COMPARATIVE GUT PHYSIOLOGY SYMPOSIUM: COMPARATIVE PHYSIOLOGY OF LOWER GUT

0198 Integrated responses to feeding, comparative aspects. J. Furness*, *University of Melbourne, Parkville, Australia.*

The optimal utilization of nutrients requires an integrated response of the gastrointestinal tract to ingested food. Broad mechanisms are similar in all mammals and involve sensing food components through olfaction, taste and specialized receptors within the stomach and intestines. The sensing of food components leads to release of gut hormones and activation of nerves, which in turn modify digestive functions. Bacteria, viruses, fungi and potentially injurious substances in foods activate tissue defense mechanisms. While the responses to nutrients lead to broadly similar changes in appetite, satiety and food-seeking behavior, gastrointestinal motility, release of digestive enzymes and induction of nutrient transporters, the requirements in different animals differ. To simplify discussion, we can divide species into ruminant foregut fermenters (such as cattle and sheep), non-ruminant foregut fermenters (e.g., kangaroo, colobus monkey), hind-gut fermenters (such as horse), and auto-enzyme dependent digesters (pig, human) that also gain nutrition from hind-gut fermentation. Ruminants are efficient digesters because the rumenal movements are able to stratify food into gas, fluid and particle components, retaining food to be digested in the forestomach and passing more fully digested material into the abomasum and duodenum, and also being able to return food from the forestomach to the mouth for mastication and limited enzyme exposure. Poultry have multiple stomachs that allow for storage, digestion and tituration, but not fermentation. Ruminants lose efficiency in that most carbohydrate is utilized by gastric bacteria and very little glucose reaches the small intestine. Thus glucose must be synthesized from short chain fatty acids produced by bacteria, whereas species such as pig and human convert carbohydrate to glucose enzymatically. Thus ruminants are more prone than other groups to enter into negative glucose balance, for example during post-partum lactation. Obligatory by-products of fermentation are carbon dioxide and methane. Foregut fermenters are also advantaged by being able to readily utilize vitamins produced by fermentation. It is thought that coprophagy by hind-gut fermenters, such as rabbits, provides access to such vitamins. Foregut fermentation also contributes to detoxification, for which hindgut fermenters and autoenzymic digesters rely primarily on the liver. With these differences in mind, it is necessary to closely consider what information can be readily extrapolated between species. Conversely, we can point to similarities in neural and hormonal signaling systems and products of digestion, achieved in different ways that are available for energy utilization and incorporation into tissues.

Key Words: digestive physiology, fermentation, glucose, comparative

0199 Expression of nutrient transporter mRNA in the jejunum of high and low efficiency steers. H. C. Cunningham[1], Z. T. L. Gray[1], S. I. Paisley[1], K. J. Austin[1], K. M. Cammack[1] and A. M. Meyer*[2], *[1]Department of Animal Science, University of Wyoming, Laramie, [2]Division of Animal Sciences, University of Missouri, Columbia.*

