Rumen fermentation and liveweight gain in beef cattle treated with monensin and grazing lush forage

EL Packer, EH Clayton and PMV Cusack

Objective  To determine the prevalence of subacute rumen acidosis (SARA) in beef cattle grazing lush pasture and the effect of monensin on reducing SARA and improving animal performance.

Design  Commercial Angus and Murray Grey steers received a monensin slow-release capsule (n = 19) or remained untreated (n = 19). Cattle grazed an oats crop or tetraploid ryegrass pasture for a total of 91 days. Rumen fluid pH, volatile fatty acids (VFA) and lactic acid concentrations and body weight data were collected prior to treatment and again 28, 56 and 91 days after treatment. Changes in measures over time were analysed using mixed model repeated measures analysis. Differences in average daily gain between treatment groups were determined.

Results  The prevalence of SARA was low during the study, with only one animal satisfying criteria for SARA at one time point. Cattle treated with monensin capsules were 11.9 kg heavier at the completion of the study compared with untreated controls (414.5 ± 3.88 kg vs 402.6 ± 4.03 kg, P < 0.04). Rumen VFA and L- and D-lactate levels did not differ between cattle treated with monensin and untreated cattle. However, the ratio of propionate to acetate plus two times butyrate was higher (P < 0.001) when cattle were treated with monensin.

Conclusions  Subacute rumen acidosis was not consistently detected under the conditions of the study. The higher body weight of cattle treated with monensin may have been due to improved energy utilisation of the pasture, indicated by increased propionate proportions in the rumen, rather than prevention of SARA.

Keywords  average daily gain; beef cattle; ionophore; subacute rumen acidosis

Abbreviations  ADF, acid detergent fibre; ADG, average daily gain; BW, body weight; CP, crude protein; DOMD, digestible organic matter in the dry matter; DM, dry matter; GC, gas chromatography; NSW DPI FQS, New South Wales Department of Primary Industries Feed Quality Service; ME, metabolisable energy; NDF, neutral detergent fibre; NFC, non-fibrous carbohydrate; NIR, near infrared reflectance spectroscopy; N, nitrogen; Pr : Ac + 2 × Bu, ratio of propionate to acetate plus two times butyrate; REML, random estimates maximum likelihood; SARA, subacute rumen acidosis; VFA, volatile fatty acids; WSC, water-soluble carbohydrates

S ubacute rumen acidosis (SARA) is a well-recognised condition, primarily occurring in dairy cattle that are fed concentrates.1 The occurrence of SARA in cattle grazing lush pasture high in water-soluble carbohydrates (WSC) and low in neutral detergent fibre (NDF) is increasingly being recognised in Australia2-3 and overseas.4-7 However, in those studies, SARA was usually associated with the feeding of small, but significant levels of concentrates in addition to pasture. The prevalence of SARA in beef cattle grazing only high quality pasture is unknown.

Monensin is a rumen modifier that reduces the risk of acidosis induced by feeding glucose or ground corn into the rumen of cattle6-8 and improves body weight (BW) gain in cattle fed pasture.9,10 Although monensin has also been used to prevent SARA that was induced by feeding high moisture corn to cattle,12 the effect of monensin in the prevention of SARA in beef cattle only grazing lush pasture without concentrates and the subsequent effect on live-weight gain has not been explored. Therefore, the aims of the current study were to determine whether or not SARA occurs in beef cattle grazing lush pasture and then determine the effect of monensin on reducing SARA and improving animal performance.

