EFFECT OF MONENSIN AND ITS METABOLITES IN BROILER LITTER ON SHEEP CONSUMING THE BROILER LITTER

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ABSTRACT

Two trials were conducted to determine the effect of monensin in broiler litter on sheep receiving the broiler litter in their diets. Broiler litter from chickens fed monensin as a coccidiostat, and from chickens receiving no coccidiostat, was included at a level of 30% in 2 sheep diets. In a further 2 treatments, monensin (15 mg kg^{-1}) was added to each of the 2 diets to give a 2x2 factorial experimental design. In the first trial, copper (20 mg kg^{-1} feed) was added to the diets. These lambs were fed individually at a slightly restricted level of intake. No differences between treatments were observed in feed intake, average daily gain or efficiency of feed utilisation or in the concentrations of zinc, iron and manganese in the liver, glutathione peroxidase in erythrocytes and creatine kinase concentrations in the plasma. Hepatic copper content and copper retention in the livers of the sheep receiving the added monensin were significantly higher ($P < 0,05$ and $< 0,01$ respectively) than in those not receiving added monensin. The aspartate transaminase and alkaline phosphatase concentrations in the plasma of these sheep were also higher ($P < 0,05$) than in those not consuming added monensin. In the second trial, the lambs were group-fed according to treatment and received the diets on an ad lib basis. The mean intakes of the groups receiving the diets with the added monensin, were lower than the intakes by the other groups. It was concluded that the monensin metabolites in broiler litter had no measurable effect on any of the parameters of monensin activity investigated in sheep, when such litter was included in sheep diets.

Key words: Monensin, sheep, broiler litter, hepatic copper

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INTRODUCTION

Monensin sodium (Elanco Product Company, Indianapolis, USA) is used widely as a coccidiostat in the diets of broilers, at a maximum recommended rate of inclusion of 120 mg kg^{-1} feed[6]. Monensin is also effective as an ionophor in the diets of sheep[11], at recommended levels of 15 to 22 mg kg^{-1}, and of cattle at a maximum of 33 mg kg^{-1} feed[6]. As an ionophor, the feeding of monensin can have various beneficial effects on the production of ruminants. One of its most important effects is an improvement in efficiency of feed utilisation in animals on high energy diets. This is quite often accompanied by a decrease in feed intake, without a concurrent decrease in bodymass gains[3]. Further beneficial effects are the control of coccidiosis in calves, control of feedlot disorders such as a reduction in the incidence of bloat[2] and acidosis especially during the period of adaptation to high concentrate diets[8].

Various metabolic changes due to the consumption of monensin have been observed[3]. Of relevance to the present study are the changes in the metabolism of minerals. Van Ryssen & Barrowman[18] found that monensin increased the retention of dietary copper in the liver of sheep. Anderson et al.[1] measured an improvement in the selenium status of sheep due to monensin. It has also been reported that the metabolism of sodium, potassium, magnesium, calcium, phosphorus and zinc in ruminants is altered by monensin[9 10 14].

At intakes higher than the recommended levels, monensin can have detrimental effects on animals. A reduced feed intake is one of the first symptoms of an excessive monensin intake[11 12]. At high intakes monensin can be very toxic. The LD_{50} of monensin for sheep is 11,9 mg kg^{-1} and for cattle 26,4 mg kg^{-1} livemass[12]. The LD_1 for a 400 kg bovine is estimated to be 2 210 mg monensin[12]. Symptoms of chronic toxicity are anorexia, skeletal muscle weakness, ataxia, decreased gain in mass and eventually death. Primary target tissues are the skeletal and cardiac muscles[15]. This is evident from elevated plasma concentrations of enzymes such as creatine kinase, which is usually associated with the breakdown of muscle tissues[13]. The supplementation of selenium and vitamin E to a selenium-deficient diet provided some protection against the harmful effect of high monensin intakes in pigs[19]. Deaths due to excessive monensin intake are usually due to inadvertent inclusion of excessive levels of monensin in ruminant diets, or the poor mixing and thus poor distribution of monensin in the feeds[11 12]. However, it was pointed out (M H Lowrey 1990, Private Veterinarian, P O Box 151, 3370, Umlaas Road, personal communication) that it is sometimes claimed that the mortalities which have occurred among ruminants consuming broiler litter, are caused by monensin or by its metabolites in litter which originated from the coccidiostat-treated broilers.