We hypothesized that small intestinal expression of nutrient transport-related genes contributes to differences in metabolic efficiency in beef cattle. The objective was to investigate jejunal expression of glucose transporter 2 (GLUT2), glucose transporter 5 (GLUT5), sodium-dependent glucose transporter 1 (SGLT1), and peptide transporter 1 (PepT1) in finishing steers classified as high and low efficiency based on residual feed intake (RFI). Hereford-Angus crossbred steers (yr 1, $n = 59$, 461 ± 4.5 kg initial BW; yr 2, $n = 75$, 412 ± 3.8 kg initial BW) from a single contemporary group in each year (birth through slaughter) were used. Steers were fed a finishing diet (yr 1, 11.4% CP, 2.0 Mcal NE_m/kg, 1.35 Mcal NE_g/kg; yr 2, 13.2% CP, 1.8 Mcal NE_m/kg, 1.19 Mcal NE_g/kg; DM basis) for 57 (yr 1) or 80 d (yr 2) using the GrowSafe system. Residual feed intake was calculated as the difference between actual and expected feed intake of each individual, where expected intake was determined by regressing ADG and metabolic midweight on actual intake. Following the intake test in each year, the 20% most efficient (low RFI, $n = 8$/yr) and 20% least efficient (high RFI, $n = 8$/yr) steers with 12th rib fat thickness $\geq$ 1.02 cm were slaughtered between 5 and 8 d after the feed intake test conclusion. At slaughter, jejunal mucosa was flash-frozen for real-time RT-PCR determination of GLUT2, GLUT5, SGLT1, and PepT1. Data were analyzed with PROC MIXED in SAS 9.2 using RFI class (high vs. low efficiency), year, and their interaction as fixed effects. Expression of SGLT1 was affected ($P = 0.02$) by the RFI class × year interaction, although there were no differences ($P \geq 0.12$) within each year. Jejunal expression of GLUT2, GLUT5, and PepT1 were not affected ($P \geq 0.18$) by RFI class; however, expression of each was greater ($P \leq 0.03$) in yr 2 than yr 1. It was previously reported in this study that jejunal expression of y^+LAT2, a basolateral membrane cationic AA transporter, was greater for low efficiency than high efficiency steers in yr 2. Additionally, low efficiency steers had greater jejunal expression of vascular endothelial growth factor (VEGF, a major regulator of angiogenesis) than high efficiency in both years. These data suggest that nutrient absorption and transport in the small intestine may play a role in whole animal feed efficiency of beef cattle.

Key Words: feed efficiency, nutrient transport, small intestine

0200 Comparative physiology of glucagon-like peptide 2—Implications and applications for production and health of ruminants. E. E. Connor*, M. P. Walker, C. M. Evock-Clover, T. H. Elsasser, and S. Kahl, [1]*USDA-ARS, Bovine Functional Genomics Laboratory, Beltsville, MD*.

Glucagon-like peptide 2 (GLP-2) is a 33-amino acid peptide derived from proteolytic cleavage of proglucagon by prohormone convertase 1/3 in enteroendocrine L-cells. Studies conducted in humans, rodent models, and in vitro indicate that GLP-2 is secreted in response to the presence of molecules in the intestinal lumen including fatty acids, carbohydrates, amino acids, and bile acids, which are detected by luminal chemosensors. The physiological actions of GLP-2 are mediated by its G protein coupled receptor expressed primarily in the intestinal tract on enteric neurons, enteroendocrine cells, and myofibroblasts. The biological activity of GLP-2 is further regulated by dipeptidyl peptidase IV, which rapidly cleaves the N terminus of GLP-2 that is responsible for GLP-2 receptor activation. Within the gut, GLP-2 increases nutrient absorption, crypt cell proliferation, and mesenteric blood flow, and decreases gut permeability and motility, epithelial cell apoptosis, and inflammation. Outside the gut, GLP-2 reduces bone resorption, can suppress appetite, and is cytoprotective in the lung. Thus, GLP-2 has been studied intensively as a therapeutic to improve intestinal function of humans during parenteral nutrition and following small bowel resection, and more recently, as a treatment for osteoporosis, obesity-related disorders, and to reduce cellular damage associated with inflammation of the gut and lungs. Recent studies demonstrate that GLP-2 has many similar biological actions and properties in ruminants as in monogastrics, including the potential to reduce intestinal nitro-oxidative stress in calves caused by parasitic diseases like coccidiosis. Due to its beneficial impacts on nutrient absorption, gut healing, and normal gut development, GLP-2 therapy offers significant opportunities to improve calf health and production efficiency. However, GLP-2 therapies require an extended time course to achieve desired physiological responses, as well as daily administration due to the hormone's short half-life. Thus, practical means of administration and alternative strategies to enhance basal GLP-2 secretion (e.g., through specific feed additives), which are more likely to achieve consumer acceptance, are needed. Opportunities to address these challenges are discussed.

