

Introduction

- Propionate is the primary precursor for gluconeogenesis, supplying 60-74% of the required carbon (Aschenbach et al., 2010).
- Increasing propionate is thought to alter glucose metabolism and consequently feed intake. This in-turn could improve feed efficiency.
- Solute carrier family 16 member 1 (*SLC16A1*) encodes for the monocarboxylate transporter 1 protein. Though not fully understood MCT 1 is thought to be responsible for propionate uptake into hepatocytes.
- Phosphoenolpyruvate carboxykinase 1 (*PCK1*) is known to control gluconeogenesis regulation within the liver. Presumably because it is closely linked to PEPCK activity and PEPCK regulates propionate in the gluconeogenesis pathway. (Greenfield et al., 2000; Al-Trad et al., 2010, Hartwell et al., 2001)
- Phosphoenolpyruvate carboxykinase 2 (*PCK2*) regulates the catalysis of oxaloacetate into phosphoenolpyruvate in the mitochondria.
- Glucose-6-phosphatase (*G6PC*) assists in transporting glucose out of the liver at the end of gluconeogenesis.
- Solute carrier family 2 member 2 (*SLC2A2*) encodes for glucose transporter 2 which assists in the transport of glucose into and out of the liver. Increased propionate uptake could lead to an increase in GLUT2 expression (Gelardi et al., 1999).

Hypothesis & Objective

Hypothesis: Supplementing propionate will increase the expression of genes involved in gluconeogenesis.

Objective: To ascertain if supplementing calcium propionate (CaP) in varying amounts would result in the increased expression of genes related to glucose metabolism in the liver.

Materials & Methods

- All animal procedures were approved by Oklahoma State University Institutional Animal Care and Use Committee.
- The study utilized cannulated Holstein steers. (n = 6) (BW: 418 ± 17.7 kg)
- A 3 × 6 Latin rectangle design was used with 15-d periods.
- The treatments were as follows:

Treatment	g Propionate/d
Control (CON)	0
Low Propionate (LOW)	100
High Propionate (HIGH)	300

Table 1. Daily treatment dosage.

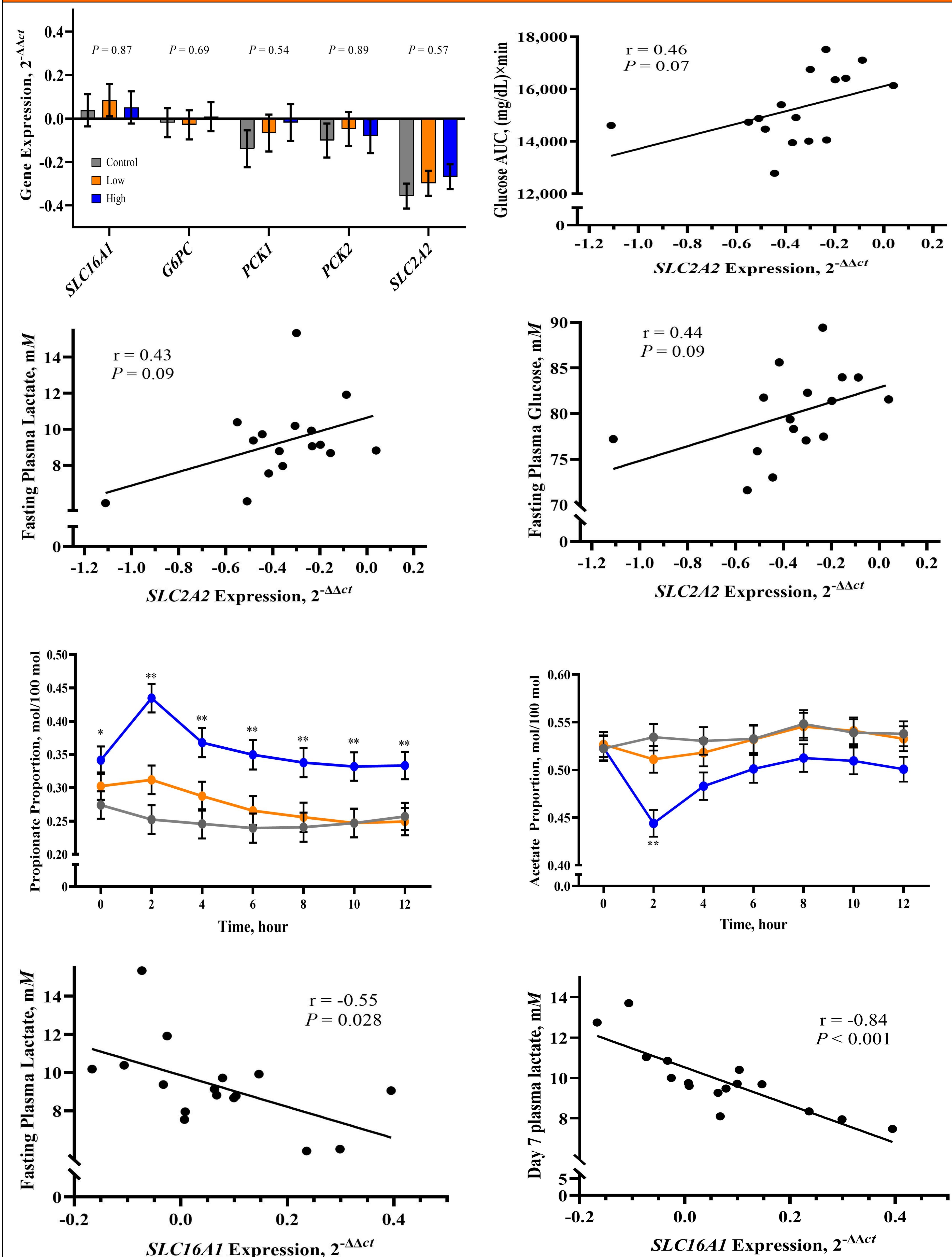
- Treatments were administered in halves twice a day through ruminal cannulas.
- Finishing ration was provided ad libitum and Insentec feeders were used to record intake. Water was unrestricted.
- Intravenous glucose tolerance tests were conducted on d14 of each period. Liver biopsies were taken on d15 of each period.
- RNA was extracted from the liver tissue, reverse transcribed for cDNA and analyzed through quantitative real-time PCR.
- Five target genes involved in gluconeogenesis were analyzed and included: *SLC16A1*, *PCK1*, *PCK2*, *G6PC*, and *SLC2A2*.
- Data were analyzed using a mixed model (SAS 9.4; SAS Inst., Cary, NC) with treatment, period and intake as fixed effects and steer as a random effect. Pearson correlations were also analyzed for gene expression ($2^{-\Delta\Delta Ct}$), plasma metabolite concentrations and variables from the IVGTT.

Ingredient	%, DM
Prairie Hay	6
Alfalfa Hay	4
Rolled Corn	60
Sweet Bran, wet	20
Liquid Supplement	5
Mineral	5

Table 2. Ingredient composition of the diet on a dry matter basis.



Results



Conclusions

These data indicate increased propionate may not have an impact on hepatic gene expression. This is not necessarily indicative of lack of change in protein expression or function. Further research could explore those factors and whether this is true at all growth stages.