It was demonstrated with the use of radio-active markers that the monensin ingested by poultry, is excreted almost quantitatively via the faeces in the form of metabolites[6]. The monensin is metabolised in the body of the bird and reaches the digestive tract in the bile. The presence of these metabolites in faeces could not be detected with the use of the standard microbial test for monensin and showed a very low biological activity[6]. The question therefore arises whether the metabolites of monensin in the broiler litter could have the same detrimental effect on animals as does the unmetabolised compound.

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Two trials were conducted to establish whether excreted monensin or its metabolites in broiler litter, have the same measurable and metabolic effects on sheep as dietary monensin when sheep consume the litter. It is assumed that if litter is included in a sheep diet at a level of 30%, a relatively high level of these metabolites should still be present in the diet.

MATERIALS AND METHODS

Two groups of broilers were reared for 6 weeks in experimental rearing pens containing wood shavings as bedding material. One group received a diet containing monensin as a coccidiostat at a level of 100 mg kg^{-1} feed while the other group received no monensin. At the end of the rearing period, the litter from these pens was removed separately, sifted and sun-dried.

Two feeding trials with sheep were conducted in which these 2 types of litter were included in their experimental rations. At least 3 weeks before the onset of the trials, all the sheep were vaccinated against botulism and dosed with a wide-spectrum anthelmintic.

Trial 1: Liver biopsies were done on SA Mutton Merino wethers, (n=38) ca 9 months of age, with a mean body mass of 29 kg. Six wethers with liver copper concentrations deviating the most from the mean of the group, were slaughtered as a pre-experimental group to determine the relationship between liver mass and empty body mass. Using a 2x2 factorial design, the remaining 32 wethers were allocated to 4 treatment groups in a randomised block according to the copper concentration in their livers. The treatments were: i) a control group (C) receiving a diet containing 30% broiler litter from the broilers which did not receive the coccidiostat in their diet; ii) the control diet (as for Group C) but with added monensin (15 mg kg^{-1} feed) (C+); iii) a group on the test diet (T) including the litter (at 30%) from the chickens receiving monensin, and iv) the test diet fortified with monensin (15 mg kg^{-1} feed) (T+). The experimental diet consisted of 300 kg broiler litter, 380 kg hominy chop, 50 kg fishmeal, 180 kg veld hay, 80 kg molasses meal, 5 kg lime and 5 kg salt per 1000 kg. Copper (as copper sulphate) was added to the rations at 20 g per 1000 kg. For the first 2 weeks of the trial, monensin was included at half the intended level. This practice enabled the lambs to adapt to monensin feeding and allowed an opportunity to minimise mortalities if treatments should prove to be toxic. The sheep were fed individually. In order to keep the metabolic changes in the body as comparable as possible, it was planned to keep feed intake per sheep as constant as possible. Therefore allocations were adjusted weekly, according to the intakes of the groups receiving the added monensin.

Feed intakes and body mass changes were recorded weekly. The sheep were observed closely for ill-health and any other symptoms of toxicity. Blood samples were collected in heparin at various stages of the trial for mineral and enzyme assays. In order to minimise any effect of the liver biopsy on enzyme levels in the plasma, the first collections were only done at 36 d after the onset of the trial. On Day 77 the lambs were slaughtered, after exsanguination. Carcass and fresh liver masses were determined. Liver samples were taken, dried at 80°C and stored for further analysis.