Key Words: cattle, glucagon-like peptide-2, gut health

0201 Differential subcellular and cellular storage of GLP-1 and PYY and its implications. J. Furness*[1], H. J. Cho[1], S. Kosari[1], and D. M. Bravo[2], [1]*University of Melbourne, Parkville, Australia,* [2]*PANCOSMA SA, Geneva, Switzerland.*

Intestinal L cells have key roles in the detection of the chemical environment in the gut lumen, to which they react by the release of hormones that influence appetite, proximal gut motility, insulin secretion and mucosal function (Furness et al., Nature Gastroenterology, 10, 729–740, 2013). Important amongst L cell hormones are glucagon-like peptide1 (GLP-1) and peptide tyrosine-tyrosine (PYY), which are products of separate genes. The conventional description of their localization is that GLP-1 and PYY are in the same storage vesicles in the same cells. However, GLP-1 and PYY have different functions, particularly in relation to insulin secretion and mucosal function. We have used super-resolution (3D-SIM) microscopy and double-labelling immunohistochemistry to investigate the subcellular localizations of the hormones, and digital scanning microscopy to investigate cell populations. Super-resolution microscopy revealed that GLP-1 and PYY are in separate storage organelles in enteroendocrine cells from mouse, rat, pig and human. The majority of the organelles were 150-170 nm or less in diameter, and are concluded to be secretory vesicles. Only 10-20% of organelles had immunoreactivity for both hormones. Even this may be an overestimate, as touching or very close vesicles may not be effectively resolved, even with super-resolution microscopy. In investigating co-localisation at the cell level, we included glucagon-like insulinotropic peptide (GIP), an incretin of K cells, in the analysis. The work shows that there is a K/L cell gradient in the mouse intestine. From the duodenum to the distal colon, there are populations of cells with GIP alone, GLP alone, PYY alone and all combinations of the three hormones. Greatest numbers of GIP cells were in the doudenum and jejunum, where 30—40% contained only GIP and the remainder also contained GLP-1. A small proportion also contained PYY. Similar patterns of overlap occurred in the proximal and distal ileum, where GLP-1 was the dominant peptide, which was often alone, or co-localised with PYY. In the large intestine the majority of cells contained both GLP-1 and PYY, but cells with only one of these and cells with all three hormones were found. The findings reveal a structural basis for the separate or preferential control of GLP-1, PYY (and possibly GIP) release. A number of physiological studies imply that there can be differential release of GLP-1 and PYY. This should be investigated further.

Key Words: enteroendocrine cells, incretins, glucagon like peptide, peptideYY

0202 The role of the microbiome in gut immune system development in newborn and mature cattle. P. J. Griebel[*1], N. Malmuthuge[2], G. Liang[2], M. Zhou[2], and L. L. Guan[2], *[1]Vaccine and Infectious Disease Organization, University of Saskatchewan, Saskatoon, SK, Canada, [2]University of Alberta, Edmonton, AB, Canada*.

There is increasing evidence in a variety of mammalian species that the commensal microbiome has diverse effects on mucosal immune system development and function. It is difficult, however, to clearly delineate the effects of the microbiome from other contributing factors, such as diet, environment, and host genetics. The bovine gastrointestinal tract (GIT) is rapidly colonized during birth and these pioneer species are then replaced by a succession of changes, involving both increased microbioal density and diversity. This succession occurs rapidly during the first week of life and then progresses much more slowly. Characterization of the microbiome in the neonatal bovine GIT at both a family and species level revealed marked bacterial variation among individual animals. Furthermore, the composition of the microbiome varied significantly when comparing ingesta- and mucosa-associated communities within individual GIT regions. The first week postpartum is also a very dynamic developmental period in the bovine GIT with significant changes in both mucosal barrier and immune function. These developmental changes were analyzed by profiling miRNAs expressed throughout the small intestine. This analysis confirmed the greatest changes in GIT development occurred during the first week of life with differential expression of miRNAs involved in regulating a broad range of GIT developmental and immunological processes. Relatively few miRNAs were differentially expressed when comparing tissues collected from 6 wk old calves and 3 wk old calves. Correlation analyses between total bacterial numbers and specific families revealed significant associations between the commensal microbiome and the expression of genes involved in regulating both mucosal barrier and innate immune function. It appears the microbiome is an important factor influencing age-dependent changes in the expression of immune function genes. These correlation analyses also suggest that regional differences in the microbiome may be associated with significant regional differences in the expression of innate immune genes. This information provides the baseline to begin analyzing the role of individual bacterial species and interactions among bacterial species in regulating mucosal immune system function in healthy animals and during enteric infections. New experimental models will be required, however, to clearly delineate the role of specific bacterial species in the complex interaction between microbiome and host.

