Recent Developments in Ruminant Nutrition 2

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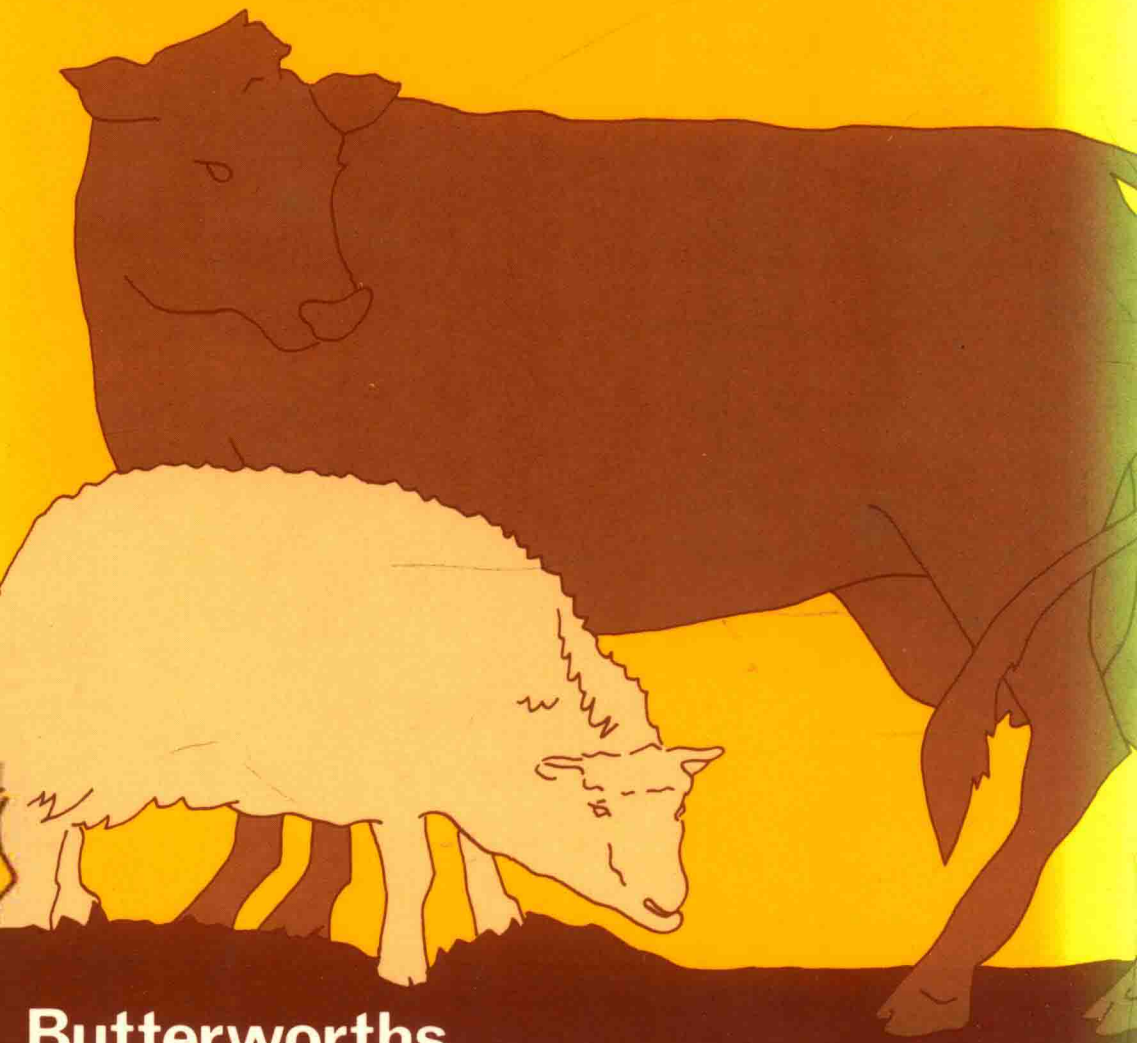