Trial 2: In a group feeding trial, ewe lambs were allocated randomly to similar experimental treatment groups as in Trial 1. However, the added copper was omitted from the diets. The lambs (5 per group) received their diets on an ad lib feeding basis in order to measure voluntary feed intake. The trial lasted for 45 days. Feed intakes per group and individual body masses were recorded weekly.

Atomic absorption spectrophotometry was used to determine the concentrations of copper, zinc, iron and manganese in the liver and feed. Sodium and potassium concentrations in plasma were obtained using flame photometry. Glutathione peroxidase (EC 1.11.1.9) was assayed at 25°C using the coupled enzyme procedure as modified by Whanger et al.[20]. Boehringer Mannheim standard kits (Boehringer Mannheim GmbH Diagnostica, West Germany) were used to estimate the aspartate transaminase (EC 2.6.1.1), alkaline phosphatase (EC 3.1.3.1) and creatine kinase (EC 2.7.3.2) concentrations in plasma. The monensin content of the diets was estimated using an anti-microbial growth assay[6].

To calculate hepatic copper retention at the end of the trial, the following procedure was carried out:

An estimate of individual liver masses at the onset of the trial was made from the body mass of each lamb and the ratio of

Table 1: **An illustration of the interpretation of a single 2x2 factorial analysis (Copper concentration in Table 6 used as example)**

		Monensin added to broiler diet		
		No	Yes	Mean
		A		B2
Monensin added to sheep diet	No	778(C)	740(T)	759
	Yes	985(C+)	835(T+)	910
		B1		
Mean		882	788	835

Least significant differences (LSD):

	P=0,05	P=0,01
for Box A	220	297
for Boxes B1 and B2	156	210

i) Box A contains the original treatment means which each came from 8 sheep. The statistical significance of the difference between any pair of these can be calculated from the LSD's, e.g. the difference between C and C+ is 207, therefore less than 220 and not significant. These means with the respective LSD's are presented in a column in subsequent tables. Differences between 2 treatments can be compared.

ii) Both the boxes designated B, contain the means of the 2 basic treatments, viz. with or without monensin as a coccidiostat in B1, and with or without added monensin in B2. The differences within each box can be compared with the LSD's, e.g. in B2 the difference is 151 therefore less than 156 and not significant at P = 0,05.

Where the means within the basic treatments were large, being significant or approaching significance, the means are presented in subsequent tables.

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liver mass to body mass as obtained from the pre-experimental slaughter group. The copper content of the livers was calculated from this estimated liver mass and the copper concentration in the biopsy samples. This was subtracted from the copper present in the liver at the end of the trial (copper concentration x liver mass) to calculate hepatic copper accumulation. The percentage dietary copper retained in the livers was obtained from total copper intake and hepatic copper accumulation.

The 2x2 factorial analyses were performed on the data with the aid of the computer programme Genstat (Lawes Agricultural Trust, 1980). To demonstrate the interpretation of results in a 2x2 factorial statistical design, a description is presented in Table 1 which can be used to evaluate the proceeding tables. In these tables the original treatment means, each from 8 sheep, are supplied and compared with the use of the LSD (least significant difference). Where combined means related to one of the 2 basic treatments were important, the means of the 16 sheep are presented with the relevant LSD's, e.g. in Tables 5 and 6.

RESULTS

Diet, feed intake and performance

The concentrations of monensin and minerals in the experimental diets are presented in Table 2.

Due to restricted feeding, no difference in feed intake among groups was observed in Trial 1. In Trial 2 the differences in

Table 2: **The concentration of monensin and minerals in experimental diets containing broiler litter (C), broiler litter with monensin added (C+), broiler litter from chickens which received monensin (T), and broiler litter from chickens which received monensin with monensin added (T+). Copper sulphate was added to all rations**

Treatment	Monensin	Copper	Zinc	Iron	Manganese
	mg kg^{-1}		mg kg^{-1} dry mass		
C	3	25,0	92	540	170
C+	26	25,6	101	612	181
T	7	25,5	100	555	175
T+	22	24,2	102	518	177

