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Effects of abomasal pectin infusion on milk production, digestion and nitrogen utilization pattern of lactating Saanen dairy goats

M. Sari*, A.A. Naserian, R. Valizadeh

Department of Animal Science, Excellence Center for Animal Science, Ferdowsi University of Mashhad, 91775-1163, Islamic Republic of Iran

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ABSTRACT

To study the effects of graded abomasal infusion of pectin on milk production, nitrogen balance and nutrient digestibilities of goats, four abomasally cannulated lactating Sannen dairy goats were used in a 4 × 4 latin square design with 14-d periods. Goats were fed the same basal diet and the treatments were the abomasal infusion of 0, 40, 80 or 120 g/d of citrus pectin. Pectin infusion resulted in linear decreases in basal ration intake from 2250 to 1985 g/d. There was insignificant decrease in total tract apparent digestibilities of dry matter and neutral detergent fiber. Decreased basal ration intake and digestibility led to decreased milk production from 1.95 to 1.75 kg/d. Milk fat content increased quadratically with increasing levels of infused pectin ($P = 0.04$), but milk fat yield was unaffected by treatments. Milk total solids and solid-not-fat were also linearly decreased by pectin infusion. There were linear decreases in urinary N and plasma urea N (PUN) with the increase in infusion of pectin. As a proportion of N intake, urinary N excretion decreased from 36.31 to 31.92%, whereas N excreted from faeces increased from 25.2 to 29.4%, as the amount of pectin infusion increased. Abomasal pectin linearly decreased faecal pH from 7.31 to 6.86 and tended to decrease faecal ammonia from 0.246 to 0.215 mg/g faecal DM. These results suggest that manipulating dairy goats diets to increase postruminal fermentation may reduce urinary N and consequently manure ammonia losses. However, decreased digestibility and milk production at the highest level of pectin infusion is suggesting that pectin may have reduced postruminal nutrient utilization.

1. Introduction

Emissions of ammonia (NH_3-N), as well as other gases and particulates, to the atmosphere are a growing concern of livestock producers, the general public, and regulators (Cole et al., 2005). Urinary nitrogen (N) is rapidly converted to ammonia during manure collection and storage, whereas faecal N is converted to NH_3 at a much slower rate. Most NH_3-N emitted from concentrated animal feeding operations is produced from the microbial hydrolysis of urinary urea to ammonium (NH_4-N) and carbon dioxide. Thus, factors that increase urinary N excretion could increase NH_3-N emissions (Erickson and Klopfenstein, 2001). However, factors such as urine pH and soil moisture (Luebes et al., 1974), or chemical composition of excreted urine (Whitehead et al., 1989) can also affect NH_3-N emissions. Developing nutritional strategies to shift N excretion from urine to faeces may reduce NH_3 from dairy manure which is implicated in reduced air quality.

Diets that increase large intestinal carbohydrate fermentation appear to shift some N from urine to faeces. Supplying fermentable substrates to the large intestine increased bacterial conversion of plasma urea nitrogen (PUN) into faecal microbial protein in sheep (Ørskov et al., 1970; Thornton et al., 1970; Mason et al., 1981), and abo-

* Corresponding author. Tel.: +98 511 8795618x20; fax: +98 511 8787430.
E-mail address: mohsensare@yahoo.com (M. Sari).

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Table 1
Ingredient composition (%) of the basal diets and nutrient contents (g/100 g DM) in the basal diet and pectin.

	Basal diet	Pectin[a]
Ingredient		
Alfalfa hay	40	
Barley	25	
Canola meal	9	
Wheat middlings	15	
Beet pulp	10	
Salt	0.2	
Vitamin and mineral permix[b]	0.4	
Limestone	0.4	
Nutrients		
NDF	35.9	–
ADF	22.3	–
EE	2.9	0.2
CP	15.9	1.1
RDP	11.80	–
RUP	4.1	–
NFC[c]	39.0	96.5
Ash	6.41	2.2
Ca	0.8	–
P	0.5	–
DM, %	90	98.2

RDP: rumen degradable protein; RUP: rumen undegradable protein; NFC: nonfiber carbohydrates; EE: ether extract; CP: crude protein.

[a] Pectin (HM slow-set pectin; TIC gums, Belcamp, MD). Nutrient composition reported as analyzed.

[b] Supplying (per kg diet): Vitamin A, 5000 i.u.; Vitamin D3, 2000 i.u.; Vitamin K, 1 mg; Cu, 10 mg; Zn, 30 mg; Fe, 50 mg; Mn, 20 mg; I, 0.1 mg; Se, 0.1 mg; Co, 0.04 mg.