Key Words: microbiome, innate immunity, miRNA

0203 The effects of intentionally-induced leaky gut on metabolism and production in lactating Holstein dairy cows. S. K. Stoakes[*1], M.Abuajamieh[1], D. B. Snider[1], M. V. Sanz Fernandez[1], J. S. Johnson[1], P. J. Gorden[1], N. K. Gabler[1], H. B. Green[2], K. M. Schoenberg[2], and L. H. Baumgard[1], *[1]Iowa State University, Ames, IA, [2]Elanco Animal Health, Indianapolis, IN.*

Presumably, intestinal barrier dysfunction negatively affects productivity, but it has never been studied in a controlled lactation experiment. Objectives were to elucidate consequences of leaky gut in otherwise healthy mid-lactation dairy cows. Twelve Holstein cows (170.0 ± 15.1 DIM, 670 ± 13 kg BW, parity 1 to 5) were enrolled in two experimental periods. Period 1 (P1) lasted 5d and served as baseline for period 2 (P2), which lasted 7d in which cows received one of two treatments I.V. twice daily: 1) sterile saline (control) or 2) γ secretase inhibitor (GSI; 1.5 mg/kg BW). GSI specifically inhibits crypt stem cell differentiation into enterocytes via disrupting Notch signaling. Control animals were pair-fed (PF) to GSI-treated cows (to eliminate the confounding effects of dissimilar feed intake). GSI administration caused a progressive reduction in DMI ($P < 0.01$; 82%) and milk yield ($P < 0.01$; 57%), but there was no treatment effect on milk components. Cows in both treatments lost a similar amount of BW (56 kg) by the end of P2. Histological analysis indicated GSI increased jejunum goblet cell area (3.3 vs. 1.0%; $P = 0.02$), tended to: deepen villous crypts ($P = 0.06$), reduce villous height ($P = 0.07$) and alter villous height to crypt depth ratio ($P = 0.08$). No treatment effects were detected in ileum or colon morphology, but manure score (a measure of fecal consistency) was decreased 36% ($P < 0.01$) in the GSI-treated vs. PF controls. By d5−7 of P2, circulating lipopolysaccharide (LPS) was increased > threefold in PF controls compared to GSI-treated cows. Plasma LPS binding protein (LPB) levels progressively increased in both treatments but were increased (42%, $P < 0.01$) in GSI-treated vs. PF controls by d5−7 of P2. By the end of P2, the LPS:LPB ratio was increased 3.6-fold ($P < 0.05$) in PF controls compared to GSI-treated cows. Haptoglobin and serum amyloid-A concentrations progressively increased (> 400 and > fivefold, respectively) similarly in both treatments. Circulating IFNγ, TNFα and IL-6 were unaffected by treatment or time. GSI-treated cows tended to have increased plasma insulin ($P = 0.07$) and decreased circulating NEFA ($P = 0.06$) vs. PF cows. For both treatments, plasma glucose decreased with time ($P = 0.05$), while BHBA progressively increased (87%; $P = 0.08$). There were no treatment differences in spleen weight, liver weight, liver moisture, or liver lipid content. In summary, based on all the data, GSI-treatment compromised intestinal integrity and markedly reduced feed intake and milk yield. Further, we have demonstrated progressive feed reduction also negatively influenced intestinal integrity.

Key Words: lipopolysaccharide, insulin

0204 Manipulating goblet cell function to protect against enteric infection. M. Wlodarska*, *University of British Columbia, Vancouver, BC, Canada.*

