Effect of Monensin on Milk Production of Holstein-Friesian Dairy Cows

R. H. Phipps*, J.I.D. Wilkinson†, L. J. Jonker†, M. Tarrant†, A. K. Jones*, and A. Hodge†
*Centre for Dairy Research, Department of Agriculture, The University of Reading, Arborfield, Reading RG2 9HX, UK
†Eli Lilly and Co. Ltd, Elanco European Animal Science Research, Basingstoke, Hants RG21 6XA, UK

ABSTRACT

We examined the effects of monensin on feed intake and milk production in Holstein-Friesian cows receiving a total mixed rations in two experiments. In experiment 1, 60 individually fed cows consumed, during wk 7 to 26 of lactation, 1 kg/d of supplement containing either 0, 150, 300, or 450 mg of monensin. In experiment 2, 98 group-fed cows also received 1 kg/d of a supplement with either 0 or 300 mg/d of monensin for two consecutive lactations. In lactations 1 and 2, treatment started at wk 8 and 3 wk prior to calving, and continued for 32 wk. In experiment 1, 150, 300, and 450 mg of monensin/d produced a small decrease in feed intake and milk yield responses of 2.8, 2.5 and 1.5 kg/d, respectively. In experiment 2, milk yield responses of 0.8 and 1.1 kg/d were recorded in lactations 1 and 2. Milk fat and milk protein content declined in experiments 1 and 2, lactations 1 and 2 by 0.46, 0.38 and 0.27%, and 0.16, 0.16 and 0.11%, respectively. Yield of milk constituents was unaffected. Efficiency of milk production was increased by 5% in experiment 1. In experiment 2, lactation 2, monensin decreased β-hydroxybutyrate and acetoacetate but increased blood glucose concentration.
(**Key words:** monensin, milk production, metabolic parameters)

Abbreviation key: Expt 1 = experiment 1, **Expt 2** = experiment 2, **L1** = lactation 1, **L2** = lactation 2, **NE** = net energy.

INTRODUCTION

Numerous workers have reported that one of the major effects of feeding monensin in ruminant diets is a reduction in molar proportions of acetate and butyrate with a concomitant increase in propionate (Jalc et al., 1991; Mackintosh et al., 1996; Sauer et al., 1989). While these changes have long been associated with improved efficiency in the beef industry, they have also been associated with a consistent reduction in milk fat content in lactating dairy cows (Lowe et al., 1991; Sauer et al., 1989). The effect of monensin on milk protein content has, however, been variable (Chalupa, 1988), with both reduced, (Sauer et al., 1989) and increased (Wade et al., 1996) values being reported. Published results also indicate that the inclusion of monensin will increase milk yield and that the response may vary from 0.5 to 2.9 kg/d (Sauer et al., 1989; Williamson et al., 1996) with increases of 0.5 to 1.5 generally being recorded (Lean and Wade, 1997).

With the exception of the study conducted in Australia (Wade et al., 1996), few studies have been conducted to examine the effect of monensin dose rate on feed intake, milk production and efficiency of feed utilization of lactating dairy cows. The aims of the current studies were to determine (a) the effects of monensin dose rate (0, 150, 300, and 450 mg/d) and (b) effects over two lactations for cows receiving either 0 or 300 mg/d of monensin on feed intake, milk production, and metabolic profiles of lactating Holstein-Friesian cows.

MATERIALS AND METHODS

Experiment (Expt) 1

Sixty individually fed multiparous Holstein-Friesian cows were used to determine the effect of monensin (Eli Lilly and Co. Ltd, Basingstoke, Hants, UK) dose rate on feed intake and milk production. There were four treatment groups receiving 0, 150, 300, or 450 mg/d of monensin. Treatments were introduced at wk 7 of lactation and continued for 20 wk. For each cow, the mean value for each variable during the treatment period was calculated from the weekly data points, and these values were analyzed using SAS. The study was a randomized block design, and the average value per cow from the last 3 wk of the pretreatment period was used as a covariate. Cows were blocked in pairs according to parity (range of three parities/block) and mean milk yield/d during wk 2 to 4 of lactation (range of 3 kg/d per block) and were then allocated to treatment according to a preprepared sequence derived from ran-