[c] NFC = 100 − (CP + ash + NDF + EE).

masal infusions of 1 kg/d of pectin were shown to reduce urinary N by approximately 10% in lactating dairy cows (Gressley and Armentano, 2005).

To our knowledge, no research has been conducted investigating the relationship between increasing hindgut fermentation and ammonia emissions from dairy goats. A greater understanding of the factors controlling NH_3-N emissions from dairy goat farms would aid in the development of prediction models and in the development of methods to control these emissions. To that end, this study was conducted to determine the effects of increasing carbohydrate fermentation in the large intestine on production and urinary nitrogen excretion in lactating Saanen dairy goats. A secondary objective of these experiments was to identify faecal characteristics that are altered by pectin infusion, with the intent of identifying a marker that could be used to quickly evaluate the differences in postruminal fermentation of practical diets.

2. Material and methods

2.1. Animals and treatments

Four multiparus lactating Saanen dairy goats (average body weight (BW) 41 ± 1.5 kg, days in milk 48 ± 2.1 and milk production 1.98 ± 0.35 kg) were used. Goats were fitted with permanent "T" type abomasal cannulae for infusing pectin and surgery was performed under general anesthesia. Goats were allowed approximately 4 weeks to recover after the surgery. Each of them was housed individually in metabolism crates with free access to water and under continuous lighting. In the metabolism cages, there were facilities for separate collection of faeces and urine. Goats were milked twice daily at 8.00 and 20.00 h and milk yield was recorded at each milking. All goats received the same basal diet (Table 1). The diet was formulated to meet or exceed all nutrient requirements for goats according to NRC (1981). The basal diet was fed to all goats in equal portions at 8.30 and 20.30 h and refusals were removed at 7.45 h weighed, and analyzed for dry matter (DM) to accurately predict dry matter intake (DMI). The basal diet was fed at 120% of intake for 10 d immediately before the start of the experiment, and ad libitum DMI was estimated as the average DMI during the final 4 d of this pre-experimental period (mean of 2410 g/d). Each goat was offered the basal diet at 95% of its estimated ad libitum intake (mean of 2290 g/d) throughout the remainder of the experiment.

Treatments were the abomasal infusion of water only (0 pectin) and water containing 40, 80 or 120 g/d pectin. High-methoxyl slow-set citrus pectin (Provisco AG, Hauptwil, Switzerland) was infused from day 6 to 14 of each period. Abomasal infusates were delivered via polyvinyl chloride tubing (3.2 mm i.d. × 0.78 cm o.d.; Down Corning, MI, U.S.A.), and peristaltic pump (model 323; Watson–Marlow, UK) delivered the 2.4 L of infusate continuously over 24 h except for about 2 h during milking. Infusion equipment was checked daily during treatment periods to ensure correct placement in the abomasum.

2.2. Sampling

Total collections of urine and faeces were conducted for last 4 d of each period. Faeces weight and urine volume were recorded twice daily and faeces and urine samples were taken twice each day. Faecal samples were composited by goat within the period according to wet daily output and a portion of each faecal sample was dried at 60 °C for later component analysis. A portion of fresh faeces was frozen at −20 °C for later N analysis and the rest was used for the determination of pH and NH_3. Approximately 25 g of fresh faeces was weighed into each of two specimen cups. To the first cup, 50 mL deionized water (pH 6.7) was added. To the second cup 10 mL of distilled water plus 0.43 mL of 50% TCA were added. Each cup was shaken vigorously for 20 s and liquids were squeezed through one layer of cheesecloth. Liquids from the first cup were measured for pH, and liquid from the second cup was frozen at −20 °C for later analysis of NH_3. Urine samples were acidified with 50% H_2SO_4 (0.02 mL/1 mL of urine) and frozen at −20 °C for later N analysis.

Blood and rumen fluid samples were taken 3 h after feeding on day 1 and 3 during each collection period. Blood samples (10 mL) were taken by jugular venipuncture. The blood was centrifuged at 2000 × *g* for 30 min, and serum was frozen at −20 °C for later analysis. The rumen fluid collection was conducted only once daily because more frequent sampling was considered to jeopardize the health of the goats. Rumen fluid was aspirated using an oral probe (Duffield et al., 2004). The first 50 mL of collected rumen fluid was discarded, and the subsequent 30 mL of rumen fluid was kept for subsequent analysis. Rumen fluid pH was measured using a 744 pH Meter (Metrohm, Herisau, Switzerland). The goats were weighed on day 8 of each period, between morning milking and feed distribution. Duplicate milk samples were taken at each milking during the final 2 d of each period. One set of duplicate samples was analyzed for fat, protein, lactose and solids-nonfat with an infrared analyzer (MilcoScan 605; Foss Electric, Hillerød, Denmark). The second set of samples was refrigerated and frozen at −20 °C until milk urea N (MUN) analysisAQ for change.

