**272 Effect of pelleted barley on performance and carcass quality of feedlot steers.** L. M. Williams*[1], J. J. McKinnon[1], V. R. Racz[1], D. A. Christensen[1], and K. Ataku[2], [1]*University of Saskatchewan*, *Saskatoon, SK, Canada*, [2]*Rakuno Gakuen University*, *Ebetsu, Hokkaido, Japan*.

Development of export markets for cereal grains necessitates shipping grain in processed forms; however, excessive processing can lead to problems such as acidosis. A trial was conducted to evaluate the performance of beef steers fed diets containing ground, pelleted barley as the concentrate. Weaned steers (N=350; 285±22kg) were assigned to one of 12 pens and fed either pelleted or rolled barley as the concentrate. During backgrounding, cattle were fed 42% concentrate (DM), consisting of 85% pelleted or rolled barley and 15% canola meal. During finishing the concentrate was fed at 86% of the diet (DM) and consisted of 94% pelleted or rolled barley and 6% canola meal. Steers were weighed every 4 weeks during growing and every 2 weeks during finishing. Ultrasound backfat (USBF) and *longissimus dorsi* (LD) area were measured monthly. Animals were slaughtered at 12mm of USBF or 625 kg. Carcass data included weight, LD area, grade fat, marbling score, and liver abscess score. Rib fat, lean and bone %, LD and fat color, and marbling fat content were determined on 8-bone rib samples from 40 randomly selected steers. Steers fed pelleted barley had lower ($P$<0.05) ADG during finishing (1.80 vs. 2.00 kg/d) and for the total trial (1.60 vs. 1.70 kg/d), as well as more ($P$<0.05) days on feed (196 vs. 186). DMI was lower ($P$<0.05) for cattle fed pelleted barley throughout the trial. Feed efficiency (kg feed:kg gain) was superior ($P$<0.05) for the group fed pelleted barley during finishing (6.03 vs. 6.21) and the total trial (6.27 vs. 6.64). Both diets produced similar carcasses, but inter-muscular fat % was higher ($P$<0.05) for the rolled barley group (60.0 vs. 57.4%), as was ($P$<0.05) grade fat (11.2 vs. 10.7 mm). Results from this study indicate that pelleted barley can be used effectively in growing and finishing diets, however further research is required to discover why intake of pelleted barley is reduced.

**Key Words:** Pelleted barley, Carcass quality, Finishing steers

## Growth and Development: Postnatal Development as a Harbinger of Future Performance

**273 Hormone and growth factor regulation of tissue remodeling in the mammary gland.** D. Flint*[1], G. Allan[1], J. Beattie[1], M. Travers[1], M. Barber[1], A. Kolb[1], C. Whitelaw[2], M. Boutinaud[3], N. Binart[4], and P. Kelly[4], [1]*Hannah Research Institute*, *Ayr, UK*, [2]*Roslin Institute*, *Midlothian, Edinburgh, UK*, [3]*INRA Unite Mixte de Recherches Sur la Production du Lait*, *Saint Gilles, France*, [4]*Inserm Unit 584, Hormone Targets*, *Faculty of Medicine Rene Descartes, Paris.*

