THE EFFECT OF MONENSIN SODIUM ON LACTATIONAL PERFORMANCE OF AUTUMN- AND SPRING-CALVING COWS

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List of Abbreviations

a.m.	(*ante meridiem*) before midday
Ac:Pr	Acetate:Propionate
ADG	average daily gain
ANOVA	Analysis of Variance
A.P.U.	Animal Physiology Unit
^{o}C	degrees Celsius
DM	dry matter
EDTA	Ethylenediaminetetraacetic acid
FCE	feed conversion efficiency
≥	greater than or equal to
g	gram
g	gravity
H^+	hydrogen ion
h	hours
ha	hectare
ID	inner diameter
IU	international unit
l	litre
K^+	potassium ion
kg	kilogram
LCFA	long chain fatty acid
≤	less than or equal to
M	Molar
MANOVA	Multivariate Analysis of Variance
ME	metabolisable energy
MJ	megajoule
MLW	metabolic liveweight
mg	milligram

ml	millilitre
mm	millimetre
N	nitrogen
Na^+	sodium ion
NADPH	nicotinamide adenine dinucleotide (phosphate)
$NaHCO_3$	sodium bicarbonate
NEFA	non-esterified fatty acid
pH	potential hydrogen
p.m.	(*post meridiem*) after midday
OD	outer diameter
VFA	volatile fatty acid
VFI	voluntary feed intake
v/v	unit volume (of solute) per unit volume (of solvent)
w/v	unit weight (of solute) per unit volume (of solvent)

LEVELS OF STATISTICAL SIGNIFICANCE

NS	Not significant $P>0.10$
+	$0.10 > P > 0.05$
*	$0.05 > P > 0.01$
**	$0.01 > P > 0.001$
***	$0.001 > P$

CHAPTER I

INTRODUCTION

GENERAL OVERVIEW

A great deal of effort has been spent attempting to increase total production and efficiency of milk production by dairy cows. Some of this effort has been expended in the study of rumen fermentation with the aim of discovering methods of manipulating rumen fermentation to increase production from dairy cows. One rumen modifier, monensin sodium, has been widely studied and is a very effective method of manipulating rumen fermentation and increasing feed conversion efficiency in growing beef cattle. Despite ample evidence concerning the effects of ionophores on weight gain in cattle, information on the effects of monensin, and other ionophores, on the milk and milk component production of lactating dairy cows is very limited. This thesis is concerned with the effects of monensin sodium on the milk production of pasture-fed lactating dairy cows.

This chapter describes the pasture-based system of dairy production used in New Zealand. The effects of monensin sodium and other related ionophores, in the context of digestion and metabolism in the lactating ruminant, are discussed. Effects of ionophores on growing beef cattle, lactating beef cows and lactating dairy cows are reviewed. The mode of action of ionophores on the ruminal microbial cells and subsequent effects on ruminal fermentation and the products supplied to the host animal are also covered. Relationships between the known effects of ionophores on ruminant digestion and the responses observed in lactating dairy cows are discussed. Finally the information which is lacking and the area of study related to this thesis are outlined.

NEW ZEALAND DAIRY PRODUCTION

A low production cost system, using pasture fed *in situ*, allows the New Zealand dairy industry to sell produce on the world market at competitive prices despite its relative isolation from the markets. Improving the efficiency of conversion of pasture to milk would enhance the competitiveness of the industry. Furthermore, most of the milk produced in New Zealand is processed and manufactured into component products of varying value on the world market. Hence altering composition of milk to enhance production of high value components could be beneficial to the New Zealand dairy farmer.

Dairy farms in New Zealand are generally classified into two groups, seasonal supply and town milk farms. In 1988 seasonal supply farms made up 93% of all dairy farms and had an average herd size of 158 cows. Town supply farms made up 7% of the total herds and had an average herd size of 143 cows (New Zealand Dairy Board 1988). In an attempt to utilize the massive investment in milk processing facilities during the period of low production (i.e. winter), several New Zealand dairy companies have recently encouraged "out of season" milk production from seasonal supply farms by paying premium prices for milk produced at this time. This also ensures a steady supply of milk for domestic consumption and for export of short shelf-life products such as yoghurt.

Commercial dairy production in New Zealand is pasture-based and seasonal in nature. Pasture dry matter (DM) accumulation and feed quality is highest during the spring season. Consequently on most farms the herd is managed in such a way as to calve during late winter/early spring in an attempt to match maximum pasture growth rate with maximum feed requirements of the herd, which occurs during early to mid lactation.

Rotational grazing, of predominantly perennial ryegrass/white clover pasture, is used to manage feed by maintaining pasture growth rates, produce pasture with a high feeding value and manipulate feed supply to meet the herd's future requirements. Stocking rates of up to 150 cows/ha/day are often used in order to increase competition for pasture and increase "harvesting efficiency". Hence cows may often not be provided with an allowance high enough to enable maximum voluntary intake, especially during periods of poor pasture growth such as winter when low soil temperatures preclude rapid pasture growth, or summer when pasture is under moisture stress and in a reproductive phase of growth. Under pasture feeding conditions there is a substantial negative energy balance in early lactation. This negative energy balance has effects on milk and milk component yields and feed conversion efficiency (FCE).

All high producing dairy cows are in negative energy balance during early lactation and pasture-fed cows appear to be in a greater state of negative energy balance compared to concentrate-fed cows. Furthermore due to its chemical composition, autumn pasture appears to be conducive to a greater negative energy balance and therefore reduced milk production when compared to spring pasture. Processes which affect digestive efficiency can increase the energy supply to the cow, reduce the magnitude of negative energy balance, and therefore increase milk production. This increased energy supply would be of benefit to all cows in early lactation, but could be of greater value to cows milking in the autumn than those milking in the spring. One possible method of increasing digestive efficiency is through the use of rumen modifiers.

RUMEN MODIFIERS

Rumen modifiers are defined as agents which modulate selected pathways of microbial metabolism (Chalupa 1977). A number of products such as antibiotics, buffers (sodium bicarbonate, or magnesium oxide), or ionophores have been used to manipulate rumen fermentation. Carboxylic polyether ionophore antibiotics are a family of chemical compounds produced by fermentation of specific bacteria. Monensin sodium is a common ionophore produced by the bacteria *Streptomyces cinnamonesis*. Narasin, tetronasin, lysocellin, laidlomycin, lasalocid, and salinomycin are similar ionophores, produced by the same family of bacteria, which have been tested or are commercially available. By 1984 there were over 70 known ionophores (Schelling 1984). Monensin sodium was approved for feeding to beef cattle in the United States in 1976. By 1978 over 80% of cattle in feedlots were fed monensin. Such rapid acceptance attests to its effectiveness at improving feed conversion efficiency in growing beef cattle (Owens 1980). After several years of widespread use ionophores continue to improve animal efficiency and performance which suggests that sensitivity of rumen microbes is relatively stable and that the pattern of resistance in the ionophore-resistant species is due to a fundamental difference between microbial cells (Russell and Strobel 1989).

Substances capable of interacting stoichiometrically with metal ions, thereby serving as a carrier by which these ions can be transported across bimolecular lipid membranes, are defined as ionophores (Ovchinnikov 1979). Ionophores are able to shield and localise the charge of ions and facilitate their movement across membranes. They are highly lipophilic substances which are toxic to many bacteria, protozoa, fungi and higher organisms and therefore fit the classical definition of antibiotics (Pressman 1976).

All ionophores have similar modes of action but monensin is the only one being tested to any extent in New Zealand. This review will therefore concentrate on monensin but other ionophores will be considered where appropriate.

EFFECTS OF RUMEN MODIFIERS ON PRODUCTION

1) Growing beef cattle

Most data on the effects of ionophores relate to research and commercial use of monensin with growing beef cattle. Goodrich et al. (1984) summarised data from trials involving nearly 16,000 cattle and used regression techniques to determine overall (across-trial) effects of monensin on animal performance. With diets containing high levels of readily fermentable carbohydrates (i.e. concentrate-based diets), ionophores generally depress voluntary feed intake (VFI), but average daily gain (ADG) is not decreased and FCE is improved. When ruminants are fed diets containing considerable β-linked carbohydrates (i.e. roughages), ionophores do not depress VFI but ADG is improved, again resulting in improved FCE (Bergen and Bates 1984). Average (± standard deviations) responses to monensin treatment were; a 1.6% (± 8.5%) increase in ADG; a 6.4% (± 5.0%) reduction in VFI; and 7.5% (± 6.5%) reduction in feed/100 kg gain (Goodrich et al. 1984). Therefore FCE was substantially increased on average but, as is clear from the standard deviations of these means, the variation in response was considerable.

Goodrich et al. (1984) found that ADG of cattle accounted for the largest amount of variation in change in ADG. In trials where ADG was low, monensin improved ADG more than in trials where ADG was high. This suggests that cattle which are inefficient in converting feed to gain respond more to monensin than highly efficient cattle, or that cattle on poorer quality diets show a better response. However, cattle with higher DM intake, and therefore a higher metabolisable energy (ME) intake of the same type of concentrate diet, responded better in terms of ADG to monensin than cattle with low DM, and therefore reduced ME, intakes. These two ideas seem contradictory at first because generally cattle that have high ADG have a correspondingly high ME intake. This apparent anomaly is likely due to the use of data which were expressed as percentages. All treatment responses were expressed as a percentage of the control group. As an example, cattle with a low ADG of 0.5 kg/day and showing a 10% response to monensin relative to the control group would have an ADG of 0.55 kg/day. However cattle gaining at 1.5 kg/day and showing the same 0.05 kg/day response to monensin, relative to the control group, would have shown only a 3% increase in ADG. Cattle fed monensin on pasture have shown increases in rate of gain of up to 17%, but VFI of monensin-treated grazing animals has not been measured (Schelling 1984).

The effect of monensin on voluntary feed intake is variable. In trials where VFI was low, monensin-fed cattle consumed more DM than controls while monensin reduced VFI when DM intake was high (Goodrich et al. 1980).

Monensin consistently reduces feed:gain ratio (FCE) and therefore must reduce energy requirements per unit gain, improve dietary energy value or both (Goodrich et al. 1984). Goodrich and Thompson (1980) reported apparent digestibilities of 71.6% and 72.8% for cattle and sheep fed control and monensin-containing diets respectively. The FCE response of monensin-treated animals was generally greater in cattle that required a large amount of feed DM/100 kg gain (i.e. in cattle that had low FCE). Maximum improvement in feed efficiency was at an ME concentration of 12.1 MJ ME/kg DM and response was reduced when diets containing ME concentrations above or below this level were fed.

2) Lactating beef cows

Several authors have studied the effect of ionophores on milk production of lactating beef cows and the subsequent growth rate of their calves. Milk production of beef cows was measured using either a milking machine or the weigh-suckle-weigh method. The information from beef cows is useful but must be treated with some caution because generations of genetic selection for different production traits in dairy and beef breeds have created vastly different animals. Dairy cows normally produce 15 to 50 litres (l) of milk per day whereas milk production from beef cows has usually been measured at between 4 and 10 l per day. Beef cows are not normally energy-limited for milk production as is the case with high yielding dairy cows, particularly in early lactation.

Milk yield of lactating beef cows was either not affected (Lemenager et al. 1978a; Hixon et al. 1982a; Hixon et al. 1982b; Griggs and Males 1988) or reduced (Rennick et al. 1982) by monensin treatment. Hixon et al. (1982a) found that lasalocid fed to beef heifers increased milk production on a low energy diet, but not on a high energy diet, and therefore suggested that ionophore supplementation to diets marginally deficient in energy may improve milk production and calf performance with beef cows. Milk composition from beef cows has not been appreciably affected by monensin treatment (Lemenager et al. 1978a; Rennick et al. 1982; Griggs and Males 1988).

Although few changes were observed in terms of milk or milk component production following ionophore treatment, increased ADG of calves suckling monensin-fed cows has been observed (Lemenager et al. 1978a; Griggs and Males 1988). This suggests that the calves of monensin-fed cows utilized milk and/or forage more efficiently or that the nutritional value of the milk was increased (although this was not detected by standard analyses for fat and protein content). In some cases calves may have had access to monensin supplement as well which would have confounded comparisons with respect to milk yield of the dam.

Voluntary feed intake of lactating beef cows is reduced (Lemenager et al. 1978a) or unaffected (Rennick et al. 1982) by monensin treatment.

3) Lactating dairy cows

Information on the effects of ionophores on dairy cattle is quite limited. In some cases the trials have been done with a small number of animals or over a very short time period, therefore the adaptation period necessary for the rumen microbial population to stabilise with treatment was inadequate. Several important variables in the data, such as stage of lactation and diet of the cows, could also have had large effects on the results.

Monensin treatment has been shown to increase milk production of pasture-fed cows (Pankhurst and McGowan 1978; Kube et al. 1988; Lynch et al. 1990). Cows fed mixed roughage and concentrate diets have also shown increased milk production when treated with monensin (Mohsen and El-Keraby 1981; Van Beukelen et al. 1984). However Pankhurst et al. (1977) found no effect of monensin treatment on milk production of cows fed *ad lib* or 85% *ad lib* cut pasture in stalls.

Milk fat content was reduced by monensin treatment (Pankhurst et al. 1977; Pankhurst and McGowan 1978; Van Beukelen et al. 1984; Kube et al. 1988). In cases where milk yield was increased and fat content decreased, monensin treatment did not change fat yield (Mohsen and El-Keraby 1981; Lynch et al. 1990).

Milk protein content was either unchanged (Pankhurst et al. 1977; Pankhurst and McGowan 1978; Kube et al. 1988; Lynch et al. 1990) or increased (Mohsen and El-Keraby 1981) in response to monensin treatment. Therefore protein yield increased or stayed constant.

Pankhurst et al. (1977) reported that monensin reduced VFI level although Pankhurst and McGowan (1978) found no change. Kube et al. (1988) found that cows fed monensin had slightly higher lactational persistency, possibly due to a reduced rate of utilization of body fat in early lactation, although body weight data were not given. Milk fat from treated cows contained a lower proportion of long chain fatty acids (LCFA), indicating reduced utilization of body reserves (Kube et al. 1988)

Several experimenters measured the effect of lasalocid on milk production of lactating dairy cows. Lasalocid reduced milk yield (Johnson et al. 1988), or had no effect (Beede et al. 1986; Dye et al. 1988; Weiss et al. 1990). Milk fat % and yield were reduced (Dye et al. 1988; Johnson et al. 1988) or unchanged (Beede et al. 1986; Weiss et al. 1990). Lasalocid generally had no effect on protein % (Beede et al. 1986; Dye et al. 1988; Johnson et al. 1988; Weiss et al. 1990) but in one case protein yield was reduced due to the reduced milk yield (Johnson et al. 1988). A latin square experimental design with three levels of lasalocid dosage and a control group was used by Dye et al. (1988). This experimental design would cause control periods to immediately follow treatment periods which could allow serious carry-over effects if the modifications to rumen metabolism caused by the ionophore did not stop immediately upon cessation of the treatment. Therefore this design could have affected the results and masked possible treatment effects.

In summary responses of dairy cows to ionophores have been variable. In most cases milk yield has remained constant or increased, fat % has decreased and protein % has remained constant. Consistent patterns of response are difficult to determine because of the wide range of experimental methods used, the relatively small number of experiments conducted and the generally small responses (where they occur at all).

NUTRIENT UTILIZATION BY THE DAIRY COW

The conversion of feed to milk by the dairy cow involves many mechanisms. Feed consumed by the cow is initially digested and fermented by the rumen bacteria, protozoa and fungi, providing nutrients primarily for microbial growth (Hungate 1966). Byproducts of this digestion and the microbial mass so generated are available as nutrients for the host animal.

Acetic, propionic, butyric, iso-butyric, valeric and iso-valeric volatile fatty acids produced as byproducts of anaerobic fermentation by the rumen microbes are absorbed directly across the rumen epithelium. Undigested food material and the microbial mass pass out of the rumen into the host's intestinal system where enzymatic digestion, similar to that of monogastric animals, takes place. A small amount of glucose (Lindsay 1978) and vitamins and minerals are absorbed in the small intestine. Protein is broken down to peptides and amino acids and absorbed into the blood stream in the small intestine. The relative quantities and proportions of these nutrients supplied to the host by fermentation, digestion, and absorption are important in determining the potential milk and milk component production of the cow. Milk and milk component yield is particularly dependent on the production of glucose, volatile fatty acids (VFA), amino acids, and LCFA from feed.

Milk volume produced by the cow is largely determined by lactose secretion of the mammary gland. Glucose is the main carbon substrate for lactose synthesis (Oldham and Emmans 1989) and propionate is the primary precursor for glucose. In a ruminant animal gluconeogenesis in the liver and kidney is the primary supplier of glucose (Oldham and Emmans 1989) because ≤ 10% of circulating glucose is actually absorbed intact from the gastrointestinal tract (Lindsay 1978). Acetate and β-hydroxybutyrate tend to be used as lipid precursors, thus they are called "lipogenic" volatile fatty acids. Propionate, iso-butyrate, valerate, and iso-valerate VFA's produced in the rumen are normally used as glucose precursors are called "glucogenic" (Lindsay 1978).