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Recent Developments in Ruminant Nutrition – 2

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Recent Developments in Ruminant Nutrition – 2

INTRODUCTION

The remarkable success of the first two volumes in this series, *Recent Developments in Ruminant Nutrition* and *Recent Developments in Pig Nutrition*, has stimulated this new text. Like the others it draws together, in one volume, important chapters from the *Recent Advances in Animal Nutrition* books which are the proceedings of the University of Nottingham Feed Manufacturers Conferences. With such a background it is inevitable that while chapters are founded on sound scientific principles they do not lose sight of the practical applications. Of great interest to the scientist and producer is the way in which performance may be improved by modifying animal function. Food is the major factor in this respect but various chapters also consider the role of season, photoperiod, hormones and growth promoters.

The selection of chapters reflects the state of the agricultural industry and its further influence on research effort. For example, the change in the payment scheme in England and Wales for the compositional quality of milk has stimulated nutritional work in that direction and a number of chapters are devoted to the manipulation of milk composition by dietary means. This, coupled with a quota scheme within the European Economic Community, has brought new pressures on farmers. In spite of this, there are those who consider that the high yielding dairy cow will still be central to milk production, with many farmers having herds producing 6500–7000 kg/lactation. With yields in excess of this, appetite is of great significance. The importance of feed intake is a theme central to many chapters. In this context the importance of grass, both *in situ* and in its ensiled form, is recognized. In addition to producing good quality silage, several authors emphasize the need to be able to predict accurately silage quality.

Consideration is also given to both intensive and forage based systems of beef production. The Agricultural Research Council schemes for calculating the nutrient requirements of beef cattle have been examined and their limitations discussed. While they are regarded as useful systems when used with intelligence and flexibility, it is suggested that comprehensive, dynamic, stochastic models of ruminant nutrition are eagerly awaited.

That the greatest emphasis should be given to cattle, as milk and meat producers, amongst the ruminants is not surprising. Although sheep are often tied to systems which do not allow great flexibility in their nutrition, there has been a considerable development in the last fifteen years in our knowledge of energy and protein requirements of the ewe. With current moves within the industry towards increased intensification and winter housing, particularly in the lowlands, this will be invaluable in planning feeding strategies.

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MANIPULATION OF RUMEN FERMENTATION

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In the ruminant animal nutrient inputs are subjected to fermentative digestion by micro-organisms and to hydrolytic digestion by the animal's own enzyme systems (*Figure 1.1*). Fermentative digestion of food fibre and the production of high quality protein from poorer nitrogen sources enables ruminants to make food for humans out of materials not directly utilized by man. Fermentation however, is accompanied by extra losses of both energy and amino nitrogen. Thus, the efficiency of producing food with ruminant animals may be optimized by properly balancing fermentative and hydrolytic digestion.

Fermentative digestion and outflows of nutrients from the rumen can be adjusted favourably by

(1) protecting dietary components from micro-organisms, and
(2) controlling the balance of microbial species or their activities (Chalupa, 1980; 1981).

Protection requires processing of specific feed components and is limited to dietary inputs which can be digested hydrolytically by the animal's enzymes. Control of microbial balance may be used with feed components that require fermentative digestion.

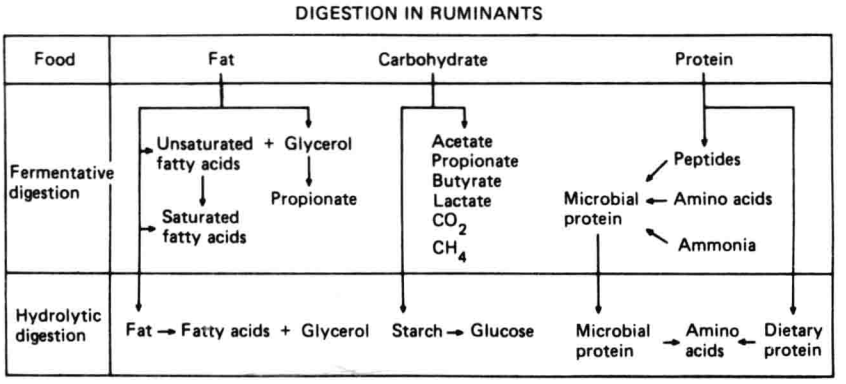

Figure 1.1 Outline of fermentative and hydrolytic digestion in ruminants

Intensive research has identified chemical agents which modulate ruminal fermentation. This chapter describes adjustments of digestion and metabolism with a variety of chemical agents, and accompanying responses in animal performance.

