The Benefits of Getting More Potassium into Lactating Cows

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Introduction

Potassium (**K**) is the principal intracellular cation of most body tissues. Potassium ions participate in many essential biological processes such as the maintenance of osmotic potential within cells, nerve impulse transmission, enzyme reactions in cellular metabolism, the maintenance of normal kidney function, and cardiac, skeletal and smooth muscle function. Because milk is an intracellular fluid, milk contains a large amount of K.

This paper reviews the responses of lactating dairy cows to increasing K concentration in the diet on milk yield and components. Because some K sources have had consistent positive effects on milk fat percentages, and milk fat percentages have been linked to the ruminal production of certain biohydrogenation intermediates, then data from several continuous culture experiments are reviewed to determine how K supplements affect biohydrogenation.

Negative K Balance in the Early Lactation Dairy Cow

Published research suggests that the early lactation dairy cow is in negative K balance (Bannink et al., 1999; Jarrett et al., 2012; Nennich et al., 2006; Silanikove et al., 1997). Potassium retention in this data set was positive for over 85% of cows in the calibration dataset; however, in a set of early lactation cows, K retention was negative for all cows (Nennich et al., 2006). Early lactation cows (less than 75 days in milk) had an average K retention of – 66 g/d (**Figure 1**). Excretion of K appears to be directly related to K intake. **Figure 2** shows the relationship of K intake and K excretion.

Potassium metabolism of cows in the early lactation dataset varied from cows in the calibration dataset. Early lactation cows tended to excrete greater amounts of K even though K intakes were similar to cows in the calibration dataset (Figure 2). Due to the greater K excretion and the greater secretion of K in milk, early lactation cows were in a negative K balance.

Potassium's role in milk production can be tied to the concept of dietary cation anion difference (**DCAD**). Potassium is a cation that raises the DCAD, which represents interaction among the macrominerals. Interacting effects among the macrominerals sodium (**Na**), K, chloride (**Cl**), and sulfur (**S**) have been observed in the pre-calving cow, but little has been written on this subject for the post-calving cow. DCAD affects the cow

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by altering its acid-base status. For a general review and broader examination of these and other related topics please see the review by Block (1994). There are differences in the response to DCAD that depends on the source of Na and K used in these studies. This difference appears to show up mainly in cows in the early lactation period.

Production Responses to K and DCAD

In 2012 we published a study that evaluated the relationship of level of K feeding in early lactation when DCAD was increased with K carbonate sesquihydrate (Harrison et al., 2012). Cows were on study from ~ 15 days in milk until ~ 85 days in milk. Diets were formulated to be similar in all nutrients except K (**Table 1**) with K levels of 1.3% and 2.1% of DM; and DCAD levels of 25 and 42 mEq/100 g of DM.

The inclusion of a higher amount of K in the early lactation diet resulted in an increase in production of milk, 3.5% fat-corrected milk, and milk fat (**Table 2**). This increase was not associated with an increase in dry matter intake (**DMI**), and therefore appears to be unrelated to energy intake.

Table 3 summarizes the evaluation we conducted on milk fat samples from this same study. Milk samples from one-half the cows in each treatment group that represented a range from low to high milk production were selected for characterization of milk fatty acids. A limited set of the milk fatty acids is shown in Table 3. The added dietary potassium carbonate decreased unsaturated and trans-fatty acids, and increased C18:0 in milk. This suggests that one mechanism for the increase in milk fat production is ruminally based.

Potassium and Heat Stress

With an increase in ambient temperature dairy cows rely on adaptive mechanisms to dissipate heat and these include: moving to shade if available, decreasing DMI, increasing water intake, and increasing evaporative loss via respiration and sweating. Mallonee et al. (1985) observed a 5 fold increase in loss of K via sweating when cows were provided shade during the hottest part of the day, 9.6 mg/m^2 vs 46.7 mg/m^2. When respiration is increased to dissipate heat, CO_2 is lost more quickly, plasma CO_2 partial pressure is lowered, and the pH of blood tends to rise. Potassium and Na are key to maintaining a blood acid-base balance, and their role is critical in times of heat stress and increased respiration rates.