The level of milk fat and protein produced by the cow is dependent on the level of precursors supplied to the mammary gland. About 50% of the fatty acids in milk fat are synthesized within the mammary gland from acetate (40%) and β-hydroxybutyrate (10%). Of the remaining 50%, most is synthesized from preformed plasma triglyceride in the blood (Oldham and Emmans 1989). Short chain fatty acids (C_4 to C_{10}) in milk are synthesized from acetate and β-hydroxybutyrate. Fatty acids in milk with 18 or more carbon atoms are synthesized from blood plasma triglycerides and fatty acids in milk with chain lengths between 10 and 18 carbon atoms originate from either source. Long chain fatty acids can be absorbed from the gut or from mobilisation of LCFA from body tissue.

Fermentation of digestion end-products such as hexoses and amino acids is accompanied by substantial losses in both energy and amino-nitrogen. Many nutritionists have attempted to minimise this loss of nutrients by altering dietary ingredients or their relative proportions. Several feed additives including monensin sodium are used in attempt to improve efficiency of ruminant digestion or reduce bloat. These agents may influence milk yield and composition by altering the balance of absorbed nutrients. Increases in acetate, glucose and protein supply have positive effects on milk yield; increases in acetate, butyrate and LCFA supply have positive effects on milk fat content; increases in propionate and protein supply have positive effects on milk protein content; and increases in propionate and glucose supply may have negative effects on milk fat content (Thomas and Martin 1988).

MODE OF ACTION OF MONENSIN

1) Mode of action on microbial cells

All living cells, including those of ruminal microbes, require maintenance of specific ionic gradients between internal and external cell fluids. Bacteria often contain much higher concentrations of solute than does the external environment (Pressman 1976). Ionophores act as proton (H^+) couriers in one direction and metal cations in the opposite direction across a membrane. Sodium (Na^+) and potassium (K^+) are examples of metal cations commonly transported by ionophores. Intact membranes are required for ionophore activity. Interference with normal ion gradients plays a dominant role in the mode of action of ionophores within the rumen (Romatowski 1979; Isichei and Bergen 1980). Monensin has been shown to dissipate proton gradients across the cell membranes causing the cell to utilize its total intracellular ATP reserves to regenerate the proton gradient in *Bacillus subtiles* (Joliffe et al. 1981) and has also been shown to dissipate the transmembrane Na^+ gradient of several bacteria (Andu et al. 1982; Kitada and Horikoshi 1982).

Gram negative bacteria are generally ionophore-resistant due to biochemical mechanisms which can overcome the reduction in transmembrane ion gradients caused by ionophores (Bergen and Bates 1984; Russell and Strobel 1989). Protozoa, fungi, and gram-positive bacteria which lack a protective outer membrane, and therefore lack the ability to overcome the ion gradient reduction, are usually sensitive to ionophores (Russell and Strobel 1989). Reports about the effects on rumen microbes agree that monensin is a metabolic inhibitor of hydrogen- and formate-producing bacteria, and stimulatory to succinate- and propionate-producing bacteria (Bergen and Bates 1984; Russell and Strobel 1989). Monensin has also been found to be inhibitory to 4 of 7 strains of lactic acid-producing bacteria but was not inhibitory to three strains that ferment lactate to propionate (Dennis et al. 1981).

The intracellular pH of most bacteria appears to be highly regulated and constant at around 7.6 to 7.8 while the pH of rumen fluid ranges from 5.7 to 7.3

(Bergen and Bates 1984). When the pH of the rumen fluid increases the transmembrane proton gradient declines and *vice versa*. The relative concentrations of Na^+ and H^+ may play an important role in the physiological activity of monensin (Bergen and Bates 1984). Addition of sodium bicarbonate ($NaHCO_3$) as a buffer to monensin-containing diets blocked the normal shift in ruminal VFA production to propionate (Weber 1979; Rogers and Davis 1982).

Most of the monensin results are from beef cattle which were fed concentrate-based diets known to cause acidic fermentation and would therefore be expected to be associated with a high transmembrane H^+ gradient and low pH in the rumen fluid. Perski et al. (1982) showed that monensin completely inhibited methane production of *Methanobacterium thermoautotrophicum* when the extracellular sodium concentration was low and the pH of the rumen fluid was 6.0. At a higher pH of 6.7 the effect was dramatically reduced and at a pH of 7.5 the effect of monensin on methane production was completely eliminated. Therefore the effect of monensin on the ruminal microbes is greatest when the pH of the rumen fluid is low and this effect is reduced as the pH of the rumen fluid increases.

In summary ionophores allow ions to flow across normal healthy cellular membranes. This action reduces the internal-external ion gradient which is necessary for normal metabolism of microbial cells. Some strains of microbes can overcome this reduction in ion gradient and are therefore resistant to ionophores. The ionophore-sensitive strains which produce hydrogen, ammonia, and lactate are reduced in number. Reduction of the ion gradient or increase in pH of the rumen fluid reduces the effectiveness of ionophores.

2) Rumen system mode of action

The basic mode of action of monensin is to modify the movements of ions across biological membranes. It is assumed that the basic mode of action results in the system modes of action which can be consolidated into seven categories. Monensin treatment has been associated with modification of:

a) Rumen volatile fatty acid production.
b) Rumen gas production.
c) Rumen protein digestion
d) Rumen dilution rate and rumen fill level.
e) Voluntary feed intake.
f) Feed digestibility.
g) Other responses.

a) Volatile fatty acid production

The most commonly observed change in rumen fermentation associated with feeding ionophores is decreased molar proportions of acetate and butyrate, with a concomitant increase in propionate production (Richardson et al. 1976; Chalupa 1977; Bergen and Bates 1984; Russell and Strobel 1989) and therefore a decreased acetate to propionate ratio (Goodrich et al. 1984; Schelling 1984). This increased propionate production at the expense of acetate, rather than a simple increase in total VFA production, has been documented by measurement of total VFA production levels in rumen fluid (Richardson et al. 1976) and by isotope dilution techniques (Van Maanen et al. 1978) in cattle. In most experiments the concentration of total VFA in rumen fluid has not been altered by monensin treatment (Richardson et al. 1976). Volatile fatty acids are absorbed in the same proportions as they are present in the rumen fluid so that the proportions present in rumen fluid are a close approximation of the ratio of VFA's absorbed by the host (Richardson et al. 1976).

Fermentation of glucose to pyruvate by rumen microbes is associated with production of hydrogen. The largest quantities of hydrogen are used in the formation of propionate and butyrate from pyruvate and in the reduction of carbon dioxide to methane (Chalupa 1980). The differences in production and utilization of metabolic hydrogen cause the efficiency of fermenting hexose to acetate, propionate, and butyrate to be 62%, 109%, and 78% respectively (Hungate 1966; Chalupa 1980). Therefore the metabolically useful energy recovered in fermentation end products can be increased by enhancing the production of propionate and to a lesser extent butyrate at the expense of acetate.

Propionate is more flexible as an energy source for the host, having the potential of being used for gluconeogenesis or for direct oxidation via the citric acid cycle (Bergen and Bates 1984; Russell and Strobel 1989). The glucose so produced can be used for oxidation or as a carbon skeleton for non-essential amino acids (Russell and Strobel 1989).

Ionophore treatment has not altered the concentration of total VFA in rumen fluid of beef cattle (Richardson et al. 1976), lactating beef cows (Lemenager et al. 1978b; Rennick et al. 1982; Griggs and Males 1988) or lactating dairy cows (Van Beukelen et al. 1984; Kube et al. 1988). A reduction in total VFA concentration was found in rumen fluid of lactating dairy cows by Pankhurst et al. (1977), Dye et al. (1988) and in a second experiment by Van Beukelen et al. (1984). The reduction in total VFA production found by Van Beukelen et al. (1984) could be a result of taking the samples five days after treatment commenced, at which time the microbial population may have been in a state of change and therefore not completely adjusted to the treatment.

Ionophore treatment has also been associated with decreased ruminal proportions of acetate and butyrate, and increased propionate in growing beef cattle (Bergen and Bates 1984), lactating beef cows (Lemenager et al. 1978b; Rennick et al. 1982; Griggs and Males 1988), and lactating dairy cows (Dye et al. 1988; Johnson et al. 1988). Weiss et al. (1990) found that on day 7 of the treatment period, lactating dairy cows fed lasalocid had a lower ruminal acetate to propionate ratio than controls. However by day 28 the effects on VFA proportions and energetic efficiency had disappeared suggesting that the effect was short lived and that the ruminal adaptation to lasalocid is less than 28 days. Dye et al. (1988) found that total VFA concentration of rumen fluid was reduced and that the proportion of acetate was reduced in lactating dairy cows treated with ionophores.

The response of ruminal VFA production to ionophore treatment has been relatively consistent in all cattle, i.e. little or no change in total VFA production, a reduction in molar proportions of acetate and butyrate and an increase in the proportion of propionate. Most of these data are from animals fed concentrates. This change in VFA production appears to be beneficial to growing beef cattle because of the increased efficiency of energy metabolism. However it is not well documented if this altered nutrient balance is advantageous to lactating dairy cows.

b) Gas production

Ionophore feeding reduces methanogenesis but ionophores are not particularly toxic to methanogens (Russell and Strobel 1989). Hydrogen- and formate-producing bacteria, protozoa and fungi are inhibited by ionophores but succinate- and propionate-producing bacteria are more tolerant (Bergen and Bates 1984; Russell and Strobel 1989). It appears that reduced methanogenesis is due to reduced production of hydrogen, the primary substrate of methanogenesis, in the rumen. All reports indicate reduced methane production in concentrate-fed beef cattle (Joyner et al. 1979; Thornton and Owens 1981; Bergen and Bates 1984) but the level of inhibition ranges from 4% to 31% (Bergen and Bates 1984).

Methane production, which can be as high as 12 l/day in a mature bovine, can represent a loss of feed energy of as much as 12% (Russell and Strobel 1989). Since the rumen is an anaerobic ecosystem, substrate oxidations must be closely coupled to reduction reactions. When methanogenesis is decreased the concentrations of other reduced products, such as propionate, increase at the expense of acetate and more feed energy is available for productive purposes (Russell and Strobel 1989).

c) Protein utilization

A considerable proportion of the protein consumed by ruminants is fermented to ammonia and volatile fatty acids by rumen microbes. The primary goal when manipulating ruminant nitrogen transactions is to increase the balance and level of amino acids entering the small intestine from the rumen (Chalupa 1977). Because ruminal ammonia production often exceeds the capacity of the ammonia-utilising species to absorb it, ammonia can accumulate in the rumen. Excess ammonia is absorbed across the rumen wall and converted to urea by the liver. Some urea is recycled back to the rumen but much of it is lost in urine.

Monensin reduces ammonia production *in vitro* and *in vivo* (Bergen and Bates 1984; Schelling 1984; Russell and Strobel 1989) and this decrease is probably associated with a reduction in the extent of ruminal protein degradation (Schelling 1984; Russell and Strobel 1989). Decreases in ammonia concentration of the rumen fluid, which are also commonly associated with monensin treatment, are consistent with depressed deamination and/or proteolysis. Ionophores inhibit the growth of proteolytic ruminal bacteria and therefore inhibit degradation of protein and peptides.

Adaptation of the microbes to ionophores may be an important factor in the efficacy of ionophores with respect to the protein-sparing effect. Poos et al. (1979) showed that, in steers adapted to monensin, less bacterial nitrogen (N) reached the small intestine and the proportion of dietary protein reaching the small intestine increased relative to that in untreated animals. Monensin increased amino acid flow on a diet containing dried brewers grains but not on a diet where urea was the only nitrogen source. However Beever et al. (1987) found that monensin had no significant effect on nitrogen digestion when steers were fed a diet of mixed perennial ryegrass and white clover, or white clover only, possibly because the adaption period allowed in that study was only 14 days.

Ionophores have been associated with reduced ruminal degradation of dietary protein when the animal is fed concentrate diets containing intact protein. Decreases in ammonia concentration of the rumen fluid, which are also commonly associated with monensin treatment, are consistent with depressed deamination or proteolysis.

d) Rumen fill and rate of passage.

Rumen fill and rate of passage play a major role in ruminant digestion. These factors have a direct effect on the extent and site of digestion, the extent of microbial fermentation and the end products of fermentation and N utilization.

Dilution rate is generally higher in animals fed forage vs concentrate diets (Chalupa 1977). Estimates of dilution rates for individual animals eating the same diet can have large variation, indicating individual differences. The many complexities of this system mode of action preclude a full understanding of the effects at this point (Schelling 1984). The whole area of study related to dilution rate and its effects on digestive products is incomplete and no clear cause and effect relationship has been determined.

Increasing the flow of the fluid fraction from the rumen, by any means such as addition of artificial saliva or poly-ethylene glycol, is associated with decreased proportions of propionate and increased acetate, butyrate and microbial amino N flow. Stoichiometric calculations indicate lower fermentative efficiency at higher dilution rates because less hydrogen is recovered in VFA (Chalupa 1977; Schelling 1984). Increasing the flow of solids which do not require pregastric digestion will reduce losses incurred in fermentation of carbohydrate and amino nitrogen. However, solids which require microbial degradation, such as fibre, may be digested less completely in the rumen. The reserve fermentation capacity of the large intestine may compensate for all or a portion of this reduced digestion (Schelling 1984). This suggests that the reduced VFI associated with monensin treatment is actually a response to the reduced rumen outflow rate.

Ionophores decrease rumen turnover rate and increase rumen fill (Schelling 1984) and may be the cause of most of the associated systems effects. Monensin consistently decreased the ruminal rate of passage, of steers and heifers fed coastal bermuda grass pasture, by an average of 15.2% (Pond et al. 1980). Rumen liquid turnover rate and solids turnover rate were reduced 31% and 44% respectively by monensin treatment (Lemenager et al. 1978b). Rumen liquid volume was reduced by monensin but rumen dry matter volume remained constant. However in a second trial when steers were limit-fed a high concentrate diet, VFI was constant yet monensin still decreased rumen turnover rate (Lemenager et al. 1978b). Faulkner et al. (1985) found that rumen outflow rate of particulate matter was increased but liquid outflow rate was not changed by monensin treatment.

Several possible explanations exist for the decreased rumen turnover rate (Schelling 1984). Rumen turnover rate may be decreased as a response to reduced VFI. However Lemenager et al. (1978b) found that rumen outflow rate decreased even when feed intake was limited and therefore constant. The associated reduction in rumen turnover rate would then be a cause for and not an effect of reduced intake.

Although a great deal remains unknown about rumen outflow rate and its specific effect on rumen products, the ruminal responses to ionophores discussed previously mimic the responses of an artificially induced reduction in rumen turnover rate very closely. The selective reduction in number of some microbes could reduce the rate of carbohydrate digestion and the extent of protein digestion in the rumen. This would reduce the rumen outflow rate and change the pattern of VFA production. The similarity in magnitude and response would point to most other responses being an effect of the altered rumen fill and turnover rate (Schelling 1984).

e) Voluntary feed intake

The effect of monensin on VFI is well documented. Monensin reduces VFI about 10.7% in cattle fed high concentrate diets and about 3% in cattle fed roughage diets (Bergen and Bates 1984; Schelling 1984). Some of the depression observed in experiments may be due to introducing cattle abruptly to monensin. Other studies indicate a 5% reduction in VFI after 112 days on the treatment (Schelling 1984). Johnson et al. (1988) found that lasalocid reduced VFI of dairy cows when added to a diets containing high fat levels.

Voluntary feed intake reduction could be due to a decrease in rate, but not necessarily extent, of ruminal digestion. If rate of digestion is reduced and passage out of the rumen via the omasal orifice is limited by particle coarseness, the increased time required to complete particle size reduction would increase rumen retention time and slow rumen turnover (Schelling 1984). The decreased energy intake may be offset by compensating factors such as:

1) increased propionate production
2) reduced methane production
3) reduced heat loss
4) reduced time grazing
5) reduced metabolic faecal energy loss

This would explain the reduction in VFI, along with the associated increase in ME values of feeds, and therefore no associated reduction in animal performance.

f) Digestibility modification

Addition of monensin reduced the apparent dry matter digestibility of harvested range grass *in vitro* with inocula from both control and monensin-adapted steers but the reduction was of greater magnitude in the control groups (Lemenager et al. 1978b). Monensin-treated steers fed a high fibre cornstalk-based diet supplemented with 20% of a high quality protein supplement had increased apparent organic matter and neutral detergent fibre digestibility, relative to controls, with intermediate levels of monensin (12.2 and 18.8 parts per million) being superior to a high level (36.6 parts per million) of monensin (Faulkner et al. 1985). Monensin did not affect digestibility of cotton fibre, dietary carbohydrate or nitrogen when incubated *in vitro* with rumen fluid from steers fed a chopped orchard grass hay diet (Dinius et al. 1976). Cellulose digestibility from cotton strips was not affected *in vivo* (Dinius et al. 1976) however monensin treatment reduced the ruminal acetate:propionate ratio. Digestibility of dry matter, crude protein, hemicellulose and cellulose were not affected (Dinius et al. 1976). File et al. (1980) found that organic matter digestibility of ground, pelleted lucerne fed to lambs was not affected by monensin although the ruminal acetate:propionate (Ac:Pr) ratio was decreased. Apparent digestibilites of DM and protein showed no change with lasalocid treatment (Johnson et al. 1988). However Dye et al. (1988) found that crude protein digestibility was increased by lasalocid treatment.