Received November 3, 1999.
Accepted July 9, 2000.
Corresponding author: R. H. Phipps; e-mail: r.h.phipps@reading.ac.uk.

dom permutations. Hence, the analysis of variance model used for each variable was:

$$y(ijk) = \mu + t(i) + b(j) + \beta x(ijk) + \varepsilon(ijk)$$

where

- y(ijk) = average value for cow k in treatment group i and block j,
- μ = overall mean,
- t(i) = effect of treatment I,
- b(j) = effect of block j,
- β = regression coefficient for pre-treatment covariate,
- x(ijk) = average pre-treatment value for cow(ijk), and
- ε(ijk) = residual error.

The treatment groups were compared using treatment contrasts in the analysis of variance model. It should be noted that further analyses were performed to investigate the repeated measures aspect of the data and, in particular, to test for the presence of an interaction between treatment group and period of lactation. It was found that any such interactions were small relative to the size of the main treatment effect. Hence, the analysis of variance models described here are considered to provide a valid assessment of the main treatment effect.

Experiment (Expt) 2

This study was conducted with group-fed cows over two consecutive lactations (**L1** and **L2**). In L1, 69 multiparous and 29 primiparous Holstein-Friesian cows received either 0 or 300 mg/d of monensin. In L2, 79 of the original cows, plus 19 cows from Expt 1 were allocated to the same treatments as they received in the previous lactation, either 0 or 300 mg/d of monensin. In L1 treatment started at wk 8 of lactation and continued until the end of wk 40 of lactation, while in L2 treatment started 3 wk prior to calving and continued until the end of wk 32 of lactation. For each cow, the mean value for each variable during the treatment period was calculated from the weekly data points, and these values were analyzed using the SAS. The study was a randomized block design and the average value per cow from the last 3 wk of the pretreatment period in L1 was used as a covariate. Cows were allocated to treatment as in Expt 1, except primiparous cows were blocked separately. The analysis of variance model can be expressed in the same way as in Expt 1, and similar further analyses were performed to validate the choice of model.

Administration of Monensin

In both Expt 1 and 2, excluding the first week of treatment, when 0.5 kg of concentrate/d was offered to allow for adaptation of rumen flora, all cows received 1 kg/d of either control or monensin-treated concentrate. The same level of monensin was used throughout the treatment periods. In Expt 1, the control and monensin-treated concentrate was provided once daily as a top dressing to the TMR, while in Expt 2, concentrates were offered via Fullwood out-of-parlor feeders.

Diets

In both experiments cows were offered a TMR ad libitum, which for the first 18 wk of lactation contained on a DM basis 13.4% grass silage, 50.4% maize silage, 21.5% concentrate, 11.8% sodium hydroxide-treated wheat grain, and 3.0% molasses. The concentrate contained 52.2% soybean meal, 35.3% rapeseed meal, 10.2% fishmeal, and 2.3% minerals. In Expt 1 and in the first lactation of Expt 2 the proportions of ingredients used in the TMR were changed at wk 19 of lactation to 37.4, 36.8, 14.4, 9.0, and 2.3% grass silage, maize silage, concentrate supplement, sodium hydroxide treated wheat grain, and molasses, respectively. The TMR was fed once daily and refusals were taken three times a week. The nutritive value of the diets is shown in Table 1.

Measurements

Milk yields were recorded automatically for all cows at each milking and were used to produce a mean daily average on a weekly basis. Milk samples were taken at two consecutive milkings once per week and analyzed for milk fat, protein, and lactose concentration by an infrared milk analyzer [Foss Electric (UK) Ltd]. Cows were weighed and body condition scored (Mulvany, 1977) before treatment start and at monthly intervals thereafter. In Expt 2 L2 all cows were blood sampled from the tail vein before calving and at wk 2, 4, 6, 8, 10, and 12 of lactation to determine concentrations of β-hydroxybutyrate, acetoacetate and glucose. Net energy (**NE**) values for milk (Tyrell and Reid, 1965) and maintenance (Van Es 1978) were estimated to calculate efficiency parameters.

