410 Genome-wide association study of horn fly resistance in organic Holstein cows. B. L. Basiel*[1], L. C. Hardie[1], B. J. Heins[2], and C. D. Dechow[1], [1]*Pennsylvania State University, University Park, PA,* [2]*University of Minnesota, St. Paul, MN.*

The objective of this research was to identify genomic regions and candidate genes for horn fly resistance in organic Holstein cattle. Observations of fly load were recorded on 640 genotyped and 506 additional pastured Holstein cows with known pedigrees on 13 organic dairies. Fly load was determined using a 0 to 4 scale based on fly coverage on one side of the body where 0 indicated few to no flies and 4 indicated high infestation. Proportions of black and white coat coloration were observed on 390 individuals from photos taken during scoring. A multi-trait model of fly load, stayability, and proportion of coat color was used in single-step genomic analysis with the BLUPF90 family of programs. Stayability was included to account for bias if fly score was correlated with early-life survival and color was included because coat color was associated with fly load. Models varied by trait and included the random effect of cow. The color model included the fixed effect of herd-year-season of birth as did the stayability model in addition to the effect of parity and the random effects of herd-year-season of parity and permanent environment. The fly score model had fixed effects of scorer, the interaction of date scored and herd, stage of lactation by week, and parity. Estimated breeding values of the traits were decomposed by GWAS into SNP effects. The association between fly score (n = 435 observations of fly score from 233 cows with color evaluated) and a SNP variant of interest detected by GWAS was evaluated by a mixed linear model containing fixed effects of SNP variant, fly scorer, the interaction of date scored and herd, and a random effect of cow. The same model was run with percentage of black or white coat color as a covariate. A 1 MB region on BTA 6 explained the most variation (1.7%) in fly score and contained most of the significant SNP identified by GWAS. The only significant SNP located on a gene in this region, Hapmap27516-BTC-042465, was located on *KIT*, a gene associated with piebaldism. Variants of the SNP significantly influenced fly score where animals with 0 copies of the variant had the lowest fly score while those with 2 copies had the highest score. The SNP effect was nullified when color was added to the model and coloration tended to influence fly score with black cows having more flies than white cows. The results suggest that *KIT* is a likely candidate gene for horn fly resistance in Holstein cattle, potentially by altering coat color.

Key Words: fly, organic, *KIT*

411 Genomic analysis of visceral fat accumulation in Holstein cows. L. C. Novo*[1], L. Cavani[1], P. Pinedo[2], P. Melendez[3], and F. Peñagaricano[1], [1]*University of Wisconsin–Madison, Madison, WI,* [2]*Colorado State University, Fort Collins, CO,* [3]*Texas Tech University, Amarillo, TX.*

Visceral fat is related to important metabolic processes, including insulin sensitivity and lipid mobilization. The goal of this study was to identify individual genes, pathways, and molecular processes implicated in visceral fat deposition in dairy cows. Data from 172 genotyped Holstein cows classified at slaughterhouse as having low (n = 77; omental fold <5mm in thickness and minimum fat deposition in omentum) or high (n = 95; omental fold >20mm in thickness and marked fat deposition in omentum) omental fat were analyzed. The identification of regions with significant additive and non-additive genetic effects was performed using a 2-step mixed model-based approach. Genomic scans were followed by gene-set analyses to reveal the genetic mechanisms controlling abdominal obesity. The association mapping revealed 4 regions located on BTA13, BTA19, BTA20 and BTA24 with significant additive effects. These regions harbor genes, such as *SMAD7*, *ANKRD55*, and the *HOXB* family, that are implicated in lipolysis and insulin tolerance. Two regions located on BTA1 and BTA6 showed marked non-additive effects. The region on BTA1 harbors genes *MRAP* and *MIS18A*, that are involved in energy balance and obesity. The gene-set analysis revealed functional terms, such as tyrosine-kinase receptors and negative regulation of fibroblast growth factor receptor 1 and 2, that could be implicated in visceral fat deposition. We further evaluated the genetic link between visceral fat and 2 metabolic diseases, ketosis and displaced abomasum. For this, we analyzed 28k records of incidence of diseases from 14k cows across lactations using a single-step genomic BLUP approach. Notably, the region on BTA20 significantly associated with visceral fat deposition was also associated with the incidence of displaced abomasum. However, the correlations between SNP effects among visceral fat and metabolic diseases were negligible. Overall, our findings suggest that visceral fat deposition in dairy cows is controlled by both additive and non-additive effects. We detected at least one region with marked pleiotropic effects affecting both visceral fat and displaced abomasum.