Changes in nitrogen digestibility associated with monensin are affected by the nature and level of protein in the diet. Increased nitrogen digestibility has been shown in sheep, goats and cattle when ionophore-containing diets were fed (Schelling 1984). However when urea was fed the results were inconsistent. Again factors such as rumen fill and rate of passage which have been shown to affect protein digestibility independent of ionophore treatment may be involved (Schelling 1984). Poos et al. (1979) found that monensin reduced dry matter digestibility and nitrogen retention. However, by days 40-46 of treatment, the values for these parameters were similar in the treatment and control groups indicating the possibility of an adaptation effect.

Goodrich and Thompson (1980), in a summary of six studies, stated that monensin increased apparent digestibility of protein by 6.5% (58.6% vs 62.4% digestibility). The greatest response to monensin in ADG, reduction in VFI, and improvement in FCE occurred in diets that contained preformed protein as opposed to non-protein nitrogen. Thus these data may also suggest a reduction in the magnitude of protein digestion in the rumen associated with monensin treatment. This would allow an increased supply of amino acids, which are commonly the most limiting nutrient to increased production.

The effects of monensin on digestibility are variable and often unfavourable during the adaption period. Several studies suggest that monensin may initially produce a negative effect on apparent digestibility followed by a slight increase, but this has not been thoroughly studied (Schelling 1984).

In vitro experiments with mixed ruminal microbes have often shown a decrease in cellulose digestion after the addition of monensin. Many *in vivo* studies showed no decline in fibre digestibility during monensin treatment although feed intake generally decreases (Russell and Strobel 1989). When intake declines the dilution rate of solid material from the rumen decreases and there is a longer period of time for digestion. Rumen cellulolytic bacteria are very sensitive to a decline in pH and therefore cellulose digestion could be enhanced by decreases in lactate production and a rise in ruminal pH associated with ionophores. This is a good example of a case where the effect *in vivo* may be quite different from that observed *in vitro* and may explain why cellulose digestion is not normally decreased *in vivo*, as expected, when ionophores are fed.

Overall it appears that monensin does result in a slight improvement in protein digestibility in many different conditions. Much of the early work was done with *in vitro* cultures which would not adapt like a normal rumen culture.

g) Other responses

Monensin appears to increase ME values of feeds by increasing hydrogen retention in propionic acid and slightly increasing DM digestibility. In addition monensin may reduce ruminal methane energy loss and reduce fasting heat production. Taken collectively these modifications of energy metabolism are sufficiently large to explain differences between control and monensin-containing diets in feed efficiency of beef cattle (Goodrich et al. 1984).

Several other responses have been commonly noted when ionophores are fed. Van Maanen et al. (1978) found that glucose turnover increased 14% when monensin was fed. Increased glucose production from the increased propionate supply has been shown to reduce the use of amino acids for gluconeogenesis in the liver (Beede et al. 1976).

Ionophores have also been associated with reduced incidence of bloat and reduced lactic acidosis (Bergen and Bates 1984). Bloat is a common disorder in ruminants and is particularly common when animals are fed pasture which is growing rapidly and contains significant quantities of legume species.

3) Monensin Mode of Action in Relation to Milk Production Responses

The responses of dairy cows, in terms of milk production, to ionophore treatment can generally be explained by the systems modes of action. However the data from lactating dairy cows are very limited and come from widely varying feeding programmes and treatment lengths. Most of the previous work has involved concentrate diets rather than the pasture feeding system used in New Zealand.

Monensin and lasalocid have been shown to increase propionate production and decrease acetate production in the rumen. The lactating dairy cow in early lactation is in a state of negative energy balance and therefore the change in VFA production could allow more metabolically useful energy to be produced from the same amount of feed. The form of this increased energy supply would have two potential benefits to milk production. Increased propionate would increase the level of gluconeogenic precursors and therefore allow hepatic glucose production to increase. This in turn could allow increased mammary lactose synthesis and increase total milk volume. A second benefit would be to reduce the need for amino acids as gluconeogenic precursors. This fact, coupled with the reduction of rumen dietary proteolysis, could allow increased amino acid availability to the mammary gland. This would explain the ability of ionophore-treated cows to increase milk volume, maintain protein content and therefore increase protein yield.

The fact that ionophores reduce acetate and butyrate production would suggest a reduction in the level of milk fat precursors. This could explain why milkfat content is normally reduced or unchanged, and therefore fat yield remains unchanged or decreases, in treated cows. A selective increase in protein but not fat yield could be financially advantageous to the New Zealand dairy industry.

AUTUMN VS SPRING MILK PRODUCTION

Autumn and winter milk production has often led to significantly lower peak production per cow during early lactation and reduced production per hectare when compared to spring and summer production (Baldwin 1989). Lowered milk production at this time has not been fully researched but decreased rates of gain of sheep and cattle on autumn pasture as opposed to spring pasture have been attributed to differences in the chemical composition of the pasture (Lonsdale and Taylor 1971; During and Weeda 1972; Joyce and Brunswick 1975). These factors may also affect milk production. Chemical analysis has shown that the largest single difference in composition is the level of water-soluble carbohydrate in the plant material (Corbett et al. 1966; Scott et al. 1976; Beever et al. 1978; MacRae et al. 1985). Autumn pastures with low soluble carbohydrate levels have been shown to produce a rumen fermentation which has a high acetate:propionate ratio compared to that of spring pastures. The reduced growth rate of sheep and cattle is attributed to inefficient utilization of metabolisable energy (ME) because of a shortage of reducing agents, such as nicotinamide adenine dinucleotide phosphate (NADPH), which are normally produced from oxidation of propionate and glucose. As a result the acetate is used in the less efficient tricarboxylic acid (TCA) cycle. Therefore any method of manipulating the rumen fermentation to increase the propionate production of pasture-fed cows in autumn may have a double benefit for the lactating dairy cow. First, the increased supply of glucose precursors would allow increased lactose synthesis and therefore milk production. Second, an increase in the level of reducing agents would allow more efficient utilization of absorbed acetate and may increase total milk fat production.

APPLICATION OF MONENSIN TO THE NEW ZEALAND SITUATION

Based on previous results it is reasonable to expect positive effects of monensin treatment on milk production in the New Zealand dairy production system. Because the amount of data from trials involving ionophore-treated cows is so limited, many questions remain. Monensin and other ionophores have well documented anti-coccidial and anti-bloat effects in all types of cattle. The bloat effect has been linked to a marked depression of rumen fluid viscosity (Bergen and Bates 1984). Monensin is presently used in dairy cows as a method of bloat control but the effect of ionophores on milk production is unclear.

The optimum dosage level for beef cattle has been well established but treatment level for dairy cows has historically been estimated from beef cattle results and therefore needs clarification. Perhaps a lower level of treatment than the common 330 mg/day would provide more subtle rumen manipulation and therefore favourable milk production results. Furthermore the most appropriate timing of ionophore treatment is not well researched. The lactating cow in early lactation is in negative energy balance to an extent that the limited improvement in efficiency provided by ionophores could be of little productive consequence. However in late lactation or the dry period this increased efficiency might be of greater benefit. Ionophores have traditionally been administered to beef cattle fed *ad lib* concentrate diets. However, grazing dairy cows are at times, such as late spring, limit fed to manage pasture quality. The effects of ionophores on milk production at different levels of feeding is not known. The effects of different feeding programmes on ruminal digestion parameters such as rumen outflow rate, methane production, and fibre digestion, are relatively well characterised for concentrate diets but not so well for fresh forage diets. It is also well known that the rumen pH, and therefore the ion gradient between the internal and external environment of the ruminal bacteria, is critical to the activity of ionophores. However it is not known if the ruminal parameters of a forage digestion are suitable for optimum ionophore activity. Analysis of a feed quality by ionophore treatment interaction on milk production would also suggest the type of supplemental feed which would best complement pasture and monensin treatment. The overall effect of treatment on milk and milk component production of ionophore-treated pasture-fed cows has not been characterised.

PURPOSE AND SCOPE OF THIS INVESTIGATION

As discussed previously, there is evidence that monensin treatment increases milk and milk protein yields while reducing or not affecting milk fat yield, at least in concentrate-fed cows. The situation is less clear with respect to growing animals, primarily because of historical limitations in the administration of rumen modifiers to this group.

Rumen modifiers must be administered to the animal in accurate and consistent doses in order to be effective. With housed animals this is commonly done by mixing the material in the feed ration, however treatment of pasture-fed dairy cows has, until recently, required daily drenching. The drenching method has the disadvantages of being labour intensive and potentially dangerous due to dosage inaccuracies. Recently the development of intraruminal controlled release capsules (CRC) has allowed accurate, consistent and safe administration of compounds which require small dosages, such as ionophores, to grazing livestock. Thus the potential now exists for widespread use of rumen modifiers in grazing animals.

The nutritive value of autumn pasture, as measured by the growth rate of sheep and cattle and by chemical analysis, has been shown to be lower than that of spring pasture. Therefore the possibility of a differential response to monensin treatment, in terms of milk and milk component production, in the two seasons does exist. A study to determine the extent of such a treatment x season interaction would ideally be carried out using a single group of cows, managed in such a way as to have equal numbers of autumn- and spring-calving cows on the same farm. It would also be necessary to have the two groups at the same physiological state, i.e. stage of lactation, when measuring milk production parameters in each season. The necessary level of resources and lead time for this type of trial however make it impractical, particularly on commercially-oriented farms which tend to have either spring- or autumn-calving herds. Alternatively pasture could be cut in one season and stored, likely by freezing, for feeding in the other season. Preservation does however affect subsequent chemical composition and digestion characteristics of forage, therefore this method is not appropriate. Thus no simple method exists of comparing interactions between monensin treatment and pasture type (autumn vs spring) in a single experiment free of confounding effects such as the different management practices on different farm types, and other seasonal effects (eg. photoperiod). The strategy adopted in this study was to measure the effect of monensin sodium treatment on milk and milk component production in autumn-calving cows on one farm and in spring-calving cows on a nearby farm. The studies were linked by examining the effect of monensin on the ruminal digestion characteristics of fresh-cut autumn- and spring-pasture, similar to that fed to the cows, in a single group of ruminally-fistulated castrated male sheep. The sheep were not lactating therefore the effect of treatment was not confounded with physiological state of the animal.

The objectives of this study were therefore to:

1. Examine the effects of monensin sodium, delivered by intraruminal controlled release capsule, on lactational performance, liveweight, condition score and blood parameters in autumn- and spring-calving cows on two separate farms.

2. Determine whether there are monensin treatment x age interactions in lactational performance, i.e. whether monensin is more effective in stimulating the performance of young vs older cows.

3. Examine interactions between the effects of monensin treatment and pasture type (autumn vs spring) on rumen digestive function and blood parameters in non-lactating sheep.

CHAPTER II

THE EFFECT OF MONENSIN SODIUM ON LACTATIONAL PERFORMANCE OF AUTUMN- AND SPRING-CALVING COWS

The Effect of Monensin Sodium on Lactational Performance of Autumn- and Spring-Calving Cows

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ABSTRACT

The effects of monensin sodium, administered at the rate of 320 mg/day for 100 days by intraruminal controlled release capsule, on lactational performance and related parameters were examined in pasture-fed lactating dairy cows. The study was conducted in two seasons, with 60 autumn- and 60 spring-calving Friesian cows divided into two balanced groups, treatment and control. Responses were measured in terms of yield of milk, fat and protein, liveweight, condition score and plasma levels of insulin, glucose, β-hydroxybutyrate, urea and non-esterified fatty acids (NEFA).

The effect of season and monensin treatment on ruminal digestion characteristics was measured in a single group of ruminally-fistulated castrated male sheep which were housed indoors in metabolism crates and fed fresh-cut autumn and spring pasture similar to that fed to the cows. Responses were assessed in terms of ruminal volatile fatty acid (VFA) molar proportions, ruminal ammonia and pH levels, feed apparent digestibility and nitrogen balance. Blood parameters, similar to those measured in the cows, were also evaluated.

Autumn pasture had significantly lower proportions of water-soluble carbohydrate ($P<0.05$), cellulose ($P<0.05$) and lignin ($P<0.05$) and increased pectin ($P<0.05$), hemicellulose ($P<0.05$) and crude protein ($P<0.10$) levels when compared to spring pasture. Voluntary feed intake by sheep of autumn pasture was lower ($P<0.001$) than that of spring pasture and was significantly ($P<0.05$) reduced by monensin treatment. Monensin treatment significantly decreased the molar proportions of acetic acid ($P<0.10$) and butyric acid

(P<0.001) and increased the molar proportions of propionic acid (P<0.001) and minor VFA's (P<0.01). Nitrogen retention of the sheep was significantly (P<0.05) reduced by monensin treatment. Plasma glucose levels were increased (P<0.10) by monensin treatment during the fourth 5-day collection period in both seasons.

The monensin treatment did not significantly affect milk or milk component yield of the cows in either season. There was a trend towards increased milk and protein yield in monensin-treated cows of the spring-calving group but this difference did not attain statistical significance. The age x monensin treatment interaction was significant (P<0.05) for milk yield of spring-calving cows, 2- and 3-year old cows having increased milk yield as compared to the remaining age groups. The spring-calving cows had significantly (P<0.05) higher plasma urea levels when treated with monensin. The autumn-calving cows showed significantly (P<0.05) higher plasma glucose levels and reduced loss of body condition (P<0.10) in response to monensin treatment.

Although monensin treatment altered ruminal VFA proportions in the sheep in a manner consistent with previous reports, these effects were not associated with improved lactational performance in the cows. Thus while monensin sodium administered by controlled release capsule may be a useful method of controlling bloat in cows it does not appear to give reliable responses in lactational performance.

INTRODUCTION

Several methods are presently available to manipulate rumen fermentation with the ultimate goal of increasing milk and milk component yield of lactating dairy cows. However most techniques necessitate daily treatment of the cow with "rumen modifiers", commonly by mixing the compounds in the feed ration. The development of intraruminal controlled-release capsule technology has allowed the treatment of grazing animals, such as pasture-fed dairy cows in New Zealand, with rumen modifiers. One such rumen modifier, monensin sodium, is now being used commercially in New Zealand to reduce bloat but effects of this treatment on lactational performance are not clear.

Data explaining the effects of ionophore treatment on weight gain of concentrate-fed growing beef cattle are abundant but information on the effects on milk and milk component production of pasture-fed lactating dairy cows is conflicting. Monensin treatment has reduced voluntary feed intake, increased average daily gain and increased feed conversion efficiency of concentrate-fed growing beef cattle (Bergen and Bates 1984). These production responses are believed to be caused by several modes of action, the most important of which is altered ruminal volatile fatty acid production i.e. an increase in propionate production at the expense of acetate and butyrate production (Schelling 1984). Monensin treatment has increased milk yield of dairy cows fed pasture (Pankhurst and McGowan 1978; Kube et al. 1988; Lynch et al. 1990), or mixed roughage and concentrate diets (Mohsen and El-Keraby 1981; Van Beukelen et al. 1984). However Pankhurst et al. (1977) found that monensin treatment did not affect milk production of cows fed *ad lib* or 85% *ad lib* cut pasture in stalls. Milk fat content was reduced by monensin treatment (Pankhurst et al. 1977; Pankhurst and McGowan 1978; Van Beukelen et al. 1984; Kube et al. 1988). In cases where milk yield was increased and fat content decreased, monensin treatment did not change fat yield (Mohsen and El-Keraby 1981; Lynch et al. 1990). Milk protein content was either unchanged (Pankhurst et al. 1977; Pankhurst and McGowan 1978; Kube et al. 1988; Lynch et al. 1990) or increased (Mohsen and El-Keraby 1981) in response to monensin treatment. Therefore protein yield increased or remained constant. This evidence suggests that monensin treatment increases milk and protein, but not fat, yield of lactating dairy cows.

Recent developments in marketing of New Zealand dairy products have encouraged increased autumn/winter milk production as compared to production during the more traditional spring/summer season. However the level of autumn/winter milk production, in terms of individual cow peak yield or milk yield per hectare (ha), has been shown to be lower than that of spring/summer production (Baldwin 1989). Causes of lower production at this time are not fully researched. However, decreased rates of gain of sheep and cattle on autumn pasture as compared to spring pasture have been attributed to differences in the pasture chemical composition. Therefore the possibility exits of differential responses to monensin treatment, in terms of milk and milk component production, in the two seasons.