Special Considerations

Our current dietary recommendations are to formulate for 1.6% K, and to increase to 1.8 to 2% for heat stress. Sodium levels can be increased to assist in achieving a DCAD of > 35 meq/100 g of DM. Sodium should not exceed 0.8% of the ration DM. There are three reasons that guidelines for Na and K are higher than NRC (2001). First, because early lactation cows eat less than mid-lactation cows, there is a need to increase nutrient concentrations to reflect reduced feed intakes. Second, most of the macro-

mineral research was conducted with low and medium producing cows; high producing cows secrete more of these minerals in milk and generate more acid in the rumen and blood. Third, the higher concentrations of Na and K represent an additional role these nutrients play in rumen buffering and acid-base balance, and recent data suggests that cows can be deficient in K and Na in early lactation.

No recommendation is given for Na because of its dependency on K and DCAD concentrations. Salt *per se* is not a required nutrient by dairy cows. However, because salt is one of the four taste sensors on the tongue we recommend a minimum of salt (~ 0.1 lb/d) in every lactation ration. Chloride should be kept to as close to the minimum NRC recommendations as possible to avoid complications due to chloride's contribution in subclinical metabolic acidosis.

Ruminal Explanation for K and Milk Fat

Increasing DCAD in diets fed to lactating cows has had positive effects on milk fat and milk fat yield. Iwaniuk and Erdman (2015) reported in a meta-analysis of 196 dietary treatments that milk fat percentage increased 0.1 for each increase in DCAD of 100 mEq/kg of DM. One explanation for the increase in milk fat percentage with increasing DCAD can be linked to ruminal fluid pH. Increasing DCAD was shown to increase ruminal fluid pH an average of 0.03-units per 100 mEq of DCAD/kg of dietary DM (Iwaniuk and Erdman, 2015). The increase in DCAD and ruminal fluid pH likely alters the types and amount of biohydrogenation (**BH**) intermediates produced by the rumen microbial population, which in turn increases milk fat. Therefore, the milk fat response to DCAD requires an understanding of 1) how BH intermediates are linked to milk fat synthesis and 2) how ruminal fluid pH is linked to the production of BH intermediates.

Biohydrogenation and Milk Fat Synthesis

Biohydrogenation of linoleic acid in the rumen begins with its conversion to conjugated linoleic acid (**CLA**). In this initial step, the number of double bonds remains the same but one of the double bonds is shifted to a new position by microbial enzymes. Normally, the double bonds in linoleic acid are separated by two single bonds, but in CLA, the double bonds are only separated by one single bond. Many types of CLA are produced in the rumen of dairy cows, but a common CLA produced from BH of linoleic acid is *cis*-9, *trans*-11 C18:2 (Jenkins et al., 2008). As BH progresses, double bonds in the CLA intermediates are then hydrogenated further to *trans* fatty acids having only one double bond. A final hydrogenation step by the ruminal microbes eliminates the last double bond yielding stearic acid as the final end product.

In cows on a typical forage diet, the major *trans* C18:1 produced in ruminal contents is *trans*-11 C18:1 (Zened et al., 2013). Most of the remaining isomers have double bonds distributed equally among carbons 9 through 16. The exact pathways for the production of these positional isomers are not known. Linoleic and linolenic acids are converted to several *trans* C18:1 and C18:2 intermediates during BH. Mosley et al. (2002) showed that the BH of oleic acid by mixed ruminal microorganisms involves the

formation of several positional isomers of *trans* C18:1 rather than only direct BH to form stearic acid as previously described.

Under certain dietary situations the rumen environment is altered and a portion of BH occurs via a pathway that produces *trans*-10, *cis*-12 CLA and *trans*-10 18:1. The *trans*-10, *cis*-12 CLA produced in the rumen travel via the blood to the mammary gland, where it inhibits the synthesis of milk fat by impairing the production of several enzymes essential for fat synthesis in the mammary gland (Jenkins and Harvatine, 2014). The *trans*-10, *cis*-12 CLA are also present in cows that produce acceptable milk fat levels, but at concentrations too low to cause milk fat depression (**MFD**).

