campus farm. Milk was put into the respective pasteurizers and inoculated with the appropriate level of pathogens. The pasteurizers were heated to the specific time and temperatures: 145F for 30 minutes for the batch/vat pasteurizer and 161F for 15 seconds for the continuous-flow pasteurizer. Pre- and post-pasteurization (0, 24, and 48 h) samples were taken from each of the triplicate runs performed for each of the two pasteurizers. The milk samples were plated onto selective media for each pathogen and incubated at 37C for the appropriate time. All of the post-pasteurization samples showed no growth for *E. coli* 0157:H7, *Salmonella sp.*, *Listeria monocytogenes*, and *Staphylococcus aureus*. The HEYM Mycobactin J slants from the milk samples for the *Mycobacterium paratuberculosis* are in week 5 of incubation. From the results obtained, pasteurization with both on-farm units (batch/vat and continuous flow) was shown to destroy *E. coli* 0157:H7, *Salmonella sp.*, *Listeria monocytogenes*, and *Staphylococcus aureus* effectively. Because it will take 16 weeks to determine a true negative for *M. paratuberculosis*, results are still pending.

Key Words: Pasteurization, *Mycobacterium paratuberculosis*, Commerical pasteurizers

607 Detection comparison of *L. monocytogenes* in yogurt and cold pack cheese using enzyme-linked immunofluorescent assays. T. M. Silk* and C. W. Donnelly, *University of Vermont, Burlington, Vermont, USA*.

Recent outbreaks of *Listeria monocytogenes* have been attributed to low levels of contamination in food products. Rapid detection methods should be sensitive and accurate at reporting the presence of this pathogen in food. In the current study, two commercially available enzyme-linked immunofluorescent assays (ELIFAs), specific for *Listeria* spp., were used for the detection of *L. monocytogenes* in yogurt and cold-pack cheese. Food products naturally contaminated, and inoculated with *L. monocytogenes* at various inoculation levels ranging from 3.0 - 0.007 MPN/g were tested. Ten to twenty replicate samples were analyzed for each inoculation level. Detection results were compared with those obtained using the current U. S. Food and Drug Administration Bacteriological Analytical Manual (BAM) method for *Listeria* detection in food. One of the ELIFAs, lacking a secondary enrichment step, performed very poorly in comparison to the BAM method. Detection agreement values decreased as inoculation levels decreased. In food products inoculated with fractional positive levels of *L. monocytogenes*, ELIFA performance produced false negative rates approaching 100% whereas the BAM method did not produce false negative rates higher than 10%. Further cultural analysis of enrichment used for ELIFAs subsequently yielded positive *L. monocytogenes* results, indicating that the enrichment used for ELIFAs may not have increased target cell levels to those needed to elicit a positive response. The inability of the enrichment to increase *Listeria* levels may be attributed to an increased acriflavine level, which may result in a failure of these procedures to recover low levels of or injured *Listeria*, which can exist in acidic foods or those containing preservatives. Better enrichment protocols focused on the recovery of low level, or injured cell populations may increase the sensitivity of detection, ultimately improving the safety of dairy foods.

Key Words: *Listeria monocytogenes*, Detection, Enzyme-linked immunofluorescent assay

Growth and Development

608 Effect of differences in pattern of prepubertal growth on response to realimentation: Relationships to reproductive development. John Klindt*, J.T. Yen, and R. K. Christenson, *USDA, ARS, U.S. Meat Animal Research Center, Clay Center, NE*.

Our previous work (Klindt et al., 2001, J. Anim. Sci. 79:2513) showed an inverse relationship between feed consumed during development and feed consumed during breeding in gilts subjected to feed restriction during development, 1/2 to 7/8 ad lib, and given ad libitum access to feed during breeding. Age at first estrus was least in the 1/2 ad lib gilts, possibly due to increased feed consumption during breeding. The current study sought to replicate the feed intakes of gilts in the previous study and measure the effect on physiological responses. Crossbred white gilts, 90.3 ± 0.5 d of age, 38.2 ± 0.7 kg BW, were assigned to receive 1/2, 5/8, 3/4, or 7/8 of calculated ad libitum feed intake (24 gilts/dietary treatment, TRT) for 12 wk. After the restriction period, all gilts were fed quantities of feed similar to those consumed by similar gilts given ad libitum access to feed in group pens previously. During realimentation, ADFI was 3.03 ± 0.06, 2.76 ± 0.08, 2.40 ± 0.07, and 2.31 ± 0.08 kg/d by gilts in the 1/2, 5/8, 3/4, and 7/8 TRT groups, respectively. On d 0, 7, 14, and 21 of realimentation, gilts were slaughtered and wts of offal and carcass components were recorded. Blood samples were collected from the gilts during the last wk of the restriction period and during realimentation for assay of serum urea, glucose, insulin, and IGF-I. Urea, glucose, insulin, and IGF-I were influenced ($P < 0.03$) by the interaction of TRT×wk of realimentation. Slaughter and carcass wts were influenced ($P < 0.01$) by the main effects of TRT and wk. Of the offal components, only liver and small intestine were influenced ($P < 0.02$) by TRT×wk. It is concluded that increased feed intake by the more severely restricted gilts during the early part of breeding/realimentation period allowed those gilts to exhibit compensatory gains, had effect on liver and small intestine wts, and stimulated acceleration of onset of first estrus in the most severely restricted gilts.