Materials and methods

Trial location  Commercial Angus and Murray Grey steers purchased from the Wagga Wagga saleyards were transported to a commercial property in the Central West of New South Wales, Australia (33°40′12.20″S 148°35′52.10″E) approximately 3 months prior to commencement of the study. Before enrolment, all animals were assessed for suitability for the study, vaccinated against clostridial diseases (Ultragav 5 in 1, Pfizer Animal Health, West Ryde, Sydney) and treated with a macrocyclic lactone anthelmintic drench (Eprinex Pour-on, Merial, NSW). Following treatment, all cattle were grazed together on non-irrigated cereal and grass crops. The cattle grazed Bimbilo oats (Avena sativa) for 56 days and then a tetraploid annual ryegrass (Lolium perenne cv Feast II) pasture for a further 35 days. All steers were treated with a hormonial growth promotant (Compudose 100, Elanco Animal Health, West Ryde) 59 days after the commencement of the study, which was defined as when the monensin treatment had been given. Each implant (30-mm rubber implant impregnated with 21.1 mg oestradiol 17β) was placed under the skin of the ear. An audit confirmed all implants were retained at the conclusion of the study.

The study was approved by the Charles Sturt University Animal Care and Ethics Committee and was compliant with the Animal Research Act 1985 (as amended) in accordance with the ethical principles that have their origins in the Australian Code of Practice for the Care and Use of Animals for Scientific Purposes.13
**Experimental design and treatments**

A randomised untreated control design was used with one treated group and one control group; 38 steers (mean BW 253.5 ± 2.2 kg) were randomly allocated to treatment group using a stratified block randomisation procedure with animals stratified to block based on breed then BW. Steers were allocated to either receive a monensin controlled-release rumen capsule (Rumensin Anti-Bloat Capsule™, Elanco Animal Health, West Ryde, Sydney, 32 g monensin per capsule, 14 Angus, 5 Murray Grey), or remain untreated (14 Angus, 5 Murray Grey). Personnel collecting samples from animals were not blinded to treatment, but the laboratory personnel undertaking VFA and lactic acid analyses were.

**Data collection**

Rumen fluid, plasma and BW data were collected prior to treatment and again on days 28, 56 and 91 following treatment. The BW of the steers was measured using Ruddweigh 700™ cattle scales (Gallahger Australia, Rydalmere, NSW) following calibration at the beginning of each weighing using a known check weight.

Rumen fluid was collected using a stomach tube14 and assessed for saliva contamination;3 if contaminated the sample was discarded and another was taken. If the second sample appeared to be contaminated, the sample was still collected, but the values were not included in the analysis of rumen fluid pH.3 Rumen fluid pH was assessed immediately after collection using an electronic pH meter (Activon Scientific Products, Thornleigh, NSW). Approximately 20 mL of rumen fluid was decanted into a glass container and placed on ice until further processing.

Rumen fluid samples were centrifuged and stored as soon as practicable, similar to the collection of rumen fluid.14 Rumen fluid, plasma and BW data were collected prior to treatment, but the laboratory personnel undertaking VFA and lactic acid analyses were.

Blood was collected by caudal venipuncture using an 18-gauge needle and a 10 mL evacuated tube containing lithium heparin (Vacutainer™, Interleuvenlaan 40, Terumo Corporation, Leuven, Belgium). Following collection, samples were mixed and immediately placed on ice, centrifuged and stored as soon as practicable, similar to the collection of rumen fluid.

Pasture samples were collected using the median quadrat technique.15 Samples of forage were cut to a height of 25 mm to represent herbage mass available to the cattle. Samples were placed in sealable plastic bags and frozen until analysis.

**Sample analyses**

The concentrations of L- and D-lactate were determined in undiluted rumen fluid and plasma using a D-lactate/L-lactate Ultraviolet method enzymatic BioAnalysis combination test kit (Roche biopharm Cat No. 1 112 821) with a Cobas Bio spectrophotometer autoanlyser.16

Volatile fatty acid concentrations were determined by capillary gas chromatography (GC) using a Shimadzu GC17A gas chromatograph with an autosampler and autoinjector. The method uses a wide bore capillary column (SGE BP21 column; 12 × 0.53 mm internal diameter (ID) and 0.5-μm film thickness, SGE International, Ringwood, VIC, Australia, P/N 054473) with a 2 × 0.53 mm ID guard column, P/N SGE RGK2.

