markers, 8 SSCP polymorphisms, 4 protein polymorphisms, and 5 erythrocyte antigen loci covering the whole genome. De-regressed breeding values were available for 29 traits, including 20 conformation, 2 fertility, 4 birth, 2 workability, and 1 longevity trait. After determining the most likely marker haplotypes for all grandsires based on the genotypes of their sons, multi-marker regression analysis of the trait data was used to scan the genome for QTL. Chromosome-wise and experiment-wise significance thresholds were determined by permutation test. The test statistic exceeded the genome-wise significance threshold with a type I error of less then 10% for the following traits and chromosomes: rump width on chromosome 1; feet and legs, foot angle, teat placement, udder, and udder depth on chromosome 6; calving difficulties on chromosome 8; teat length on chromosome 13; rate of stillbirth on chromosome 18, and milking speed and temperament on chromosome 29. All QTL on chromosome 6, that exceeded the genome-wise significance threshold, were located around 88 cM. QTL for rump width on chromosome 1 may have an effect on calving difficulties. QTL for udder traits on chromosome 6 and 13 may affect somatic cell score and mastitis resistance. If there are no unfavorable correlations with other economic traits, marker assisted selection using markers associated with these QTL can be applied.

Key Words: whole genome scan, quantitative trait loci, conformation and functional traits

ASAS/ADSA Growth and Development: Conjugated Linoleic Acid (CLA) in Milk Production, Growth, and Health

799 Conjugated linoleic acid (CLA) and lipid metabolism in lactating cows. D. E. Bauman*[1], L. H. Baumgard, B. A. Corl, E. Matitashvili, D. G. Peterson, J. W. Perfield II, and M. A. Madron, [1]*Cornell University*.

The uniqueness of CLA in ruminant fat relates to biohydrogenation of unsaturated fatty acids by rumen bacteria. The major CLA isomer is *cis*-9, *trans*-11 and recent work with animal models has established that this isomer is anticarcinogenic when provided as a natural component of food. Although some CLA is of rumen origin, the major source in lactating cows is endogenous synthesis via Δ^9-desaturase from *trans*-11 C18:1, another biohydrogenation intermediate. Thus, fat content of CLA is a function of rumen outflow of CLA and *trans*-11 C18:1, plus tissue activity of Δ^9-desaturase. Investigations of these aspects have identified dietary manipulations, regulatory processes and animal differences that can impact the CLA content of ruminant fat. Under certain conditions, the initial step in linoleic acid biohydrogenation is altered so the isomerization results in production of *trans*-10, *cis*-12 CLA. This CLA isomer affects lipid metabolism during growth and lactation. Across a range of diets a curvilinear relationship exists between the increase in milk fat content of *trans*-10, *cis*-12 CLA and the reduction in milk fat yield. Thus, rumen biohydrogenation can result in the formation of *trans*-10, *cis*-12 CLA, and possibly other unique biohydrogenation intermediates that are potent inhibitors of milk fat synthesis; we refer to this as the "biohydrogenation theory" of milk fat depression. *Trans*-10, *cis*-12 CLA is a potent inhibitor of milk fat synthesis with 3.5 g/d (0.016% dietary dry matter) resulting in a 25% reduction. Yields of all milk fatty acids are reduced, but effects are especially pronounced on those fatty acids originating from de novo synthesis and Δ^9-desaturase activity. Although specific mechanisms are not well defined, recent investigations indicate that exogenous *trans*-10, *cis*-12 CLA causes substantial reductions in mammary tissue mRNA abundance for key enzymes associated with de novo fatty acid synthesis, uptake of preformed fatty acids, fatty acid transport and esterification, and plasticity of milk fat.

Key Words: CLA, fat, milk fat

800 The use of rumen-protected conjugated linoleic acid to reduce milk fat percentage in lactating dairy cattle. M.A. Sippel*[1], J.P. Cant[1], and R. Spratt[2], [1]*University of Guelph, Guelph, Ontario,* [2]*Agribrands Purina Canada, Woodstock, Ontario*.