Mucus production by goblet cells serves as one of the crucial mucosal defenses at the interface between the eukaryotic and prokaryotic cells and yet the immunoregulatory pathways involved remain uncharacterized. The inner mucus layer of the intestine functions as a barrier, which serves to minimize microbial translocation, prevents excessive immune activation, and decrease infection. Here we have described methodology to alter the thickness of the inner mucus layer through treatment with antibiotic or a phytochemical. We showed that the antibiotic metronidazole caused a significant thinning of the inner mucus layer accompanied by a dramatic change in the microbial community structure. In contrast, treatment with the phytochemical eugenol resulted in a significant thickening of the inner mucus layer that was accompanied by a change in the microbial community. These changes in community structure were complimentary; sequencing showed that groups depleted by metronidazole treatment were more abundant with eugenol treatment. To investigate how changes in the integrity of the inner mucus layer affect intestinal defense, Citrobacter rodentium (Cr) was used to examine susceptibility to enteric-induced colitis. Metronidazole-induced reduction in mucus thickness correlated with exacerbated severity of Cr-induced colitis. Thickening of the inner mucus layer with eugenol treatment resulted in protection from Cr-induced colitis. Further, we identified a novel innate immune pathway involved in regulation of goblet cell function and mucus layer production. The NLRP6 inflammasome was shown to regulate mucus secretion and deficiency in any component of the NLRP6 inflammasome resulted in impaired goblet cell function preventing mucin granule exocytosis and mucus layer formation. Abrogated mucus secretion led to increased invasiveness and pathology of Cr infection. Mechanistically, NLRP6 deficiency led to stalled autophagy in goblet cells, providing a link between inflammasome activity, autophagy, mucus exocytosis, and antimicrobial barrier function.

Key Words: mucus, goblet cell, *Citrobacter rodentium,* phytochemical, eugenol

0205 Nutritional immunology in swine. Y. Liu*[1], D. M. Bravo[2], and J. Pettigrew[1], [1]*University of Illinois at Urbana-Champaign, Urbana,* [2]*PANCOSMA SA, Geneva, Switzerland.*

The immune system of pigs is vital as its proper functioning protects the pig from disease and health. It also causes inflammation, which contributes to the animal's ability to fight off infection but also inhibits growth performance by reducing feed intake and diverting amino acids and nutrients away from growth to the immune response. It is now clear that reducing inflammation would benefit pig health. We have shown that several plant extracts can do just that, as shown here. Our in vitro study reported that several plant extracts (anethol, capsicum oleoresin, carvacrol, cinnamaldehyde, eugenol, garlicon, and turmeric oleoresin) suppressed ($P < 0.05$) pro-inflammatory cytokines' secretion from lipopolysaccharide-stimulated porcine alveolar macrophages, which indicates the in vitro anti-inflammatory effects of these plant extracts. Results from an in vivo *Escherichia coli* (*E. coli*) challenge study showed that feeding capsicum oleoresin, garlicon, or turmeric oleoresin reduced ($P < 0.05$) diarrhea of *E. coli*-challenged pigs. Feeding these 3 plant extracts also decreased inflammatory responses of *E. coli*-challenged pigs, as indicated by reduced ($P < 0.05$) white blood cell numbers, serum pro-inflammatory cytokines, and acute phase proteins when pigs were fed plant extracts compared with pigs fed control diet. A potential mechanism of action is that plant extracts may enhance gut mucosa health and attenuate the overstimulation of the immune system. The microarray data from the same in vivo study indicated that these plant extracts counteracted ($P < 0.05$) the effects of *E. coli* by reducing the expression of genes involved in antigen presentation or other biological processes of immune responses. Another in vivo study was conducted with porcine reproductive and respiratory syndrome virus (PRRSV) challenge. The results of this study indicated that feeding these 3 plant extracts to nursery pigs enhanced the pigs' immune responses to a PRRSV challenge and may help alleviate negative impacts of infection, as indicated by reducing ($P < 0.05$) viral load and serum concentrations of inflammatory mediators, and shortening ($P < 0.05$) the time of fever in PRRSV-infected pigs. In conclusion, feed additives, such as certain plant extracts, may potentially improve pig health and disease resistance by modulating inflammation.

Key Words: immunology, pigs, plant extracts

0206 Mucosal IgA responses to members of the gut microbiota in healthy vs. malnourished Malawian children. A. Kau*, *Center for Genome Sciences & Systems Biology, St. Louis, MO*.