2.3. Sample analyses

Feed and faecal samples were dried at 55 °C and ground through a 1-mm screen. Ground feed and faecal samples were analyzed for DM, OM, CP, NDF and ADF. Milk and urine were analyzed for total N. Rumen and faecal samples were analyzed for NH_3.

Dry matter was determined by drying overnight in a 103 °C forced-air oven. Dry samples were ashed overnight at 550 °C in a muffle furnace. All nitrogen determinations were made using a Tecator automated Kjeldahl system with Na_2SO_4 and $CuSO_4$ as catalysts and collecting the distillate in a boric acid solution (AOAC, 1990). The NDF and ADF of diet and faeces were analyzed by the detergent system, using the sequential procedure of Van Soest et al. (1991), with sodium sulfite and thermostable amylase and were corrected for ash. For ammonia N determination, a 5-mL sample of filtered rumen fluid was acidified with 5 mL of 0.2N HCl and frozen. Rumen fluid and faecal samples were thawed, centrifuged (25,000 × *g* at 4 °C, 20 min), and the supernatant was analyzed for NH_3 (Chaney and Marbach, 1962). Faecal output was measured directly and digestibility calculations included infused pectin as a component of intake. Composite milk samples analyzed for MUN enzymatically (modified Berthelot reaction) on a Chemspect 150 analyzer (Bently instrument Chaska, MN). Plasma glucose

Table 2
Effect of abomasal pectin infusion on intake and digestibility.

Item	0	40	80	120	S.E.M.	Linear	Quadratic
	Treatment (level of pectin, g/d)[a]						
Basal DM intake[b]	2250	2215	2190	1985	79.4	<0.01	0.72
Total DM intake[c]	2250	2255	2270	2105	78.2	0.23	0.09
	Total intake of nutrients[c] (g/d)						
OM	2100	2108	2121	1975	75.5	0.42	0.12
NDF	825	817	805	737	9.3	0.09	0.65
ADF	496	488	483	447	10.1	0.15	0.85
EE	63	61	61	58	1.3	0.41	0.56
CP	360	355	353	319	3.9	0.13	0.82
NFC	875	900	931	890	10.9	0.33	0.11
	Apparent digestibility[d] (%)						
DM	65.69	65.01	63.28	60.58	0.6	0.14	0.65
OM	68.03	67.18	64.15	63.72	0.6	0.16	0.61
ADF	49.57	48.35	48.01	47.57	1.6	0.35	0.59
NDF	52.45	52.51	49.60	48.70	1.9	0.13	0.30

[a] Treatments: 0 = abomasal infusion of saline only; 40 = abomasal infusion of 40 g/d pectin in saline; 80 = abomasal infusion of 80 g/d pectin in saline, 120 = abomasal infusion of 120 g/d pectin in saline.
[b] Intake of the basal ration only, not including pectin infused.
[c] Intake of the basal ration plus pectin infused.
[d] Digestibility calculations include infused pectin.

(Trinder, 1969), triglycerides (McGowan et al., 1983), cholesterol (Allain et al., 1974) and urea (Talke and Schubert, 1965) were determined by using commercial kits according to manufacture's instructions (Zist-Shimi Co., Tehran, Iran) in the blood samples.

2.4. Statistical analyses

Data were analyzed as a 4 × 4 latin square design using the General Linear Models procedure of SAS (1999) according to the following model: $Y_{ijk} = \mu + T_i + P_j + C_k + e_{ijk}$; where Y_{ijk} is the dependent variable, μ is the overall mean, T_i is the fixed effect of treatment, P_j is the fixed effect of period, C_k is the random effect of goat, and e_{ijk} is the random residual error. The residual error term was used to test for the significance of goat, period, and treatment. In addition, the linear and quadratic effects of pectin infusions were determined, using the residual error term. Cubic effect was tested and found not significant. Significance was declared at $P < 0.05$ and a trend was declared at $P < 0.15$.