RESULTS

Feed Intake and Milk Yield

In Expt 1, the use of monensin resulted in a small nonsignificant reduction in DMI (Table 2). As cows were group fed in Expt 2, individual feed intake measurements were not obtained.

Table 1. Composition of the TMR used in experiment 1 (Expt 1) and experiment 2 (Expt 2), lactation 1 (L1) and lactation 2 (L2).

Total Mixed Ration	Year 1 (Expt 1 and Expt 2 L1)		Year 2 (Expt 2 L2)
Composition of DM (g/kg DM)	1 to 18[1]	19 to 40[1]	1 to 40[1]
Corrected oven DM (g/kg)	389	312	360
CP	192	179	201
NDF	361	434	396
Water soluble carbohydrates	31	22	45
Starch	247	83	N/A[2]
Oil (acid hydrolysis)	33	36	24
Total ash	69	75	63
Metabolizable energy (MJ/kg DM)	11.2	10.6	12.0
Calcium	5.5	5.2	5.1
Phosphorus	4.4	4.4	4.1
Potassium	1.7	1.8	1.3
Magnesium	1.9	1.8	1.7
Sodium	3.0	3.4	3.2
Manganese	62	60	77
Zinc	47	43	41
Copper	13	10	14

[1]Lactation weeks.

[2]N/A = Not available.

The milk yield response of 2.8 and 2.5 kg/d that occurred when cows received 150 and 300 mg/d of monensin were both significant ($P < 0.05$) when compared with the control cows. Although the use of the highest dose rate (450 mg/d) increased milk yield by 1.5 kg/d it was not significantly different from either the control or the two lower dose rates. In Expt 2, monensin increased milk yield in L1 and L2 by 0.8 and 1.1 kg/d ($P > 0.05$) when compared with the controls (Table 3). The pattern of milk production during L2 is shown in Figure 1.

Milk Composition

Milk fat. During the 20-wk study period in Expt 1, the use of 150, 300, and 450 mg/d of monensin reduced ($P < 0.01$) the milk fat content from 3.90 to 3.58, 3.44,

Table 2. Mean DMI and milk production values (adjusted by covariance for 3 wk start of trial) when cow received 0, 150, 300, or 450 mg/d of monensin (Eli Lilly and Co. Ltd, Basingstoke, Hants RG21 6XA, UK) in experiment 1.

	Monensin (mg/d)				
	0	150	300	450	SED[2]
DMI (kg/d)	19.4	19.3	18.8	18.9	0.60
Milk yield (kg/d)	25.0[b]	27.8[a]	27.5[a]	26.5[ab]	1.25
Composition (%)					
Fat	3.90[a]	3.58[b]	3.44[b]	3.44[b]	0.129
Protein	3.39[a]	3.24[b]	3.25[b]	3.23[b]	0.056
Lactose	4.59	4.62	4.59	4.59	0.039
Constituent yield (g/d)					
Fat	971	981	929	892	46.9
Protein	854	901	897	858	39.7
Lactose	1145[b]	1279[a]	1260[ab]	1214[ab]	59.0
Gain					
BW (kg)	10.7[a]	16.8[ab]	19.7[ab]	38.6[b]	11.7
BCS	0.69	0.86	0.58	0.57	0.187
Efficiency measures					
Milk yield (kg)/DMI (kg)	1.248[b]	1.441[a]	1.453[a]	1.423[a]	0.060
NE[1] milk/NE intake	0.576	0.609	0.607	0.578	0.0246
NE efficiency	0.849	0.885	0.890	0.864	0.0230

[ab]Means within row with different superscripts differ ($P < 0.05$).

[1]Net energy.

[2]SED = Standard error of the difference between two treatment means.

Table 3. Mean milk production values (adjusted by covariance for 3 wk before start of trial in lactation 1) when cow received either 0 or 300 mg/d of monensin (Eli Lilly and Co. Ltd, Basingstoke, Hants RG21 6XA, UK) in experiment 2, lactations 1 and 2.