For GC analysis, the carrier gas was helium with a total flow rate of 48.0 mL/min, a split ratio of 5:1 and a column flow of 7.84 mL/min. The inlet temperature was 155°C, inlet pressure was 19 kPa and injection volume was 1 μL. The oven temperature was set at 80°C for 2 min, increasing 6°C/min to 122°C, 12°C/min to 144°C, then increasing 40°C/min to 180°C, held at this temperature for 2 min before increasing 40°C/min up to 220°C, which was then maintained to give a total run time of approximately 20 min. The flame ionisation detector temperature was set at 200°C with the following gas flow rates: hydrogen, 40 mL/min, instrument air, 500 mL/min and nitrogen make-up gas, 30 mL/min.

Sample VFA peaks were identified by comparing their retention times with those of a standard mixture of genuine VFA (Sigma Aldrich) and quantified using Shimadzu Class GC10 Version 1.62 and Microsoft Excel using 4-methylvaleric acid as the internal standard. All results were calculated as ppm and converted to mmol/L for subsequent analyses. The ratio of propionate to acetate plus two times butyrate (Pr:Ac + 2 × Bu) was also calculated from molar concentrations.

Pasture mass was estimated according to methods described in the median quadrat technique.19 A sub-sample of approximately 150 g of each pasture sample was dried in an oven at 80°C for 24 h to determine dry matter (DM). Proximate analyses (% DM) were determined using near infra-red reflectance spectroscopy (NIR) with a Bruker multi-purpose analyser (MPA, Bruker Optik GmbH, Ettlingen, Germany) and OPUS software (version 5.1) with calibrations developed by the NSW Department of Primary Industries Feed Quality Service (NSW DPI FQS). Calibrations were based on the following methods including: neutral detergent fibre (NDF) and acid detergent fibre (ADF) analysed sequentially24 using the filter bag method (Ankom® 200/220 fibre analyser, ANKOM technology, Macedon, NY, USA); nitrogen (N) using the Dumas combustion method with a Leco CNS 2000® analyser (Leco, St Joseph, MI, USA);13 ash by heating a sample in a muffle furnace at 550°C for 6 h;13 digestible organic matter in the dry matter (DOMD) using the pepsin cellulase digestibility assay.20 In addition, WSC concentrations were determined on oven-dried samples using the alkaline ferricyanide method (Technicon Industrial Method Number. 302-73A)21 and total lipid concentrations were determined by solvent extraction using Goldfischer apparatus22 with petroleum ether as the solvent. Crude protein (CP) was estimated from N (CP (% DM) = N (% DM) × 6.25), non-fibrous carbohydrate (NFC) was estimated by difference of components (NFC = 100 – (NDF + Ash + CP + Lipid))23 and metabolisable energy (ME) was estimated from DOMD (ME (MJ/kg DM) = 0.203 × DOMD (%) – 3.001).24

**Statistical analyses**

Changes in all measures over time between animals given monensin controlled-release capsules and untreated controls were analysed by repeated measures analysis using the MIXED Model procedure in
the SAS statistical program. The restricted maximum likelihood (REML) estimation used animal as the individual experimental unit and animal within treatment group as a random effect. The most appropriate covariance structure for each analysis was determined by reference to the Schwarz’s Bayesian Information Criterion (BIC). The analysis examined the fixed effects of treatment, breed and time as well as the interaction between fixed effects. A sub-group analysis also determined the fixed effect of average daily gain (ADG) above or below the median gain of 1.69 kg/day for all animals. Baseline values were analysed as covariates and, where significant, were included in the final model. An alpha of 0.05 was used for all statistical tests. Differences in ADG over the experimental period were determined using the general linear model procedure in the SAS program. Concentrations of VFAs and rumen fluid pH were also compared with previously published values indicating SARA. Four primary criteria were used to indicate the occurrence of SARA, including rumen pH < 6.2, total VFA > 94 mmol/L, butyrate > 12 mmol/L and isovalerate > 1.3 mmol/L. The relationship between rumen VFA and pH were analysed by Pearson correlation using SPSS Version 14.0 for windows.