Childhood malnutrition is a major contributor to childhood morbidity and mortality worldwide. While inflammatory conditions, such as recurrent infection and environmental enteropathy, have been implicated in pathogenesis, the role of the interaction between the host immune system and the gut microbiota in shaping the outcomes remains poorly understood. To identify microbes that were targeted by the host gut mucosal immune response, we developed a flow cytometry-based method to recover and characterize viable gut microbes based on their binding to immunoglobulin A (IgA). Fecal microbiota from Malawian twins discordant for a form of severe acute malnutrition (Kwashiorkor) were transplanted into different groups of adult germ-free C57BL/6J mice that were fed a representative Malawian diet deficient in macro- and micronutrients. IgA-targeted microbes in the fecal micro-

biota of these 'kwashiorkor-Malawian diet–fed' (KM) mice were purified by FACS and transferred to a second group of germ-free mice, also fed the Malawian diet. KM-IgA+ consortia produced (i) dramatic weight loss; (ii) pronounced gut barrier dysfunction manifest by sepsis, and histopathologic changes that were most severe in the colon but also manifest in the small intestine where disruption of the intracellular pattern of epithelial cell adhesion molecule (EpCAM) staining and evidence of extrusion of cells along the length of villi rather than just at the apical region was evident, and (iii) high mortality phenotype in recipient animals. This phenotype was both diet-dependent and microbiota dependent: we did not observe it in mice fed a macro- and micronutrient replete diet or those receiving IgA targeted taxa from mice harboring the healthy co-twin's microbiota. The barrier dysfunction and mortality phenotypes transmitted by the IgA+ consortium could be mitigated through the administration of IgA-targeted microbes from a mouse colonized with a healthy microbiota, including *Akkermansia muciniphila* and *Clostridium scindens*. Applying this FACS-based approach directly to the fecal microbiota of two cohorts of Malawian children, we found that members of Enterobacteriaceae are prominent targets of IgA responses in individuals with severe acute malnutrition. Targeting to other bacterial taxa, including members of Veillonellaceae and Lactobacillaceae, were correlated with the degree of stunting. These findings indicate that this approach for identifying and quantifying mucosal immune responses to members of the fecal microbiota has potential diagnostic and therapeutic applications to childhood malnutrition, and perhaps other diseases affecting the gut mucosal immune system.

Key Words: gut microbiota, gut immunity, nutritional immunology

---

0207 Gut immune system: A new frontier for nutritional modulation of gut health.

H. Lillehoj*, *ARS USDA, Beltsville, MD.*

The gut represents a continuously evolving ecosystem consisting of trillions of commensal bacteria living in symbiosis with the host. This host-microbe interplay plays a crucial role in host physiological development and health. There is increasing evidence that shows a dynamic interaction between the gut microbiota and the development and function of the host immune system. Particularly, the intestinal microflora influences diverse aspects of host metabolic and immunological functions and this "crosstalk" with the various immune component of mucosal immunity, comprising cellular and soluble elements, is critical in maintaining gut homeostasis and gut health. Various chronic inflammatory conditions and metabolic diseases are closely associated with altered symbiotic relationship. Furthermore, probiotics, when used for the treatment of diseases caused by the dysregulation of the immune system, can exert a beneficial immune response. In this regard, as shown in our recent studies, the dietary immunomodulation of gut immunity in broiler chickens using natural dietary supplements, such as TLR ligands, DFMs and plant-derived phytochemicals that interact with innate sensing molecules to stimulate innate immunity, is a promising alternative strategy that can be applied to many infectious diseases where traditional prevention methods show limitations. Furthermore, application of high-throughput functional genomics tools in delineating detailed immune mechanisms associated with alternative disease control strategies will lead to enhanced understanding of how different alternative strategies function. As we move into the 21st Century and the demands for animal food products increase to meet the nutritional needs of a growing world population, developing drug-free alternative strategies to prevent and control animal diseases and to maintain gut homeostasis is a global issue and a critical component of our long-term efforts to alleviate poverty and world hunger.

Key Words: gut health, innate immunity, antibiotic alternatives

---

0208 Effect of dietary supplementation of *Capsicum* extract on immune responses, blood cell counts, blood chemistry, and oxidative stress markers in lactating dairy cows.