	Lactation 1			Lactation 2		
Monensin (mg/d)	0	300	SED*	0	300	SED[1]
Milk yield (kg/d)	21.4	22.2	0.54	27.2	28.3	1.01
Composition (%)						
Fat	4.45[a]	4.07[b]	0.073	4.45[a]	4.18[b]	0.078
Protein	3.64[a]	3.48[b]	0.032	3.55[a]	3.44[b]	0.046
Lactose	4.67	4.63	0.028	4.66	4.62	0.024
Constituent yield (g/d)						
Fat	947	900	27.8	1195	1178	42.1
Protein	775	776	19.5	949	973	30.4
Lactose	1007	1035	27.7	1270	1308	48.4
Gain						
BW (kg)	71	72	9.7	57	55	7.3
BCS	0.2	0.3	0.16	0.4	0.5	0.12

[ab]Subcolumn means within row with different superscripts differ ($P < 0.05$).

[1]SED = Standard error of the difference between two treatment means.

and 3.44%, respectively. Milk fat content was unaffected as dose rate was increased from 150 to 450 mg/d. In Expt 2 in both L1 and L2 the use of monensin also resulted in a large and immediate reduction in milk fat concentration (Figure 2). When averaged over the study periods, 300 mg/d of monensin reduced milk fat in L1 ($P < 0.001$) from 4.45 to 4.07% and from 4.45 to 4.18% in L2 ($P < 0.01$). Although milk fat yield for treated cows was consistently lower when compared with controls, the decrease was not significant ($P > 0.05$). When averaged over the entire study, the reduction in milk fat yield in L1 and L2 was 46 and 17 g/d, respectively.

Milk protein. When compared with the control, the use of 150, 300, and 450 mg/d monensin reduced ($P < 0.05$) milk protein in Expt 1 from 3.39% to 3.24, 3.25, and 3.23%, respectively (Table 2). Milk protein content was unaffected as dose rate was increased from 150 to 450 mg/d. There were no significant treatment effects on yield of milk protein. In Expt 2 monensin reduced milk protein concentration in L1 ($P < 0.001$) and L2 ($P < 0.05$) from 3.64 to 3.48% and 3.55 to 3.44%, respectively (Table 3, Figure 3), when compared with the controls. Table 3 also shows that even though monensin significantly reduced milk protein concentration, yield of milk protein was unaffected ($P > 0.05$).

Body Weight, BCS, and Metabolic Profile

The increase in BW of 38.6 kg recorded during the treatment period for the cows receiving 450 mg/d of monensin in Expt 1 was higher ($P < 0.05$) than the 10.7 kg recorded for the control cows (Table 2). Intermediate values were noted for cows fed the lower dose rates.

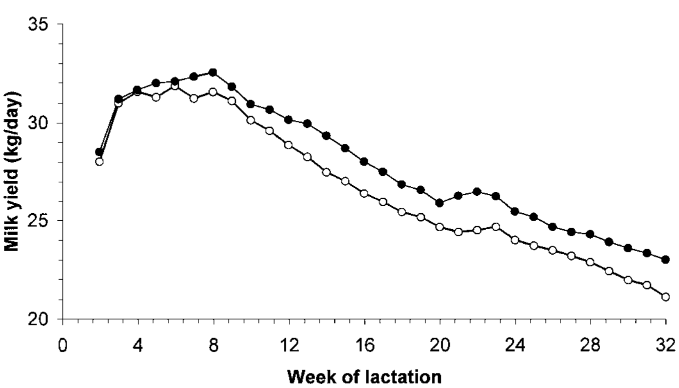

Figure 1. Mean milk yield (kg/d), adjusted for block and pretreatment covariate, of Holstein-Friesian cows receiving 0 (Control ○) or 300 (Treated ●) mg/d of monensin (Eli Lilly and Co. Ltd, Basingstoke, Hants RG21 6XA, UK) in experiment 2 lactation 2.