The use of conjugated linoleic acid (CLA) has been shown to reduce milk fat secretion in dairy cattle. A rumen-protected source of CLA is required for commercial feed applications. To test the ability of different rumen-protected CLA sources to induce milk fat depression, four Holstein cows (avg. 77 DIM, 40.9 kg/d milk, parity 2.25) were randomly assigned to treatments in a 4 x 4 Latin Square design. Treatments were a control diet (0 g CLA/d), CaCLA salt providing 50 g CLA/d, GRAS-coated providing 50 g CLA/d, and liquid CLA oil providing 50 g CLA/d. Periods were 3 weeks of adjustment and 1 week of sampling. The CaCLA and liquid CLA oil caused 23.4 and 23.9% depressions in milk fat content, respectively, without affecting milk, protein or lactose yields, or dry matter intake. In a second experiment, CaCLA was fed to provide 0, 25, 50, 75 or 200 g CLA/d. Ten Holstein cows were divided into two 5 x 5 Latin Squares by parity number; square 1 multiparous (avg. 110 DIM, milk yield 35.9kg/d, parity 2.3), square two primiparous (avg. 103 DIM, milk yield 29.3kg/d). Periods were 3 weeks of adjustment, followed by 1 week of sampling. Milk fat content averaged 3.48, 2.84, 2.53, 2.47 and 2.19 % as CaCLA intake increased from 0 to 200 g CLA/d. Milk yield was reduced by 2.19 kg/d on the 200g CLA/d treatment relative to the control. There was no effect of treatment on dry matter intakes or milk protein and lactose percentages. There was no significant difference in response due to parity. Feeding the CaCLA salt source to provide 25 to 75 g CLA/d was effective in decreasing milk fat percentage without affecting other production variables.

Key Words: conjugated linoleic acid, milk fat, rumen protection

801 Milk fat synthesis in dairy cows is progressively reduced by increasing amounts of *trans*-10, *cis*-12 conjugated linoleic acid (CLA). Lance H. Baumgard*, Jodi K. Sangster, and Dale E. Bauman, *Cornell University*.

CLA supplements containing a variety of isomers reduce milk fat yield in a number of species. We have recently identified *trans*-10, *cis*-12 as the CLA isomer responsible for inhibiting milk fat synthesis in dairy cows (Baumgard et al. Am. J. Phys. 278:R179). Our objectives were to establish a dose-response relationship between *trans*-10, *cis*-12 CLA and milk fat synthesis, and relate effects on milk fatty acid composition to the potential mechanism of action. Multiparous Holstein cows in late lactation were used in a 4 x 4 Latin square design where treatments consisted of four doses of *trans*-10, *cis*-12 CLA: 1) 0.0 g/d, 2) 3.5 g/d, 3) 7.0 g/d, and 4) 14.0 g/d. Over the 5d treatment intervals doses were continuously infused into the abomasum as a convenient experimental means to avoid possible alterations by rumen microbes. Milk fat yield was decreased 25, 33, and 50%, and milk fat concentration was reduced 24, 37 and 46% when cows received 3.5, 7.0 and 14.0 g/d of *trans*-10, *cis*-12 CLA, respectively. Feed intake, milk yield and milk protein content and yield were unaffected by treatment. Milk fat content of *trans*-10, *cis*-12 CLA averaged < 0.1, 1.5, 3.2 and 7.0 mg/g from cows receiving 0.0, 3.5, 7.0 and 14.0 g/d of *trans*-10, *cis*-12 CLA. Comparison of milk fat composition and synthesis revealed that reductions were most extensive for *de novo* synthesized fatty acids (short and medium chain) when cows received the two highest doses, but at the low dose (3.5 g/d) decreases in *de novo* synthesized fatty acids and preformed fatty acids were similar. Changes in milk fatty acid composition also indicated that Δ^9-desaturase was inhibited at the two high doses of *trans*-10, *cis*-12 CLA, but relatively unaffected by the low dose. Overall, results indicate that *trans*-10, *cis*-12 CLA is a potent inhibitor of milk fat synthesis. A hyperbolic dose response curve was observed and even the low dose of *trans*-10, *cis*-12 CLA (0.016% of dietary intake) dramatically inhibited milk fat synthesis (25%).

Key Words: CLA

802 Mechanisms for conjugated linoleic acid-mediated reduction in fat deposition. Harry Mersmann*, *USDA/ARS Children's Nutrition Research Center, Department of Pediatrics, Baylor College of Medicine*.

Potential mechanisms for the decreased fat deposition observed after oral administration of conjugated linoleic acids (CLAs) to mice, rats, hamsters, humans, and pigs will be reviewed. Most mechanisms are based on experiments with rodents or rodent-derived cells. In intact mice, there is an increased metabolic rate, but not in rats or sows. There is a decreased respiratory quotient in mice and rats, suggesting increased fat oxidation. Bovine milk-fat synthesis is decreased. Rat adipocyte size is smaller, but cell number is unchanged. In mice, there is increased adipocyte apoptosis. In 3T3-L1 preadipocytes, a clonal cell line derived from rodents, CLAs decrease proliferation. Differentiation