Key Words: abdominal fat, genomic scan, metabolic diseases

412 Hepatic mitochondrial function in 2 Holstein genotypes during early and mid-lactation in a pasture-based system. M. Garcia-Roche*[1,2], D. Talmón[1], A. Mendoza[3], C. Quijano[2], A. Cassina[2], and M. Carriquiry[1], [1]*Departamento de Producción Animal y Pasturas, Facultad de Agronomía, Universidad de la República, Montevideo, Uruguay,* [2]*Departamento de Bioquímica, Facultad de Medicina, Universidad de la República, Montevideo, Uruguay,* [3]*INIA La Estanzuela, Colonia, Uruguay.*

To assess differences in hepatic mitochondrial function between Holstein genotypes, multiparous cows of New Zealand (NZH, n = 10) and North American (NAH, n = 10) origin (512 ± 19 vs. 563 ± 29 kg body weight, 3.1 ± 0.1 body condition score (BCS), fall calving) were used. Cows grazed a mixed pasture of *Medicago sativa* and *Dactylis glomerata* (8.7 ± 0.7 kg DM/ha in one grazing session during early lactation and 16.3 ± 1.1 kg DM/ha in 2 sessions during mid-lactation) and were supplemented with 7.3 ± 0.4 kg DM/d of concentrate and 4.4 ± 0.5 kg DM/d of corn silage and forage reserves during early lactation or 6.0 ± 0.4 kg DM/d of concentrate during mid-lactation. Liver biopsies were collected and cryopreserved at 21 and 180 ± 17 d postpartum (DPP) and mitochondrial function was assessed measuring oxygen consumption rates using: glutamate/malate (G/M), succinate (succ), palmitoyl-CoA/carnitine (p-CoA/car) and palmitoyl-carnitine (p-car). Data were analyzed using a mixed model that included DPP, genotype and their interaction as fixed effects. Milk yield was 5.3 kg/d higher ($P < 0.01$) for NAH vs. NZH cows but solid corrected milk yield did not differ ($P = 0.49$). Cow BCS was higher for NZH than NAH (2.74 vs. 2.67 ± 0.03, $P < 0.05$). Milk, fat and protein yield decreased from early to mid-lactation. State 3 respiration increased with the progress of lactation for all substrates (11 vs. 14 ± 1 for G/M, 34 vs. 45 ± 6 for succ, 6.2 vs. 10.3 ± 0.8 for p-CoA/car and 7 vs. 12 ± 1 pmolO_2/min/mg for p-car $P < 0.05$). Although maximum respiratory rate was not affected by DPP when G/M was used, it had a 1.7-fold increase ($P < 0.01$) from early to mid-lactation when p-car and p-CoA/car were used. State 3 and maximum respiratory rate in succinate-driven respiration were affected by the interaction between DPP and genotype ($P < 0.05$) as it was 2-fold greater for NZH vs. NAH cows at 180 DPP. Also, maximum respiratory rate when p-CoA/car was used tended to be higher for NAH than NZH (12 vs. 9 ± 1.4 pmolO_2/min/mg, $P = 0.09$). Our results point out to early-lactation mitochondrial function impairment and a preference

for gluconeogenic vs. fatty acids as ATP synthesis precursors for NZH and NAH, respectively.

Key Words: grazing, dairy

413 The effect of the interleukin-10 receptor alpha gene on bovine mammary epithelial cells infected with *Mycobacterium avium* ssp. *paratuberculosis*. A. Fong*[1], M. M. M. Muniz[1], U. K. Shandilya[1], A. Sharma[1], F. S. Schenkel[1], N. A. Karrow[1], and C. F. Baes[1,2], [1]*Centre for Genetic Improvement of Livestock, Department of Animal Biosciences, University of Guelph, Guelph, ON, Canada,* [2]*Institute of Genetics, Vetsuisse Faculty, University of Bern, Bern, Switzerland.*

Johne's disease is a chronic wasting disease caused by the bacterium *Mycobacterium avium* subspecies *paratuberculosis* (MAP). It is highly contagious and eventually leads to death. Testing for Johne's disease in dairy cattle is often done with a milk ELISA test, as it is non-invasive, has a quick turn-around time, and is low-cost. However, it has low sensitivity (~30%), which can lead to false-negative results and it may not detect early stages of infection. There is currently no cure for Johne's disease and no vaccine is available in Canada. A vaccine has been approved for use in the United States of America however, it does not provide complete protection against the disease. In combination with improvements in management and vaccine development, genetic selection can further enhance control of this disease, as heritability for resistance to Johne's disease has been estimated to be around 6%. One candidate gene of particular interest for this disease is the gene encoding for the interleukin-10 receptor subunit α (IL10Rα). This is an anti-inflammatory cytokine that aids in tissue repair by regulating the antimicrobial activity of macrophages. The expression of IL10Rα is upregulated in response to MAP bacteria and this is suspected to help the survival of the bacteria. A bovine mammary epithelial cell line (MAC-T) was created with the gene encoding IL10Rα knocked out (IL10RαKO), using the CRISPR/cas9 method. This cell line was exposed to live MAP bacteria for 48 h, and thereafter mRNA was extracted from both infected and uninfected cells. The differentially expressed genes (DE) have been compared between the IL10RαKO cell line and the wild type MAC-T cell line. There are 561 DE genes between infected and uninfected IL10RαKO cells, 1613 DE genes between infected IL10RαKO cells and infected wild type MAC-T cells, as well as 1487 DE genes between uninfected IL10RαKO cells and uninfected wild type MAC-T cells. Gene ontology for the DE genes, as well as copy number variant detection will be performed. The results of this study will help to better understand how Johne's disease is affected by the IL10Rα gene, how it affects the animal, and how to incorporate resistance into the genomic evaluation.

Key Words: Johne's, IL10Rα, genetics