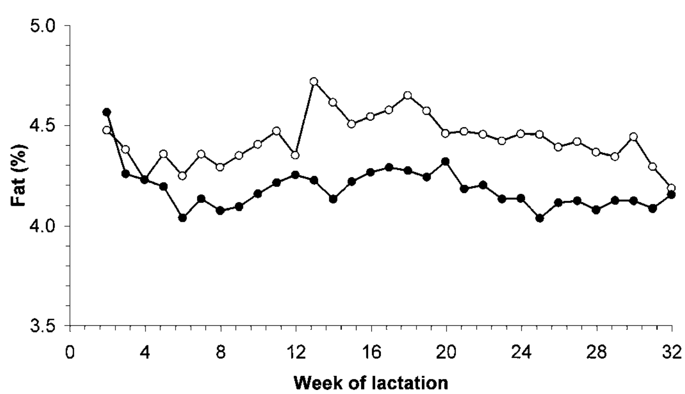

Figure 2. Mean milk fat concentration (%), adjusted for block and pretreatment covariate, of Holstein-Friesian cows receiving 0 (Control ○) of 300 (Treated ●) mg/d of monensin (Eli Lilly and Co. Ltd, Basingstoke, Hants RG21 6XA, UK) in experiment 2 lactation 2.

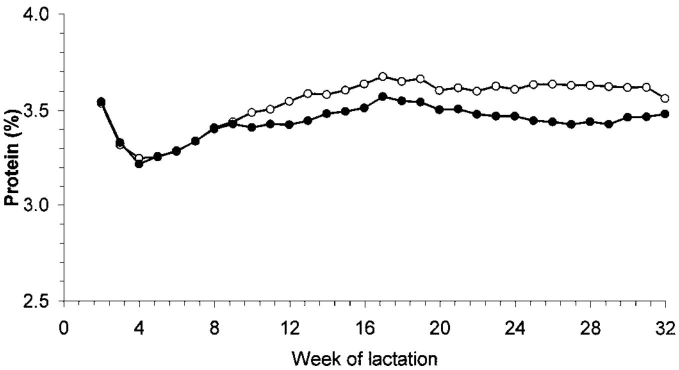

Figure 3. Mean milk protein concentration (%), adjusted for block and pre-treatment covariate, of Holstein-Friesian cows receiving 0 (Control ○) or 300 (Treated ●) mg/d of monensin (Eli Lilly and Co. Ltd, Basingstoke, Hants RG21 6XA, UK) in experiment 2 lactation 2.

The higher BW gain of the cows receiving the highest dose rate was not reflected in BCS, which was similar for all treatment groups. In Expt 2 there were no significant treatment effects on either BW or BCS (Table 3).

In Expt 2, L2 the use of monensin before calving decreased the β-hydroxybutyrate concentration in wk 2 to 4 ($P < 0.001$) and 6 to 8 ($P < 0.01$) postcalving (Table 4). Monensin also reduced ($P < 0.05$) acetoacetate values in wk 2 to 4 postcalving and increased glucose values in wk 2 to 4 ($P < 0.05$) and 6 to 8 ($P < 0.001$) postcalving. However, no differences were detected in these metabolites in wk 10 to 12.

Efficiency of Feed Utilization

Table 2 shows that the use of monensin increased ($P < 0.01$) milk yield/kg of DMI, with no significant effect of dose rate. Although not statistically significant, there was a 5% increase in NE milk/NE intake and NE efficiency.

DISCUSSION

The current study supports earlier trials, which established that the inclusion of monensin in dairy cow diets would increase milk yield (Lean and Wade, 1997). The likely mechanism of action to support additional milk yield is that monensin increased the supply of glucogenic precursors resulting from changes in pattern of rumen fermentation. The milk yield responses of 2.8, 2.5, and 1.5 kg/d in Expt 1 to the inclusion of 150, 300, and 450 mg/d of monensin in the diet were similar to 3.1, 2.5, and 1.6 kg/d recorded in Australia using the same dose rates (Wade et al., 1996). Although there were no significant differences between dose rates in either study, they both indicate a marked reduction in milk yield response at the highest dose rate. The milk yield responses of 0.8 and 1.1 kg/d recorded in Expt 2 L1 and L2 are similar to those generally reported in the literature, which range between 0.5 to 1.5 kg/d (Lean and Wade, 1997; McGuffy and Giner-Chavez, 1998).