3. Results

3.1. Intake and digestion

Nutrient intakes including pectin are presented in Table 2. The basal diet DMI decreased linearly ($P < 0.01$) and intake of the basal ration plus infused pectin tended to quadratically increase then decreased ($P = 0.09$).

Table 3
Effect of abomasal pectin infusion on milk yield and composition.

Item	Treatment (level of pectin, g/d)[a]				S.E.M.	Linear	Quadratic
	0	40	80	120			
Milk yield (kg/d)	1.95	1.90	1.90	1.75	0.09	0.04	0.59
4% FCM yield (kg/d)	1.79	1.76	1.76	1.60	0.08	0.10	0.62
Fat							
%	3.46	3.55	3.50	3.43	0.10	0.37	0.04
g/d	66.30	66.71	66.50	60.10	2.1	0.25	0.18
Protein							
%	3.01	3.10	3.05	3.01	0.06	0.92	0.75
g/d	58.70	58.90	57.95	52.68	1.9	0.15	0.37
Lactose							
%	4.63	4.65	4.60	4.62	0.03	0.81	0.54
g/d	90.28	88.35	87.78	80.85	2.4	0.11	0.25
TS							
%	11.65	11.90	11.76	11.67	0.70	0.24	0.09
g/d	227.2	226.1	223.44	204.26	8.2	0.05	0.23
SNF							
%	8.25	8.34	8.26	8.24	0.58	0.46	0.15
g/d	160.87	158.46	156.94	145.95	3.5	0.04	0.45
MUN (mg/dl)	15.61	15.12	13.85	13.47	0.75	0.12	0.29

FCM: fat corrected milk; TS: total solids; SNF: solid-not-fat; MUN: milk urea nitrogen.
[a] Treatments: 0 = abomasal infusion of saline only; 40 = abomasal infusion of 40 g/d pectin in saline; 80 = abomasal infusion of 80 g/d pectin in saline, 120 = abomasal infusion of 120 g/d pectin in saline.

Table 4
Effect of abomasal pectin infusion on intake and excretion of nitrogen (g/d).

Item	Treatment[a]				S.E.M.	Linear	Quadratic
	0	40	80	120			
Intake	58.95	58.07	57.49	53.05	0.51	0.04	0.62
Faeces N	14.74	15.10	16.09	15.39	0.24	0.23	0.10
Urine N	21.39	20.21	18.64	16.95	0.35	0.04	0.54
Milk N	9.19	9.22	9.07	8.25	0.51	0.42	0.11
Retained N	13.61	13.49	13.65	12.40	0.82	0.13	0.35
Milk N as % of intake	15.60	15.85	15.76	15.56	0.65	0.48	0.21
Faeces N as % of intake	25.2	26.5	28.8	29.4	0.81	0.04	0.88
Apparent N digestibility	73.88	72.56	70.81	69.58	0.44	0.08	0.30
Urine N as % of intake	36.31	34.79	32.39	31.92	0.52	0.05	0.69
Urine N/(fecal N + urine N)	59.20	57.25	53.69	52.42	1.63	0.03	0.84
Retained N as % of intake	23.05	23.21	23.80	23.34	0.91	0.37	0.25

[a] Treatments: 0 = abomasal infusion of saline only; 40 = abomasal infusion of 40 g/d pectin in saline; 80 = abomasal infusion of 80 g/d pectin in saline, 120 = abomasal infusion of 120 g/d pectin in saline.

Table 5
Effect of abomasal pectin infusion on ruminal fluid pH and ammonia N concentration as well as concentration of plasma constituents.

Item	Treatment[a]				S.E.M.	Linear	Quadratic
	0	40	80	120			
Rumen fluid							
pH	6.51	6.56	6.47	6.53	0.09	0.36	0.75
Ammonia N (mg/dl)	25.3	25.0	23.8	24.5	0.9	0.18	0.46
Plasma							
Glucose (mg/dl)	71	69	70	73	2.8	0.51	0.48
Urea N (mg/dl)	23	21	20	15	0.87	0.07	0.86
Triglycerides	23	25	22	22	0.7	0.58	0.22
Cholesterol	105	107	103	106	2.4	0.83	0.30

[a] Treatments: 0 = abomasal infusion of saline only; 40 = abomasal infusion of 40 g/d pectin in saline; 80 = abomasal infusion of 80 g/d pectin in saline, 120 = abomasal infusion of 120 g/d pectin in saline.

Apparent total tract digestibilities of DM, OM, ADF and NDF are presented in Table 2. Abomasal pectin infusion tended to linearly decreases total tract apparent digestibility of dry matter ($P = 0.14$) and neutral detergent fiber ($P = 0.13$).