The '*trans*-10 shift' in BH pathways is not a risk for MFD unless it is accompanied by a bottleneck at the terminal step of the pathway. Without a bottleneck, excess *trans*-10, *cis*-12 CLA is quickly and extensively converted to the *trans*-10 C18:1 intermediate, never accumulating to levels needed for MFD. With a bottle neck at the terminal step, there is excess accumulation of *trans*-10, *cis*-12 in the rumen leading to MFD. This can be seen by the associated increase in the *trans*-10 18:1 content of milk fat, which is indicative of the complex changes in ruminal BH pathways characteristic of MFD. Although *trans*-10 18:1 does not directly inhibit mammary synthesis of milk fat (Lock et al., 2007), it is relatively easy to analyze compared to *trans*-10, *cis*-12 CLA and other CLA isomers. Therefore, in general, this fatty acid can serve as a surrogate marker for the type of alterations in rumen BH that characterize diet-induced MFD.

The bottom line is that the type of feed the cow consumes affects rumen conditions, which in turn affects the amount and type of CLA produced. Since *trans*-10, *cis*-12 CLA overproduction in the rumen leads to MFD, excess *trans*-10, *cis*-12 CLA and therefore MFD can be controlled by paying close attention to several key nutritional risks.

Ruminal Fluid pH and Biohydrogenation Intermediates

Factors that can result in marked changes in ruminal fluid pH through any 24-h period include: dietary carbohydrate profile and rates of degradation of the carbohydrate fractions as affected by source, processing, and moisture; physically effective NDF (**peNDF**) supply as affected by source and particle size; and production of salivary buffers as a function of peNDF supply and source (Shaver, 2005). Despite our general understanding of these factors, the degree and duration of low ruminal fluid pH required to cause sufficient flux of unsaturated fatty acids through alternative pathways of ruminal BH is not known. Although data are limited, changes in ruminal fluid pH are most likely associated with MFD because they cause a change in the bacterial population favoring alternative BH pathways. Ruminal pH has independent effects on both extent of BH as well as on the profile of BH intermediates.

Martin and Jenkins (2002) examined the continuous culture incubations that were conducted at dilution rates of 0.05 and 0.10/h with pH values of 5.5 and 6.5, and 0.5 and 1.0 g/L of mixed soluble carbohydrate. They found that the most influential environmental factor on both extent of BH and *trans* FA profile was culture pH At pH

5.5, the concentration of *trans*-C18:1 and CLA were significantly reduced resulting from reduced extent of BH from linoleic acid. Similar effects were observed by Troegeler-Meynadier et al. (2003). Low amounts of CLA from reduced extent of BH at pH 6.0 could be due to low isomerase activity or to high reductase activity. Moreover, they found that low pH (pH 6.0) resulted in lower amount of *trans*-11 C18:1 at all incubation times compared with higher pH (pH 7.0), but concentration of *trans*-10 C18:1 were higher at 16 to 24 h of incubation indicating a shift in BH intermediates. Low pH inhibited initial isomerization and the second reduction (*trans*-11 C18:1 to stearic acid), leading to an accumulation of *trans*-11 C18:1 in ruminal cultures (Troegeler-Meynadier et al., 2006). Choi et al. (2005) reported that *cis*-9, *trans*-11 CLA are produced at pH higher than 6.2 by rumen bacteria, but *trans*-10, *cis*-12 CLA are produced more than *cis*-9 *trans*-11 CLA at lower pH. They concluded that *trans*-10, *cis*-12 CLA producing bacteria may be more aero and acid-tolerant than *cis*-9, *trans*-11 CLA producing bacteria.

Qiu et al. (2004) reported that reduced ruminal fluid pH can affect microbial populations, especially cellulolytic bacteria. Total cellulolytic bacteria numbers are reduced, accompanied by reduced acetate-to-propionate ratio and altered BH when pH was low. The ruminal fluid pH also influenced fungal growth and metabolism. Culturing ruminal fungi at pH 6.0 and pH 7.0 slowed BH compared with pH 6.5. CLA production was increased by pH 7.0 compared to pH 6.0 and pH 6.5. Therefore, optimum pH was 6.5 and 7.0 for BH and CLA production, respectively, by ruminal fungi (Nam and Garnsworthy, 2007).