Early studies established that the use of monensin in dairy cows reduced the molar proportions of acetate and butyrate and increased that of propionate and that these changes were associated with depressed milk fat concentration (Sauer et al., 1989). Although studies described by Lowe et al. (1991) and Ramanzin et al. (1997) have shown that milk fat depression ranged from 0.14 to 0.56%, the mean declines noted in Expt 1 and Expt 2 L1 and L2 of 0.46, 0.38 and 0.27% is close to the average values reported in the literature. The ability to manipulate milk composition is important in the existing European milk quota structure, as there is a milk fat base concentration of approximately 4.0% above which farmers are penalised.

A recent review of literature (Mackintosh, 1998) noted that in 12 out of 15 dairy cows studies, milk protein content of monensin-treated cows was lower than the untreated cows, but significant differences were generally not established. This was attributed to low cow numbers and the lack of statistical power (Lean and Wade 1997). In contrast the reduction in milk protein content of 0.16, 0.16, and 0.11% recorded in Expt 1 and Expt 2 L1 and L2 respectively, were all significant. The reduction in milk protein concentration may

Table 4. Mean values for β-hydroxybutyrate, acetoacetate (μmol/L) and glucose blood values (mmol/L) during the pretreatment period and for wk 2 and 4, wk 6 and 8 and wk 10 and 12 postcalving when cow received either 0 or 300 mg/d of monensin (Eli Lilly and Co. Ltd, Basingstoke, Hants RG21 6XA UK) in experiment 2, lactation 2.

	β-Hydroxybutyrate (μmol/L)			Acetoacetate (μmol/L)			Glucose (mmol/L)		
Monensin (mg/d)	0	300	RSD[1]	0	300	RSD	0	300	RSD
Pretreatment	490	382	277	128	100	101	3.21[b]	3.39[a]	0.38
Wk 2 and 4	524[a]	380[b]	178	100[a]	81[b]	41	3.38[b]	3.53[a]	0.28
Wk 6 and 8	489[a]	395	136	62	55	30	3.54[b]	3.69[a]	0.22
Wk 10 and 12	489	487	128	61	63	28	3.63	3.70	0.23

[a,b]Subcolumn means within row different superscripts differ ($P < 0.05$).

[1]Residual standard deviation.

be a consequence of the increased milk yield resulting in a dilution effect.

The improvement in efficiency of feed utilization recorded in Expt 1 with cows receiving 150 and 300 mg/d of monensin supports a recent study (McGuffy and Giner-Chavez, 1998), and is attributed to its effect on rumen fermentation with increased propionate and reduced methane production.

When monensin was introduced into the diet before calving (Expt 2 L2) 300 mg/d of monensin significantly reduced β-hydroxybutyrate and acetoacetate while increasing glucose plasma concentration. These changes are consistent with increased propionate and reduced acetate and butyrate concentration in the rumen (Sauer et al., 1989). Similar changes were reported in a large-scale field study conducted in Canada (Duffield et al., 1999) and are indicative of improved energy status in early lactation, and are associated with a reduced incidence of clinical and subclinical ketosis (Duffield et al., 1999; Erasmus et al., 1993; Jonker et al., 1996). The results from Expt 2 L2 support these earlier studies and indicate the potential role of monensin in the management of the transition cow.

CONCLUSIONS

Monensin produced a small decrease in DMI, an increase in milk yield that ranged from 0.8 to 2.8 kg/d, a significant depression in both milk fat and milk protein content, but little change in yield of milk constituents. In early lactation there was a significant decrease in β-hydroxybutyrate and acetoacetate concentration and an increase in blood glucose indicating the potential of monensin as a management tool for the transition cow.

REFERENCES

Chalupa, W. 1988. Manipulation of rumen fermentation. Pages 1–18 *in* Recent Developments in Ruminant Nutrition. W. Haresign and D.J.A. Cole, ed. Nottingham Univ. Press, Nottingham, UK.