Commercial use of the intraruminal controlled-release capsule has facilitated the consistent and accurate administration of small quantities of compounds such as rumen modifiers to grazing livestock. The objective of this study was therefore to examine the effect of monensin treatment, administered by intraruminal controlled-release capsule, on the lactational performance of autumn- and spring-calving cows fed pasture.

MATERIALS AND METHODS

Experiment 1: Autumn-calving cows

Sixty multiparous Friesian cows aged between two and eleven years were selected from a single dairy herd at No. 1 Dairy Farm, Massey University. These animals were randomly assigned to either treatment or control groups. The groups were balanced for age, breeding index and production index, as well as pre-trial period liveweight, condition score, milk yield, fat yield and protein yield. The groups had equal numbers of cows aged 2,3,4,5 and ≥ 6 years

The sixty experimental animals were run together with a herd of 110 cows under commercial conditions at No. 1 Dairy Farm, Massey University. The cows were grazed on mixed perennial ryegrass/white clover pastures and had access to ample clean water at all times. Cows were milked at 0530 and 1530 h daily.

The treatment group was administered monensin sodium (320 mg/day) by intraruminal controlled release capsule (Rumensin Anti-Bloat Capsule, Elanco Products, Auckland, New Zealand), with an expected payout of 100 days, commencing on April 17, 1989. Treatment commenced at an average of 47 days post-calving.

Milk yield and composition were measured at two-weekly intervals commencing 1 day before treatment. At each sampling, evening and morning milk yields were recorded using "Tru-test" milk meters and composite p.m./a.m. samples were taken. Cows were weighed, condition scored and blood sampled every 4 weeks, commencing at 0900 h immediately after the morning milking of test day. Condition score of each animal was assessed by averaging values of three independent scorers at the time of weighing at weeks 0 and 12 relative to treatment, using the scoring system described by Holmes and Wilson (1984).

Blood was sampled by tail venipuncture at the time of weighing. An 8 millilitre (ml) blood sample was withdrawn into EDTA vacutainers ("Neotube", Nipro Medical Industries Ltd, Tokyo) and immediately placed on ice. Plasma was harvested by centrifugation at 3000 *g* and 4^{O}C for 20 minutes. The plasma was then transferred into duplicate vials and stored at -12^{O}C.

Experiment 2: Spring-calving cows

Experiment 2 was carried out with an identical protocol to experiment 1 with the following exceptions.

Experiment 2 began on September 7 1989 at Massey No. 4 Dairy Farm. The total herd size on the farm was 260 cows and the average days in lactation at commencement of the trial was 49.

Experiment 3: Sheep fed fresh cut pasture in autumn and spring

Twelve 1-year-old castrated male sheep were selected from the flock of ruminally fistulated animals at the Sheep and Beef Cattle Research Unit, Massey University. The animals had been fistulated at 6 months of age and during the experiment had 65 millimetre (mm) inner diameter (ID) cannulae. The sheep were randomly divided into two groups, treatment and control, which were balanced for liveweight.

The sheep were housed indoors at the Animal Physiology Unit (A.P.U.), Massey University during the two experimental periods which began on April 14, 1989 and September 6, 1989. Each study consisted of a 10 day adjustment period followed by four consecutive 5-day collection periods. The animals were kept in individual metabolism crates, tethered around the neck with a collar but otherwise were allowed free movement in the crate. The flooring of the metabolism crates consisted of a series of expanded mesh grates and a solid galvanised pan which allowed the daily collection of urine and faeces in separate fractions.

The treatment group was administered monensin sodium ("Rumensin", Elanco Products, Auckland, New Zealand) in the form of a premix which contained 10% active monensin sodium. Individual dosages were weighed out into tissue paper and calculated so they were equivalent to the dosage received by the cows based on a "gram of active ingredient per kg $LW^{0.75}$". Equal portions of the treatment were administered a.m. and p.m. by passing the paper pellet containing the premix into the rumen via the rumen cannula.

All feeding, feed refusal weighing and faeces and urine collection was done at 1600 h daily. The diet, of perennial ryegrass/white clover pasture, was obtained from paddocks at Massey No. 1 Dairy Farm (autumn) and Massey No. 4 Dairy Farm (spring) respectively. Paddocks from which the pasture was cut were selected to be of similar composition to those being grazed by the cows in experiments 1 and 2 respectively. Fresh pasture was cut daily at 1400 h with a sickle bar mower, raked into piles and transported to the A.P.U. in large bins.

The sheep were fed approximately 40% of their dry matter allocation in their individual feed bin. The remaining 60% of the feed allocation was placed on an individual overhead belt feeder which was calibrated to deliver an equal portion of fresh pasture into the feed bin each hour. Therefore the animals had access to fresh feed continuously. Total dry matter allocation was offered at approximately 1.10 times the previous day's consumption. Feed refusals were weighed each day and a 200 g sample was oven dried for 24 hours at 90^{o}C to determine refusal dry matter content. Each day a selection of random samples from the feed offered were mixed and sub-sampled. Duplicate 200 g samples were oven dried for 24 hours at 90^{o}C to determine dry matter content. A subsample of each days feed was pooled into a bulk sample for the five day collection period. This bulk sample was subsampled, freeze dried and ground through a 1 mm screen for subsequent analysis.

Faeces were collected from each animal and frozen in a plastic bag. The faeces were bulked for each five day collection period, then at the end of each period allowed to thaw and mixed. From this a 300 g sample was oven dried for 72 hours at 90^{o}C to determine faecal dry matter content. A 300 g sample was also freeze dried, ground through a 1 mm screen and stored for subsequent analysis. The 24 hour urine production was collected into a bucket containing 100 ml 50% (v/v) sulfuric acid (H_2SO_4) to reduce the pH below 3.0 and eliminate nitrogen loss by volatilization. Buckets were removed at 1600 h daily and the total daily urine volume measured in a graduated cylinder. A 5%

subsample was bulked over each five day collection period for each animal and frozen.

Rumen fluid was sampled at 1000 h on the day immediately prior to the start of monensin treatment and then at the end of each five day period. Rumen fluid was sampled through a rumen sampler which consisted of a hollow brass cylinder 100 mm long, 15 mm outer diameter (OD) with a series of 2 mm holes along its length. The brass section was attached to a plastic 3 mm OD tube which was 270 mm long. Rumen fluid samples were taken by attaching a 30 ml syringe to the plastic tube and drawing fluid through the rumen sampler. The first 15 ml of rumen fluid was discarded. Duplicate 10 ml samples were added to 2.5 ml of 1 M H_2SO_4 saturated with magnesium sulphate, centrifuged for 20 minutes at 2000 *g* and stored at -20^{o}C until analysed for ammonia. For VFA analysis, duplicate 5 ml samples were added to 1 ml of metaphosphoric acid/formic acid (18.75% w/v / 25% v/v) protein precipitant. One ml of 0.52% v/v isocaproic acid internal standard was added to one of the duplicates for use as an internal standard and 1 ml distilled water was added to the other for use as a control sample. Both samples were centrifuged at 2000 *g* for 20 minutes. The pH of rumen fluid was determined immediately on a PHM 61 Laboratory pH metre (Radiometer, Copenhagen Ltd.) after calibration with pH 7.0 and pH 4.0 buffers.

Blood was sampled by jugular venipuncture at 0800 h on the morning of rumen fluid sampling. An 8 ml blood sample was withdrawn into EDTA vacutainers ("Neotube", Nipro Medical Industries Ltd, Tokyo) and immediately placed on ice. Plasma was harvested by centrifugation at 3000 *g* and 4^{o}C for 20 minutes. The plasma was then transferred to duplicate vials and stored at -12^{o}C.

Chemical analyses

Milk samples were analysed for fat and protein content using an infra-red milk analyser ("Milko-scan", N. Foss Electric, Denmark).

Blood plasma was analysed for concentrations of : insulin by a double antibody radioimmunoassay (Flux et al. 1984) using bovine insulin (Sigma

Catalogue No. I-5500, 23.4 IU/mg) for iodination and reference standards; glucose, by the autoanalyser method of Rosevear et al. (1969); NEFA, using the modified colourimetric method described by McCutcheon and Bauman (1986); β-hydroxybutyrate using the method of Williamson and Mellanby (1974); and urea by the autoanalyser method of Marsh et al. (1965). Intra- and inter-assay coefficients of variation were : insulin 8.9%,12.2%; glucose 0.5%,1.0%; NEFA 2.6%,3.8%; β-hydroxybutyrate 2.5%,4.8%; and urea 1.0%,1.3%.

All nitrogen analyses were done using the Kjeldahl method on a Kjeltec Auto 1030 Analyser. Carbohydrate analysis of the feed samples was by the sequential carbohydrate extraction method of Bailey (1967). The concentrations of hemicellulose and cellulose were determined by photometric measurement of reducing sugars in neutralised extracts of their respective individual fractions.

VFA analyses were carried out by gas chromatography (Carlo-Erba HRGC 5300) on a "007" bonded phase capillary column (Quadrex Corp. U.S.A.). Integration of chromatogram peaks of molar proportions of VFA's was calculated using the "Maxima 820" computer package (Waters, Division of Millipore. U.S.A.).

Statistical analyses

For the purposes of the analyses, cows in experiments 1 and 2 were classified according to their age and treatment group (monensin vs control). Effects of these treatments (treated as fixed effects) and their first order interactions, on milk yield, fat yield, protein yield, liveweight, condition score and plasma insulin and metabolite concentrations were tested by repeated measures analysis of variance. Analyses were conducted separately for each experiment. Although included in the model, the terms age, time and their interaction are not discussed because they did not relate to hypotheses which the experiment was designed to test. Where interaction effects were significant the corresponding main effects are not reported because they cannot be tested (Tables 1,3,4).

Sheep in experiment 3 were classified according to their treatment group (monensin vs control) and season. Effects of season, treatment group, collection/sampling period and their first order interactions on dry matter intake, dry matter intake per unit metabolic liveweight, feed digestibility, nitrogen balance, rumen fluid VFA molar proportions and blood insulin and metabolite concentrations were initially tested by repeated measures analysis of variance with collection/sampling time as the repeated measure. None of the interactions between collection/sampling period and the other main effects were significant (i.e. effects of season and monensin treatment were constant across the four collection or sampling periods). Accordingly, data were pooled across the collection/sampling periods and univariate analysis applied to data averaged across the whole 20-day period after initiation of monensin treatment. Parameters that had pre-treatment values were analysed using this value, nested within season, as a covariate adjustment. If the covariate was found to be non-significant it was removed from the model. In the presence of significant interaction effects, main effects are not reported because they cannot be tested. All statistical analyses were conducted using the generalised linear models (G.L.M.) procedure of the S.A.S. statistical package (S.A.S. 1985).

RESULTS

Experiment 1: Autumn calving-cows

Milk production, liveweight and condition score

The effects of monensin treatment on milk yield, fat yield, protein yield, liveweight and condition score of autumn-calving dairy cows in experiment 1 are shown in Figure 1 and Figure 2. Yields of the production parameters decreased as the lactation continued. Liveweight and condition score both showed decreases as lactation progressed.

Monensin treatment did not affect the lactational performance over the entire experimental period. However there was a monensin treatment by age and a monensin treatment by time interaction on fat yield ($P<0.10$, Table 1). ANOVA of fat yield at each individual time showed that monensin treatment significantly ($P<0.05$) reduced fat production only at two and ten weeks after initiation of treatment. Although there was a significant age by treatment interaction on fat yield, the response did not change in a regular manner with age suggesting that the interaction was a chance effect (Table 2). Monensin treatment significantly ($P<0.10$) reduced the extent of loss of body condition during the experiment but had no effect on liveweight (Figure 2 and Table 1).

Table 1. Significance of effects of monensin treatment, monensin treatment by age interaction and monensin treatment by time interaction, on milk yield (MY), fat yield (FY), protein yield (PY), liveweight (LW) and condition score (CS) of cows, experiment 1.

Variable	MY	FY	PY	LW	CS
Monensin (M)	NS	N/A	NS	NS	+
M x Age	NS	+	NS	NS	NS
M x Time	NS	+	NS	NS	NS

N/A not applicable

Table 2. Effects of monensin treatment on fat yields (kg/day) of cows classified by age group, experiment 1.

Age	Control	Treatment	% Change[a]
2	.523	.407	-22.0
3	.440	.514	+17.0
4	.557	.582	+4.5
5	.708	.644	-9.0
≥6	.645	.586	-9.0

[a] Response to monensin expressed as a percentage of the control group yield

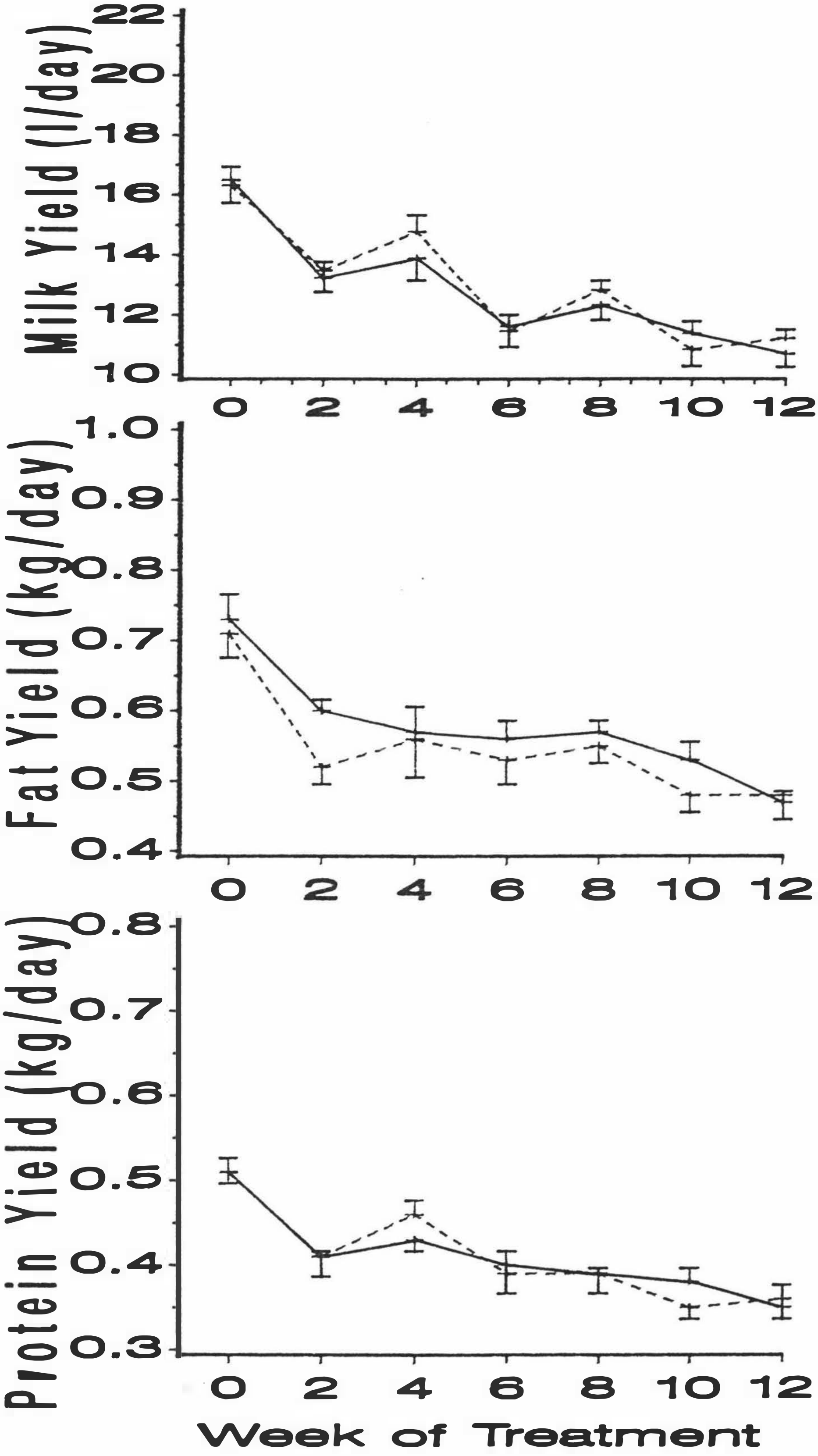

Figure 1. Yield of milk (upper panel), fat (middle panel) and protein (lower panel) of 30 control (——) and 30 monensin-treated (- - - -) cows at each sampling time, experiment 1. Weeks of treatment are expressed relative to the day of monensin capsule administration, day 0. Vertical bars represent standard errors about the mean.