Supplemental K Effects on Biohydrogenation Intermediates

Reports of increased milk fat yields following the addition of K to the diet raised questions if K altered ruminal BH and the type of CLA produced. A series of continuous culture experiments were run at Clemson University to determine if increasing K concentration in the culture contents was associated with a decline in the production of the *trans*-10, *cis*-12 isomer linked to MFD. The first experiment (Jenkins et al., 2014) consisted of four dosage levels of a 10% K_2CO_3 (w/w) stock solution (0, 10.6, 21.2, and 32 mL) injected directly into the fermenters twice daily immediately after each feeding (fermenters were fed 60 g of 1:1 forage to concentrate in two equal portions at 0800 and 1630 h). Distilled water was also injected (32, 21.4, 10.8, and 0 mL, respectively) to maintain a total injection (K_2CO_3 + water) volume of 32 mL/d. The K added was 0, 0.6, 1.2, and 1.8 g/d or 0 (K0), 1% (K1), 2% (K2), or 3% (K3) of the daily feed. Because aqueous solutions of K_2CO_3 are strongly alkaline, pH was expected to increase with increasing dosage of K_2CO_3. To determine if any changes in BH and fermentation could be attributed to effects on pH, a fifth treatment (NaOH) consisted of injection of sufficient 10% NaOH (w/w) each day to match the K3 pH.

As expected, pH averaged over the three sampling days increased ($P < 0.05$) linearly with increasing K, but remained in the 6.0 to 6.4 range (**Table 4**). Culture pH were similar for the K3 and NaOH treatments. Increasing K had effects on VFA proportions but not total VFA concentrations. As K addition to the cultures increased, there were linear decreases ($P < 0.05$) in propionate but increases ($P < 0.05$) in acetate

and acetate to propionate ratio. Addition of NaOH could not duplicate the VFA changes seen for K_2CO_3. K addition also affected the pattern of BH intermediates. As K addition increased, the daily production in mg/d of *trans*-11 18:1 and *cis*-9, *trans*-11 CLA both increased ($P < 0.05$) linearly. Conversely, K addition decreased ($P < 0.05$) *trans*-10 C18:1 but had no effect on *trans*-10, *cis*-12 CLA. The addition of K caused a shift in BH intermediates consistent with the improvement in milk fat % observed in previous lactation trials. Changes in BH intermediates also were caused by the NaOH treatment suggesting K might shift BH by elevating pH.

A second continuous culture experiment (Jenkins et al., 2014) was run to examine the effects of K in culture contents that had elevated *trans*-10, *cis*-12 CLA concentrations induced by feeding high fat. Six treatments were arranged as a 2 x 3 factorial with two levels of added soybean oil (0 and 4%) and 3 levels of added K (0, 1.5, and 3%). Potassium was introduced by injection of a 10% K_2CO_3 (w/w) stock solution (0, 16, and 32 ml/d) directly into the fermenters twice daily immediately after each feeding. Distilled water was also injected (32, 16, and 0 mL/d, respectively) to maintain a total injection (K_2CO_3 + water) volume of 32 mL/d. The K added was 0, 0.9, and 1.8 g/d or 0 (K0), 1.5% (K1.5), or 3% (K3) of the daily feed. Cultures on the low fat diet were fed 60 g of basal diet per day. Cultures on the high fat diet were fed 60 g of basal diet plus 2 g of soybean oil (mixed as a complete diet) for a total of 62 g of feed per day.

Similar to the first experiment, increasing K caused an increase ($P < 0.05$) in culture pH regardless of diet fat content (**Table 5**). Addition of K also affected VFA as in the first experiment, but differently depending on dietary fat content. For the low fat diet, increasing K again increased ($P < 0.05$) acetate and acetate to propionate ratio, and reduced ($P < 0.05$) propionate concentration. However, K had little effect on VFA when dietary fat content was high. As expected, the 4% added soybean oil increased ($P < 0.05$) *trans*-10, *cis*-12 CLA production from an average of 4.3 mg/d for the low fat diets to 53.8 mg/d for the high fat diets. Regardless of fat content in the diet, increasing K reduced ($P < 0.05$) *trans*-10, *cis*-12 CLA production supporting earlier results that K enhances milk fat content by re-directing the pathways of BH back to normal. As K decreased ($P < 0.01$) *trans*-10, *cis*-12 CLA, it also increased ($P < 0.05$) the production of *cis*-9, *trans*-11 CLA that is typical of normal BH.