J. Oh*[1], S. Walusimbi[1], F. Giallongo[1], H. L. Weeks[1], T. W. Frederick[1], A. N. Hristov[1], J. L. Pate[1], R. J. Elias[2], L. Tao[2], and E. H. Wall[3], *[1]Department of Animal Science, The Pennsylvania State University, University Park, [2]Department of Food Science, The Pennsylvania State University, University Park, [3]Pancosma, Geneva, Switzerland*

The objective of this experiment was to investigate the effects of dietary *Capsicum* extract (CE) on T cell phenotypes, phagocytotic and oxidative burst activity of neutrophils, blood cell counts, blood chemistry, and oxidative stress markers in lactating dairy cows. Eight multiparous Holstein cows (DIM, 50 ± 9.6 d; BW, 591 ± 32.6 kg), including 3 ruminally-cannulated, were used in a replicated 4 × 4 Latin square design with 25-d periods. Treatments were 0 (CON), 250, 500, and 1000 mg CE/cow/d, in which the principal active compounds were capsaicin and dihydrocapsaicin. The CE was mixed with a small portion of the TMR and topdressed. Compared with CON, CE did not affect concentration of cluster of differentiation antigen (CD) 4 positive, $CD8^+$, $CD25^+$, and $\gamma\delta^+$ cells. The phagocytosis of neutrophils tended to quadratically increase ($P = 0.07$) with CE. Relative to CON, total white blood cells, neutrophils, and eosinophils were linearly increased ($P = 0.04$, 0.01, and 0.03, respectively) with CE supplementation. Treatments had no effect on lymphocytes, monocytes, and basophils. Red blood cells quadratically increased ($P = 0.04$) with CE. Hemoglobin was higher ($P < 0.01$) for CE than CON and responded quadratically to CE level of supplementation. Platelets were lower for CE than CON and linearly decreased ($P = 0.04$) with CE supplementation. Glucose, creatinine, al-

bumin, and total protein in blood plasma were not affected by CE. Blood urea N was increased ($P = 0.02$) by CE relative to CON and blood plasma P concentration tended to be lower ($P = 0.09$) for CE than CON. Although there was no effect of CE on oxygen radical absorbance capacity (ORAC) and thiobarbituric acid reactive substances (TBARS), CE tended to decrease ($P = 0.09$) 8-isoprostane relative to CON (14.7 vs. 16.5 pg/mL, respectively). In conclusion, dietary supplementation of CE did not affect T cell phenotypes and neutrophil activities in this study. However, CE increased total white blood cells, neutrophils, and eosinophils, and tended to decrease 8-isoprostane. It is suggested that CE may facilitate cells with function in innate immunity and reduce blood oxidative stress markers in lactating dairy cows.

Key Words: capsicum extract, immune response, oxidative stress

---

0209 Host-microbiome interactions during gut development across species: the role of milk.

T. B. McFadden*, *University of Missouri, Columbia.*

In humans, the benefits to babies of consuming whole colostrum and breast milk are now universally recognized. As a result, colostrum and breast milk are in high demand and are even donated and banked for use by mothers who are unable to lactate but still want to feed their babies breast milk instead of formula. Despite numerous studies on the functions and properties of human colostrum and breast milk, we have only scratched the surface in describing the specific bioactive factors and the mechanisms by which they protect the baby and promote GIT development, as well as programming future health and function. The same is true of all mammals as all mammalian offspring rely on mammary secretions (colostrum and milk) to provide nutrients and bioactive factors necessary for survival and subsequent growth and development. Although young of various species can be reared successfully on milk from other species, it is generally accepted that homologous milk provides the optimal match of components to requirements of the young. From this perspective, there is much potential to gain understanding from comparative studies of the unique relationships between milk composition and requirements of the suckling young across various species. In addition, recent recognition of the role of the gut microbiome as a key determinant of immunological and digestive system development opens new avenues by which milk components can have long term effects on future health and vitality of offspring. This paper will review current knowledge of milk composition and bioactive components and the effects they have on the young of various species. In addition, the effects of these components and temporal changes in the gut microbiome will be considered.

Key Words: gut microbiome, milk