Key Words: Gilts, Puberty, Growth

609 The effect of carbohydrate source on intestinal morphology of weaned pigs. M.A.M. Spreeuwenberg*[1], J.M.A.J. Verdonk[2], M.W.A. Verstegen[3], and A.C. Beynen[4], [1]*Nutreco, Boxmeer,* [2]*IDTNO, Lelystad,* [3]*Wageningen University,* [4]*Utrecht University, The Netherlands*.

Epithelial cells need energy to maintain gut integrity as measured with histology. It is hypothesised that with increasing the number of glucose molecules bound together, glucose availability and thereby gut integrity decreases: glucose > lactose > starch. A total of 42 newly weaned barrows (26 ± 0.8 d of age, 7.8 ± 1.0 kg) was used. On the day before weaning (d -1) all pigs were weighed and assigned to 7 experimental groups (n=6). The groups differed in diet and day of dissection. On the day of weaning (d 0), dissection was performed on 1 group. The remaining groups were fed 1 of 3 diets in which glucose, lactose or starch were iso-energetically exchanged, supplying 24% of the energy. The animals received a liquid diet (meal: water = 2:1) based on net energy requirement for maintenance (M, kcal = $78 \times BW^{0.75}$). Energy offered to the pigs increased from 0.5×M at d 0, 1.0×M at d 1, 1.5×M at d 2, 2.0×M from d 3-9. At d 0, 3 and 10 selected pigs were weighed and euthanized. Tissue samples for histology were taken at 0.5 m (prox.) and 3.5 m (mid) distal of the ligament of Treitz. Dry matter intake, body weight gain, villus height and crypt depth did not differ between diets. Dry matter intake was 59 28.0 g/pig/d from d 0-3, 173 ± 67.0 from d 3-7 and 257 ± 33.1 from d 7-10. At d 3, villus height was decreased compared to d 0. At d 10, villus height reached pre weaning levels for the lactose diet at the prox and for all diets at the mid small intestine. Crypt depth was increased at d 10 compared to d 0 and 3. It was concluded that dietary carbohydrate source does not affect intestinal morphology.

Day		0	3			10			
Diet			glucose	lactose	starch	glucose	lactose	starch	SEM
Villus height (μm)	prox[1]	394^{a}	274^{bc}	278^{c}	272^{bc}	315^{b}	360^{ab}	298^{b}	32.5
	mid[1]	337^{a}	236^{bc}	200^{c}	252^{bc}	293^{ab}	304^{a}	311^{ab}	32.4
Crypt depth (μm)	prox[2]	166^{a}	175^{a}	188^{a}	185^{a}	288^{b}	291^{b}	2 88^{b}	14.6
	mid[2]	157^{a}	172^{a}	193^{a}	179^{a}	254^{b}	252^{b}	256^{b}	15.6

different letters within a row differ: 1, $P<0.10$; 2, $P<0.05$. Comparisons: between diets within day and between days for the same diet.