**Results**

Available pasture herbage mass (pasture greater than 25 mm height, or approximately 1200 kg DM/ha) did not fall below 1000 kg DM/ha, so pasture availability was unlikely to have limited feed intake and growth of the cattle (Table 1). Over the 56-day period that cattle grazed the oats crop, quality decreased, as indicated by the observed changes in ME and CP. At day 56 of the experiment the cattle were introduced to the ryegrass pasture, which had a lower NDF and higher ME, CP and WSC content than the previous oats crop (Table 1).

The mean BW of cattle over the experimental period was not significantly different for cattle treated with monensin capsules compared with untreated animals (Table 2). The BW of all cattle increased

### Table 1. Yield and proximate analysis of pasture offered to cattle during the experiment

<table>
<thead>
<tr>
<th>Experimental day</th>
<th>Yield (kg DM/ha)</th>
<th>DM (%)</th>
<th>ADF</th>
<th>NDF</th>
<th>Ash</th>
<th>CP</th>
<th>WSC</th>
<th>Lipid</th>
<th>NDF : NFC Ratio</th>
<th>ME (MJ/kg DM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day –1</td>
<td>1350.3</td>
<td>22.90</td>
<td>17.51</td>
<td>35.91</td>
<td>9.53</td>
<td>18.68</td>
<td>27.71</td>
<td>3.54</td>
<td>1.12</td>
<td>11.53</td>
</tr>
<tr>
<td>Day +14</td>
<td>3309.6</td>
<td>16.67</td>
<td>22.11</td>
<td>43.10</td>
<td>10.42</td>
<td>19.24</td>
<td>20.03</td>
<td>3.20</td>
<td>1.81</td>
<td>11.24</td>
</tr>
<tr>
<td>Day +28</td>
<td>1256.7</td>
<td>15.10</td>
<td>23.27</td>
<td>42.69</td>
<td>9.13</td>
<td>12.33</td>
<td>26.53</td>
<td>2.42</td>
<td>1.28</td>
<td>10.88</td>
</tr>
<tr>
<td>Day +42</td>
<td>1778.8</td>
<td>24.96</td>
<td>21.53</td>
<td>40.95</td>
<td>8.75</td>
<td>10.81</td>
<td>31.55</td>
<td>1.78</td>
<td>1.09</td>
<td>10.86</td>
</tr>
<tr>
<td>Day +56 Oats</td>
<td>1093.2</td>
<td>29.47</td>
<td>21.49</td>
<td>39.92</td>
<td>9.54</td>
<td>8.76</td>
<td>30.18</td>
<td>1.60</td>
<td>1.00</td>
<td>10.46</td>
</tr>
<tr>
<td>Day +56 Ryegrass</td>
<td>2448.7</td>
<td>22.00</td>
<td>14.50</td>
<td>30.42</td>
<td>9.18</td>
<td>17.59</td>
<td>32.83</td>
<td>2.19</td>
<td>0.75</td>
<td>11.92</td>
</tr>
<tr>
<td>Day +70</td>
<td>2266.3</td>
<td>22.70</td>
<td>20.27</td>
<td>37.04</td>
<td>8.77</td>
<td>13.79</td>
<td>32.36</td>
<td>1.88</td>
<td>0.96</td>
<td>10.89</td>
</tr>
<tr>
<td>Day +91</td>
<td>1036.7</td>
<td>33.28</td>
<td>28.76</td>
<td>49.54</td>
<td>10.17</td>
<td>12.13</td>
<td>17.09</td>
<td>1.25</td>
<td>1.85</td>
<td>9.15</td>
</tr>
</tbody>
</table>

DM, dry matter; ADF, acid detergent fibre; NDF, neutral detergent fibre; CP, crude protein; WSC, water-soluble carbohydrate; NFC, non-fibrous carbohydrate \[(100 – (NDF + Ash + CP + Lipid))\]; ME, metabolisable energy.