3.2. Milk yield and composition

Pectin infusion significantly decreased milk yield ($P = 0.04$) and tended to decrease 4% FCM yield ($P = 0.10$) linearly by up to 0.2 and 0.19 kg/d respectively. Milk fat percentage increased ($P = 0.04$) in a quadratic matter but milk fat production remained unchanged (Table 3). The largest decrease in milk production resulted in the highest infusion rate. Milk protein percentage and protein secretion remained unchanged. Pectin infusions had no significant effect on milk lactose percentage but tended to decrease lactose production ($P = 0.11$). Milk concentrations of total solids (TS) and solids-not-fat (SNF) remained unchanged but both TS and SNF production linearly decreased with pectin infusion ($P = 0.05$ and $P = 0.04$, respectively).

3.3. Nitrogen balance

The increase in pectin infusion up to 120 g/d linearly ($P = 0.04$) reduced N intake by 6 g/d (Table 4). Pectin infusion tended to increase faecal N quadratically ($P = 0.1$) and decreased urine N linearly ($P = 0.04$). Pectin linearly increased the fraction of intake N that was excreted in the faeces ($P = 0.04$), linearly decreased the fraction of intake N that was excreted in the urine ($P = 0.05$) and proportion of urinary N in total urinary and faecal N ($P = 0.03$). Increasing levels of pectin infusion did not affect milk N, retained N and retained N as a fraction of N intake.

Table 6
Effect of abomasal pectin infusion on fecal characteristics.

Item	Treatment[a]				S.E.M.	Linear	Quadratic
	0	40	80	120			
DM, g/d	775	781	836	830	13.6	0.13	0.19
DM, %	55	51	49	53	1.5	0.25	0.09
pH	7.31	7.18	7.12	6.86	0.06	0.05	0.54
NH_3 mg/g fecal DM	0.246	0.237	0.218	0.215	0.017	0.11	0.69

[a] Treatments: 0 = abomasal infusion of saline only; 40 = abomasal infusion of 40 g/d pectin in saline; 80 = abomasal infusion of 80 g/d pectin in saline, 120 = abomasal infusion of 120 g/d pectin in saline.

3.4. Ruminal and faecal characteristics and blood metabolites

There were no significant effects of treatments on rumen pH and rumen NH_3. Pectin infusions did not affect basal plasma contents of glucose, triglyceride and cholesterol but decreased plasma urea N ($P = 0.07$, Table 5).

Increasing pectin infusion tended to linearly increase faecal DM output ($P = 0.13$, Table 6) from 775 to 830 g/d, and faecal DM content was quadratically affected ($P = 0.09$) with pectin infusion. Abomasal pectin resulted in linear decrease in faecal pH ($P = 0.05$) and faecal NH_3 tended to be affected ($P = 0.11$) with the lowest faecal NH_3 for the greatest level of pectin infusion.

4. Discussion

4.1. Intake and digestion

In the present study, basal diet DMI was linearly influenced by pectin infusion. Goats with 0 and 40 g/d pectin infusion had the highest feed intakes. The reduction in basal ration intake suggests that postruminal pectin itself or intestinal pectin degradation products reduced intake. Although data are lacking in dairy goats the study in dairy cows (Gressley and Armentano, 2005) showed similar results. According to the results from monogastric studies, dietary inclusion of viscose fiber such as pectin reduced voluntary energy intake due to slower gastric emptying and intestinal transit times (Burton-Freeman, 2000). Mertens (1994) suggested that not flow capacity of undigested feed residues or decreasing flow through the abomasum or intestines, limits intake. If pectin increased abomasal and postruminal digesta viscosity, it might have decreased abomasal emptying and consequently reduced intake.

The slight, although not statistically significant, decrease in total tract apparent digestibility of DM, OM and NDF agrees with the results of Gressley and Armentano (2005) in which pectin tended to numerically reduce digestibilities of DM, OM, ADF and NDF in dairy cows. Pectin may have limited digestion and consequently energy available to the animal from the basal diet. This suggests that pectin may have had a negative impact on the dietary energy utilization of the animal either by increased hindgut fermentation or by some other impact of pectin on intestinal digestion. However, except for starch, pectin effects on digestibility were not observed in one of the studies conducted by Gressley and Armentano, 2005.