Key Words: pig, morphology, small intestine

610 Dietary betaine (Betafin) and porcine somatotropin (Reporcin) have additive effects upon growth performance in restrictively-fed boars. D Suster[1], M Mottram[2], B.J. Leury[3], R.H. King[1], and F.R. Dunshea*[1], [1]*Natural Resources and Environment, Werribee, Vic 3030, Australia,* [2]*Feedworks, Hamilton, Qld 4007, Australia,* [3]*Institute of Land and Food Resources, University of Melbourne, Vic 3010, Australia.*

Twenty individually-penned entire male pigs (initial weight 64 kg) were used to investigate the interactions between dietary betaine (Betafin, Bet) and porcine somatotropin (Reporcin, pST) treatment. The study was a 2x2 factorial design with the respective factors being dietary betaine (0 or 1.5 g/kg) and pST (0 or 5 mg/d of Reporcin). Pigs were fed 2.7 kg/d of a protein adequate diet containing 3.38 MCal DE/kg and 1.1 g/kg lysine until they were slaughtered 31 days later. An Hologic QDR4500 Dual Energy X-ray Absorptiometer (DXA) was used to estimate body composition of pigs at the beginning and end of the study. Both pST and betaine increased daily gain and lean tissue deposition and decreased P2 back fat and the effects were to a large extent additive. Despite the reductions in P2 backfat, neither pST nor betaine had any effect on fat deposition. While these feed intakes were approximately 80% of ad libitum for individually-housed pigs, they are probably at the high end of feed intakes observed in group-housed pigs under commercial conditions. Therefore, it is likely that in situations where energy intake is limiting the potential for lean tissue deposition, betaine alone, or in combination with pST, can increase growth performance and lean tissue deposition.

pST, mg/d	0	0	5	5			P-value	
Betaine, g/kg	0	1.5	0	1.5	LSD	Bet	pST	BetxpST
ADG, g/d	1067	1162	1308	1379	116.3	.049	<.001	.77
FCR	2.55	2.38	2.11	2.06	.213	.16	<.001	.41
P2, mm	13.8	12.6	12.2	11.8	1.52	.081	.014	.52
Lean, g/d	673	752	863	915	83.9	.032	<.001	.65
Fat, g/d	207	216	196	205	27.8	.34	.26	.97

Key Words: Betaine, Porcine somatotropin, Growth

611 The somatotropin (ST)/insulin-like growth factor (IGF) system is not affected by an infectious disease challenge in growing pigs. W.T. Oliver*[1], G.W. Almond[1], S.A. Mathews[1], J.A. Brown[1], and R.J. Harrell[1], [1]*North Carolina State University, Raleigh, NC.*

Respiratory diseases account for considerable economic loss in the swine industry due, in part, to reduced growth performance. The objectives of this experiment were: 1) to determine the effect of porcine reproductive and respiratory syndrome virus (PRRSv) and *M. hyopneumoniae* (*M. hyo.*) on basal IGF-I concentrations, and 2) to determine if exogenous ST can stimulate the IGF system in health-challenged pigs. Barrows were weaned from the sow at 10 d of age and reared in isolated facilities to achieve high-health status. Pigs (2 replicates of 15) were randomly assigned to one of three treatment groups: 1) Non-infected, *ad libitum* intake (C); 2) Challenged with PRRSv and *M. hyo.*, *ad libitum* intake (HC); and 3) Non-infected, pair-fed to HC pigs (PF). HC pigs were infected with *M. hyo.* during jugular catheterization surgery and with PRRSv 8 d later (d 0). On d 14 of infection, pigs within a treatment group were randomly assigned to receive either 0 or 120 mg porcine STkg body weight^{-1}day^{-1} for 4 days. Only infected pigs had lung lesions typical of *M. hyo.* on d 21. Initial body weight did not differ (33.91.0; P>0.64), but C pigs were heavier than both the HC and PF groups on d 21 (53.21.4 vs. 45.81.8 and 46.81.5, respectively; P<0.01). From d 0 to 21, C pigs gained 91931 g/d compared to 67840 and 60133 for HC and PF pigs, respectively (P<0.01). Infection with PRRSV and *M. hyo.* reduced feed intake beginning on d 7 of infection (P<0.04), with a maximum reduction of 50 % (P<0.01) on d 16. Disease status did not affect feed conversion (G:F, P>0.10), but PF pigs had a 17 % lower G:F than HC pigs (P<0.02). Basal circulating IGF-I levels were 32222 ng/mL, regardless of treatment (P>0.15). ST administration increased circulating IGF-I 2.5-fold (P<0.01), regardless of treatment. These results indicate that circulating IGF-I under basal conditions or stimulated with ST was not responsible for the growth reduction found in PRRSv and *M. hyo.* infected pigs.