### Table 2. Mean body weight, rumen fluid pH, L- and D-lactate and plasma L- and D-lactate in samples collected from cattle treated with monensin slow-release capsules or untreated

<table>
<thead>
<tr>
<th>Treatmenta</th>
<th>Control</th>
<th>Monensin</th>
<th>Treatment</th>
<th>Time</th>
<th>Treatment × Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean study body weight (kg)</td>
<td>352.20 (± 3.57)</td>
<td>358.90 (± 3.48)</td>
<td>0.19</td>
<td>&lt;0.01</td>
<td>0.20</td>
</tr>
<tr>
<td>Rumen Fluid</td>
<td>pH</td>
<td>7.13 (± 0.04)</td>
<td>7.08 (± 0.04)</td>
<td>0.46</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td></td>
<td>L-lactate (mmol/L)</td>
<td>0.22 (± 0.02)</td>
<td>0.20 (± 0.02)</td>
<td>0.64</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td></td>
<td>D-lactate (mmol/L)</td>
<td>0.32 (± 0.03)</td>
<td>0.31 (± 0.03)</td>
<td>0.92</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Plasma</td>
<td>L-lactate (mmol/L)</td>
<td>2.59 (± 0.41)</td>
<td>2.46 (± 0.41)</td>
<td>0.82</td>
<td>0.12</td>
</tr>
<tr>
<td></td>
<td>D-lactate (mmol/L)</td>
<td>0.07 (± 0.01)</td>
<td>0.06 (± 0.01)</td>
<td>0.57</td>
<td>0.57</td>
</tr>
</tbody>
</table>

aValues are least squares means ± standard error of the least squares means.

bPre-treatment weights were included in the analysis as a covariate.
(P < 0.001) over the course of the study (Figure 1) and cattle treated with monensin capsules were 11.9 kg heavier (P = 0.04) at the completion of the study compared with untreated cattle (414.5 kg ± 3.9 kg vs 402.6 kg ± 4.0 kg). The mean BW of cattle was not significantly different between Angus and Murray Grey steers and the interaction between treatment and breed was also not significant (P = 0.06). The ADG over the duration of the study was numerically greater in cattle treated with monensin compared with untreated controls, however, this difference was not statistically significant (1.73 ± 0.06 kg/d vs 1.62 ± 0.06 kg/d, P = 0.16).

A sub-group analysis of cattle above or below the median ADG of 1.69 kg/day indicated that the effect of monensin was greater when the ADG of steers was higher. The BW of steers at the completion of the experiment was higher in animals treated with monensin compared with untreated controls when the ADG of the steers was greater than 1.69 kg/day (Figure 2). However, when the ADG of steers was equal to, or below, 1.69 kg/day, the effect of monensin was not significant (P > 0.50). Rumen fluid pH and total rumen lactic acid and VFA concentrations were not different in steers treated with monensin with either high or low ADG (data not shown, P > 0.50).

Rumen fluid pH of all cattle decreased after introduction to the oats crop (P < 0.001), but did not differ between cattle treated with monensin capsules and those not treated (P = 0.50, Table 2). Additionally, the change in rumen fluid pH over the study period was not significantly different in cattle treated with monensin compared with cattle left untreated (group ¥ time, P = 0.15).

Following the introduction of the cattle to the oats crop, total concentration of rumen VFA (P < 0.001) and the molar percentage of propionate (P < 0.001) increased (Table 3). Total concentrations of rumen VFA did not differ between cattle treated with monensin capsules and untreated cattle (P = 0.44); however, the concentration of propionate and the ratio of Pr:Ac + 2 ¥ Bu was higher in cattle treated with...
Table 3. Total concentration and molar proportions of volatile fatty acids in rumen fluid collected from cattle treated with monensin controlled-release capsules or untreated