Apparent N digestibility linearly decreased with pectin infusion. One method to assess potential differences in postruminal fermentation is to look for the differences in apparent N digestibility (Ørskov et al., 1970). Pectin digested in the hindgut would primarily be fermented to VFA as in the rumen, but microbial protein produced would not be absorbed as amino acids and would increase endogenous protein loss and decrease apparent nitrogen digestibility (Owens et al., 1986; Mason et al., 1981), shifting nitrogen excretion from the urine to the faeces (Heijnen and Beynen, 1997).

4.2. Milk production and composition

Relatively few studies have measured effects of increasing hindgut fermentation on milk production and composition. Factors affecting the response would include resulting change in DMI and nutrient digestibility. In the current study, the reduction in basal DMI is thought to be primarily responsible for the reduction in milk yield by increasing levels of pectin infusion. In contrast with our result, the reduction in milk yield was not observed by Gressley and Armentano (2005). Milk fat percentage was affected quadratically with pectin infusion. In cows, abomasal pectin decreased milk fat content (Gressley and Armentano, 2005), and abomasal inulin decreased milk fat yield (Gressley and Armentano, 2007). The effect of pectin infusion on milk fat was unexpected but these results suggest that postruminal fermentation may play a role in milk fat synthesis. Pectin infusion tended to linearly decrease MUN. It was hypothesized that postruminal pectin fermentation would increase the conversion of plasma urea N to bacterial protein. Consequently, MUN was predicted to decrease with abomasal pectin. However, MUN was not significantly affected by treatment, suggesting that the changes in the blood urea pool due to pectin infusion were not large enough to affect MUN.

4.3. Nitrogen balance

At the same basal ration intake, pectin infusion was expected to increase intestinal microbial protein synthesis from blood urea N, thereby increasing faecal N and decreasing urinary N. Pectin infusion linearly reduces N intake, and regardless of pectin effects, one would expect this drop in N intake to reduce urinary N and to a lesser extent faecal N (Wright et al., 1998).

The N balance data indicate that when goats receive pectin, partitioning of N is affected by treatments. The increase in faecal N excretion that accompanied the infusion of pectin, combined with the decrease that occurred in the urinary N of goats, clearly indicates that there was an increase in microbial nitrogen synthesis. The results cannot show whether the nitrogen incorporated into bacterial cells arose from the ammonia formed during protein degradation in the hindgut or from an influx of nitrogen from the blood or from both, although pectin infusion linearly decreased PUN. In both instances the results would be a change in the route of nitrogen excretion from the urine to the faeces. In agreement with our data, abomasal infusions of 1 kg/d of pectin significantly decreased urinary N by 27 g/d and increased faecal N by 22 g/d in lactating cows (Gressley and Armentano, 2005). Based on faecal purine output, Gressley and Armentano (2005) showed that faecal bacteria were responsible for approximately 77% of the 22 g/d increase in faecal N with abomasal pectin infusion. In another experiment, increasing hindgut fermentation of dairy cows with 1 kg/d inulin infusion into abomasum resulted in increased faecal N by 23 g/d and decreased urinary N by 24 g/d (Gressley and Armentano, 2007), indicating that different rapidly fermentable substrates led to similar shifts in N excretion. It was shown by Mason et al. (1981) with sheep that cecal infusions of 50 g/d of pectin

increased faecal N from 6.1 to 7.6 g/d and decreased urinary N from 11.4 to 10.1 g/d. Shifts of N from urine to faeces have also been observed in sheep infused into the terminal ileum with starch (Ørskov et al., 1970) or glucose (Thornton et al., 1970) or into the cecum with starch (Mason et al., 1981; Surra et al., 1997). Similar findings were also observed by Reynolds et al. (2001) who, in lactating dairy cows received abomasal infusion of 0 to 1200 g/d wheat starch, reported significant reduction in urinary N due to increased intestinal fermentation. These results demonstrate that if rapidly fermentable carbohydrates reach hindgut, this can increase postruminal bacterial growth and change the pattern of N excretion.

Although retained N tended to linearly decrease with pectin infusion, fraction of intake N that was retained remained similar among treatments. Gressley and Armentano (2005) reported that pectin infusion tended to decrease N retention by 56 g/d in their first experiment. They hypothesized that abomasal pectin may have reduced postruminal digestion of rumen microbial or dietary protein, consequently reducing N retention. On the other hand, pectin may have reduced N retention by increasing endogenous true protein secretion or reducing intestinal reabsorption of endogenous N. However, in another experiment from the same study, pectin effects on N retention were not observed. Similarly, increasing hindgut fermentation by abomasal inulin infusion did not affect retained N (Gressley and Armentano, 2007).