of these preadipocytes is diminished by CLA in two laboratories, but increased in a third. In contrast to the rodent-derived cells, CLAs did not inhibit proliferation in either porcine or human preadipocytes. In both porcine and human preadipocytes, CLAs acutely increased lipid deposition, but lipid content quickly reached a plateau. Peroxisome proliferator-activated receptor gamma (PPARg) and PPAR alpha, key transcription factors in adipocyte differentiation and lipid metabolism require an activating ligand; CLAs are ligands for both PPARs. The concentration of PPARg mRNA increases during adipocyte differentiation. In both porcine and human CLA-treated preadipocytes, the increase in PPARg mRNA concentration was minimal, suggesting differentiation was not markedly increased. The primary CLA isomer in ruminant tissues is cis 9, trans 11-CLA. Most synthetic CLA preparations contain a considerable amount of trans 10, cis 12-CLA, in addition to 9,11-CLA. The 10,12-CLA is responsible for the body composition changes in mice and for the decreased bovine milk-fat synthesis. The two CLA isomers equally reduced lipid deposition in porcine preadipocytes, whereas there is both evidence for a preferential effect of 10,12-CLA or no isomer distinction in human preadipocytes.

Key Words: Conjugated linoleic acid, Fat deposition, Adipocyte differentiation

803 Dietary conjugated linoleic acid (CLA) influence the lipogenic enzyme activities in adipose tissue and liver of rabbit. C. Corino[1], J. Mourot[2], G. Pastorelli[1], and V. Bontempo*[3], [1]*University of Milan/Italy,* [2]*INRA, Saint-Gilles/France,* [3]*University of Molise, Campobasso/Italy.*

A study was conducted to determine the effect of conjugated linoleic acid (CLA) synthesized from sunflower oil on lipogenic enzyme activities of adipose tissues and liver of rabbit. Thirty-six NZW rabbits, half male and half female, averaging 1.80 kg LW, allotted within weight and sex to a randomised complete experimental design, were fed ad libitum conventional pelleted diets supplemented with different CLA or sunflower oil levels: 0.5 % sunflower oil (C), 0.25 % sunflower oil and 0.25 % CLA (T1), 0.5 % CLA (T2). CLA oil contains 65 % CLA isomers (Conlinco, Inc., Detroit Lakes, Minnesota 56502 USA). The rabbits were slaughtered at 2.8 kg LW. Acetyl-CoA-carboxylase (CBX) activities, expressed as nmol $H{\cdot}CO_3{}^-$ incorporated $min^{-1}g^{-1}$were lower T than in C animals both in perirenal (T1= 3.14; T2= 6.34; C= 7.62; P< 0.01) and interscapolar (T1= 5.40; T2= 4.98; C= 8.51; P< 0.01)adipose tissues. Glucose-6-phosphate-dehydrogenase (G6PDH), expressed as μmol NADPH formed $min^{-1}g^{-1}$ resulted higher in perirenal adipose tissue of rabbits fed CLA (T1= 12.88; T2= 15.33; C= 9.3; P< 0.01). The lipogenic activities of liver was not influenced by the diet. The present study indicates that lipogenic enzyme activities are modified by dietary CLA and that are differently regulated on adipose tissues and liver of the rabbit. These results are consistent with the lower fatness of CLA fed rabbits and with the different lipogenic activities of tissues in rabbit.

Key Words: Dietary conjugated linoleic acid, Rabbit, Lipogenic enzyme

804 Performance and lipid deposition in broilers fed conjugated linoleic acid. L. Badinga*, K. T. Selberg, C. W. Comer, and R. D. Miles, *University of Florida, Gainesville Florida.*

With the broiler industry's major thrust in further processing, a major research challenge is to identify dietary components that reduce carcass fat deposition at economically reasonable levels of dietary protein. The objective of this study was to determine if a diet supplemented with conjugated linoleic acid (CLA) alters hepatic lipid metabolism in broiler chicks. Ninety-six 1-d-old male chicks of a commercial strain were weighed and allocated randomly to either CLA (n = 48) or control (n = 48) diets. On the basis of 63.6% CLA in the CLA source obtained from BASF Germany, the CLA mix was utilized at 7.86% of the diet to meet the assigned CLA concentration of 5.0%. The control diet was supplemented with corn oil to adjust the concentrations of total fats at 7.86% in both diets. At 3 wk, birds were weighed and feed intake measured. Twenty-four birds were randomly selected from each dietary treatment group and killed by cervical dislocation. Samples of livers and thigh muscles were collected and stored at -80C until subsequent determination of dry matter, fat and protein contents. Broilers fed CLA had smaller body weights (692.2 $\pm$ 7.6 g < 862.2 $\pm$ 7.6g) and grew at slower rates (30.9 $\pm$ 0.4 g/d < 39.3 $\pm$ 0.4 g/d) than chickens fed corn oil. Although CLA feeding significantly reduced feed intake (913.6 $\pm$ 10.8 g < 1043.8 $\pm$ 10.8 g), the overall feed conversion was better for the control (feed:gain = 1.27 $\pm$ 0.01) than the treated (F:G = 1.41 $\pm$ 0.01) group. The total liver lipid content was 25% lower in CLA-fed birds compared with corn oil-fed birds. CLA feeding increased lean tissue (+ 1.6%) and reduced fat (- 30%) contents in thigh muscle. This resulted in significant increase (+ 45%) in lean : fat ratio in broilers fed CLA. Results provide convincing evidence that CLA supplementation increases lean tissue deposition at the expense of fat accretion in broilers and may provide a novel strategy for decreasing carcass fat and enhancing the quality of poultry meat.