<table>
<thead>
<tr>
<th>Volatile fatty acid (molar proportion, %)</th>
<th>Treatmenta</th>
<th>P-values</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
<td>Monensin</td>
</tr>
<tr>
<td>Acetate</td>
<td>65.23 (±0.32)</td>
<td>63.18 (±0.32)</td>
</tr>
<tr>
<td>Propionate</td>
<td>18.04 (±0.33)</td>
<td>20.52 (±0.33)</td>
</tr>
<tr>
<td>Iso-butyrate</td>
<td>1.19 (±0.05)</td>
<td>1.24 (±0.05)</td>
</tr>
<tr>
<td>Butyrate</td>
<td>12.67 (±0.26)</td>
<td>12.15 (±0.26)</td>
</tr>
<tr>
<td>Iso-valerate</td>
<td>1.83 (±0.06)</td>
<td>1.95 (±0.06)</td>
</tr>
<tr>
<td>Valerate</td>
<td>0.97 (±0.04)</td>
<td>0.95 (±0.04)</td>
</tr>
<tr>
<td>Pr:Ac + 2 × Bu b</td>
<td>0.20 (±0.01)</td>
<td>0.23 (±0.01)</td>
</tr>
<tr>
<td>Total (mmol/L)</td>
<td>67.65 (±2.63)</td>
<td>70.55 (±2.63)</td>
</tr>
</tbody>
</table>

*Values are least squares means ± standard error of the least squares means.

bPr : Ac + 2 × Bu = ratio of propionate to acetate plus two times butyrate.

monensin compared with cattle left untreated (P < 0.001) (Figure 3). Rumen fluid pH was negatively related to rumen fluid total VFA concentration (r² = 0.74, P < 0.001) (Figure 4).

Only one animal (untreated) fulfilled the four criteria for SARA at any time-point over the study period and the four criteria were only met on one occasion by that animal. In addition, only seven animals (18.4%) fulfilled any two of the four primary criteria for SARA at any time during the study, including four untreated animals and three animals treated with monensin, indicating that there was a very low occurrence of any parameters associated with SARA.

The concentration of rumen L-lactate (P = 0.64) or D-lactate (P = 0.92) was not different between those animals that received monensin capsules and those that did not (Table 2). The concentration of plasma L-lactate (P = 0.82) or D-lactate (P = 0.57) was also not different between those animals that received monensin capsules and those that did not (Table 2).

The higher BW of cattle at the conclusion of the study following treatment with monensin is consistent with previous research showing a positive effect of monensin in cattle grazing pasture.10,11 The higher BW may have been due to an improvement in energy utilisation of pasture, which is consistent with the observed higher proportion of rumen propionate following treatment with monensin.29 It is also possible that reduced energy loss from methane also contributed to the improved BW, as monensin can reduce in vivo methane production by up to 16% when cattle graze high-quality pasture.30

All cattle in the current study received an oestradiol 17-β growth promotant 59 days after receiving monensin capsules. In previous trials, the effect of monensin and oestradiol have been additive.11,31 Therefore, the continued improvement in BW gain over the last
32-day period of the trial in cattle treated with monensin compared with untreated controls was likely to be due to a continued effect of monensin alone and not due to an interaction between monensin and oestradiol.

The lack of an observed effect of monensin on rumen pH is not unexpected, given the variation in response to monensin seen in other studies. The effect of monensin on rumen pH appears to be greatest when acid accumulation potential is high, particularly lactic acid. As there was no significant accumulation of rumen lactic acid and rumen pH was not low enough to be indicative of SARA or acute acidosis, the lack of effect of monensin may be expected. In the current study, rumen pH was associated with total rumen VFA concentration and the lack of effect of monensin on total VFA concentration is consistent with the lack of an effect on rumen pH.

The effect of monensin is generally greater when pasture quality is poor and the ADG of controls is low and the response to monensin is lower when pasture quality is high. In the current study, however, the response to monensin was greatest when ADG was highest (Figure 2), similar to effects observed with lasalocid. If cattle with higher ADG were selectively grazing pasture with higher digestibility, it is possible that rumen lactic acid accumulation would be higher and the response to monensin in preventing this acid accumulation would be greater. However, there were no indications that faster-growing animals had a differential accumulation of rumen VFA or lactic acid from grazing pasture of different quality in the current study and the improved BW with monensin in steers with higher ADG was not related to changes in rumen fluid pH or lactic acid and VFA concentrations.