4.4. Ruminal and faecal characteristics and blood metabolites

Pectin infusion did not affect rumen pH or NH_3. Across treatments, the rumen NH_3 values were above the 10.0 mg of NH_3-N/dL required for maximal microbial protein synthesis in vivo (Reynal et al., 2005). In cows, abomasal pectin infusion tended to decrease rumen NH_3 and urinary purine derivative excretion (Gressley and Armentano, 2005), but no significant response of rumen NH_3 to abomasal inulin infusion was observed in the experiment of Gressley and Armentano (2007). Overall, urea recycling to the rumen was apparently unaffected by increasing intestinal fermentation, suggesting that manipulating dairy diets to increase hindgut fermentation and reduce manure ammonia volatilization should not reduce N recycling to the rumen for microbial protein production.

Pectin infusion did not significantly affect plasma glucose, triglycerides and cholesterol but linearly decreased PUN. Reductions in PUN attributable to increased faecal microbial protein production were observed when cows were infused abomasally with 1 kg/d inulin (Gressley and Armentano, 2007), or when sheep were given up to 90 g/d of cecal glucose infusions (Thornton et al., 1970).

A secondary objective for this experiment was to identify whether postruminal pectin digestion was reflected by the changes in faecal characteristics. In the future, those characteristics might be used as markers to predict relative differences in postruminal fermentation. For example, faecal pH was decreased linearly by abomasal pectin, so faecal pH might be one such marker. Consequently, faecal pH, DM percentage and NH_3 were measured as potential markers of pectin digestion.

In agreement with our findings, in cows, increasing hindgut fermentation by abomasal pectin (Gressley and Armentano, 2005) or inulin (Gressley and Armentano, 2007) infusion increased faecal DM output, but no differences were found in faecal DM percentage and faecal pH (Gressley and Armentano, 2005). However, in another study abomasal starch infusion in lactating cows has been shown to reduce faecal pH, and this was attributed to postruminal fermentation of the infused starch (Reynolds et al., 2001). Consistent with our results, the reduction in faecal NH_3 with pectin infusion in dairy cows (Gressley and Armentano, 2005) supports the hypothesis that postruminal pectin increased conversion of blood urea N into microbial protein. Circulating urea diffuses into the large intestine and is converted to ammonia by gut microbes. Pectin appears to have increased microbial uptake of available NH_3-N.

5. Conclusions

Abomasal pectin infusion increased faecal N and decreased urinary N and PUN. As a proportion of N intake, urinary N excretion decreased from 36.31 to 31.92%, whereas N excreted from faeces increased from 25.2 to 29.4%, as the infused amount of pectin increased. Reduced urinary N should increase the environmental stability of manure N and reduce manure ammonia losses. We had hypothesized that these changes would be due to large intestinal bacteria utilizing energy from pectin to convert PUN into faecal microbial protein. Abomasal pectin caused significant decrease in basal DMI and numeric decreases in total tract apparent digestibility of DM and NDF, suggesting that small intestine expose to soluble and highly fermentable fiber may have negatively influenced nutrient intake and digestion. In total, these results suggest that feeding diets that increase postruminal carbohydrate fermentation may reduce ammonia volatilization from manure.

References

Allain, C.C., Poon, L.S., Chan, C.S.G., Richmond, W., Fu, P.C., 1974. Enzymatic determination of total serum cholesterol. Clin. Chem. (20), 470–475.
AOAC, 1990. Official Methods of Analysis, 15th ed. Association of Analytical Chemists, Washington, DC.
Burton-Freeman, B., 2000. Dietary fiber and energy regulation. J. Nutr. (130), 272S–275S.
Chaney, A.L., Marbach, E.P., 1962. Modified reagents for determination of urea and ammonia. Clin. Chem. (8), 130–132.
Cole, R.N., Clark, R.N., Todd, R.W., Richardson, C.R., Gueye, A., Greene, L.W., McBride, K., 2005. Influence of dietary crude protein concentration and source on potential ammonia emissions from beef cattle manure. J. Anim. Sci. (83), 722–731.
Duffield, T., Plaizier, J.C., Fairfield, A., Bagg, R., Vessie, G., Dick, P., Wilson, J., Aramini, J., McBride, B., 2004. Comparison of techniques for measurement of rumen pH in lactating dairy cows. J. Dairy Sci. (87), 59–66.
Erickson, G.E., Klopfenstein, T.J., 2001. Managing N inputs and the effect on losses following excretion in open-dirt feedlots in Nebraska. Sci. World J. 1 (S2), 830–835.
Gressley, T.F., Armentano, L.E., 2005. Effect of abomasal pectin infusion on digestion and nitrogen balance in lactating dairy cows. J. Dairy Sci. (88), 4028–4044.