Insulin-like growth factor binding protein-5 (IGFBP-5) production increases dramatically during forced involution of the mammary gland in rats, mice and pigs. Growth hormone (GH) increases production of the survival factor IGF-I, whilst prolactin enhances the effects of GH by inhibiting IGFBP-5 synthesis which would otherwise prevent the actions of IGFs. A causal relationship between IGFBP-5 and cell death was demonstrated in transgenic mice expressing IGFBP-5 specifically in the mammary gland. DNA content in the mammary glands of transgenic mice was decreased as early as day 10 of pregnancy and remained so during the first 10 days of lactation. The concentration of caspase-3 was increased in transgenic animals whereas the concentrations of two pro-survival molecules Bcl-2 and Bcl-xL were decreased. Furthermore, IGF receptor- and Akt-phoshorylation were both inhibited. We also demonstrated that the effects of IGFBP-5 could be mediated in part by IGF-independent effects involving the plasminogen system, and matrix metallo-proteinases (MMPs). Treatment with prolactin was able to inhibit early involutionary processes in normal mice but was unable to prevent this in mice over-expressing IGFBP-5, although it was able to inhibit expression of MMPs. Thus IGFBP-5 simultaneously inhibits IGF action and activates the plasminogen system, thereby coordinating cell death and tissue remodelling processes. The ability to separate these properties, using mutant IGFBPs, is currently under investigation. We have also developed a mouse model of diet-induced obesity which shows numerous abnormalities relating to mammary gland function. Animals ate approximately 40% more calories, gained weight at three times the rate of controls and exhibited reduced conception rates, increased peripartum pup mortality and impaired lactogenesis. Despite access to high energy diets, the obese animals mobilised even more adipose tissue during lactation than their lean counterparts. Obese animals also exhibited marked abnormalities in ductal branching morphogenesis and alveolar development of the mammary gland, which may partially explain the delay in differentiation evident during lactogenesis.

**Key Words:** IGF, Proteases, Obesity

**274 Effects of modified calf growth on mammary development, endocrine physiology, and performance.** M. Vestergaard*[1], S. Purup[1], M. S. Weber Nielsen[2], Y. R. Boisclair[3], and K. Sejrsen[1], [1]*Danish Institute of Agricultural Sciences*, *Tjele, Denmark*, [2]*Michigan State University*, *East Lansing*, [3]*Cornell University*, *Ithaca, NY*.

The purpose of rearing heifers comprises a utilization of the genetic potential of the animal to achieve the most favorable body and mammary gland development by optimizing feeding and rearing conditions. Our research has led to the concept of a 'critical period' before puberty, where reproductive and thus mammary development can be negatively affected by high levels of nutrition. Since then focus has been devoted to exploring the possibilities of promoting a high rate of gain without negatively affecting mammary gland growth and milk yield potential. In vivo studies on the effects of nutrition, somatotropin axis activity, and steroid hormone activity or pubertal stage on mammary development have shown that classical endocrine factors, such as somatotropin and estrogen, are involved in the regulation of normal growth and development, but their role in mediating the effects on mammary development is less clear. Most evidence suggests that the key regulation takes place locally in the mammary gland. Investigations on local regulation of mammary development have included functional receptor studies in specific tissues and in vitro cell culture experiments using tissue and serum from in vivo experiments. The results indicate that locally produced IGFs and IGF-binding proteins play a role, but many other factors, such as TGFβ and leptin, likely also contribute. The importance of other body tissues and the interaction between the mammary gland and other tissues as well as the cross-talk within the mammary gland are less well-studied. However, both the endocrine actions of various growth factors, such as the IGFs, which target most tissues, and the tissue-specific expression and production of important paracrine and endocrine factors have to be considered to further elucidate the complex regulation of mammary gland and body development. We expect that future research will have to focus more on the interactions and synergism among different types of tissues during calf development.

**Key Words:** Cattle, Mammary, Endocrinology

**275 Tissue proteolytic enzymes: Modifiers of muscle and adipose tissue.** G. Hausman*, *USDA ARS*, *Athens, GA*.

A fundamental aspect of tissue remodeling is the breakdown and degradation of connective tissue and extracellular matrix (ECM) proteins. Degradation or proteolysis of ECM proteins is implicated in cell attachment, cell migration, ECM invasion, angiogenesis and release and processing of membrane bound cytokines and growth factors. Extracellular proteolysis involves several families of proteolytic enzymes, including the plasminogen activator (PA) - plasmin system, the adamalysins (ADAMs) family and the matrixin matrix metalloproteinases