Table 3: **Mean feed intakes and performances of sheep on diets containing broiler litter (C), broiler litter with monensin added (C+), broiler litter from chickens which received monensin (T), and broiler litter from chickens which received monensin with monensin added (T+)**

	Trial 1					Trial 2		
		ADG*		EFU**		ADG*		EFU**
Treatment	Feed intake kg d^{-1}	Carcass g d^{-1}	Live g d^{-1}	Carcass	Live	Feed intake kg d^{-1}	Live g d^{-1}	Live
C	1,69	121	179	14,1	9,6	2,00	173	11,5
C+	1,68	122	192	14,0	8,9	1,68	160	10,5
T	1,70	122	197	13,9	8,7	1,93	229	8,5
T+	1,66	120	187	14,0	9,3	1,61	160	10,0
LSD*** P = 0,05	0,10	18	31	1,4	1,4	-	43	-

*ADG - average daily gain
**EFU - efficiency of feed utilisation (kg feed kg^{-1} gain)
***LSD - least significant difference

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feed intake between the groups receiving the metabolites and those without the metabolites (Treatments T versus C and T+ versus C+) were negligible, while those receiving the added monensin (Treatments C+ and T+) consumed substantially less feed compared to the other 2 groups respectively (Table 3). It was not possible to compare the intakes in Trial 2 statistically, because group feeding was applied.

The mean final body masses, average daily body and carcass gains and efficiency of feed utilisation (kg feed consumed divided by kg gain) calculated for body and carcass mass gains (initial carcass mass estimated from live mass at onset of trial and dressing percentage of pre-experimental slaughter group), did not differ significantly between treatments in Trial 1 (Table 3). In Trial 2, sheep on the T treatment showed a higher ($P < 0,05$) gain in body mass than sheep on the other treatments (Table 3).

Blood analyses

The concentration of sodium (149, 148, 149 and 151 mmol l^{-1}) and potassium (4,7; 4,8; 5,0 and 4,9 mmol l^{-1}) in plasma for treatments C, C+, T and T+ respectively, did not differ significantly between treatments. The differences between treatments per collection in erythrocyte glutathione peroxidase activity and plasma creatine kinase, were not significant (Table 4). However, both glutathione peroxidase and creatine kinase levels increased between the collection on Day 36 and that on Day 77. When extra monensin was added to the diets (Treatments C+ and T+), the mean concentration of aspartate transaminase and alkaline phosphatase (Table 5) in plasma were significantly ($P < 0,05$) higher than in the 2 treatments without the added monensin.

Liver

Mean iron, zinc and manganese concentrations in the livers did not differ significantly due to treatments. These concentrations for treatments C, C+, T and T+ were: 228, 224, 241 and 224 mg iron kg^{-1} dry matter (DM); 169, 163, 182 and 159 mg zinc kg^{-1} DM and 13,9; 14,2; 13,3 and 13,9 mg manganese kg^{-1} DM respectively.

The livers of the groups receiving added monensin in the diets (C+ and T+) had a higher ($P < 0,05$) total hepatic copper content and copper accumulation (Table 6) than the groups without the added monensin. The percentage dietary copper retained in the livers of the groups receiving added monensin was significantly higher ($P < 0,01$) than for the other groups (Table 6).

The mean liver mass and liver mass expressed as a percentages of carcass mass, did not differ significantly between treatments. Fresh liver mass as a percentage of carcass mass in treatments C, C+, T and T+ was 3,2; 3,3; 3,3 and 3,6% respectively.

DISCUSSION

Accepting that metabolites from monensin cannot be detected in poultry manure[6], the concentration of unmetabolised monensin in the litter of the birds receiving the coccidiostat, can be calculated from the monensin in diet T (based on the 30% litter in the experimental diets). This amounted to 23 mg monensin per kg litter. Some of this might have originated from feed spilled onto the bedding material. Donoho[6] reported that chickens excreted less than 10% of the orally-administered monensin as "parent" monensin via the faeces. The presence of 3 mg monensin per kg feed in the Control diet, is probably a false positive reading which showed up in the anti-microbial growth assay for monensin. Such a false positive reading, plus sampling errors, might have reduced the reliability of the monensin concentrations presented.