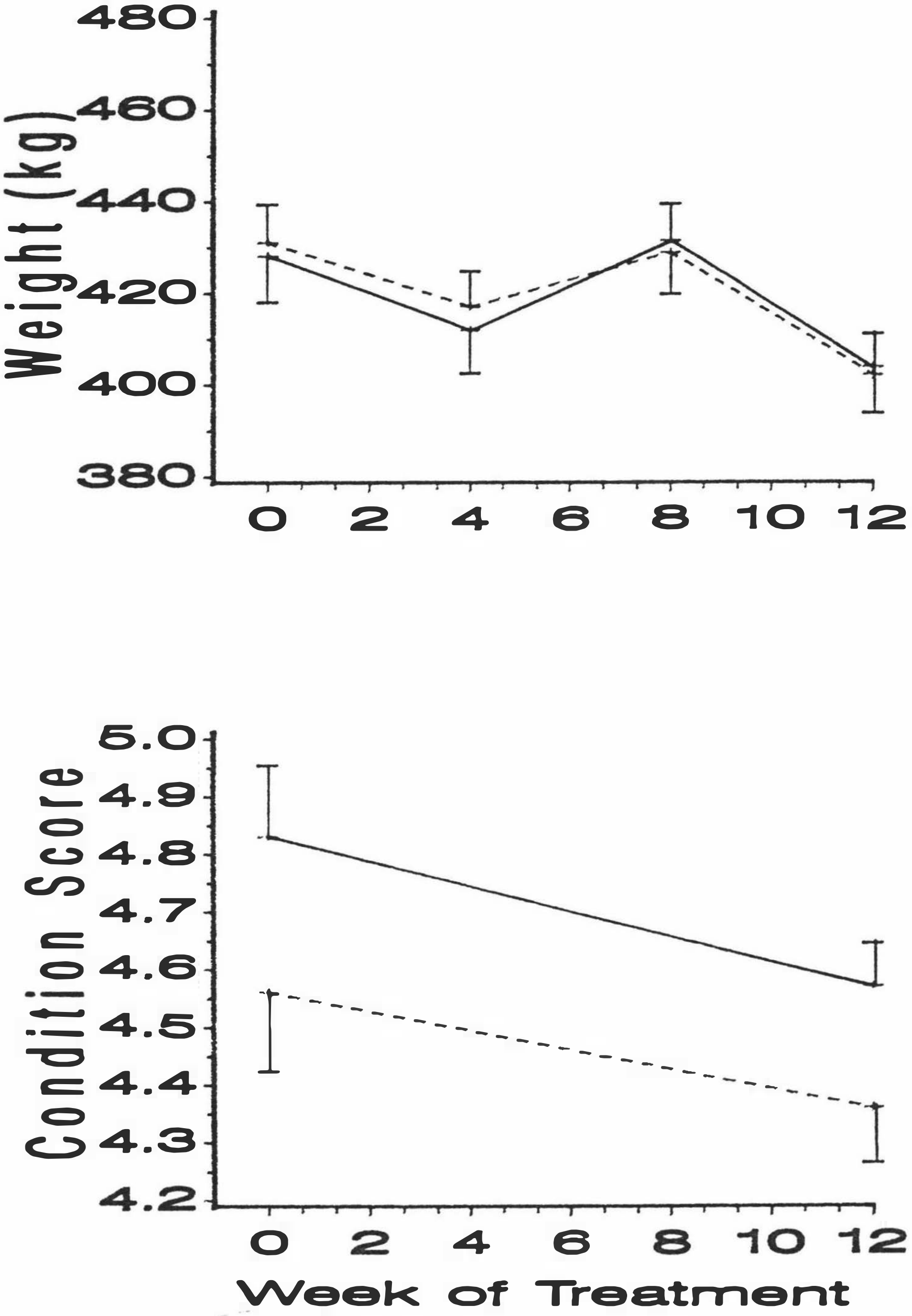

Figure 2. Liveweight (upper panel) and condition score (lower panel) of 30 control (——) and 30 monensin-treated (- - - -) cows at each sampling time, experiment 1. Weeks of treatment are expressed relative to the day of monensin capsule administration, day 0. Vertical bars represent standard errors about the mean.

Plasma hormone and metabolite levels

The effects of monensin treatment on plasma levels of insulin, glucose, β-hydroxybutyrate, urea and NEFA of lactating dairy cows in experiment 1 are shown in Figure 3 and Figure 4. Insulin and glucose levels increased as the lactation progressed however β-hydroxybutyrate and urea levels generally decreased with time. NEFA levels were inconsistent over time.

Monensin treatment significantly ($P<0.05$) increased glucose levels (Table 3). There was a significant ($P<0.05$) monensin treatment by time interaction on β-hydroxybutyrate level and ANOVA at each time showed that monensin treatment significantly ($P<0.10$) reduced the β-hydroxybutyrate level only at 4 weeks after the start of treatment.

Table 3. Significance of effects of monensin treatment, monensin treatment by age interaction and monensin treatment by time interaction, on plasma insulin (IN), glucose (GL), β-hydroxybutyrate (BHB), urea (UR) and NEFA levels of cows, experiment 1.

Variable	IN	GL	BHB	UR	NEFA
Monensin (M)	NS	*	N/A	NS	NS
M x Age	NS	NS	NS	NS	NS
M x Time	NS	NS	*	NS	NS

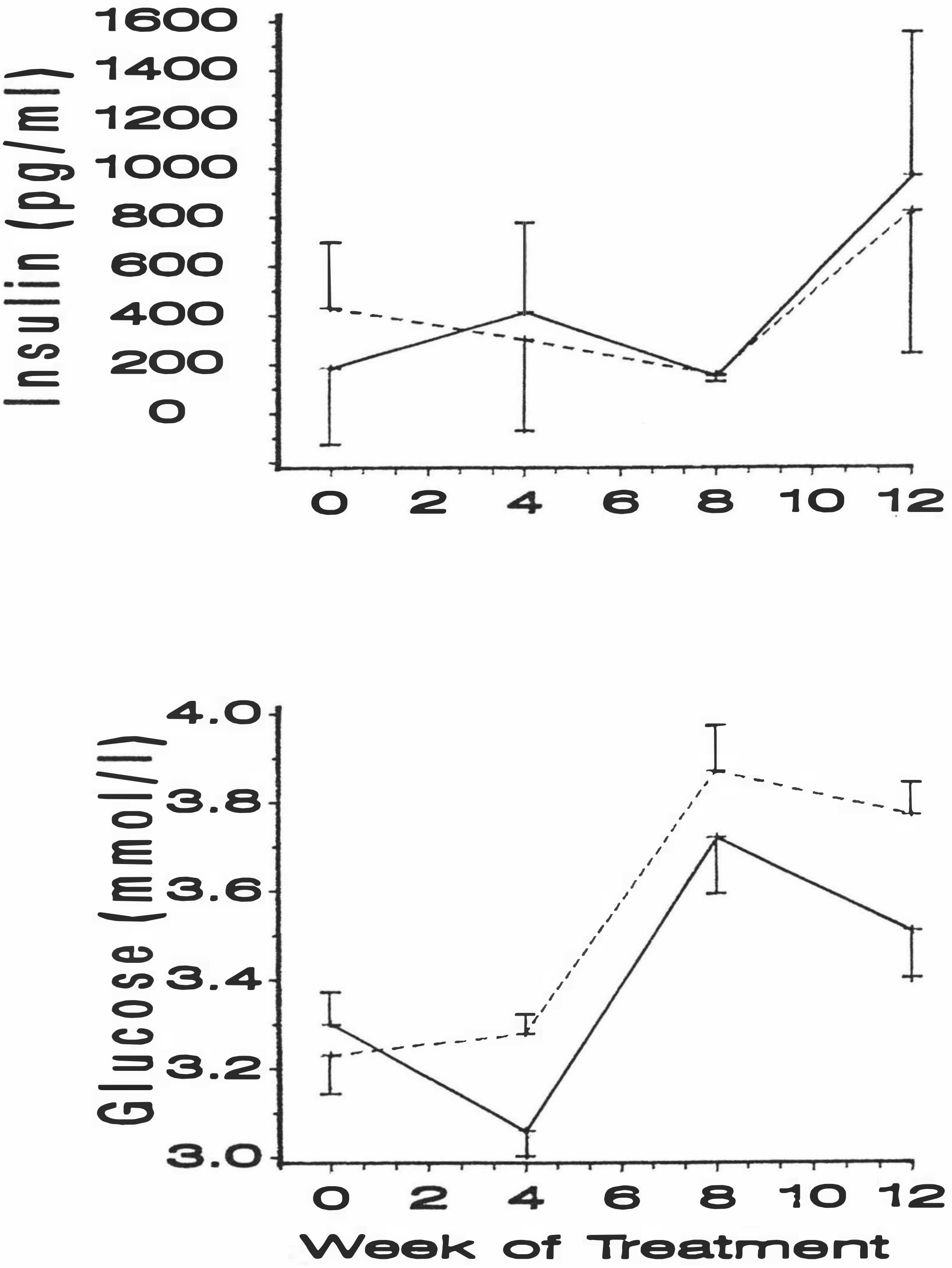

Figure 3. Plasma insulin (upper panel) and glucose (lower panel) levels of 30 control (——) and 30 monensin-treated (- - - -) cows at each sampling time, experiment 1. Weeks of treatment are expressed relative to the day of monensin capsule administration, day 0. Vertical bars represent standard errors about the mean.

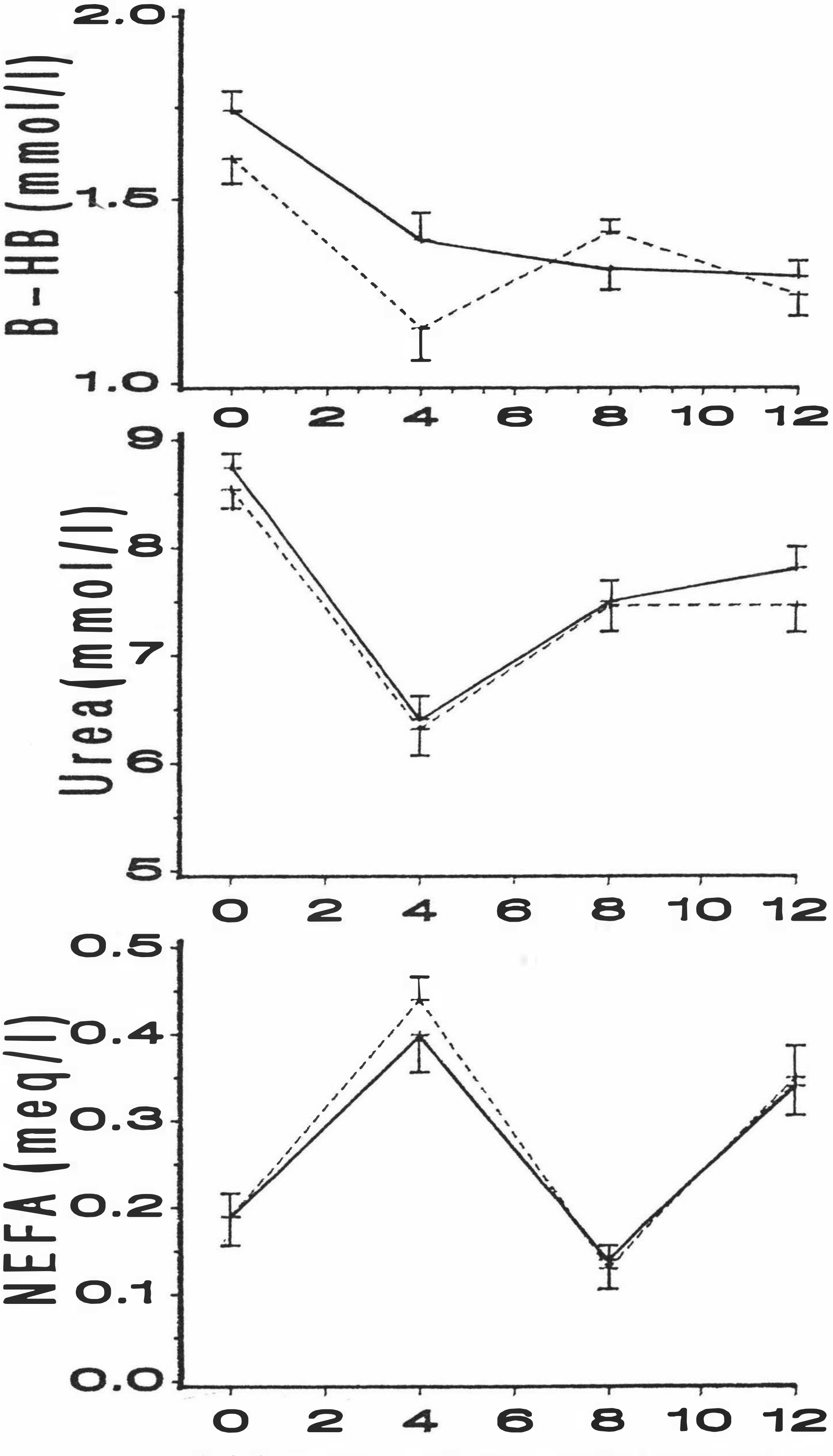

Figure 4. Plasma β-hydroxybutyrate (upper panel), urea (middle panel) and NEFA (lower panel) levels of 30 control (——) and 30 monensin-treated (- - - -) cows at each sampling time, experiment 1. Weeks of treatment are expressed relative to the day of monensin capsule administration, day 0. Vertical bars represent standard errors about the mean.

Experiment 2: Spring-calving cows

Milk production, liveweight and condition score

The effects of monensin treatment on milk yield, fat yield, protein yield, liveweight and condition score of spring-calving dairy cows in experiment 2 are shown in Figure 5 and Figure 6. As in experiment 1, the yield of milk and milk components decreased as the lactation progressed. Liveweight tended to increase and condition score decreased slightly with time.

These parameters were all unaffected by monensin treatment over the entire experimental period. There was a significant monensin treatment by age interaction on milk yield ($P<0.05$) and fat yield ($P<0.10$) (Table 4). Table 5 shows that both 2- and 3-year old cows had a small but positive increase in milk yield in response to monensin treatment. However a consistent response across ages was not apparent. Likewise while the age by monensin treatment interaction on fat yield was significant, the response did not change consistently across ages (Table 6). This suggests that both of the interactions were chance effects.

There was also a significant ($P<0.01$) monensin treatment by age interaction on condition score. The mean condition score of each age group (Table 7), again shows that the response did not change across ages consistently, suggesting that the interaction was a chance effect.

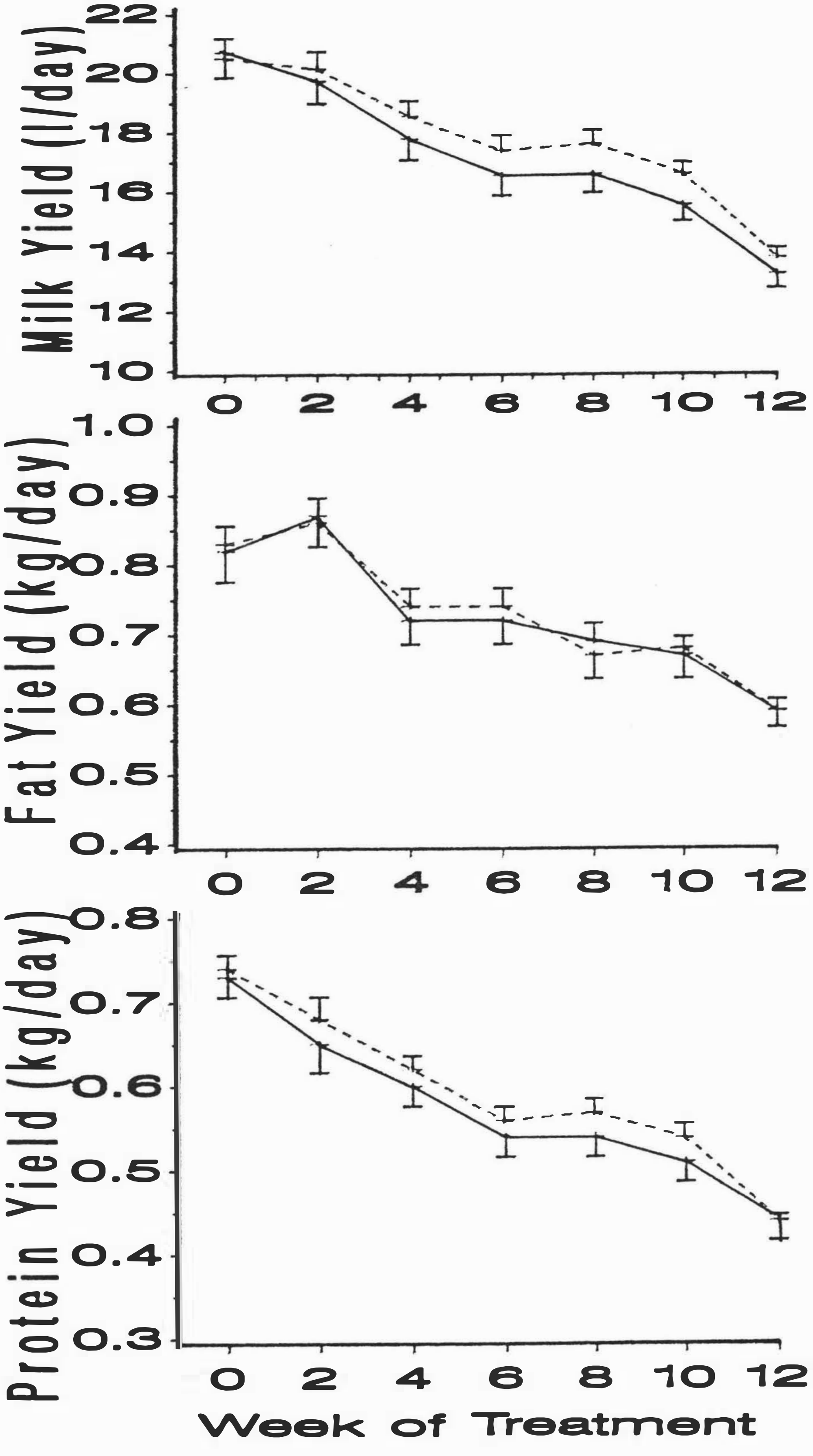

Figure 5. Yield of milk (upper panel), fat (middle panel) and protein (lower panel) of 30 control (——) and 30 monensin-treated (- - - -) cows at each sampling time, experiment 2. Weeks of treatment are expressed relative to the day of monensin capsule administration, day 0. Vertical bars represent standard errors about the mean.