(MMPs) and their tissue inhibitors (TIMPs). The MMP family is the most prominent of these protease families and MMP-2, MMP-9, TIMP-1 and TIMP-2 are the most studied of the MMP regulatory system. The non-ECM substrates of MMPs and limitations of in vivo studies of MMP protein levels and activities will be discussed. Expression of MMPs is critical for development since deficiencies in both MMP-2 and MMP-14 is lethal and MMP-9 deficiency results in transient abnormal bone development. Many MMPs including MMP-2 and MMP-9 and TIMPs are expressed in skeletal muscle, isolated myogenic cells, isolated muscle fibroblasts, adipose tissue and adipose tissue stromal-vascular cells in a depot dependent manner. The influence of MMP and TIMP knockouts, MMP inhibitors and PA- plasmin system knockouts and over expression on adipose tissue development will be reviewed. The role of extracellular proteolysis in myogenesis is discussed including the evidence that MMPs and the PA- plasmin system components mediate myoblast migration and fusion. Studies of MMP involvement in the initial phase of muscle angiogenesis will also be reviewed. The ramifications of the influence of PA inhibitor-1 (PAI-1) on migration of preadipocytes and associated endothelial cells will also be discussed. Finally, evidence that MMPs and, in particular, MMP-9 mediate adipocyte differentiation in vitro will be reviewed.

**Key Words:** Adipose Tssue, Muscle, Extracellular Proteolysis

**276 Tumor necrosis factor-α (TNF-α) decreases media content of epithelial cell-derived insulin-like growth factor binding proteins (IGFBP) in part through increased proteolytic degradation of IGFBP-3.** T. H. Elsasser*[1], T. J. Caperna[1], J. L. Sartin[2], C. Li[1], and S. Kahl[1], [1]*USDA, Agriculture Research Service*, *Beltsville, MD*, [2]*Auburn University*, *Auburn, AL.*

Acute proinflammatory stress is marked by significant alterations in metabolic capacity in animals mediated in part by decreased plasma and tissue levels of insulin-like growth factor-1 (IGF-1) and the respective IGFBP-2 and -3. To determine if a part of the localized tissue regulation of IGF-1 action might encompass the cellular presentation of IGFBP patterns, cultured Madin-Darby bovine kidney epithelial cells (~95% confluent, $10^6$ cells/well, 1.0 mL RPMI 1640 serum-free media) were challenged with physiologically relevant concentrations of recombinant bovine TNF-α (1 or 100 nM) in the presence and absence of the IGFBP-3 stimulator forskolin (F), a cyclic AMP pathway effector. IGFBP-2 and -3 media contents were assessed by radioligand blot; protease activity against IGFBP-3 was measured by adding recombinant human IGFBP-3 to media samples, incubating for 30 min, and measuring the residual human IGFBP-3 by Western blot using anti-human IGFBP-3 devoid of crossreactivity with bovine IGFBP-3. Where F promoted a 50% decrease in media IGFBP-2 ($P<0.02$) and a 22-fold increase in media IGFBP-3 ($P<0.001$) content, respectively, the addition of TNF-α not only further reduced IGFBP-2 but also decreased F-stimulated IGFBP-3 media content by 70% ($P<0.005$). As suggested by the decrease in human IGFBP-3 band intensity, F increased media BP-3 protease activity by as much as 75%; TNF-α further increased media protease activity as evidenced by a >95% decrease in human IGFBP-3 band density. The data are consistent with the concept that TNF-α-associated increases in cell-derived protease activity may affect endocrine functions of IGF-1 by degrading IGFBPs leading to a functional decrease in the size and distribution of IGFBP-2- and -3-bound pools of IGF-1.