Duffield, T. .F., D. Sandals, K. E. Leslie, K. Lissemore, B. W. McBride, J. H. Lumsden, P. Dick, and R. Bagg. 1999. Efficacy of monensin for the prevention of subclinical ketosis in lactating dairy cows. J. Dairy Sci. 81:2866–2873.

Erasmus. L. J., P. M. Botha, G. D. Lindsay, J. A. d'Assonville, and M. D. Viljoen. 1993. Effect of monensin supplementation and BST administration on productivity and incidence of ketosis in dairy cows. Proc. 7th World Conf. Anim. Prod. Edmonton, Canada 2:413–414.

Jalc, D., M. Baran, T. Vendrak, and P. Siroka. 1991. The effect of monensin on the fermentation of feed with different hay and concentrate proportions in (Rusitec) rumen pouch. Vet. Medicina 36:29–38.

Jonker, L. J., J.I.D. Wilkinson, and M. Tarrant. 1996. Alleviated nutrient imbalance by monensin premix (Rumensin®): reduced risk of ketonaemia in dairy cows. Pages 364–366 *in* Proc. 19th World Buiatrics Conf. Edinburgh, Scotland.

Lean, I. J., and L. Wade. 1997. Effects of monensin on metabolism, production, and health of dairy cattle. Pages 50–70 *in* Usefulness of Ionophores in Lactating Dairy Cattle. K. L. Leslie, ed. Univ. Guelph, Canada.

Lowe, L. B., G. J. Ball, V. R. Carruthers, R. C. Dobos, G. A. Lynch, P. J. Moate, and S. C. Valentine. 1991. Monensin controlled-release intraruminal capsule for control of bloat in pastured dairy cows. Aust. Vet. J. 68:17–20.

Mackintosh, E. D., R. H. Phipps, J. D. Sutton, and J.I.D. Wilkinson. 1996. Effect of monensin on *in vitro* semi-continuous rumen fermentation in the rumen simulation technique (Rusitec). Anim. Sci. 62:680. (Abstr.).

Mackintosh, E. D. 1998. The effect of monensin on *in vitro* rumen fermentation and *in vivo* rumen and total tract digestion and milk production in the dairy cow. Ph.D. Diss., The University of Reading, UK.

McGuffy, R. K., and B. Giner-Chavez. 1998. Lactation performance of dairy cows receiving monensin and a sustained release formulation of methionyl bovine somatotropin. J. Dairy Sci. 81(Suppl. 1):261. (Abstr.)

Mulvany, P. 1977. A body condition technique for use with British Friesian cows. Anim. Prod. 24:157–158.

Ramanzin, M., L. Bailoni, S. Schiavon, and G. Bittante. 1997. Effect of monensin on milk production and efficiency of dairy cows fed two diets differing in forage to concentrate ratios. J. Dairy Sci. 80:1136–1142.

SAS/STAT User's Guide Version 6. 1992 SAS Institute Inc., Cary, NC.

Sauer, F. D., J.K.G. Kramer, and W. J. Cantwell. 1989. Antiketogenic effects of monensin in early lactation. J. Dairy Sci. 72:436–442.

Tyrell, H. F., and J. T. Reid. 1965. Prediction of the energy value of cow's milk. J. Dairy Sci. 48:1215–1223.

Van Es, A.J.H. 1978. Feed evaluation for ruminants. 1. The system in use from May 1977 onwards in the Netherlands. Livest. Prod. Sci. 5:331–354.

Wade, L., B. Granzin, and B. Lowe. 1996. The effect of monensin in dairy cow rations on milk production and in reducing the severity of ketosis. Pages 289–290 *in* Proc. 19th World Buiatrics Conference, Edinburgh, Scotland.

Williamson, N. B., D. P. Hayes, and D. U. Pfeiffer. 1996. The influence of treatment with monensin capsules on milk production and reproductive performance of pasture fed dairy cows. Pages 377–380 *in* Proc. 19th World Buiatrics Conference, Edinburgh, Scotland.