Evidence of an effect of monensin on the sheep was observed in Trial 2, in the treatments where monensin was added (Treatment C+ and T+) to the diets. Feed intake was not restricted in this trial and voluntary feed intake in these 2 treatments was lower than in the others. This is in accordance with most other studies[3]. Monensin metabolised by the broilers, did not depress the feed intake of the sheep. In both trials, efficiency of feed utilisation did not differ between treatments. An improved efficiency of feed utilisation is considered to be one of the main beneficial effects of monensin in ruminants[3], although this is quite often not observed in sheep[18]. The significantly higher gain in mass of the sheep in Treatment T of Trial 2 is difficult to explain. In Trial 1, the average daily gain in Treatment T was also higher, though not statistically different, from that in Treatment C.

The increased copper content and copper retention in the livers of the sheep receiving the added monensin, is further evidence of an effect of monensin on mineral metabolism. This agrees with the observation by Van Ryssen & Barrowman[18] that monensin enhanced hepatic copper retention in sheep. From this study it is clear that the metabolites of monensin in the diets had no effect on copper metabolism while the added monensin increased copper accumulation in the liver. Significantly higher plasma concentrations of aspartate transaminase and

Table 4: **Mean serum concentrations of glutathione peroxidase and creatine kinase in sheep on diets containing broiler litter (C), broiler litter with monensin added (C+), broiler litter from chickens which received monensin (T), and broiler litter from chickens which received monensin with monensin added (T+)**

	Glutathione peroxidase nmol NADPH mg^{-1} Hb			Creatine kinase U l^{-1} plasma	
	day			day	
	36	58	77	36	77
C	182	267	324	118	168
C+	190	279	299	124	197
T	196	266	307	88	168
T+	230	249	318	127	187
LSD* P=0,05	41	34	54	59	64
P=0,01	55	45	73	80	86

*LSD = least significant difference

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Table 5: **Plasma concentrations of aspartate transaminase and alkaline phosphatase in sheep on diets containing broiler litter (C), broiler litter with monensin added (C+), broiler litter from chickens which received monensin (T), and broiler litter from chickens which received monensin with monensin added (T+)**

	Aspartate transaminase*** U l^{-1} Day 36		Aspartate transaminase*** U l^{-1} Day 77		Alkaline phosphatase*** U l^{-1} Day 36		Alkaline phosphatase*** U l^{-1} Day 77	
	Indiv.	Mean*	Indiv.	Mean*	Indiv.	Mean*	Indiv.	Mean*
C	138		171		587		554	
		137		164		717		614
T	136		157		847		674	
C+	199		224		921		852	
		291		318		914		843
T+	383		412		903		835	
LSD**								
P=0,05	178	126	198	140	245	173	210	148
P=0,01	240	170	267	189	330	233	282	200

*means to compare with or without added monensin
**LSD - least significant difference
***means to compare with or without monensin as a coccidiostat not significantly different

alkaline phosphatase accompanied the higher hepatic copper concentrations of sheep receiving the added monensin. This may indicate that these lambs were closer to the haemolytic crisis stage of copper toxicity than those in the other two treatments. Todd & Thompson[16] reported that the concentration of enzymes which may indicate liver damage, starts to rise at about 8 weeks before the onset of a haemolytic crisis. However, in the present investigation, elevated concentrations of these enzymes were observed in all the lambs from the first blood collection on Day 36, without any further increases occurring towards the end of the trial. This does not support the suggestion that some lambs were approaching a haemolytic crisis. Van Ryssen & Barrowman[18] suggested that sheep may be close to the haemolytic crisis when their liver masses expressed as a percentage of body mass were smaller than in sheep not approaching the crisis. In the present investigation, liver mass relative to carcass mass did not differ between treatments, implying that the crisis was probably not imminent.