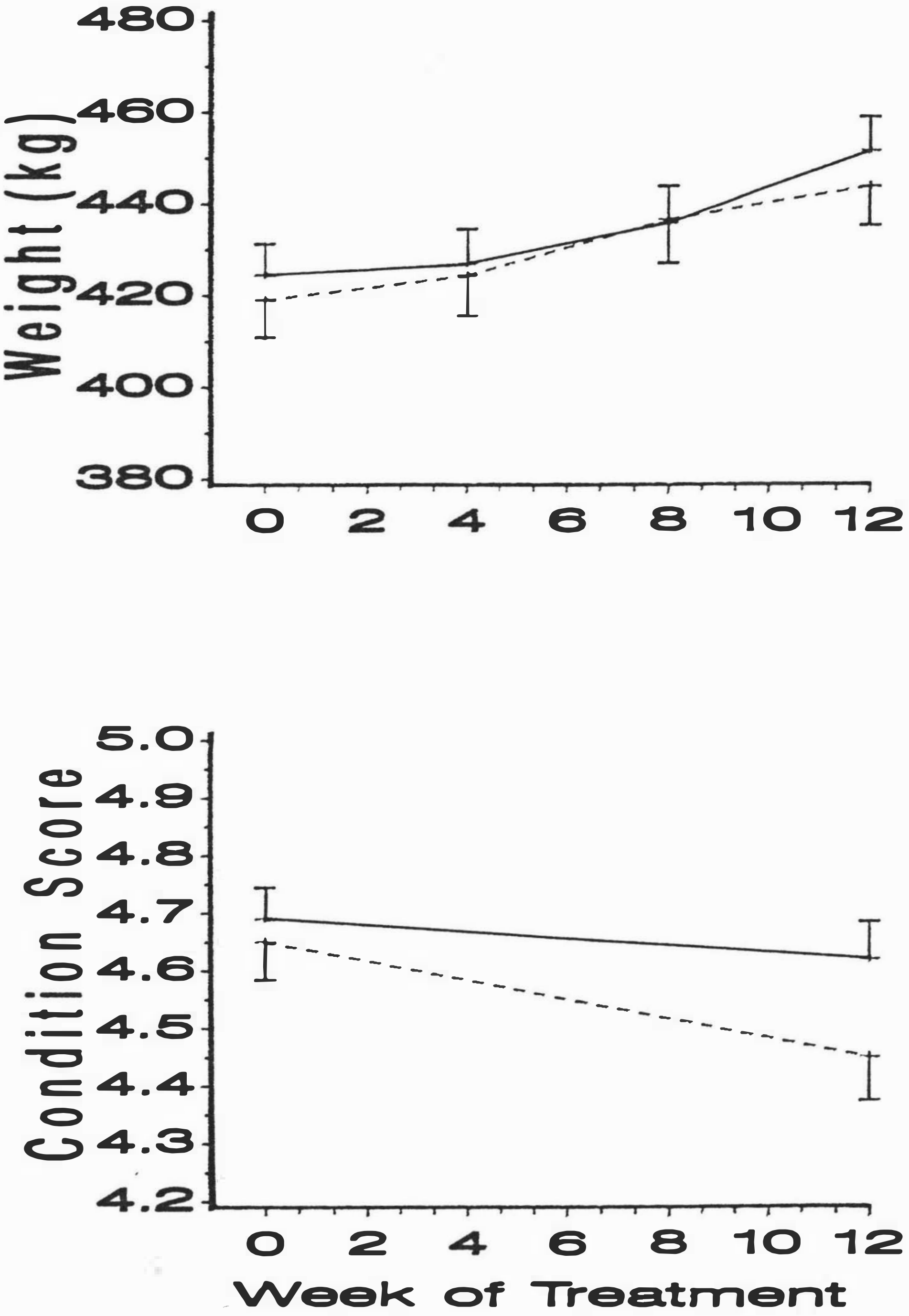

Figure 6. Liveweight (upper panel) and condition score (lower panel) of 30 control (——) and 30 monensin-treated (- - - -) cows at each sampling time, experiment 2. Weeks of treatment are expressed relative to the day of monensin capsule administration, day 0. Vertical bars represent standard errors about the mean.

Table 4. Significance of effects of monensin treatment, monensin treatment by age interaction and monensin treatment by time interaction, on milk yield (MY), fat yield (FY), protein yield (PY), liveweight (LW) and condition score (CS) of cows, experiment 2.

Variable	MY	FY	PY	LW	CS
Monensin (M)	N/A	N/A	NS	NS	N/A
M x Age	*	+	NS	NS	**
M x Time	NS	NS	NS	NS	NS

Table 5. Effects of monensin treatment on milk yields (kg/day) of cows classified by age group, experiment 2.

Age	Control	Monensin	% Change[a]
2	14.77	15.03	+1.8
3	14.76	15.22	+3.2
4	19.11	19.13	0.0
5	19.25	17.44	-9.4
≥6	18.03	21.85	+21.1

[a] Response to monensin expressed as a percentage of the control group yield

Table 6. Effects of monensin treatment on fat yields (kg/day) of cows classified by age group, experiment 2.

Age	Control	Monensin	% Change[a]
2	.598	.661	+11.5
3	.690	.597	-13.5
4	.815	.785	-3.7
5	.760	.765	+0.6
≥6	.762	.853	+11.9

[a] Response to monensin expressed as a percentage of the control group yield

Table 7. Effects of monensin treatment on condition score of cows classified by age group, experiment 2.

Age	Control	Monensin	% Change[a]
2	4.43	4.64	+4.7
3	4.77	4.40	-7.8
4	4.99	4.44	-11.0
5	4.44	4.83	+8.8
≥6	4.63	4.45	-3.9

[a] Response to monensin expressed as a percentage of the control group condition score.

Plasma hormone and metabolite levels

The effects of monensin treatment on plasma levels of insulin, glucose, β-hydroxybutyrate, urea and NEFA of lactating dairy cows in experiment 2 are shown in Figure 7 and Figure 8. Insulin level was variable and glucose decreased rapidly between 4 and 8 weeks of treatment. β-hydroxybutyrate and urea increased steadily until week 8 of treatment and then appeared to decrease rapidly. NEFA concentrations were relatively consistent across time. Monensin treatment significantly ($P<0.05$) increased plasma urea levels but had no effect on the concentrations of insulin or the other metabolites (Table 8).

Table 8. Significance of effects of monensin treatment, monensin treatment by age interaction and monensin treatment by time interaction, on plasma insulin (IN), glucose (GL), β-hydroxybutyrate (BHB), urea (UR) and NEFA levels of cows, experiment 2.

Variable	IN	GL	BHB	UR	NEFA
Monensin (M)	NS	NS	NS	*	NS
M x Age	NS	NS	NS	NS	NS
M x Time	NS	NS	NS	NS	NS

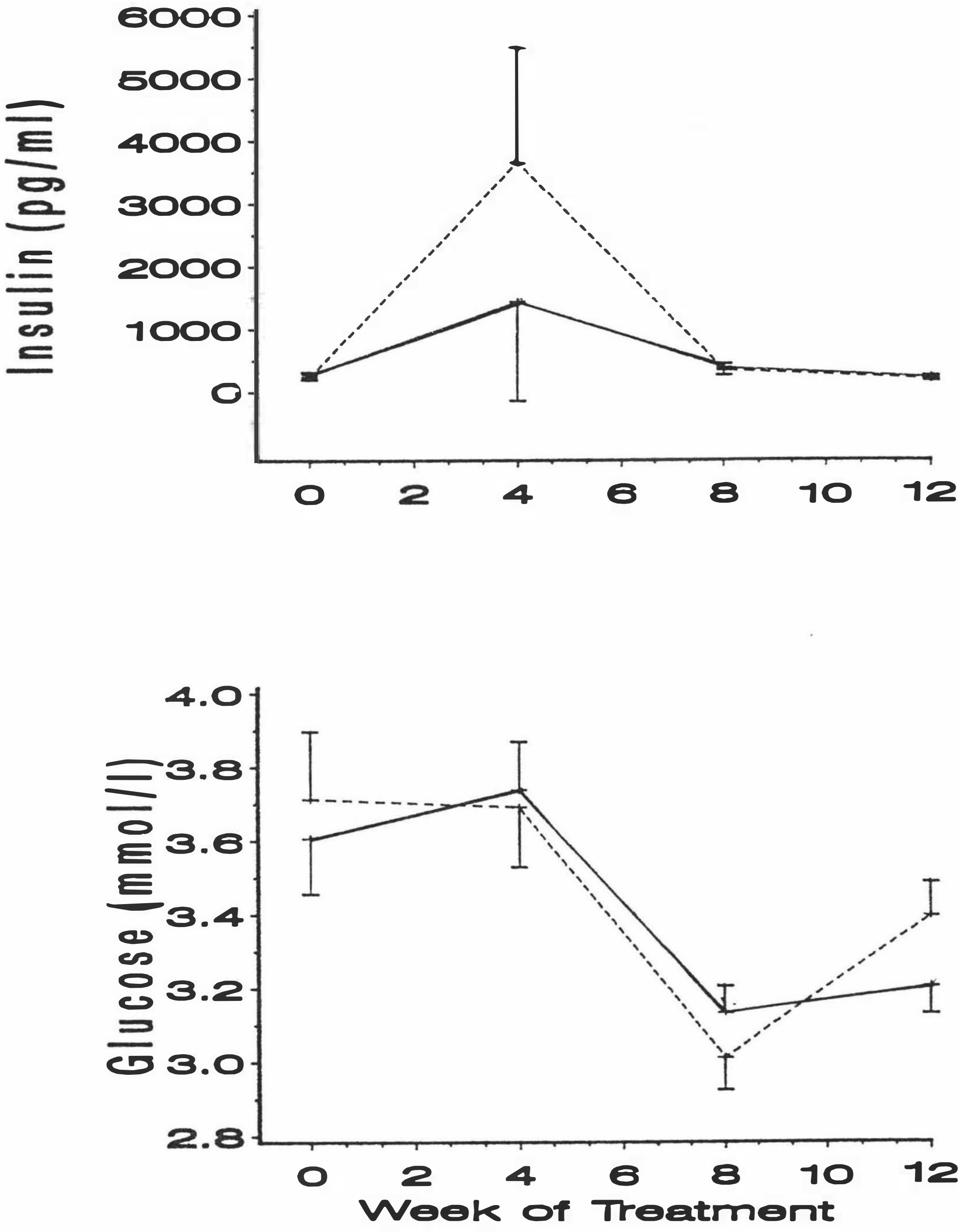

Figure 7. Plasma insulin (upper panel) and glucose (lower panel) levels of 30 control (——) and 30 monensin-treated (- - - -) cows at each sampling time, experiment 2. Weeks of treatment are expressed relative to the day of monensin capsule administration, day 0. Vertical bars represent standard errors about the mean.

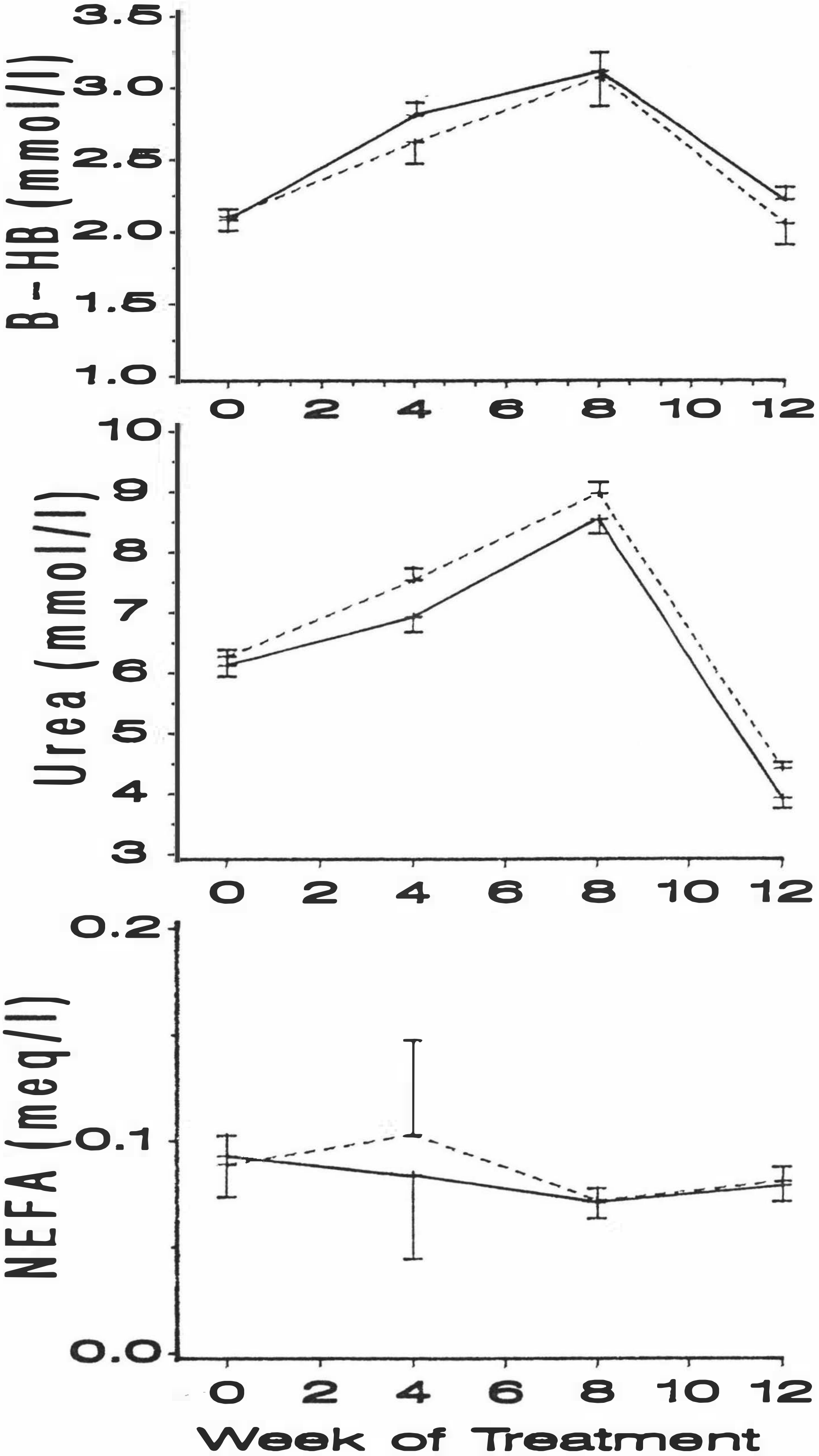

Figure 8. Plasma β-hydroxybutyrate (upper panel), urea (middle panel) and NEFA (lower panel) levels of 30 control (——) and 30 monensin-treated (- - - -) cows at each sampling time, experiment 2. Weeks of treatment are expressed relative to the day of monensin capsule administration, day 0. Vertical bars represent standard errors about the mean.

Experiment 3: Sheep fed fresh cut pasture in autumn and spring

Pasture chemical analysis

The effects of season on the chemical analyses of pasture cut from paddocks grazed by the cows and fed to sheep during the autumn and spring are shown in Table 9. Season had a significant effect on all chemical components but did not affect the gross energy value of the pasture. The pectin ($P<0.001$), hemicellulose ($P<0.05$) and crude protein ($P<0.10$) contents were significantly higher in autumn pasture than in spring pasture. The water-soluble carbohydrate ($P<0.05$), cellulose ($P<0.05$) and lignin ($P<0.05$) contents were significantly lower in the autumn pasture compared with spring pasture.