Survey of chemical agents

Chemical agents that favourably adjust fermentative digestion are listed in *Table 1.1*. Best documented are responses to ionophores, halogen-containing chemicals, avoparcin and diaryliodonium compounds.

There are at least 76 known polyether ionophores (Olentine, 1982). Those listed consistently decrease ruminal acetate:propionate ratio.

Halogen-containing compounds are potent inhibitors of methanogenesis. Halogenated methane analogues are 1000 times more effective than long-chain

Table 1.1 CHEMICAL AGENTS THAT ADJUST FERMENTATIVE DIGESTION[a]

Ionophores	Herbicides and Insecticides
Monensin	Cotoran
Lasalocid	Dalapon
Salinomycin[b]	2,4-D
ICI 139603[c]	M_{15}
Laidlomycin[d]	DDT
Narasin[e]	Ronnel
Polyether A	Others
Halogen-containing	Nonylphenol ethoxylate
Methane analogues	Buquinolate
Alcohols	Hydroxamates
Aldehydes	Na^+, K^+, Co^+, Zn^+, Fe^{2+}, Cu^{2+}[i]
Acids	Long-chain fatty acids
Esters	Reducing dyes
Amicloral	Fumarate
Benzol-1,3 dioxins[f]	Sulphate, Sulphite
Antibiotics	Nitrate, Nitrite
Avoparcin	Antipain[j]
Thiopeptin	Leupeptin[j]
Actaplanin[g]	Pepstatin[j,k]
Capreomycin disulphate	Chymostatin[j]
Oxamycin	Merthiolate[j]
Diaryliodonium[h]	Iodoacetate[j]
Diphenyliodonium chloride	*p*-Chloromercuric benzoate[k]
Diphenyliodonium bromide	PMSF[j,k]
4,4′-dimethyldiphenyliodonium chloride	Dithiothreitol[j,k]
4,4′-difluorodiphenyliodonium chloride	Trypsin inhibitors[j]
	Orotic acid[l]

[a]Most of the chemical agents listed were discussed by Chalupa (1980), Demeyer and Van Nevel (1975) and Prins (1978). Those not discussed previously are referenced in footnotes [b] to [i].
[b]Fontenot, Webb and Lucas (1980).
[c]Davies, Rowe and Broome (1982).
[d]Spires and Algeo (1982).
[e]Potter, Cooley and Richardson (1979).
[f]Davies, Rowe and Broome(1982).
[g]Pendulum *et al.* (1983).
[h]Chalupa *et al.* (1983a,b).
[i]Wallace 1983).
[j]Brock, Forsberg and Buchanan-Smith (1982).
[k]Kopecny and Wallace (1982).
[l]Bueno and Ralison (1982).

fatty acids, but these chemicals are too volatile to be used as feed additives. Consequently, attention has been directed to non-volatile chemicals.

Antibiotics commonly fed to enhance growth do not adjust fermentative digestion sufficiently for this mechanism to account fully for responses. However, both avoparcin and actoplanin do decrease the acetate:propionate ratio. Thiopeptin, capreomycin disulphate and oxamycin inhibit production of lactate. Diaryliodonium chemicals effectively suppress ruminal degradation of amino acids and may also adjust proportions of volatile fatty acids.

Many herbicides and insecticides affect microbial growth and fermentative digestion. Most contain halogens and may act by initially inhibiting methanogenesis.