Differences in background or genetic merit of animals may have contributed to the differential response to monensin in the current study. Although previous authors have suggested that a lack of response to monensin when ADG is high may be due to animals reaching their upper genetic limit for gain, this has not been proven. The ADG of cattle in the current study (1.73 and 1.62 kg/day for cattle treated with monensin and controls respectively) was higher than the range of growth rate where monensin was found to have a lower effect (0.8–0.9 kg/day). An alternative hypothesis is that higher potential ADG may allow a greater positive response to treatment to occur. Further work is required to determine the mechanisms involved with variations in response to treatment with ADG, particularly, interactions between cattle background and pasture quality.

There is little agreement as to what rumen pH is indicative of SARA. A rumen pH of less than 5.5 has been defined as indicating SARA and pH of less than 5.8 marginal. However, organic matter digestion is reduced when pH is less than 6.2 and, together with other research, a rumen pH below 6.2 appears to be indicative of SARA in grazing dairy cattle in Australia. Therefore, a rumen pH of less than 6.2 in combination with other criteria including rumen VFA was used in the current study to indicate SARA.

In addition, SARA should be viewed as a herd rather than an individual animal problem. Samples of rumen fluid should be taken from at least 12 animals per herd and 30% of sampled animals must have rumen parameters below minimum values for the herd to be diagnosed with SARA. According to these classifications, cattle from both treatment groups in the current study did not satisfy criteria for SARA. SARA may be more likely induced when cattle graze forage that is consistently higher inWSC with a lower NDF:NFC ratio than was seen in the current study (Table 1), such as in high-quality legume dominant pastures or pastures in a higher rainfall area or under irrigation.

Pasture analyses (Table 1) showed that forage samples with high WSC and low NDF were broadly accompanied by high CP. A high proportion of this lush forage CP could be expected to be highly rumen degradable, leading to increased concentrations of rumen ammonia. It is possible that the increased ammonia buffered a decrease in rumen pH from increased total VFA. Further studies on rumen dynamics of grazing cattle could include measurement of rumen ammonia concentrations.
While SARA was not consistently detected during the experiment, there were some indications that lower rumen pH values might have occurred but were not observed. Pasture NDF was low and WSC high in comparison to other studies of SARA on pasture. Ru- men pH could have been lower after introduction to the ryegrass than that observed in the cattle exiting the oats crop. The timing of rumen fluid sampling in future research is an important consideration to measure the possible short-term effects from changes in forage characteristics and previous studies suggest that lowest rumen pH occurs between 5 and 8 h post feeding. Rumen fluid pH may have been lower after introduction to the new forage crop, so rumen fluid collection should be timed accordingly in any further studies.

Previous studies report rumen pH values indicative of SARA on pastures with lower WSC and higher NDF than those measured in the current study, but those cows were also fed concentrates daily. Whilst the amounts of concentrate fed were small (1.67 to 1.83 kg/day), these concentrations may depress rumen pH in grazing cattle to a greater extent than observed in the current study. In addition, it is possible that lactate concentrations could be higher with concentrate feeding, thereby further reducing rumen pH.

Conclusions

The higher BW of cattle treated with monensin at the conclusion of the current study was consistent with the observed increase in rumen propionate, which may have improved energy utilisation. Although care needs to be taken with interpretation due to the relatively small numbers of animals in the study, the effect of monensin appeared to be greater when ADG was higher and future work should further examine the relationship between monensin and ADG in low- and high-performing cattle. Subacute rumen acidosis was not consistently detected in the grazing beef steers under the conditions observed in the current study. Collecting rumen fluid samples immediately after a diet change may be more likely to identify short-term decreases in rumen pH and further exploration of SARA under conditions more favourable to forage crop growth where fibre is low and WSC is high appears warranted.

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References

Isabel Schwilk (née Gant) will be fondly remembered as a devoted member of the profession whose unstinting dedication and affection for the animals in her care touched the hearts of a generation of students.

Isabel’s undergraduate years coincided with the heyday of Australia’s reputation for research into livestock husbandry and when students were under tutelage from some of the most enduring names in the history of the Sydney veterinary school including Bain, Carne, Gordon and Gunn.