Table 9. Chemical characteristics (mean ± standard deviation) of autumn and spring pasture fed to sheep, experiment 3.

Component[a]	Autumn	Spring	Significance
Soluble carbohydrate	7.72±1.18	13.80±1.18	*
Pectin	10.05±0.35	3.45±0.35	***
Hemicellulose	13.66±0.46	10.06±0.46	*
Cellulose	8.75±0.60	13.11±0.60	*
Lignin	4.16±0.58	8.55±0.58	*
Gross energy	18.67±0.14	18.43±0.14	NS
Crude protein	26.63±1.25	23.20±1.25	+

[a] All components, except gross energy, are expressed as a percent of total dry matter. Units of gross energy are MJ/kg DM. Crude protein was calculated as % nitrogen x 6.25.

Voluntary feed intake, feed apparent digestibility and nitrogen balance

Table 10 shows the mean values of voluntary feed intake (VFI), voluntary feed intake per unit metabolic liveweight (liveweight$^{0.75}$) (VFI/MLW), apparent digestibility and nitrogen balance pooled across the four 5-day collection periods in control and monensin-treated sheep fed autumn or spring pasture in experiment 3.

Table 10. Mean (± standard deviation) of voluntary feed intake (VFI), voluntary feed intake per unit metabolic liveweight (VFI/MLW), apparent digestibility (A Dig) and nitrogen balance (N Bal) in control and monensin-treated sheep fed autumn or spring pasture, experiment 3.

Group[a]	VFI[b]	VFI/MLW[c]	A Dig[d]	N Bal[e]
Autumn control	720±150	50.0±10.0	.81±.04	7.4±5.3
Autumn treatment	690±190	48.0±12.0	.81±.08	5.6±5.8
Spring control	1110±160	63.0±11.0	.83±.03	19.5±4.8
Spring treatment	1020±160	59.0± 6.0	.87±.03	16.1±2.9

[a] Groups of 6 animals.
[b] Voluntary feed intake in g DM per day.
[c] VFI/MLW g DM/kg liveweight$^{0.75}$/day.
[d] Apparent digestibility of pasture DM
[e] Nitrogen balance, g of nitrogen per day

Season had a significant effect on VFI ($P<0.001$), VFI/MLW ($P<0.001$) and nitrogen balance ($P<0.001$), all of these parameters being lower with autumn compared to spring pasture. Apparent digestibility of the autumn pasture was significantly ($P<0.01$) lower than that of spring pasture. Monensin treatment also significantly ($P<0.05$) reduced VFI, VFI/MLW and nitrogen balance (Table 11). Nitrogen intake ($P<0.001$) and faecal N output ($P<0.001$) were significantly increased with spring pasture compared to autumn pasture (Tables 12 and 13). Monensin treatment significantly reduced ($P<0.05$) faecal nitrogen loss however neither season or monensin treatment had an effect on urinary N output.

Table 11. Significance of effects of season, monensin treatment and season by monensin treatment interaction on voluntary feed intake (VFI), voluntary feed intake per unit metabolic liveweight (VFI/MLW), apparent digestibility (A Dig) and nitrogen balance (N Bal) in control and monensin-treated sheep fed cut pasture in autumn and spring, experiment 3.

Variable	VFI	VFI/MLW	A Dig	N Bal
Covariate	**	+	NE[a]	NE
Season (S)	***	***	**	***
Monensin (M)	*	*	NS	*
S x M	NS	NS	NS	NS

[a] Not estimated

Table 12. Mean (± standard deviation) of nitrogen (N) intake, urinary nitrogen output and faecal nitrogen output, in control and monensin-treated sheep fed cut pasture in autumn and spring, experiment 3.

Group[a]	N intake	Urine N	Faecal N
Autumn control	29.1±7.0[b]	17.1±4.4	4.5±1.0
Autumn treatment	27.1±8.5	17.5±4.6	4.0±1.4
Spring control	42.0±5.9	15.8±3.3	6.7±1.3
Spring treatment	37.2±5.0	15.8±4.2	5.3±1.3

[a] Groups are 6 animals.

[b] Mean ± standard deviation of nitrogen flows measured in g/day.

Table 13. Significance of effects of season, monensin treatment, and season by monensin treatment interaction on nitrogen (N) intake, urinary nitrogen output and faecal nitrogen output of sheep fed cut pasture in autumn and spring, experiment 3.

Variable	N intake	Urine N	Faecal N
Season (S)	***	NS	***
Monensin (M)	NS	NS	*
S x M	NS	NS	NS

Rumen volatile fatty acid production

The effects of season and monensin treatment on the molar proportions of acetic, propionic, butyric and minor VFA's in the rumen fluid (pooled across sampling times) of sheep are shown in Table 14. Acetic acid constituted the largest molar proportion VFA in the rumen fluid with propionic measuring approximately 30% of that of acetic. The three minor VFA's, iso-butyric, iso-valeric and valeric, are grouped together.

Table 14. Mean (± standard deviation) ruminal molar proportions of acetic, propionic, butyric and minor volatile fatty acids in control and monensin-treated sheep fed cut pasture in autumn and spring, experiment 3.

Group[a]	Acetic	Propionic	Butyric	Minor
Autumn control	68.5±2.7	18.2±1.6	8.8±1.7	4.4±1.0
Autumn treatment	66.9±3.8	22.0±4.3	7.0±2.0	4.2±0.8
Spring control	66.3±2.3	20.2±1.7	10.0±1.2	3.5±0.8
Spring treatment	64.6±3.2	24.2±3.8	8.2±1.7	2.9±0.7

[a] Groups are 6 animals

Season had significant effects on molar proportions of butyric ($P<0.01$) and propionic acid ($P<0.10$), both being increased in the spring compared to autumn (Table 15). Monensin treatment significantly decreased the molar proportions of acetic acid ($P<0.10$), butyric acid ($P<0.001$) and the minor VFA's ($P<0.01$) and increased the molar proportion of propionic acid ($P<0.001$).

Table 15. Significance of effects of season, monensin treatment and season by monensin treatment interaction on molar proportions of acetic, propionic, butyric and minor volatile fatty acids in control and monensin-treated sheep fed cut pasture in autumn and spring, experiment 3.

Variable	Acetic	Propionic	Butyric	Minor
Covariate	+	NS	NS	NS
Season (S)	NS	+	**	NS
Monensin (M)	+	***	***	**
S x M	NS	NS	NS	NS

Rumen production of lipogenic and glucogenic VFA's

The molar proportions of lipogenic (acetic and butyric) and glucogenic (propionic, iso-butyric, valeric and iso-valeric) VFA's, and the ratio of lipogenic to glucogenic VFA, in rumen fluid of the sheep is shown in Table 16.

Table 16. Molar proportions (mean ± standard deviation) of lipogenic (acetic and butyric) and glucogenic (propionic, iso-butyric, valeric and iso-valeric) VFA's, and the ratio of lipogenic to glucogenic VFA, in rumen fluid of control and monensin-treated sheep fed cut pasture in autumn and spring, experiment 3.

Group	Lipogenic	Glucogenic	Lip:Glu
Autumn control	77.5±.69	22.5±.70	3.46±.18
Autumn treatment	73.2±.69	26.9±.76	2.72±.17
Spring control	76.6±.65	23.4±.65	3.27±.17
Spring treatment	73.2±.76	26.8±.76	2.74±.20

Season had a significant effect on each of these parameters. The level of lipogenic VFA's, as a proportion of the total VFA in rumen fluid, was higher in the autumn than in the spring. Monensin treatment significantly ($P<0.01$) increased the proportion of glucogenic VFA and reduced ($P<0.001$) the proportion of lipogenic VFA. As a result the ratio of lipogenic to glucogenic volatile fatty acids was also significantly ($P<0.001$) decreased (Table 17).

Table 17. Significance of effects of season, monensin treatment and season by monensin treatment interaction on the ruminal proportions of lipogenic and glucogenic VFA and the ratio of lipogenic to glucogenic VFA of control and monensin-treated sheep fed cut pasture in autumn and spring, experiment 3.

Variable	Lipogenic	Glucogenic	Lip:Glu
Season (S)	*	+	+
Monensin (M)	***	**	***
S x M	NS	NS	NS

Rumen ammonia level and pH

The mean concentrations of ammonia and the pH level of rumen fluid from control and monensin-treated sheep fed cut pasture in autumn and spring are given in Table 18.

Table 18. Mean (± standard deviation) ruminal ammonia and pH levels of control and monensin-treated sheep fed cut pasture in autumn and spring, experiment 3.

Group[a]	Ammonia[b]	pH
Autumn control	18.2±1.9	6.8±.07
Autumn treatment	18.6±2.1	6.7±.08
Spring control	17.3±2.0	6.6±.07
Spring treatment	17.7±1.9	6.4±.08

[a] Groups are six animals

[b] Ammonia measured in mg NH_3-N/l rumen fluid

Season had a significant ($P<0.01$) effect on pH, autumn pastures tending to produce rumen fluid of a higher pH than spring pastures. Monensin treatment did not affect either parameter (Table 19).

Table 19. Significance of effects of season, monensin treatment and season by monensin treatment interaction on ruminal ammonia concentration and pH of control and monensin-treated sheep fed cut pasture in autumn and spring, experiment 3.

Variable	Ammonia	pH
Season (S)	NS	**
Monensin (M)	NS	NS
S x M	NS	NS

Plasma hormone and metabolite levels

The effects of monensin treatment on plasma levels of insulin, glucose, β-hydroxybutyrate, urea and NEFA in sheep fed cut pasture in autumn and spring are shown in Figure 9 and Figure 10. All parameters were adjusted for the pre-treatment value (time 0) nested within season. Insulin appeared to decrease as the trial progressed with the exception of the control group in the spring which had one higher level at the end of the third sampling period. Glucose levels were relatively consistent across the sampling times. β-hydroxybutyrate levels were quite variable across sampling times. Urea and NEFA level both tended to decrease with time in the autumn and increase with time in the spring.

Glucose levels were significantly lower (P<0.01), urea level was significantly (P<0.001) higher and β-hydroxybutyrate levels were significantly (P<0.10) lower in the autumn than in the spring (Table 20).

Table 20. Significance of effects of season, monensin treatment and season by monensin treatment interaction on plasma insulin (IN), glucose (GL), β-hydroxybutyrate (BHB), urea (UR) and NEFA levels of sheep fed cut pasture in autumn and spring, experiment 3.

Variable	IN	GL	BHB	UR	NEFA
Covariate	**	NS	+	***	***
Season (S)	NS	**	+	***	NS
Monensin (M)	NS	NS	NS	NS	NS
S x M	NS	NS	NS	NS	NS

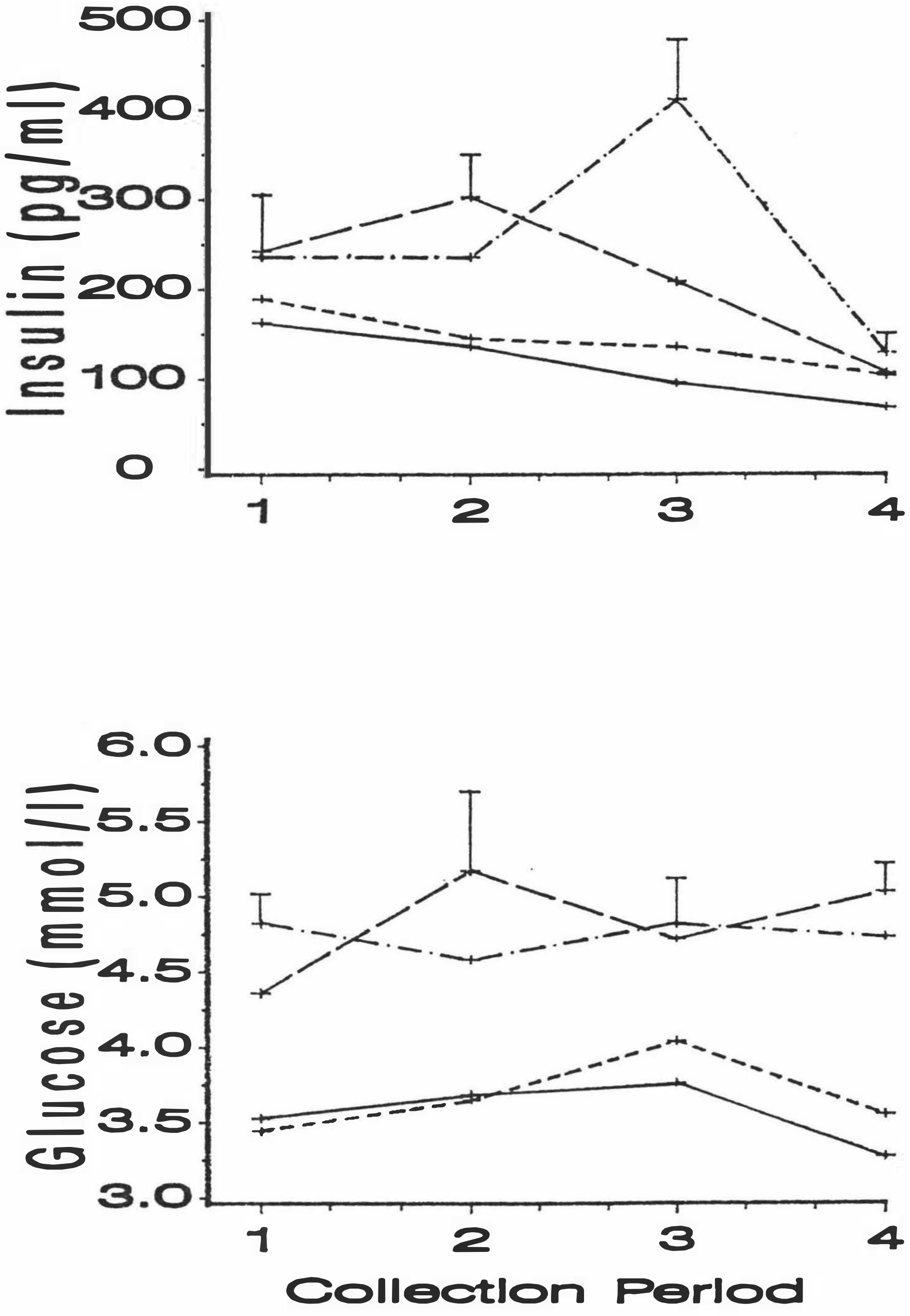

Figure 9. Plasma insulin (upper panel) and glucose (lower panel) levels of 6 control sheep fed autumn pasture (——), 6 monensin-treated sheep fed autumn pasture (- - - -), 6 control sheep fed spring pasture (- • - • -) and 6 monensin-treated sheep fed spring pasture (———), experiment 3. Vertical bars represent pooled standard errors about the mean.