Nonylphenol ethoxylate is toxic towards protozoa. The coccidiostat buquinolate decreases acetate:propionate ratio. The cations listed (*Table 1.1*) inhibit ureolytic activity, but the most potent inhibitors are the hydroxamates. Copper-containing compounds may be effective regulators of ruminal proteases. Long-chain fatty acids, reducing dyes, fumarate, sulphate, sulphite, nitrate, and nitrite increase propionate production, in most cases by inhibiting methanogenesis. Antipain, leupeptin, pepstatin, chymostatin, merthiolate, iodoacetate, *p*-chloromercuric benzoate, PMSF, dithiothreitol and trypsin inhibitors suppress ruminal proteolysis; they may provide insight towards development of new methods for regulating ruminal degradation of dietary protein. Orotic acid recently was reported to increase *in vitro* production of volatile fatty acids.

At the present time, monensin and lasalocid are the only rumen-active chemical agents approved in the USA by the Food and Drug Administration as feed additives for cattle. Claims for monensin (Rumensin) include improved feed efficiency in confined cattle fed for slaughter and increased weight gain in slaughter, stocker and feeder cattle on pasture that weigh more than 182 kg. Tylosin can be fed with Rumensin to reduce the incidence of liver abscesses. Rumensin can be added to certain types of liquid feeds fed to cattle in confinement. Lasalocid (Bovatec) only is cleared for use in feedlot cattle. The claim is for improvement of both gain and feed efficiency (Olentine, 1982).

Effects of chemical agents on fermentative and hydrolytic digestion

FERMENTATION IN THE RUMEN

Adjustments in fermentation *in vitro* caused by ionophores, halogen-containing chemicals, diaryliogonium chemicals and avoparcin are summarized in *Table 1.2*. All inhibit methanogenesis and so spare this loss of energy. The inhibition is usually greater with halogen-containing chemicals than with ionophores, diaryliodonium chemicals or avoparcin. Since halogen inhibition of methanogenesis is accompanied by accumulation of substantial quantities of hydrogen, not all of the spared energy is recovered in volatile fatty acids (Chalupa, 1980; 1981). The four types of chemical agents reduce acetate production and enhance propionate production. Ionophores also decrease production of butyrate and lactate. Halogen-containing and diaryliodonium chemicals increase production of butyrate but effects on lactate production are uncertain.

In vivo, the ruminal acetate:propionate ratio is usually decreased when animals are supplemented with ionophores, halogen-containing chemicals, and avoparcin

Table 1.2 EFFECTS OF TYPES OF CHEMICAL AGENTS ON RUMEN DIGESTION AND FERMENTATION *IN VITRO*[a,b]

	Chemical type			
	Ionophores	*Halogen-containing*	*Diaryl iodonium*	*Avoparcin*
Fermentation				
Methane	−	−	−	−
Hydrogen	0	+	0	0
Acetate	−	−	−	−
Propionate	+	+	+	+
Butyrate	−	+	+	0
Lactate	−	?	?	?
Digestion				
Organic matter	−	0	?	?
Protein	−	?	?	?
Amino acids	−	−	−	−
Cellulose	−	?	?	?
Starch	0	?	?	?
Micro-organisms				
Total yield	−	0	?	?
Growth efficiency	−	0	?	?

[a]Compiled from data by:
Barao, Bates and Bergen (1983)
Bartley *et al.* (1979)
Chalupa (1980)
Chalupa (1981)
Chalupa *et al.* (1983a)
Chalupa, Corbett and Brethour (1980)
Davies *et al.* (1982)
Davies, Rowe and Broome (1982)
Dennis, Nagaraja and Bartley (1981)
Fontenot, Webb and Lucas (1980)
Froetschel *et al.* (1983)
Fuller and Johnson (1982)
Ingle, Dalrymple and Kiernan (1978)
MacGregor (Chapter 19)
MacGregor and Armstrong (1983)
Nagarja *et al.* (1981)
Olentine (1982)
Spires and Algeo (1982)
Stanier and Davies (1981)
Slyter (1979)
Van Nevel and Demeyer (1977, 1979)
Wallace, Cheng and Czerkawski (1980)
Wallace, Czerkawski and Breckenridge (1981)
Whetson, Davis and Bryant (1981)
[b]0 = no change; + = increase; − = decrease; ? = no or insufficient information