But Isabel had a lifelong passion for veterinary science that didn’t just spring from this rich learning environment. Among the surviving output from her studies is a workbook filled with exquisite drawings depicting the histological structure of all major organs, revealing her innate love of biology, sensitivity to the beauty of nature and a depth of commitment, which defined her long working life.

After graduating, Isabel married Owen Schwilk and they settled on a small property at Castle Hill where Isabel began work in a nearby veterinary practice. During their five years at Castle Hill, the Schwilks developed an interest in cattle breeding and eventually decided the Poll Shorthorn was their breed of choice, establishing the Adair Poll Shorthorn stud from a small breeding nucleus. In 1965, Owen and Isabel took the major decision to purchase ‘Nandillyan Ponds’, a property of around 700 ha outside Orange. They immediately began a program of ensuring water security and pasture improvement while expanding their breeding herd with stock from several sources. Isabel applied her veterinary training in the selection of breeders so that conformation (and ease of calving in particular) became the chief criterion. The Adair name went on to achieve notable successes, including top priced sales at the Dubbo and Sydney Royal shows. To Isabel, however, the stud was far more than a livestock enterprise. The names of her favourite bulls quickly became known to visitors and Adair’s first home-bred sire, Astronomer, saw out his days in a pampered retirement under her loving care.

In the early 1980s, Isabel suspected a health problem among the cattle and although a diagnosis proved elusive, she doggedly pressed ahead with an investigation until, assisted by colleagues at the now disbanded Orange Regional Veterinary Laboratory, bovine Johne’s disease was confirmed. This crushing news inevitably brought severe restrictions on the sale and movement of their animals for a number of years. The stoic professionalism with which Isabel and Owen confronted this crisis, however, will stand as a powerful testimony of a selfless commitment to their many friends in the cattle industry and to one another. Ultimately, the disease was not only eradicated from their herd, but they were also honoured with a BIA Fellowship Award for their dedication, commitment and generosity to the Australian Beef Industry, recognising among other things their involvement in the control of bovine Johne’s disease.

To many, Isabel’s greatest legacy is her contribution to the training of those in the profession she loved so much. For over 30 years, veterinary students would be welcomed at Nandillyan Ponds for cattle practical work during university vacations. An extraordinary tally of over 100 students enjoyed this opportunity to gain firsthand experience under Isabel’s exacting but enthusiastic mentorship.

‘Prac work’ at the Schwilks became a priceless link between rural practice and an increasingly urbanised student body. Significantly, most of these students were women, even though they only accounted for a minority of veterinary enrolments at that time. Isabel never sought formal recognition but in 2006 she was among the female veterinarians honoured by the University of Melbourne with an award named to mark the centenary of Australia’s first woman veterinary graduate (also an Isabelle) – the Belle Bruce Reid Medal.

Isabel slowed a little with age, but her strength of spirit did not diminish and neither did the value she placed on the company of those in the profession she loved so much. For over 30 years, Tomany, Isabel’s greatest legacy is her contribution to the training of those in the profession she loved so much. For over 30 years, veterinary students would be welcomed at Nandillyan Ponds for cattle practical work during university vacations. An extraordinary tally of over 100 students enjoyed this opportunity to gain firsthand experience under Isabel’s exacting but enthusiastic mentorship. ‘Prac work’ at the Schwilks became a priceless link between rural practice and an increasingly urbanised student body. Significantly, most of these students were women, even though they only accounted for a minority of veterinary enrolments at that time.

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Isabel died on Boxing Day 2010 after enjoying Christmas lunch with family at her beloved Nandillyan Ponds.

Although never afraid to assert an opinion on a topic about which she was passionate, Isabel’s natural candour was fundamental to her character and most fittingly captured by the words of Shakespeare in an epitaph from her family: to thine own self be true; and...thou canst not then be false to any man.

Isabel is survived by Owen, her children Jim, Meredith and Iain, daughter-in-law Libby and grandsons Andrew and Timothy.

Malcolm France, Ian Lean and Deborah Racklyeft