**Key Words:** IGF Binding Proteins, Stress, Tumor Necrosis Factor-α

**277 Effects of diet and bST on expression of leptin and leptin-receptor in mammary parenchyma of heifers.** B. J. Lew*[1,2], J. S. Liesman[1], M. D. S. Oliveira[2], and M. J. VandeHaar[1], [1]*Michigan State University*, *East Lansing*, [2]*Sao Paulo State University (UNESP)*, *Jaboticabal, SP, Brazil.*

Increasing growth rates in prepubertal heifers decrease age at puberty and subsequent milk production. Administration of bST before puberty increases parenchymal tissue and decreases adipose tissue within the udder. Our objective was to examine the effects of a high energy, high protein diet combined with injection of bST on leptin and leptin-receptor (Ob-R) gene expression in mammary parenchyma. The mammary tissue used was collected in a previous experiment conducted in 1994 (Radcliff et al., 1997). In the experiment, Holstein heifers were randomly assigned to one of four treatments - low or high diet (0.8 or 1.2 kg of BW gain/d, respectively), with or without bST administration (25 ug/kg BW/d) - from 120 d of age until the early luteal phase of the fifth estrous cycle. Total RNA was extracted from parenchymal tissue of 32 heifers (8/treatment) and gene expression profile for leptin (GGGTGATTTCAGAGCCTTTGG-F; AGAATCCAGGGAGCATCATGAAGGCTACAG-R) and Ob-R (GGGCACATCCAAGCATTAAAA-F; GGCCGGCATCAAAGCTTT-R) was tested in qRT-PCR, against glucoronidase β (GUS; TGTCATCGCACACAGAGCAA-F; CACAAAATCCAGGTGAGAAGCTT-R) as a control. The results were calculated using the ΔΔCt method and analyzed as a 2 by 2 factorial experiment. High diet increased leptin mRNA 56% ($P<0.03$) and decreased leptin receptor 18% ($P<0.03$) while bST treatment decreased leptin mRNA 74% ($P<0.01$) and leptin receptor 23% ($P<0.01$). The interaction of diet and bST was not significant ($P>0.25$) for either leptin or leptin receptor mRNA. The mechanism by which a high energy diet before puberty decreases subsequent milk production is not understood, but we suggest that perhaps this increase in mammary leptin expression may be a part of it. In conclusion, a high energy diet increases and bST administration decreases expression of leptin in mammary parenchymal tissue of prepubertal heifers, consistent with a possible inhibitory role of leptin in mammary development.

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**Key Words:** Mammary Gland Development, Leptin

**278 Beta-adrenergic receptor agonist-induced skeletal muscle hypertrophy is fiber type-specific through differential involvement of the MAPK signaling pathway.** H. Shi*, A. Ricome, K. Hannon, A. Grant, and D. Gerrard, *Purdue University*, *West Lafayette, IN.*

Beta adrenergic (BA) receptor agonists induce skeletal muscle hypertrophy and antagonize atrophy. The molecular mechanisms controlling these phenomena are not, however, well known. Here we report that BA exerts a distinct muscle fiber type-specific hypertrophy that is preferentially restricted to fast-twitch fibers. Moreover, we show herein that pharmacologically or genetically attenuating ERK signaling in muscle fibers results in decreases ($P < 0.05$) in fast but not slow fiber type-specific reporter gene expression in response to BA exposure in vitro and in vivo. Consistent with these data, forced expression of MAPK phosphatase 1 (MKP-1), a nuclear protein that dephosphoryates ERK1/2, in fast-twitch skeletal muscle ablates ($P < 0.05$) the hypertrophic effects of BA-feeding (clenbuterol, 20 ppm in water) in vivo. Further analysis showed that BA-induced phosphorylation and activation of ERK occurs to a greater ($P < 0.05$) extent in fast myofibers than in slow myofibers. Analysis of the distribution pattern of phosphoERK1/2 in slow and fast muscles revealed that ERK1/2 is more activated in fast- than in slow-twitch muscles. These data suggest that the increased abundance of phosphoERK1/2 in fast-twitch myofibers than in their slow-twitch counterparts may account for, at least in part, the fiber-type-specific hypertrophy induced by BA stimulation. Given that other muscle hypertrophy and/or atrophy models respond in a muscle fiber-type specific manner, it seems logical that fast myofibers are pivotal in the adaptation of muscle to various environmental cues and that the mechanism underlying this change is partially mediated by the MAPK signaling cascade.

**Key Words:** Beta Agonist, Skeletal Muscle Hypertrophy, MAPK Signaling