Various studies show that monensin alters the metabolism of sodium and potassium in the body[9 14]. Starnes et al.[14] and Kirk et al.[9] did not observe any changes in serum potassium and sodium

Table 6: **Hepatic copper concentrations in sheep which were fed broiler litter (C), broiler litter with monensin added (C+), broiler litter from chickens which received monensin (T), and broiler litter from chickens which received monensin with monensin added (T+)**

	Hepatic copper*** Concentration mg kg^{-1}DM		Hepatic copper*** Total content mg		Hepatic copper*** Accumulation mg		Hepatic copper*** Retention %	
	Indiv.	Mean*	Indiv.	Mean*	Indiv.	Mean*	Indiv.	Mean*
C	778		158		153		4,7	
		757		159		153		4,7
T	740		159		154		4,6	
C+	985		207		202		6,1	
		910		196		191		5,9
T+	835		185		180		5,8	
LSD**								
P=0,05	220	156	44	32	45	32	1,24	0,87
P=0,01	297	210	61	43	61	43	1,66	1,18

*means to compare with or without added monensin
**LSD - least significant difference
***means to compare with or without monensin as a coccidiostat not significantly different

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concentrations, in agreement with the present study. However, Starnes et al.[14] quoted other studies in which monensin reduced serum potassium concentrations. Kirk et al.[10] found that monensin increased the retention of zinc in the body of sheep, although levels in the liver did not change. In the present investigation, liver zinc levels did not differ between treatments. In the present investigation, monensin did not have any effect on iron levels in the liver. This is in agreement with the observations of Elsasser[7], but not with those of Van Ryssen & Barrowman[18] who measured a reduction in hepatic iron concentration due to monensin. The observation by Van Ryssen & Barrowman[18] that liver manganese concentrations are elevated in monensin-fed sheep was not substantiated in the present study.

A glutathione peroxidase activity in excess of 40 nmol NADPH mg^{-1} haemoglobin indicates that the animal is consuming sufficient selenium to meet its requirements[1]. Therefore, the high levels of this enzyme in the present study, preclude the possibility that a selenium deficiency could accentuate the effects of monensin toxicosis[19]. Anderson et al.[1] reported that monensin improved the selenium status of sheep. On Day 36, in the present trial, the groups receiving the added monensin (C+ and T+) had higher glutathione peroxidase levels than treatments C and T respectively, although the differences were not statistically significant. On Days 58 and 77 differences between treatments were minimal, which is in agreement with Costa's[4] observation that monensin has no effect on the selenium status of animals. Whanger et al.[20] observed a continuous increase with time in the blood glutathione peroxidase concentrations of sheep receiving high levels of selenium in the diet. From the increases in enzyme concentrations between Days 36 and 77, it can be concluded that the sheep diets contained more than sufficient selenium to meet their requirements.

The fact that creatine kinase concentrations in plasma did not differ significantly between treatments may indicate that monensin, whether derived from litter or from addition to the diet at recommended levels, had no damaging or toxic effects on muscle tissue[13 15]. The relatively high creatine kinase levels measured in all treatments at the end of the trial is difficult to explain.

From this investigation, it may be concluded that the monensin fed to poultry as a coccidiostat had been metabolised to such an extent that it became metabolically inactive. It showed no effect in any of the parameters of monensin activity measured in the sheep. These results suggest that the feeding of monensin as a coccidiostat to poultry does not pose any risk to sheep consuming the litter as part of their diet. This supports the evidence of Donoho[6] that the metabolism of monensin in the body of poultry results in the destruction of most of its biological activity. Furthermore, the results suggest that statements in the popular press[5 17] that coccidiostats which are excreted in the litter of broilers can cause deaths in ruminants, especially if the same product is included in the ruminant rations as an ionophore, are too generalised. They may possibly apply to certain coccidiostats, but should exclude monensin.

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