Key Words: Swine, Somatotropin, Disease

612 Myostatin gene expression in nursery pigs infected with *Mycoplasma hyopneumoniae* (Mh) and Porcine Reproductive and Respiratory Syndrome Virus (PRRSV). J. Escobar*[1], T.L. Toepfer[1], W.G. Van Alstine[2], D.H. Baker[1], and R.W. Johnson, [1]*University of Illinois, Urbana, IL,* [2]*Purdue University, W. Lafayette, IN.*

Respiratory pathogens such as PRRSV and Mh markedly depress growth and purportedly reduce protein accretion (PA) in pigs. It is not known, however, if myostatin (MSTN)-a negative regulator of muscle mass-is increasingly expressed by muscle during infection. The objectives of this study were to determine if PRRSV and Mh-alone or in combination-increase circulating levels of inflammatory cytokines, decrease growth performance and increase steady-state levels of MSTN mRNA in muscle. Thirty-two pigs were subjected to one of four treatment combinations (2 × 2 factorial) of Mh [intratracheal inoculation with Friis or P5722-3 (3 ml 10^7 cfu/ml) at 4-wk of age] and PRRSV [intranasal inoculation with DMEM or VR-2385 (5 ml 10^5 50% $TCID_{50}$) at 6-wk of age]. Pigs were killed 7 d after PRRSV inoculation. Interleukin (IL)-1β and IL-6 were measured in sera collected 7 d after PRRSV inoculation using porcine-specific ELISAs. At sacrifice, triceps (T) and biceps femoris (BF) muscles were dissected, weighed and samples were taken for MSTN gene expression analysis. Whole-body protein was determined to estimate PA. Two-way ANOVA of IL-1β, IL-6, PA, ADG, FI, T and BF weights, and MSTN mRNA detected a main effect of PRRSV ($P < 0.001$), but neither the Mh effect nor the Mh × PRRSV interaction were significant. PRRSV induced a marked increase ($P < 0.001$) in IL-1β, IL-6 and MSTN mRNA, and a concomitant decrease ($P < 0.001$) in FI, ADG, PA and weight of BF and T muscles. MSTN mRNA expression and serum levels of IL-1β and IL-6 were negatively correlated (r = -0.4 to -0.8, $P < 0.001$) with FI, ADG, PA and weight of BF and T muscles. Collectively, these findings suggest that the decrease in pig performance in PRRSV-infected pigs may be due to an increase in metabolically active inflammatory cytokines and MSTN gene expression.

Key Words: Inflammatory cytokines, Myostatin, Mycoplasma and PRRSV

613 The role of JAK2 in terminal differentiation in C2C12 myoblasts. S. Miller and J.M. Reecy*, *Iowa State University.*

Satellite cell proliferation and differentiation is critical to postnatal skeletal muscle hypertrophy. Understanding the molecular mechanisms underlying hypertrophy is necessary to facilitate treatment of muscle wasting diseases and to promote increased muscle accumulation in meat animals. Previous research in our lab has suggested that JAK2 plays a role in skeletal muscle hypertrophy. Microarray analysis of gene expression in skeletal muscle after three days of work overload demonstrated increased JAK2 expression (8.7-fold). Janus kinases (JAKs) are receptor-associated tyrosine kinases that are generally required for cytokine receptor superfamily signaling. We hypothesize that JAK2 was required for skeletal muscle differentiation. To elucidate the role of JAK2 in differentiation, C2C12 myoblasts were plated at a density of 20,000 cells/cm2. The cells were allowed to differentiate for 72 hours in serum-restricted (2% horse serum) media. Tryphostin AG490, a JAK2 specific inhibitor, was used to block JAK2 signaling during differentiation. AG490 was added 0, 24 hours and 48 hours after the addition of differentiation media. Cells were collected 72 hours after induction of differentiation. Control cells cultured without AG490 were collected at 0 and 72 hours. Creatine kinase (CK) levels were used to quantify the level of terminal differentiation. Results indicate that blocking JAK2 prevented myoblast differentiation (0 and 24 hours) and arrested differentiation in immature myotubes (48 hours). We also tested to see if differentiation could be recovered after incubation with AG490. C2C12 myoblasts were incubated with AG490 in differentiation media for 48 hours. After 48 hours these cells were washed and changed to a normal differentiation media. Cells were collected 24, 48 and 72 hours after the addition of normal differentiation media and CK levels were assayed. These cells were able to form myotubes and had significantly increased CK levels over cells continuously incubated with AG490 (p<0.0001). However, these cells had significantly lower CK levels then the control cells cultured without AG490 (p<0.0001). From these results it appears that JAK2 plays a critical role in the differentiation of skeletal muscle.