(Chalupa, 1980; 1981; Chalupa *et al.*, 1981; Froetschel *et al.*, 1983). This mainly is the result of increased production of propionate (Van Maanen *et al.*, 1978; Prange, Davis and Clark, 1978; Froetschel *et al.*, 1983; Shell *et al.*, 1983). At least with monensin, production of acetate is not decreased but rather increases slightly (Shell *et al.*, 1983). The increased production of propionate with monensin is primarily through the succinate pathway (Rowe, Davies and Broome, 1981; Wallace, Czerkawski and Breckenridge, 1981).

DIGESTION IN THE RUMEN

In vitro, ionophores decrease digestion of organic matter, protein and cellulose but usually not starch (*Table 1.2*). Information on effects of avoparcin, halogen-containing, and diaryliodonium chemicals is not available except for a report of no change in digestion or organic matter in fermenters medicated with the methane inhibitor, ICI 111075 (Stanier and Davies, 1981).

In contrast with *in vitro* observations, ruminal digestion *in vivo* of organic matter and cellulose is not normally decreased by monensin (Allen and Harrison, 1979; Tolbert *et al.*, 1979). This is probably the result of an increased retention time of both solids and liquids in the rumen (Adams *et al.*, 1981; Allen and Harrison, 1979; Lemanger *et al.*, 1978; Ricke *et al.*, 1983; Tolbert *et al.*, 1979). Increases in retention time (Chalupa *et al.*, 1981) and organic matter digestion (McGregor and Armstrong, 1982) have also been observed in animals supplemented with avoparcin.

NITROGEN TRANSACTIONS IN THE RUMEN

The four categories of chemical agents listed in *Table 1.2* decrease utilization of amino acids, but the response appears least with halogen-containing chemicals (Chalupa, 1981). Rumen micro-organisms from animals supplemented with monensin have lower activities of proteolytic and deaminase enzyme systems (Barao, Bates and Bergen, 1983). Accumulation of free amino acids in the rumen of animals supplemented with 4,4′dimethyldiphenyliodonium chloride suggests suppression of amino acid utilization *in vivo* by diaryliodonium chemicals (Chalupa *et al.*, 1983; Horton, 1979).

In agreement with *in vitro* data, ruminal digestion of protein, flow of microbial protein to the small intestine and the efficiency of microbial growth are decreased by ionophores, monensin and ICI 139603 (Isichei and Bergen, 1980; Muntifering, Theurer and Noon, 1981; Poos, Hanson and Klopfenstein, 1979; Rowe, Davies and Broome, 1983). On the other hand, avoparcin induced no changes in ruminal degradation of dietary protein or flow of amino acids to the small intestine (*see* Chapter 19; MacGregor and Armstrong, 1982).

Table 1.3 EFFECTS OF IONOPHORES ON FLOWS OF DIETARY, MICROBIAL AND NON-AMMONIA NITROGEN TO THE ABOMASUM OR DUODENUM

			Nitrogen (% of control)		
Ionophore	*Species*	*Diet*	*Dietary*	*Microbial*	*NAN*
ICI 139603[a]	Sheep	Grain	129	94	105
Monensin[b]	Steers	Urea	155	67	95
		Brewers' grains	137	68	102
Monensin[c]	Steers	Grain	136	89	104
		Corn silage	133	80	100
Monensin[d]	Steers	Grain	115	86	97

[a]Rowe, Davies and Broome (1983).
[b]Poos, Hanson and Klopfonstein (1979).
[c]Isichei and Bergen (1980).
[d]Muntifering, Theurer and Noon (1981).