Additional continuous culture experiments were run to determine if changes in BH intermediates seen for K_2CO_3 in the first two experiments could be duplicated with either KCl or with Na_2CO_3. Culture pH still increased ($P < 0.05$) from K_2CO_3 addition but not from KCl addition (**Table 6**). No changes in VFA, CLA, or *trans* monenes were observed following the addition of KCl. Carbonate effects on culture pH, VFA, and CLA were identical regardless if added as K_2CO_3 or as Na_2CO_3 (**Table 7**).

Conclusions

Early lactation cows can suffer from negative K balance due to greater K excretion, greater secretion of K in milk, and increased perspiration losses during heat stress.

With the inclusion of a higher amount of K in the early lactation diet, some studies showed an increase in production of milk, 3.5% FCM, and milk fat, which was not associated with an increase in DMI. The positive lactation responses to supplemental K supports the role of K ions in many essential biological processes such as the maintenance of osmotic potential within cells, nerve impulse transmission, enzyme reactions in cellular metabolism, the maintenance of normal kidney function, and cardiac, skeletal and smooth muscle function. Potassium supplementation also has increased milk fat percentages, which can be explained in part by reduced ruminal synthesis of biohydrogenation intermediates known to inhibit milk fat synthesis. The lowering of biohydrogenation intermediates that inhibit milk fat synthesis is likely mediated through the alkalizing effects of some K supplements to increase ruminal fluid pH.

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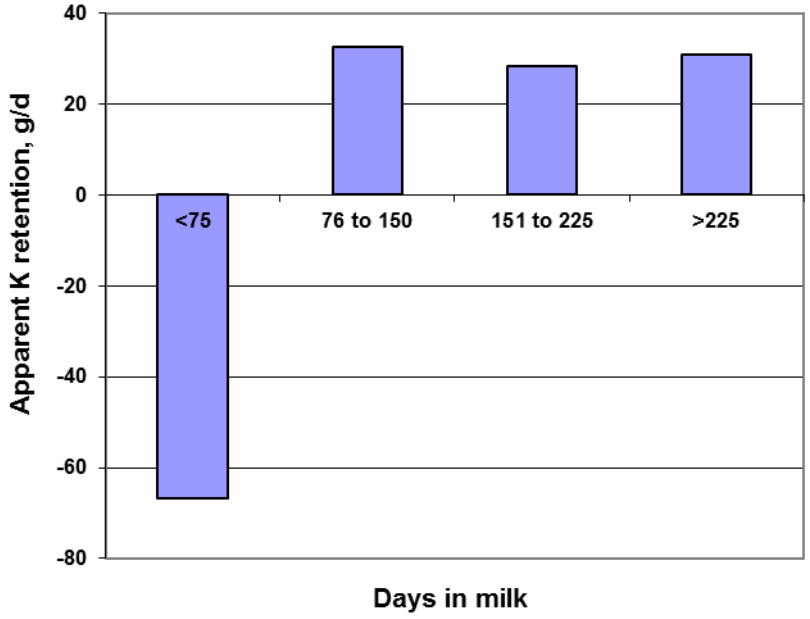

Figure 1. Apparent potassium retention of lactating cows at various days in milk.