Key Words: Muscle Differentiation, myoblast, JAK2

614 Quantification of myogenin-positive satellite cells from bovine skeletal muscle. J.S. Scheffler*, N.T. Mesires, and M.E. Doumit, *Michigan State University, East Lansing, MI.*

Skeletal muscle fiber nuclei accumulate as a result of satellite cell proliferation, differentiation and incorporation into muscle fibers. Myogenin, a muscle-specific transcription factor, is an early marker for muscle cell differentiation. Our objectives were to determine the relationship between bovine semitendinosus (ST) muscle size and DNA concentration, and to quantify the proportion of myogenin-positive satellite cells in isolates from ST muscles. Holstein steers weighing 219 ± 13 kg and 503 ± 17 kg were used (n = 4 animals per group). Semitendinosus muscles removed at harvest from light and heavy steers weighed 810 ± 44 g and 1670 ± 233 g, respectively. Muscle protein was quantified using the biuret procedure and DNA was quantified by a fluorometric assay. Heavy steers had a higher ST muscle protein concentration (257 vs 190 mg/g muscle; P<0.05) and tended to have a higher ST muscle DNA concentration (795 vs 698 μg/g muscle; P<0.08) than light steers. No difference in protein to DNA ratio was observed between weight groups, indicating that ST muscle growth is accompanied by proportional increases in protein and DNA. Satellite cells were isolated from the left ST muscle of each animal. Cells from three animals per group were separated from cellular debris using Percoll gradient centrifugation, and adsorbed to glass coverslips for fluorescent immunostaining. Satellite cells were distinguished from non-myogenic cells by positive staining for neural cell adhesion molecule (NCAM). The proportion of NCAM-positive cells (satellite cells) in isolates was similar for light and heavy steers (62 vs 66%, respectively; P>0.1). Similarly, the proportion of myogenin-positive satellite cells did not differ between light and heavy steers (P>0.1). Less than 10% of the satellite cells isolated from all steers were myogenin-positive. These data suggest that the proportions of differentiating satellite cells do not differ between light and heavy steers. Alternatively, changes in the rate of satellite cell incorporation into myofibers may offset differences in the proportion of differentiating satellite cells.

Key Words: Satellite cell, Myogenin, Bovine

615 Development of the Callipyge phenotype during early post-natal growth in lambs. R. D. Sainz*[1], J. S. Cubbage[1], M. Dally[1], F. C. Castro[1], and B. Freking[2], [1]*University of California, Davis, CA , USA,* [2]*US Meat Animal Research Center, Clay Center, NE, USA.*

In order to determine the time-course of expression of muscle hypertrophy, several skeletal muscles were obtained by dissection of normal (N) and Callipyge (C) lambs from birth to 14 weeks of age in two experiments. In Exp. 1, genotype was established using the OY15 marker, and in Exp. 2 lambs were produced by use of homozygous semen from N or C rams. Body weights (kg) were similar (P>0.05) during week 1 (5.2 and 5.5), week 3 (13.0 and 13.7), and week 8 (22.9 and 18.8), but differed (P<0.001) at week 14 (28.4 and 44.2) for N and C lambs, respectively. Body weight-adjusted weights of *longissimus dorsi* (LD), *semimembranosus* (SM), *semitendinosus* (ST), *infraspinatus* (IS) and *supraspinatus* (SS) were similar (P>0.05) in N and C lambs during weeks 1 and 3 of age. Muscles were heavier (P<0.001) in C as compared to N lambs beginning in week 8 (LD) and 14 (SM and ST). As seen in previous studies, IS and SS muscles were similar in N and C lambs at all ages. Preliminary data indicate that DNA concentrations in each muscle were similar between genotypes and ages, suggesting that the increased muscle growth seen in C lambs was accompanied by increased incorporation of satellite cell nuclei. Skeletal muscles that are affected by the Callipyge mutation begin to express the phenotype between 8 and 14 weeks of age.