In experiments summarized in *Table 1.3*, flow of dietary nitrogen increased 15 to 55%, and flow of microbial nitrogen decreased 6 to 33%, whereas flow of non-ammonia nitrogen was not affected. Because 10 to 20% of the nitrogen in microbial cells is in the form of nucleic acids, changing proportions of feed and microbial protein available for absorption in the small intestine could improve the animal's protein economy by increasing the supply and perhaps the balance of amino acids. In contrast with monensin, the methane inhibitor ICI 111075 increased the proportion of microbial nitrogen in the duodenum (Davies, Rowe and Stanier, 1982).

MECHANISMS OF RESPONSES IN THE RUMEN

Chemical agents may control rumen microbial metabolism by:

(1) selective toxic effects upon certain types of microbes,
(2) control of specific enzyme systems, and
(3) alterations in uptake of metabolites by cells.

Monensin and lasalocid cause changes in the composition of the microbial population and in activities of key enzyme systems. Monensin and lasalocid adjust the balance of microbial species by selecting against major acetate and hydrogen producers such as *Ruminoccus* species and *Bacterioides fibrisolvens* and by selecting for major producers of propionate such as *Anaerovibrio lipolytica*, *Bacterioides succinogenes*, *Bacterioides ruminocola*, *Megasphera elsdenii* and *Selenomonas ruminantium* (Chen and Wolin, 1979; Henderson, Stewart and Nekrep, 1981). Lasalocid and monensin also inhibit most of the lactate-producing bacteria but do not influence either lactate-producers that also produce succinate as a major end-product nor the major lactate fermenters (Dennis, Nagaraja and Bartley, 1981). The microbial population in fermenters medicated with monensin had increased activities of succinate dehydrogenase and alkaline phosphatase but decreased activities of acetate kinase and formate-hydrogen lyase (Van Nevel and Demeyer, 1977; Wallace, Czerkawski and Breckenridge, 1981).

Avoparcin possesses a strong affinity for cell walls of Gram-positive bacteria and disrupts peptidoglycan synthesis by inhibiting the incorporation of N-acetyl glucosamine (McGahren *et al.*, 1980). Avoparcin decreases growth of many types of Gram-positive ruminal bacteria but has few undesirable effects on Gram-negative bacteria (Stewart, Crossley and Gassow, 1983). This suggests that avoparcin decreases the acetate:propionate ratio like monensin and lasalocid. However, the ability of many Gram-positive bacteria to adapt to avoparcin suggests that high concentrations might be needed to adjust the balance of microbial species in the rumen.

Neither avoparcin (Stewart, Crossley and Garrow, 1983) nor monensin (Henderson, Stewart and Nekrep, 1981) are toxic towards methanogenic bacteria. Reductions in methanogenesis are thus indirect and probably the result of reductions in the supply of hydrogen (Van Nevel and Demeyer, 1977).

Suggested mechanisms of action of halogen-containing chemicals include a selective toxic effect upon methanogenic bacteria (Prins, 1965) and an irreversible reaction of halogenated methane analogues with reduced vitamin B_{12} derivatives to inhibit cobamide-dependent methanogenesis (Demeyer and Van Nevel, 1975; Wolfe, 1971). However, vitamin B_{12} derivatives may not be required cofactors in ruminal methanogenesis (Prins, 1978).

Less is known about other chemical agents. Because most amino acid utilization occurs intracellularly (Stuart *et al.*, 1977), diaryliodonium chemicals might protect amino acids from degradation by preventing transport into bacterial cells. Accumulation of alanine when amino acid utilization is inhibited with diaryliodonium chemicals (Broderick and Balthrop, 1979; Chalupa *et al.*, 1983a) suggests that alanine may be involved in deamination of other amino acids, possibly being formed by transamination with pyruvate. If so, diaryliodonium chemicals may be potent in preventing degradation of alanine to ammonia, acetate and carbon dioxide. This pathway involves the generation of reducing equivalents, hence diaryliodonium chemicals may function like diphenyleneiodonium salts to inhibit generation of NADH (Broderick and Balthrop, 1979).