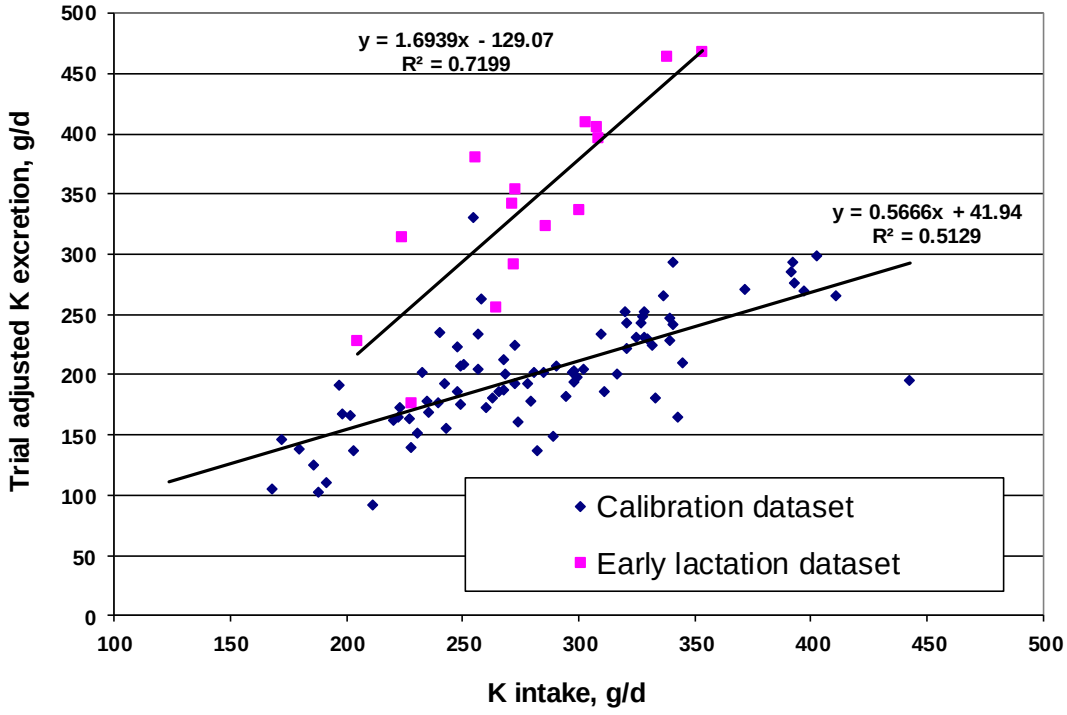

Figure 2. The relationship of potassium intake and potassium excretion for cows in the calibration and early lactation datasets.

Table 1. Summary of nutrient composition of diets in early lactation DCAD study.

% of DM	Control	DCAD+
DM	60.1	59.4
CP	16.1	16.1
ADF	19.8	19.3
NDF	35.0	34.7
Ash	7.0	8.6
Ca	0.69	0.66
P	0.37	0.36
Mg	0.43	0.45
K	1.28	2.07
DCAD[1]	32	53

[1] DCAD (mEq/100 g of DM) = (Na + K) – (Cl + S).

Table 2. Body weight, dry matter intake, milk production, and milk component production in early lactation DCAD study.

Item	Control	DCAD+	*P* <
BW, kg	669	674	0.49
DMI, kg/d	26.2	26.8	0.20
Milk, kg/d	39.3	40.8	0.01
ECM, kg/d	41.3	44.3	0.24
3.5% FCM, kg/d	42.2	46.1	0.09
Fat, kg/d	1.55	1.75	0.03
True protein, kg/d	1.16	1.14	0.12

Table 3. Milk fatty acid composition in early lactation DCAD study.

Item, % of total FA	**DCAD**	**Con**	***P* <**
C16:1	1.32	1.47	0.03
C18:0	14.2	12.6	0.02
t6,t8 C18:1	0.31	0.36	0.03
t9 C18:1	0.26	0.29	0.07
t10 C18:1	0.4	0.68	0.03
t11 C18:1	1.05	1.43	0.11
t12 C18:1	0.55	0.61	0.09
c9, t11 CLA	0.34	0.44	0.03

Table 4. Changes in pH, VFA, and biohydrogenation intermediates in continuous cultures dosed with increasing amounts of K_2CO_3.

	Treatment[1]					
	K0	K1	K2	K3	NaOH	SE
pH d8-10[a]	6.01	6.22	6.25	6.38	6.29	0.12
VFA, mol/100 mol						
Acetate[ab]	48.2	48.7	52.0	52.1	48.7	1.0
Propionate[ab]	36.2	35.6	32.2	32.9	36.7	1.4
Ac/Pr[ab]	1.34	1.37	1.66	1.60	1.33	0.09
Total VFA, mM	103.5	95.2	98.4	95.1	95.4	6.0
BH intermediates, mg/d						
t10 C18:1	537.6	499.8	461.1	538.2	575.8	38.4
t10, c12 CLA	11.6	11.3	7.9	7.6	13.2	2.6
c9, t11 CLA[a]	2.3	4.9	7.1	6.8	6.8	1.1

[a] Linear response of K0 through K3 ($P < 0.05$).
[b] K3 and NaOH differ ($P < 0.05$).
[1] K_2CO_3 injected into culture flasks to provide the equivalent of 0, 1, 2, and 3% added K. The NaOH treatment used injections of NaOH into fermentation flasks to maintain the same pH as the K3 treatment.