Key Words: skeletal muscle, Callipyge, growth

616 Leptin reduces feed intake and increases serum fatty acid concentrations in growing pigs, but does not regulate acetyl Co-A carboxylase activity or PPARα expression in adipose tissue. K. M. Ajuwon*[1], J. Kuske[1], O. Adeola[1], D. L. Hancock[2], D. B. Anderson[2], and M. E. Spurlock[1], [1]*Purdue University, West Lafayette, Indiana,* [2]*Elanco Animal Health, Inc., Greenfield, Indiana.*

To determine the effects of leptin on selected serum hormone and metabolite concentrations, we administered leptin twice daily intramuscular to barrows at 53 kg body weight at a dose of 0.05 mg kg $^{-1}$ day^{-1} for 14 days. In addition, a control group was allowed ad-libitum feed intake and injected with vehicle, and a third group was injected with vehicle and had their feed intake limited to that of the group injected with leptin. Blood samples were collected on days 1, 7 and 14 for serum metabolite and hormone analyses. Relative to the control group, the mean daily feed intake of the pigs treated with leptin was depressed by 32 % (P < 0.0001). Consistent with the lower feed intake, growth rates were reduced by 50% and 44 % in the leptin-treated and pair-fed pigs, respectively (P < 0.0001). Serum insulin-like growth factor 1 (IGF-1) was decreased in both the leptin-treated and pair-fed groups (15 % reduction, P < 0.01), but there was no treatment effect on serum growth hormone (GH) concentrations (P<0.26). Serum urea nitrogen concentrations were lower in the pigs injected with leptin than in the control and pair-fed pigs (15% reduction, P < 0.02). Although serum NEFA concentrations were higher(80% increase, P < 0.0001) in pigs injected with leptin than in the other groups, there was no effect of leptin on serum glycerol concentrations (P>0.05). The expression (mRNA abundance) of PPARα in adipose tissue, liver or skeletal muscle was not different among treatments (P > 0.05). The activity of acetyl Co-A carboxylase (ACC) was reduced by 56% due to intake restriction, but there was no further reduction attributable to leptin. These data confirm that exogenous leptin regulates feed intake in the pig, and indicate that leptin influences lipid metabolism in vivo. However, the regulation of PPARα expression by leptin previously reported in other *in-vitro* and *in-vivo* models was not evident in the pig, at least under the conditions of the present study.

Key Words: Leptin, Adipose, Pig

617 The effect of conjugated linoleic acid on the differentiation and proliferation of porcine stromal-vascular cells. T.D. Brandebourg* and C.Y. Hu, *Oregon State University, Corvallis.*

Feeding conjugated linoleic acid (CLA) decreases carcass fat in growing pigs. However the underlying mechanisms by which CLA inhibits fat accretion are poorly understood. The objective of this study was to examine the effect of CLA on adipocyte growth in pigs by determining the effect of CLA administration on the differentiation and proliferation of cultured porcine stromal-vascular cells. On d -1, stromal-vascular cells were isolated from 2-day-old crossbred pigs and plated in medium containing 10% fetal bovine serum (FBS). Cells were treated with either ligand or carrier (DMSO) from d 0 to d 8 in the presence of induction media (10 % FBS, 100 nM insulin, 10 ng/ml transferrin and 500 ng/ml hydrocortisone). In separate experiments (n=6), CLA, cis-9, trans-11 CLA, or a linoleic acid (LA) control were administered at concentrations of 25, 50, or 100 μM. Since trans-10,cis-12 CLA was toxic to cells in this system at concentrations greater than 25 μM, it was administered at concentrations of 6.25, 12.5 and 25 μM. Differentiation was evaluated by measuring sn-glycerol-3-phosphate dehydrogenase (GPDH; EC 1.1.1.8) activity on d 8. CLA significantly decreased GPDH activity (nmol/(min*mg protein) versus carrier by 15% at 25 μM (p<.001), 37% at 50 μM (p<.0001), and 70% at 100 μM (p<.0001). Administration of the trans-10, cis-12 CLA isomer decreased GPDH activity 50% at 6.25 μM (p<.0001), 73% at 12 μM (p<.0001) and 84% at 25 μM (p<.0001). The cis-9,trans-11 isomer failed to affect GPDH activity at any concentration. However as expected, LA significantly increased GPDH activity at all concentrations. In order to study the effect of CLA on the proliferation of porcine stromal-vascular cells, cells were treated as described above and cleavage of the tetrazolium salt, WST-1, by mitochondrial dehydrogenases was measured on d 2. Only the 25 μM trans-10, cis-12 CLA treatment significantly decreased WST-1 (p<.05). These data suggest that CLA inhibits fat accretion in growing pigs in part by inhibiting fat cell differentiation and perhaps to a lesser extent by decreasing stromal-vascular cell proliferation.

Key Words: CLA, Adipose Tissue, Differentiation