TOTAL TRACT DIGESTION

In most cases, total tract digestion of organic or dry matter is either not affected or increased slightly when cattle and sheep are supplemented with ionophores, avoparcin, halogen-containing chemicals or diaryliodonium chemicals (Allen and Harrison, 1979; Armstrong, 1983; Chalupa *et al.*, 1983b; Davies, Rowe and Stanier, 1982; Dinius, Simpson and Marsh, 1976; Horton, 1980a,b; MacGregor and Armstrong, 1982; Mathers and Miller, 1982; Muntifering, Theurer and Noon, 1981; Muntifering *et al.*, 1980; Ricke, Berger and Fahey, 1981; Rust *et al.*, 1978; Tolbert and Linchtenwaler, 1978; Wedegaertner and Johnson, 1983).

Increases in organic matter digestibility are mainly the result of an increased digestibility of nitrogen. Avoparcin acts in the small intestine to increase uptake of amino acids (*see* Chapter 19; MacGregor and Armstrong, 1982). Increased digestibility of nitrogen may also reflect decreased excretion in faeces of microbial cells resulting from decreased microbial growth in the rumen and perhaps the large intestine (Armstrong, 1983).

If ruminal digestion is depressed, increased digestion in the small intestine may occur provided the diet can be digested hydrolytically. Thus, Muntifering, Theurer and Noon (1981) concluded that monensin caused a greater portion of the nitrogen and starch in a diet of whole maize to be digested in the intestines than in the rumen. Amylase activity may increase in response to increases in flow of starch to the intestine (Clary, Mitchell and Little, 1967) and is higher in animals supplemented with monensin (Van Hellen *et al.*, 1977).

Effects of chemical agents on utilization of dietary energy and protein

Reductions in feed:gain ratio without changes in carcass characteristics (discussed later) reflect increased retentions of dietary energy and protein. Slaughter balance and metabolism balance experiments indicate that supplementation with monensin increases the combined net energy (NE_m + NE_g) value of diets (Byers, 1980; Garrett, 1976; Lofgreen, 1976; Wedegaerntner and Johnson, 1983). Whether NE_m or NE_g are affected similarly or to different extents has not been resolved and appears to be partly dependent upon methods of calculation.

Partition of energy and protein in animals supplemented with monensin and amicloral is presented in *Table 1.4*. Both chemical agents promoted increases in retention of energy and protein.

Table 1.4 ENERGY AND NITROGEN PARTITIONING IN ANIMALS SUPPLEMENTED WITH MONENSIN AND AMICLORAL

Parameter	*Monensin*			*Amicloral*	
	Response (% of control)				
Adaptation (days)	7[a]	7[a]	21[b]	3[c]	30[c]
Species	sheep	sheep	steers	sheep	sheep
Diet	50% grain		80% grain	60% grain	
Energy					
Faeces	98	93	90	101	79
Digested	101	103	104	100	103
Urine	92	84	99	—	—
Methane	74	69	74	36	88
Methane + hydrogen	74	69	74	49	89
Metabolized	105	108	107	105	104
Heat	102	105	104	100	100
Retained	111	115	119	136	170
Nitrogen					
Faeces	97	98	88	93	69
Digested	102	101	107	101	107
Urine	92	87	99	100	92
Retained	127	138	120	117	173

[a]Joyner *et al.* (1979). Monensin at 10 and 20 mg/kg diet.
[b]Wedegaertner and Johnson (1983). Monensin at 3 mg/kg$^{0.75}$ body weight.
[c]Johnson (1974). Amicloral at 2 g/kg diet.

Increases in energy retention were not the result of improvements in the utilization of metabolizable energy as heat increment was increased by monensin and not affected by amicloral. The lack of improvement in the efficiency of metabolizable energy usage with chemical agents that reduce ruminal acetate:propionate ratio is in agreement with studies that report equivalent efficiencies of acetate and propionate utilization (Bull *et al.*, 1970; Johnson, 1972; Orskov *et al.*, 1979). Both chemical agents increased the amount of metabolizable energy by decreasing faecal and methane energy losses and, in the study of Joyner *et al.* (1979), also by decreasing the loss of energy in urine. With amicloral some energy also was lost as hydrogen and inhibition of methanogenesis decreased to only 12% by day 30. Halogen-containing chemicals often do not sustain fermentative adjustments (Chalupa, 1980).