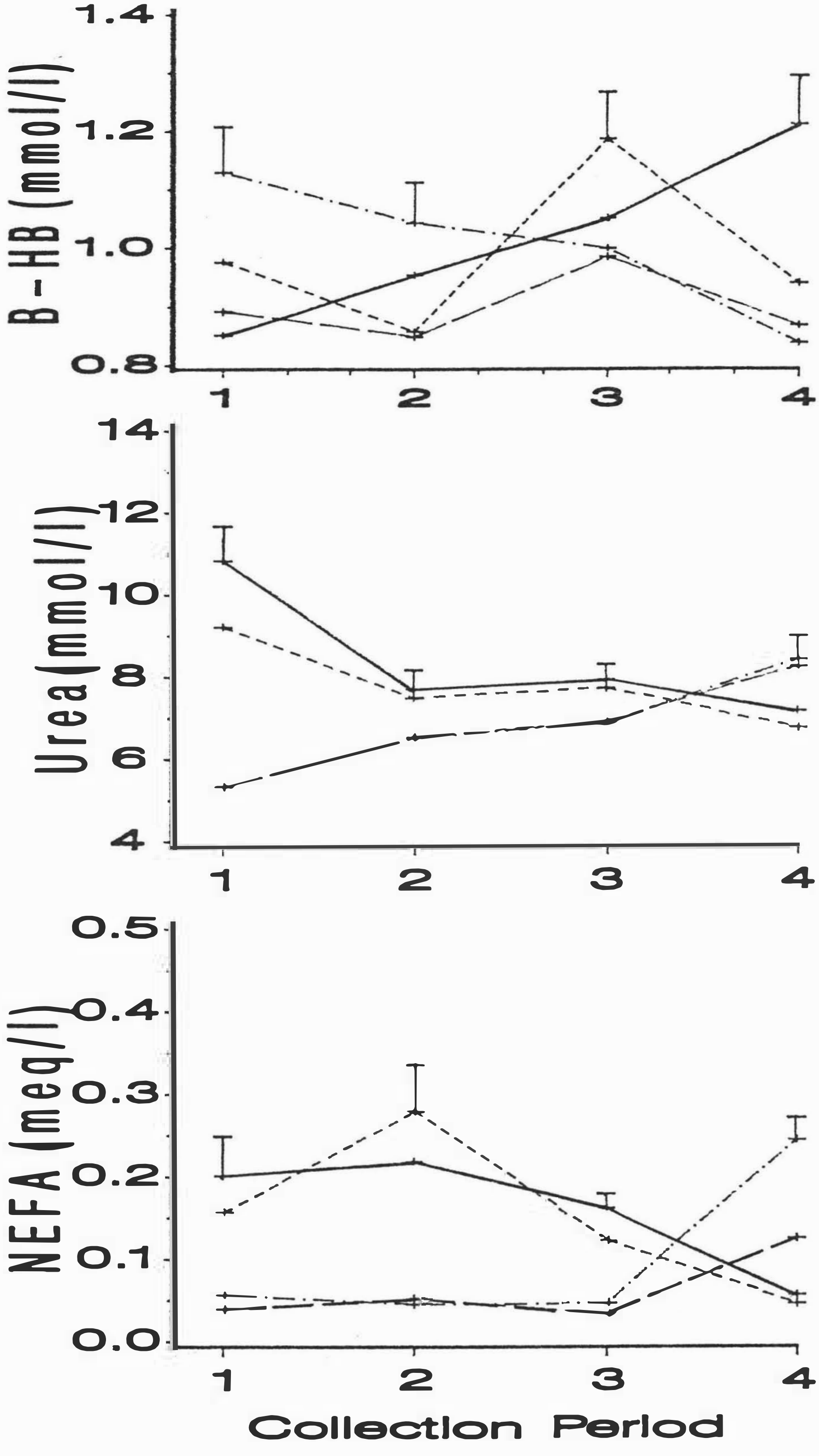

Figure 10. Plasma β-hydroxybutyrate (upper panel), urea (middle panel) and NEFA (lower panel) levels of 6 control sheep fed autumn pasture (——), 6 monensin-treated sheep fed autumn pasture (- - - -), 6 control sheep fed spring pasture (-• - • -) and 6 monensin-treated sheep fed spring pasture (———), experiment 3. Vertical bars represent pooled standard errors about the mean.

DISCUSSION

The purpose of this study was to determine the effect of monensin sodium, delivered by intraruminal controlled release capsule, on the lactational performance of autumn- and spring-calving pasture-fed dairy cows. Of particular interest was the possibility that the effectiveness of the monensin treatment in stimulating lactational performance might vary between the seasons.

Autumn and spring pasture derived from the dairy farm paddocks and fed to sheep, showed marked differences in chemical analysis due to season. Water-soluble carbohydrate levels were lower in autumn pasture than in spring pasture as was found by previous authors (Corbett et al. 1966; Scott et al. 1976; Beever et al. 1978; MacRae et al. 1985). However if pectin is included with water soluble carbohydrate in rapidly digestible carbohydrate (R) (i.e. water soluble carbohydrate + pectin) the proportion of R in pasture of both seasons was similar. The proportion of structural carbohydrate (S) (i.e. hemicellulose + cellulose + lignin) was higher in spring pasture than in autumn pasture, in contrast to previous results (Corbett et al. 1966; Beever et al. 1978; MacRae et al. 1985). Hence the ratio of structural carbohydrate to rapidly digestible carbohydrate was higher (1.83) in spring pasture than in autumn pasture (1.46), again in contrast with previous results (Corbett et al. 1966; Beever et al. 1978; MacRae et al. 1985). Unfortunately comparison of results of various laboratories is difficult due to variation in the analytical methodology.

Season had dramatic effects on the digestive parameters of the pasture measured in the sheep. The VFI of spring pasture was approximately 26% higher than that of autumn pasture. Such variation across season could account for the previously observed difference in performance of grazing sheep (Lonsdale and Taylor 1971; During and Weeda 1972; Joyce and Brunswick 1975). Apparent digestibility of spring pasture was also higher than that of autumn pasture. This suggests a positive relationship between apparent digestibility and VFI as has been reported previously (Holmes and Wilson 1984). Increased digestibility is associated with an increase in the rate of ruminal degradation of feed and therefore an increase in the rate of ruminal outflow. Hence the animal can increase VFI when rumen capacity limits intake. The variation in apparent digestibility across season could not have been predicted from the carbohydrate analysis as the proportion of S was higher in spring pasture than in autumn pasture. Digestibility of individual carbohydrate components was not measured in this study however previous work (Corbett et al. 1966; Beever et al. 1978) found actual digestibility, in the reticulo-rumen, of hemicellulose and cellulose from spring pasture to be significantly higher than that of the same components in autumn pasture. It is also possible that the variation in the ratio of hemicellulose : cellulose across seasons (spring 0.77, autumn 1.56) could explain the increased apparent digestibility of spring pasture and therefore increased VFI of spring pasture, even thought the ratio of S:R carbohydrate was higher in spring pasture. Chemical analysis of pasture, using presently available methods, does not provide a reliable indication of the intake and/or digestibility of pasture across seasons.

The higher ratio of S:R carbohydrate found in spring vs autumn pasture would suggest that spring pasture should produce a rumen fermentation with a higher Lip:Glu VFA ratio (Van Soest 1982; Preston and Leng 1987). However this study found that spring pasture produced a rumen fermentation with increased molar concentrations of propionate and reduced molar proportions of acetate and butyrate and therefore a decreased Lip:Glu VFA ratio, similar to previous reports (Corbett et al. 1966; Beever et al. 1978). Relationships between the chemical composition of pasture and ruminal VFA proportions clearly require further study.

The nitrogen balance of spring pasture fed sheep was higher than that of autumn pasture fed sheep, apparently as a result of the dramatically increased VFI and concomitant increase in nitrogen intake. While faecal N output was also higher with spring pasture compared with autumn, the digestibility of N was similar (autumn control 0.85, spring control 0.84). Rumen ammonia levels also remained relatively consistent across seasons. Therefore the observed increase in nitrogen balance associated with spring pasture could have been due to an increased rumen bypass and absorption of protein (microbial and/or dietary protein) in the small intestine as was found by MacRae et al. (1985).

The utilization of absorbed N also appeared to differ between seasons. While the urinary N output of the sheep was similar across seasons, urinary N output expressed as a percentage of N intake was 59% for the autumn pasture fed sheep and 38% for the spring pasture fed sheep. Autumn pasture also had a higher ratio of protein to carbohydrate, compared to spring pasture. Autumn pasture probably had a lower metabolisable energy value, due to lower digestibility, relative to spring pasture. These differences, together with the marked increase in VFI, in favour of the spring pasture could account for the observed difference in nitrogen balance across season. Seasonal variation in nitrogen digestion, similar to that observed in the sheep across season, may also be manifested in the lactating dairy cow, and could explain higher milk yields associated with spring pasture compared to autumn pasture.

Rumen fluid pH was lower when the diet was spring pasture (mean 6.50) compared to that of autumn pasture (mean 6.75). Both of these values are higher than the optimum pH value for ionophore activity, considered by Bergen and Bates (1984) and Perski et al. (1982) to be approximately 6.0. Therefore the pH of rumen fluid typical of ryegrass/white clover digestion may be too high (basic) to facilitate maximum ionophore responses.

The increased proportion of glucogenic VFA associated with spring pasture could allow increased glucose production compared to autumn pasture. Plasma glucose levels of the sheep were significantly lower with autumn pasture as compared to spring pasture suggesting a positive relationship across seasons between ruminal propionate production and plasma glucose levels. Of the remaining blood metabolites measured in the sheep, only β-hydroxybutyrate and urea levels were effected by season. Plasma β-hydroxybutyrate levels were lower with autumn pasture which corresponds to the reduced ruminal molar proportion of butyrate observed compared to that of spring pasture. Plasma urea levels were also lower with autumn pasture although urinary N output was unchanged as compared to spring pasture.

Monensin treatment has been shown to consistently reduce VFI of growing beef cattle (Bergen and Bates 1984; Schelling 1984), and has been associated with increases in apparent digestibility of feed although the effect on digestibility has been variable. With the sheep monensin treatment reduced VFI in both seasons but did not alter apparent digestibility of the pasture. Ruminal VFA proportions showed the characteristic response to ionophore treatment found previously (Bergen and Bates 1984; Schelling 1984, Russell and Strobel 1989) with the proportion of propionate being increased and the proportions of acetate and butyrate being decreased in response to monensin treatment. Therefore the ratio of ruminal Lip:Glu VFA was also decreased by monensin treatment of the sheep. Thus monensin treatment could be associated with increased glucose production of grazing animals fed ryegrass/white clover pasture. The treatment had consistent effects on ruminal VFA proportions in both seasons and therefore changes in chemical composition of the pasture, at least within the range of values found in this experiment, did not influence the effectiveness of monensin treatment in altering ruminal VFA proportions. The lack of a collection period by treatment interaction on VFA proportions suggests that the treatment acted quickly (within five days) and was consistent over the total twenty-day experimental period.

The two experiments with dairy cows are strictly speaking not comparable because they were carried out on different farms. However the previously discussed practical constraints, and the fact that the two farms were in very close proximity to each other, does allow some general comparison. Table 21 shows the average milk, fat and protein yields per day and the mean plasma insulin and hormone levels of the 30 control cows of experiment 1 and 2. The lactational parameters were all higher in experiment 2 (spring) than in experiment 1 (autumn). Thus the increased nutritional value of spring pasture compared to autumn pasture, as measured with the sheep, was also associated with higher milk and milk component yields of dairy cows. Of the blood metabolites β-hydroxybutyrate and NEFA appeared to exhibit seasonal differences. Plasma β-hydroxybutyrate levels were higher in spring which could be associated with the increased ruminal butyrate production detected in the sheep with spring pasture compared to autumn pasture. NEFA levels were higher in the cows fed autumn pasture as would be expected if these cows were in greater negative energy balance and were therefore mobilizing larger amounts of body tissue as compared to the cows of experiment 2 (spring). Therefore it again appears that the lactational performance of the cows in the two seasons was closely related to the digestive parameter changes of the sheep and could be accounted for, to large extent, by the chemical composition of the pasture and/or the VFI.

Table 21. Mean (± standard deviation) yield of milk, fat, and protein and the plasma levels of metabolites of the 30 control cows in experiment 1 (autumn) and 2 (spring).

Parameter	Experiment 1	Experiment 2
Milk (l/day)	13.6±2.11	18.1±3.12
Fat (kg/day)	0.61±0.13	0.77±0.13
Protein (kg/day)	0.44±0.08	0.60±0.11
Insulin (pg/ml)	420.8±1020.1	562.5±1090.6
Glucose (mmol/l)	3.42±0.32	3.43±0.28
β-hydroxybutyrate (mmol/l)	1.45±0.23	2.54±0.33
Urea (mmol/l)	7.61±0.74	6.31±0.58
NEFA (meq/l)	0.269±0.83	0.085±0.05

Milk and milk component yields of autumn-calving cows in experiment 1 were not affected by monensin treatment. Milk fat yield of the treatment group was reduced at two sample periods, however milk and protein yields remained unchanged. If the high ruminal pH level found in the sheep is characteristic of the rumen fermentation of dairy cows fed ryegrass-clover pastures, the monensin treatment effect could have been substantially reduced compared to the effect when concentrate diets have been fed and could explain the lack of response of pasture-fed cows in terms of lactational performance in either season. However monensin treatment was associated with an increased plasma glucose level in the autumn-calving cows, as was also found with the sheep fed autumn pasture, possibly from an associated increase in the level of ruminal propionate of the monensin-treated cows. The loss of body condition, as measured by condition score, was slightly lower in the treatment group of experiment 1. The combination of reduced liveweight loss, together with the absence of a change in lactational performance, would suggest that monensin treatment slightly increased the efficiency of energy recovery from feed. This has been clearly demonstrated with growing ionophore-treated concentrate-fed beef cattle (Bergen and Bates 1984; Goodrich et al. 1984; Schelling 1984; Russell and Strobel 1989).

In experiment 2 (spring), monensin treatment again failed to produce significant changes in the lactational parameters even though the graphs appear to show a trend of increased milk and protein yield. The mean increase in milk yield attained in experiment 2 (0.81 l/day) was of similar magnitude to that found previously (1.01 l/day) with monensin-treated spring-calving cows (Lynch et al. 1990). The mean increase in protein yield (0.09 kg/day) of the spring-calving monensin treatment group, although not significant, was of greater magnitude than the increase (0.03 kg/day) found by Lynch et al. (1990) under a very similar experimental situation. The age by monensin treatment effect on milk yield did show a moderate increase in milk yield of 2 and 3 year old cows, which are still growing and might therefore have increased glucose requirements, but not the mature (4,5,≥6 year old) aged cows. This could be explained if increased propionate and therefore glucose production occurred in the treated cows compared to the controls. Plasma glucose levels of the treatment cows however were not observed to increase with monensin treatment. The large metabolic need for glucose, for lactose production during lactation, could have precluded a measurable increase in plasma glucose level of the monensin treatment group even if increased glucose production did occur. The younger 2 and 3 year old cows may however have gained some small advantage from monensin treatment. In contrast, the age by monensin treatment effect on fat yield did not appear to show any consistent response of treatment in physiologically different (age) cow groups.

Monensin treatment was associated with increased plasma urea levels in the spring-calving cows in experiment 2 and a reduction in N retention of the spring pasture-fed sheep. One possible explanation is that monensin treatment could have been associated with increased ruminal protein degradation, and therefore increased plasma urea levels, however this would be in direct contrast to previous findings where monensin reduced ruminal protein degradation in beef cattle (Bergen and Bates 1984; Schelling 1984), or did not change ruminal protein degradation in sheep (Beever et al. 1987). Determination of a ruminal protein sparing effect of monensin would require a more detailed experimental protocol including measurement of protein entering the duodenum. Alternatively protein absorbed by the cow, as amino acids, in excess of the host animal's actual amino acid requirements could be catabolised by the liver (gluconeogenesis) to provide additional energy, and reduce the magnitude of negative energy balance. Gluconeogenesis, using acids as substrate, produces urea as a byproduct. Thus the increased plasma urea levels associated with monensin treatment of the cows in experiment 2 could be associated with reduced ruminal protein degradation and increased amino acids supplied to the cow.

Previously the volume of information about the comparative nutritive value of spring vs autumn dairy pasture was small. This study has contributed some information in this area, however better understanding of nutrition of the pasture-fed lactating dairy cow requires detailed information in several remaining areas. As is well known by many researchers, present chemical analysis methods, particularly of pasture, appear to be inadequate when the information is to be used to predict ruminal digestion parameters and the level of nutrients and blood metabolites supplied to the host animal. Research to match improved chemical analysis with observed *in vivo* results is necessary. A computer model utilising such improved chemical analysis could accelerate identification of key areas which are limiting growth and performance of grazing animals. For example the expected response of a growing or lactating animal to a change in diet such as from spring to autumn pasture could be estimated. Similarly estimation of the chemical composition of the appropriate supplement to be fed with pasture in order to optimise growth rate or lactational performance could increase performance of all grazing livestock.

Knowledge related to effects of monensin sodium on lactational performance of pasture-fed cows has also been increased. Again a number of related areas of study deserve further consideration. Monensin treatment may provide positive lactational performance if the level of feed allocation and hence VFI is increased. Lactating dairy cows allocated several levels of pasture intake, either as grazed pasture or as cut pasture fed in stalls, and treated with monensin would provide information in this area. Treatment of non-lactating cows with monensin may increase the conversion efficiency of feed to liveweight, as is common with beef cattle, which could increase lactational performance during the subsequent lactation and therefore increase the FCE of pasture to milk on a total farm basis. Increased glucose production associated with monensin treatment may support increased milk yields if cows are treated prior to parturition. The rumen microbial system would then be fully adjusted to treatment when glucose requirements were the highest, during early lactation. A possible related benefit could be reduced incidence of ketosis. An experiment with several monensin dosage levels would aid in determination of the dose-response relationship with respect to lactational performance of lactating cows. Subsequent trials involving ionophore treatment of pasture fed-livestock should have as part of the experimental design, a strict regime of rumen fluid sampling, by stomach tube if ruminally fistulated animals are not available, to determine the start- and end-time of ruminal parameter changes caused by ionophore treatment. This would also allow identification of reduced monensin effectiveness due to high ruminal pH, if such an effect does exist.

In summary monensin treatment did not positively or negatively affect the lactational performance of autumn- or spring-calving cows fed fresh pasture. Monensin may increase milk and protein, but not fat, yield in some specific circumstances. The primary value of the product to the New Zealand dairy farming industry appears to be its ability to control bloat. Hence the farmer should judge the value of ionophore treatment respective to the value of bloat control, but cannot automatically assume that improved lactational performance will also result from monensin treatment.

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