Gressley, T.F., Armentano, L.E., 2007. Effects of low rumen-degradable protein or abomasal fructan infusion on diet digestibility and urinary nitrogen excretion in lactating dairy cows. J. Dairy Sci. (90), 1340–1353.
Heijnen, M.-L.A., Beynen, A.C., 1997. Consumption of retrograded (RS3) but not uncooked (RS2) resistant starch shifts nitrogen excretion from urine to feces in cannulated piglets. J. Nutr. (127), 1828–1832.
Luebes, R.E., Davis, K.R., Laag, A.E., 1974. Diurnal fluctuation and movement of atmospheric ammonia and related gases from dairies. J. Environ. Qual. (3), 265–269.
Mason, V.C., Kessank, P., Ononiwu, J.C., Narang, M.P., 1981. Factors influencing faecal nitrogen excretion in sheep. 2. Carbohydrate fermentation in the caecum and large intestine. Z. Tierphysiol., Tierernä hr. Futtermittelkde. 45, 174–184.
McGowan, M.W., Artiss, J.D., Strandbergh, D.R., Zak, B., 1983. A peroxidase-coupled method for the colorimetric determination of serum triglycerides. Clin. Chem. (29), 538–542.
Mertens, D.R., 1994. Regulation of forage intake. In: Fahey Jr., G.C., Collins, M., Mertens, D.R., Moser, L.E. (Eds.), Forage Quality, Evaluation, and Utilization. American Society of Agronomy. Crop Science Society of America, Soil Science Society of America, Madison, WI, pp. 450–493.
NRC, 1981. Nutrient Requirements of Goats. National Academy of Science, Washington, DC.
Ørskov, E.R., Fraser, C., Mason, V.C., Mann, S.O., 1970. Influence of starch digestion in the large intestine of sheep on caecal fermentation, caecal microflora and faecal nitrogen excretion. Br. J. Nutr. (24), 671–682.
Owens, F.N., Zinn, R.A., Kim, Y.K., 1986. Limits to starch digestion in the ruminant small intestine. J. Anim. Sci. (63), 1634–1648.
Reynal, S.M., Broderick, G.A., Bearzi, C., 2005. Comparison of 15N, total purines, AA profiles, and urinary excretion of purine derivatives as markers for quantifying microbial protein flow from the rumen of lactating dairy cows. J. Dairy Sci. (88), 4065–4082.
Reynolds, C.K., Cammell, S.B., Humphries, D.J., Beever, D.E., Sutton, J.D., Newbold, J.R., 2001. Effects of postrumen starch infusion on milk production and energy metabolism in dairy cows. J. Dairy Sci. (84), 2250–2259.
SAS Institute, 1999. SAS User's Guide: Statistics. SAS Institute Inc., Cary, NC.
Surra, J.C., Guada, J.A., Balcells, J., Castrillo, C., 1997. Effects of post-ruminal fermentation on the faecal and urinary excretion of purines. Anim. Sci. (65), 383–390.
Talke, H., Schubert, G.E., 1965. Enzymatic urea determination in the blood and serum in the Warburg optical test. Klin. Wochenschr. (43), 174–175.
Thornton, R.F., Bird, P.R., Somers, M., Moir, R.J., 1970. Urea excretion in ruminants. III. The role of the hind-gut (caecum and colon). Aust. J. Agric. Res. (21), 345–354.
Trinder, P., 1969. Determination of glucose in blood using glucose oxidase with an alternative oxygen acceptor. Ann. Clin. Biochem. (6), 24–27.
Van Soest, P.J., Robertson, J.B., Lewis, B.A., 1991. Methods for dietary fiber, neutral detergent fiber, and nonstarch polysaccharides in relation to animal nutrition. J. Dairy Sci. (74), 3583–3597.
Whitehead, D.C., Lockyer, D.R., Raistrick, N., 1989. Volatilization of ammonia from urea applied to soil: influence of hippuric acid and other constituents of livestock urine. Soil Biol. Biochem. (21), 803–808.
Wright, T.C., Moscardini, S., Luimes, P.H., Susmel, P., McBride, B.W., 1998. Effects of rumen-undegradable protein and feed intake on nitrogen balance and milk protein production in dairy cows. J. Dairy Sci. (81), 784–793.