Increased retention of dietary nitrogen was the result of decreased losses of nitrogen in faeces or urine. Similar responses were obtained when growing steers were supplemented with diaryliodonium chemicals (Chalupa *et al.*, 1983b).

Responses of animal performance to chemical agents

IONOPHORES

Effects of monensin, lasalocid and salinomycin on animal performance in experiments conducted in the USA are summarized in *Table 1.5*. These data are intended to depict types of responses and should not be used to compare the magnitude of responses with different ionophores. All cause some decrease in feed intake but improve the efficiency of converting feed into liveweight gain. Correlation analysis revealed that one-half of the responses of gain were explained by variations in feed intake.

Table 1.5 EFFECTS OF IONOPHORES ON CATTLE PERFORMANCE IN STUDIES CONDUCTED IN THE USA[a]

Ionophore/diet	*No. trials*	*Performance* (% of control)		
		Intake	*Gain*	*Intake/gain*
Monensin				
Feedlot[b]	19	94	102	92
Feedlot[c]	6	95	99	95
Pasture[b]	12	—	117	—
Greenchop[b]	3	98	123	85
High forage[d]	12	97	114	91
Lasalocid				
Feedlot[e]	16	96	103	94
High forage[f]	6	96	105	93
Salinomycin				
Feedlot[g]	5	98	106	90

[a]Improvements (%) were calculated using performance of cattle receiving 5.5 to 33 mg ionophore/kg feed.
[b]Chalupa (1977).
[c]Witt, Owens and Gill (1980).
[d]Muller, Potter and Stewart (1978).
[e]Givens *et al.* (1982).
[f]Bartley *et al.* (1979); Berger, Ricke and Fahey (1981); Brown *et al.* (1982); Gutierrez, Schake and Byers (1982).
[g]Berger and Fahey (1983); Owens *et al.* (1982); Turgeon, Brink and Lucas (1983); McClure *et al.* (1980).

Results from 35 experiments conducted in nine countries in Europe showed that monensin decreased feed intake by 4%, gain was increased by 5% and feed:gain ratio was improved by 9% (Hawkridge, 1980). In 12 pasture studies in Europe, monensin at 200 mg/head daily increased gain by 14% (Wilkinson *et al.*, 1980).

Reductions in feed intake may be due to flavours of chemicals or to animals regulating consumption to maintain body-energy balance. Comparison of responses in feedlot versus greencrop and high forage experiments indicates that the extra usable energy derived from manipulating digestive processes was decreasing consumption in animals that were eating to regulate body energy balance. In contrast, the extra energy was used for additional gain when the feeding drive was restricted by other factors.

Rumensin flavour was found to cause an immediate aversion with a concentrate diet but post-ingestion aversion with a roughage diet (Baile *et al.*, 1979). A feed intake stimulant, elfazepam, alleviated depression in feed consumption caused by monensin in steers fed an alfalfa-grass hay diet (Dinius and Baile, 1977). It had no effect, however, in steers fed diets consisting of straw, urea and minerals (Coombe *et al.*, 1979).

Comparative responses of feedlot steers to monensin and lasalocid at 33 mg/kg diet in four experiments are summarized in *Table 1.6*. Compared to control diets monensin decreased feed intake by 2%, increased gain by 4%, and improved feed:gain ratio by 6%. Compared to monensin, lasalocid had similar effects upon intake, but gain was increased by 3% and feed:gain ratio was improved by 4%.

In cattle supplemented with monensin or lasalocid, additive responses are obtained with many other chemical agents, such as amicloral, ICI 11075 and 4-4′dimethyldiphenyliodonium chloride, antibiotics such as chlorotetracycline and oxytetracycline and tylosin and anabolic agents such as synovex, melengestrol