Table 5. Changes in pH, VFA, and biohydrogenation intermediates in continuous cultures fed a low or high fat diet in combination with three concentrations of added K_2CO_3.

	0% Fat			4% Fat			
	0	1.5	3	0	1.5	3	SEM
pH d8-10 [ab]	5.99	6.32	6.36	5.91	6.13	6.17	0.10
VFA, mol/100 mol							
Acetate [bc]	46.6	56.1	57.2	50.7	52.1	50.3	2.5
Propionate [abc]	34.7	25.8	22.4	33.5	31.3	32.0	2.0
Ac/Pr [abc]	1.35	2.21	2.59	1.58	1.72	1.60	0.19
Total VFA, mM	76.7	69.7	71.6	79.9	85.8	79.1	7.1
BH intermediates, mg/d							
trans-18:1[abc]	320.9	140.0	132.3	883.9	773.7	444.7	69.0
t10, c12 CLA [ab]	6.9	3.4	2.7	65.8	44.7	50.9	3.6
c9, t11 CLA [b]	2.6	5.7	7.0	2.7	6.4	8.3	1.0

[a] Fat effect ($P < 0.05$).
[b] K effect ($P < 0.05$).
[c] Fat x K interaction ($P < 0.05$).

Table 6. Changes in pH, VFA, and biohydrogenation intermediates in continuous cultures fed a low or high fat diet in combination with two sources of added K.

	0% Fat			3% Fat			
	0% K	K_2CO_3	KCl	0% K	K_2CO_3	KCl	SE
pH d10 [b]	6.36^{x}	6.57^{y}	6.35^{x}	6.33^{x}	6.47^{y}	6.21^{y}	0.071
4 hVFA, mol/100 mol							
Acetate [ab]	51.8^{y}	53.9^{x}	50.3^{y}	49.3^{y}	52.6^{x}	51.1^{y}	0.89
Propionate [ab]	30.2^{x}	26.3^{y}	30.6^{x}	32.0^{x}	28.8^{y}	31.4^{x}	1.10
Ac/Pr [ab]	1.72^{y}	2.06^{x}	1.68^{y}	1.56^{y}	1.83^{x}	1.60^{y}	0.062
Total VFA, mM	76.2^{xy}	67.3^{y}	79.0^{x}	83.7^{x}	67.4^{y}	79.0^{x}	6.29
BH intermediates, mg/d							
t10-18:1[ab]	25.6	17.8	24.3	221.2^{x}	143.6^{y}	196.5^{x}	15.9
t11-18:1 [abc]	69.4	104.5	65.4	130.8^{y}	272.3^{x}	148.6^{y}	16.9
t10, c12 CLA [a]	2.14	2.21	2.08	37.3	36.4	41.7	2.34
c9, t11 CLA [abc]	3.09	5.80	3.60	7.25^{y}	16.00^{x}	8.97^{y}	1.00

[a] Fat effect ($P < 0.05$).
[b] K effect ($P < 0.05$).
[c] Fat x K interaction ($P < 0.05$).
[xy] Means within a fat level with the same letter were not different ($P < 0.05$).

Table 7. Changes in pH, VFA, and biohydrogenation intermediates in continuous cultures fed a low or high fat diet in combination with two sources of added K.

	Treatment				
	CON	MIX	KCO3	NaCO3	SE
pH [a]	6.05	6.40	6.31	6.36	0.11
VFA, mol/100 mol					
Acetate [a]	58.85	64.57	65.50	66.57	2.62
Propionate [a]	27.24	23.00	22.70	21.23	1.57
Ac/Pr [a]	2.12	2.89	2.93	3.15	0.42
BH intermediates, mg/d					
t-10 18:1[a]	504.1	256.2	232.8	266.0	32.8
t-12 18:1[a]	7.66	0.46	0.92	3.70	1.88
c9, t11 CLA [a]	8.37	11.07	11.39	12.57	1.19
t10, c12 CLA [b]	19.73	12.10	12.38	13.97	3.64

[1]1:1 mix of K carbonate and Na carbonate
[a] CON differed from others ($P < 0.05$).
[b] CON differed from others ($P < 0.10$).

SESSION NOTES