Key Words: Broiler, Fat, Conjugated linoleic acid

805 Conjugated linoleic acid (CLA) in growth and development:Mechanisms involving immunity and prostanoids. Mark Cook*[1], [1]*University of Wisconsin-Madison.*

Mechanisms of immune-induced wasting or decrease rates of gain suggested that during the immune response, the cytokines, interleukin 1 (IL-1) and tumor necrosis factor (TNF) are released to up regulate immune and inflammatory cell activation. However, collateral damage caused by these cytokines includes decreases in rates of gain, muscle wasting, reduced feed efficiency, and anorexia, among other physiological changes. Effective approaches to reduce cytokine-induced suppression of growth have focused on removal of the immune stimulus or immune suppression. Both strategies improve animal growth, but with apparent drawbacks. Evidence that cytokines induced wasting of muscle through the stimulation of prostaglandin synthesis in skeletal muscle led us to explore an alternative method of growth stimulation in an immune challenged environment. CLA is a structural derivative of the precursor for prostanoids reported to be involved in muscle wasting and immune response down regulation. When chicks, mice, and rats were fed CLA, then immune challenged with endotoxin or TNF, significantly less depression of growth and feed intake was observed compared to linoleic acid fed animals. Even end-stage disease wasting associated with an autoimmune disease was reduced 50%. Studies showed that CLA actually enhanced a number of immune endpoints relative to controls. To determine if CLA was having an effect on prostanoid synthesis, a sensitized guinea pig (pg) model was used. A method involving the use of HPLC tandem mass spectrometry allowed us to examine antigen-induced release of 15 different lipid mediators, including prostaglandins (PG) and leukotrienes (LT), from lung, bladder, and trachea. Basal (nonstimulated) PG and LT were not affected in CLA fed pg in all tissues tested. While antigen challenge increased the release of all PG and LT in control fed gp, all PG and LT release was inhibited in all tissues from CLA fed gp. CLA regulation of antigen-induced PG and LT was not due to fatty acid changes in the phospholipids, and did not effect the antigen-induced increases of cyclooxygenase-2 (COX-2) protein in the tissue. We postulate that CLA is regulating COX-2 enzyme activity. COX-2 regulation could explain the beneficial effects observed in CLA fed animals.

Key Words: Conjugated linoleic acid , Prostanoid, Immunity

806 Dietary conjugated linoleic acid (CLA) influence the immune response in weanling piglets. V. Bontempo*[1], C. Corino[2], D. Sciannimanico[2], and S. Magni[1], [1]*University of Molise, Campobasso/Italy,* [2]*University of Milan/Italy.*

The aim of this study was to determine the influence of conjugate linoleic acid (CLA) supplementation of piglets diets on some immune response parameters. Forty-two weaned piglets, 45 days old and 12.44 1.96 kg L.W., were fed diets containing different levels of CLA and sunflower oil (SFO): 0 % CLA and 1% SFO (C),; 0.5 % CLA and 0.5 % SFO (T1); 1 % CLA and 0 % SFO (T2). CLA oil contained 65 % CLA isomers (Conlinco, Inc., Detroit Lakes, Minnesota 56502 USA). Blood samples were withdrawn from all the piglets at 0 d and 28 d from the beginning of the experiment and analysed for serum lysozyme (Lys), total Immunoglobulin of G class (IgG), and α-1 acil glycoprotein (AGP). Lys (T2= 1.86 μg/ml, T1= 1.77 μg/ml and C= 1.39 μg/ml, SEM= 0.09; P< 0.01) and IgG (T2= 2075 mg/dl, T1= 1930 mg/dl and C= 1548 mg/dl; P= 0.05) were improved by CLA dietary supplementation. No dietary effects were observed on AGP. These results support the view that CLA may influence the immune response. The lack of effects on AGP may be related to a reduction of PGE_2 production which down-regulates cytokine release and AGP release.

Key Words: Dietary conjugated linoleic acid, Weanling piglets, Immune response