

Litter (%)	Day of collection			
	1	28	63	81
100	27 ± 10.7	16 ± 4.8	24 ± 6.5	24 ± 7.2
92.5	28 ± 24.6	13 ± 6.1	27 ± 6.0	25 ± 5.7
85	31 ± 29.0	24 ± 9.5	32 ± 4.3	26 ± 3.2

Differences among means not significant.

Table 5: Number of sheep fed broiler litter with or without molasses with histopathological lesions in the myocardium ($n = 6/\text{treatment}$)^a.

Broiler litter (%): Molasses (%):	100 0	92.5 7.5	85 15
Myofibre hypertrophy	5	3	4
Myofibre atrophy	5	3	4
Hyaline degeneration	3	3	4
Hyaline necrosis	3	2	4
Attempted regeneration	3	3	4
Interstitial fibrosis	1	2	4
Incidental lesions			
Sarcocysts	4	1	3
Congestion	2	2	5

^aDistribution of the lesions was random and severity was classified as mild.

statistically significant.

Mineral concentrations in the plasma did not reflect any treatment effect. Average concentrations ± SD (mmol/l) over the experimental period were: 2.15 ± 0.12 Ca; 2.48 ± 0.394 inorganic P; 12.6 ± 2.49 Cu; 0.987 ± 0.076 Mg; 145 ± 3.6 Na; 5.2 ± 0.29 K; 16.8 ± 1.66 Zn. The Ca:P ratio in urine varied widely and did not show any consistent pattern. However, in many

of the sheep, irrespective of treatment, the concentration of P in urine was higher than its respective Ca concentrations.

Myocardial pathology was mild and similar for all the treatment groups (Table 5). The hepatic pathology was mild and consisted of hyaline degeneration and single-cell necrosis, considered to be non-specific, and triaditis, an incidental finding usually associated with parasites.

Renal pathology was restricted to 1 case of tubular degeneration and 11 of intertubular congestion.

When myocardial pathology was observed, the litter was screened for ionophores. Narasin, at a concentration of 10 mg/kg, was measured in the broiler litter.

DISCUSSION

The crude protein concentration of 180 g/kg DM, although less than the average South African value, was well within the levels of crude protein in litter fed to animals. However, this was substantially lower than the 300 g dietary protein/kg feed that reportedly caused the liver damage in cows¹⁸. A direct comparison of results is therefore not possible. The plasma urea N in the present trial is well above the normal range of 80–200 mg/l, indicating that excess ammonia must have been absorbed from the rumen. The normal concentrations of plasma albumin, bilirubin and hepatic enzyme activity in plasma indicate negligible hepatic pathology. Rankins *et al.*¹⁷ questioned the observation of Silanikove and Tiomkin¹⁸, and suggested that the observed liver damage might have been due to Cu toxicity. Unfortunately Silanikove and Tiomkin¹⁸ did not report the Cu concentrations in the broiler litter used or in the livers of their cows. In the present study the Cu concentration in the litter was 22 mg/kg DM. Copper concentration in the livers of our sheep (<400 mg/kg) was well below toxic levels¹². Van Ryssen *et al.*²⁰ concluded from the survey of mineral concentrations in poultry manure that copper sulphate is at present not included in broiler rations in South Africa. Consequently, in South Africa, Cu toxicosis due to ingestion of litter is unlikely, contrary to the situation in the USA¹⁷. Angus *et al.*¹ reported serious liver damage in sheep consuming diets containing up to 60 % manure from laying hens. Their findings included an accumulation of fluid in the abdomen and marked histological changes in the liver. No explanation could be given and the presence of a toxic substance was suggested. They also described kidney damage in sheep on high intakes of broiler litter, again of unknown cause¹. The possibility of Cu toxicity was considered and ruled out. The causes of the above lesions were obviously not present in our experimental diets.

The elevated plasma CK activity may indicate muscle damage. However, elevated activity was measured throughout the trial and may be unrelated to the broiler litter treatments. Although the

myocardial lesions resembled those of ionophore-associated poultry litter toxicity, as described by Bastianello *et al.*⁴, they were considered very mild in this batch of sheep. The ionophore antibiotic, narasin, which was present in the broiler litter, is not registered in South Africa as an ionophore for ruminants. However, our findings suggest that the level of narasin in the feed (10 mg/kg) was not high enough to induce severe myocardial pathology or influence the animals' ability to survive on the particular diet. Since the purpose of feeding high levels of litter was to keep the animals alive, *i.e.* at a maintenance or even submaintenance level of nutrition, these histological findings were probably of minor significance.

The inclusion of molasses in the diets resulted in a significant increase in feed intake. This increased intake was reflected in improved daily mass gains and a higher final body mass in the respective groups. The higher body mass in the molasses-fed sheep must have been due partly to higher gut content in these groups, because differences in carcass mass between treatments were less pronounced. If the objective is to keep the sheep alive, the inclusion of molasses may be unnecessary, except if the intake of the pure litter is unsatisfactory. In fact, lower litter intakes than recorded in the present trial should be sufficient for the survival of the sheep. Where litter with a crude protein content higher than that in the present study is used, the addition of molasses may have a more pronounced effect, *e.g.* in detoxifying ammonia.

The ratio of PD:C in urine can be used as an indication of relative rumen microbial protein synthesis in the animal⁷. Although this ratio increased slightly with an increase in molasses inclusion in the diet, variations within treatments were wide, and differences were not significant. From urinary PD:C ratios, Chen *et al.*⁷ estimated rumen microbial nitrogen productions in sheep of less than 5 to more than 20 g N/d. At PD:C ratios of between 15 and 30, Chen *et al.*⁷ estimated a rumen microbial nitrogen production of 6–12 g/d. In the current trial a PD:C ratio of *ca.* 23 was measured, indicating a relatively low microbial production of less than 12 g/d. The low PD:C ratios observed at 28 d may reflect incomplete adaptation of the microbes to the diets at that stage. This was evident in relatively low initial DM intakes which showed an increase for

the first 6 weeks in the 100 % litter diet. Further studies are required to confirm this apparent long adaptation period required by the sheep or the rumen microorganisms before they can utilise broiler litter effectively.

The concentrations of minerals in the broiler litter were lower than the means for South African samples, but well within the range of variation measured²⁰. The cows used by Silanikove and Tiomkin¹⁸ were hypocalcaemic and hyperphosphataemic when they consumed broiler litter and reacted positively to calcium borogluconate injections. The authors suggested that the apparent imbalances in Ca and P metabolism were due to the severe liver damage in the cows. Although the dietary Ca:P ratio in the present study was normal at 2:1, the P intake was well above requirements for sheep¹⁶. The high concentration of P in urine relative to that of Ca signifies that the high P intake through litter could cause urinary calculi in wethers.

It is concluded that the feeding of dry broiler litter of the quality used in the present investigation did not affect the health of the sheep when fed for a period of *ca.* 80 days. Therefore, in an emergency feeding situation of limited duration, it seems feasible to feed broiler litter to ruminants, even at restricted levels of intake. However, the necessary precautions must be taken because broiler litter has many potential problems as an animal feed^{9,10}.

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